

Serotonin reduces apnea and could be a clue to understanding sudden infant death syndrome

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Serotonin, a neurotransmitter in the brain, shortens periods of apnoea (temporary cessation of breathing) and promotes inspiration, according to a study published today in *Experimental Physiology*.

The researchers found that when injected into a specific part of the brain (the brain stem) serotonin shortens apnoeic events by interacting with a specific serotonin receptor, the 5-HT₃ receptor, which, in healthy babies, is highly expressed in a region of the brainstem associated with the control of apnoeas and regular breathing.

Although safe sleeping environments and reduction of behaviours associated with an increased risk of SIDS have reduced the number of events, SIDS and Asphyxia (lack of oxygen) remain among the most common causes of death in infants between the age of one month and one year. There is currently no effective treatment for SIDS or Asphyxia.

Previous research has shown that the brain stem of human babies who died of SIDS was deficient in serotonin and serotonin receptors. Subsequent investigations demonstrated that babies who died of asphyxia also have deficits in the brain stem serotonin system. To test whether serotonin could shorten apnoea, or to see whether blocking serotonin receptors would indeed prolong apnoea, the scientists conducted a range of experiments.

The team used rat pups for their study and induced apnoea by putting a small amount of water in the upper airway to induce a reflex apnoea, the laryngeal chemoreflex. Previously, researchers suspected that the laryngeal chemoreflex (a protective reflex of the airways that causes apnoea and abnormally slow heart rate) is the starting point of SIDS. They used very small amounts of serotonin and other drugs that interact with serotonin receptors and injected them into a part of the brainstem involved in the control apnoea and regular breathing. When serotonin was injected into this specialised part of the brainstem, the laryngeal chemoreflex was shortened from about 10 to 2 seconds. This shortening only occurred when a specific type of serotonin receptor was activated, the 5-HT₃ receptor.

Prof James Leiter, from the Geisel School of Medicine at Dartmouth and lead investigator of the study explained, "Serotonin is important in arousing infants and restoring regular breathing to end apnoeic events when regular breathing is interrupted. Apnoeic events are common in babies, even in healthy babies. Babies seem to be more susceptible to reflexes that suppress breathing, and they need internal processes that stop these apnoeas and restore normal breathing.

"As serotonin seems to be important in the processes that stop apnoea and restore normal breathing, the idea has emerged that the serotonergic deficiency, which many babies who die of SIDS or die of asphyxia seem to have, prevents effective arousal and recovery of normal breathing after apnoeic events.

"Many of our experiments have replicated previous human findings and tended to confirm in animals what was already known or suspected in human babies. This is the first time I think that animal studies have taken the lead in SIDS research, so I found the work quite rewarding. But there is always more to do. First, our colleagues are trying to determine whether the 5-HT₃ receptor is altered in babies who died of

SIDS. At the same time, we are trying to find out where the serotonin might originate from in other parts of the brain. We are looking in regions that are already known to be part of the arousal process to see if activation of these areas can shorten the laryngeal chemoreflex.

"Also, we are trying to create conditions during pregnancy in rats that simulate what we believe occurs in human babies during pregnancy that makes these babies susceptible to apnoea by interfering with their [serotonin](#) system during development. Finally, we are trying to figure out how best to adapt drugs for use in [human babies](#) that interact with reflex apnoea and arousal responses so that they may be studied and used effectively to prevent SIDS and asphyxial deaths in babies."

More information: Donnelly WT, Bartlett D, Leiter JC (2016) Serotonin in the solitary tract nucleus shortens the laryngeal chemoreflex in anesthetized neonatal rats. [DOI: 10.1113/EP085716](https://doi.org/10.1113/EP085716)

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