Robert Dumaine, J Clin Exp Cardiolog 2017, 8:11 (Suppl)
DOI: 10.4172/2155-9880-C1-082




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## Role of nicotine in cardiac arrhythmias leading to sudden infant death syndrome

**Goal of the Presentation:** We will present evidence showing that in- utero exposure to nicotine creates a substrate for arrhythmias leading to sudden infant death syndrome (SIDS). Our goal is to raise awareness against the use of nicotine replacement therapies in pregnant women.

**Background:** Sudden infant death syndrome (SIDS) is the leading cause of death in the first year of life. In-utero exposure to tobacco smoke is observed in 85% of SIDS cases and considered the highest risk factor. Therefore, nicotine replacement therapies are viewed as healthier alternatives to tobacco consumption and often prescribed to women who wish to quit smoking during pregnancy. However, of the 3000 toxic or carcinogenic compounds known to be present in tobacco smoke only tobacco glycoprotein (TGP) and nicotine were consistently linked to SIDS. While TGP triggers an anaphylactic response 3, only nicotine is associated to cardiac arrhythmias in newborns 4-10. Evidence linked SIDS to a failed coordination of the cardiovascular and respiratory systems during the postnatal development of the heart thus causing cardiac arrhythmias and sudden death 11-13. Among the hypothesis to explain SIDS is the failure of the newborn heart to accelerate at the onset of apnea and trigger awakening during sleep. In this talk we will present data showing that in-utero exposure to nicotine delayed the development of the heart conduction system and reduced the cardiac response to epinephrine. More specifically, our data show nicotine reduced the innervation of the sinoatrial node and the response of the cardiac sodium current responsible for triggering the ventricular action potential and conduction of the electrical impulse within the heart.

**Conclusion & Significance:** Our results are consistent with the hypothesis that SIDS babies lack the cardio-respiratory reflex that accelerates the heart at the onset of apnea and may explain why some newborn infants do no awake during sleep apnea. The data provide a basis to explain the bradycardia and conduction anomalies observed in resuscitated SIDS infants and arrhythmias leading to crib death. Finally, our data raise awareness on the use of nicotine replacement therapies in pregnant women.

### Biography

Robert Dumaine initiated and managed the Cardiac molecular genetic program at the Masonic Medical Research Laboratory (NY) from 1996-2004 and was Director of the department of Physiology and Biophysics at the University of Sherbrooke from 2004-2009. He is now full Professor at the department of Pharmacology and Physiology at the Univ. de Sherbrooke Qc. Canada. His expertise is on cardiac arrhythmias linked to potassium and sodium ion channel defects. His major contribution includes the discovery of arrhythmogenic mechanisms causing inherited and acquired forms of long QT syndrome, short QT syndrome and Brugada syndrome. In collaboration with P. Schwartz laboratory he published the first study linking SIDS to cardiac sodium channel defects. He pioneered research on the role of non-cardiac sodium channels in heart function and recently showed that overexpression of neuronal sodium channels in the heart could explain the QT prolongation and some of the arrhythmias observed in SUDEP and SIDS.

**Notes:**

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