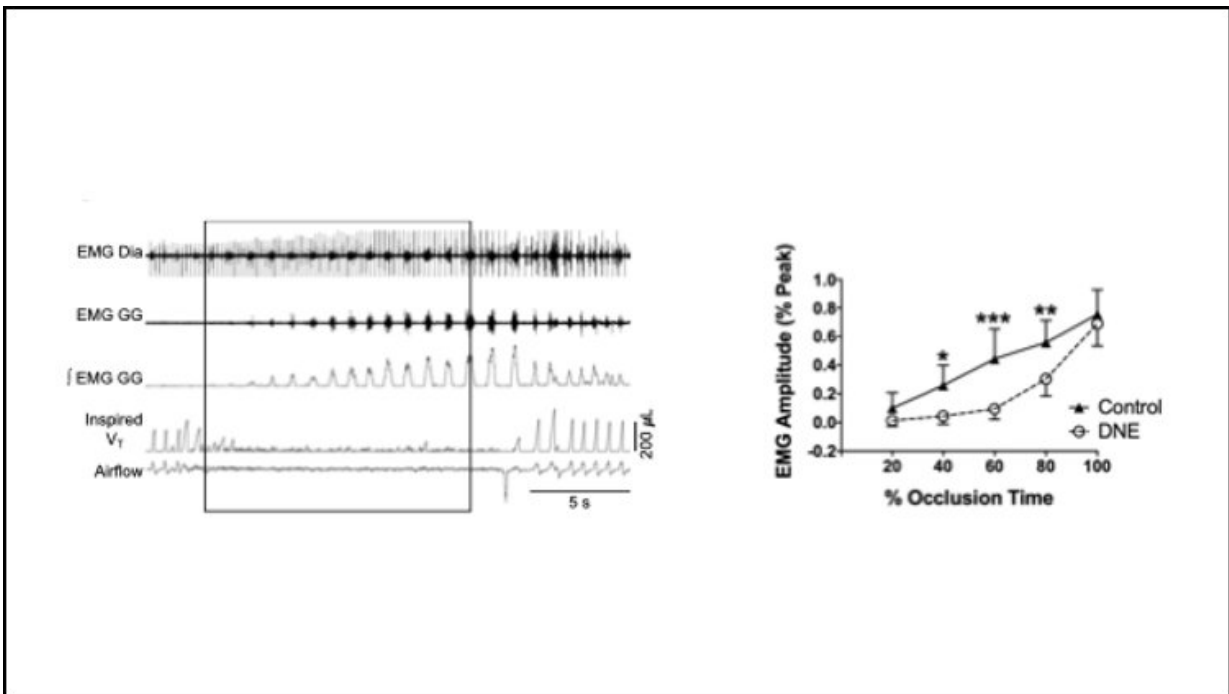


Fetal nicotine exposure harms breathing in infants

November 11 2019



Whole muscle electromyographic activity in control and nicotine-exposed rats.
Credit: Wollman et al., *eNeuro* 2019

Exposure to nicotine during development inhibits the function of neurons controlling the tongue, according to research in newborn rats recently published in *eNeuro*. This impairment may be a factor in sudden infant death syndrome in humans.

The hypoglossal nerve activates the muscles of the tongue. Wollman et al. exposed rats to nicotine in the womb and during the first few days of life. The team then measured the electrical activity of hypoglossal neurons during normal breathing and when breathing is stressed.

Normally, when breathing is obstructed, such as a blocking an animal's nostrils, a flood of excitatory neurotransmitters activate the hypoglossal neurons to move the tongue and open the airway. At rest, hypoglossal neurons in the rats exposed to nicotine behaved normally. However, during the breathing stressor test, the neurons from nicotine-exposed rats did not respond as robustly as those in the control rats, leading to difficulty breathing.

The researchers posit fetal and early life [nicotine](#) exposure in human babies could weaken their respiratory reflexes, increasing their susceptibility to [sudden infant death syndrome](#) in the face of a [breathing](#) obstruction.

More information: Developmental Nicotine Exposure Alters Synaptic Input to Hypoglossal Motoneurons, and is Associated With Altered Function of Upper Airway Muscles, *eNeuro*, [DOI: 10.1523/ENEURO.0299-19.2019](#)

Provided by Society for Neuroscience

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