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| **ISA ISPID  Abstract Submission  Nº: 185**   |  | | --- | | Topics: **SIDS/SUID** | | Type: **Oral** | | **Apoptotic (caspase-3 and TUNEL) and Interleukin 1b expression in the Sudden Infant Death Syndrome (SIDS) hippocampus and subiculum** | | **Vivekanandarajah, Arunnjah**1; **Waters, Karen A**1; **Machaalani, Rita**1 *1 - University of Sydney.* | | **Introduction** Previous work in a Canadian population of SIDS infants1 showed that apoptosis (defined as cell death), detected by labelling for DNA breaks (TUNEL), is increased in the CA4 of the hippocampus and the subiculum. Recent work2 has shown that other abnormalities exist within the SIDS hippocampus making it a site worthy of further investigation. Interleukin 1b (IL-1b) is an excitotoxic cytokine that mediates inflammatory reactions, and is increased under hypoxic conditions and subsequently induces apoptosis3. To compare neuronal expression of the specific apoptotic markers TUNEL, active caspase 3 (Casp-3) and IL-1b expression in the Dentate Gyrus, and Cornu Ammonis (CA) 1, CA2, CA3, CA4 regions of the hippocampus, the subiculum and the entorhinal cortex in SIDS and non SIDS infants.  **Material and Methods** Immunohistochemistry was performed for IL-1b expression and double Immunohistochemical analysis for Casp-3 and TUNEL colocalisation on infants diagnosed with SIDS (n=23) and age matched non SIDS (n=9). Qualitative quantitation was performed blinded to the study group using the Leica DM 6000 Nikon Upright microscope. Analysis by the two common risk factors of sleep position and cigarette smoke exposure were further performed.  **Results** SIDS infants had increased TUNEL expression in the subiculum (p=0.024) and increased IL-1b expression in the CA1 region (p=0.040) compared to non-SIDS. No significant changes were seen for active caspase-3. By risk factor analyses, SIDS infants found in the prone sleep position had greater Casp-3 expression in the CA3 region (p=0.016) and IL-1b in the CA1 region (p=0.032). SIDS infants with a history of cigarette smoke exposure had greater Casp-3 expression in the CA2 (p=0.012), CA1 (p=0.019) and subiculum (p=0.002) and TUNEL/Casp-3 colocalisation in the CA3 region (p=0.031).  **Conclusions** The preliminary findings of this study support the findings of previous research of neuronal loss in the hippocampus of SIDS and provide evidence for the first time of a role of IL-1b, thus suggesting the potential role of the CA1 and subiculum in altered neural circuit activity in these infants which may be contributing to the developmental vulnerability in these infants. The finding that the prone sleep position increased IL-1b in the CA1 suggests a hypoxic mechanism behind this increase. Further, cigarette smoke exposure seems to add its own effect seemingly inducing the active apoptotic pathway across most of the hippocampus. Current work will be looking at these markers in our larger dataset and performing in depth correlation analyses with other risk factors. **Funding:** Miranda Belshaw Foundation and The SIDS Stampede.   **References:** 1. Waters, KA, et al., 1992. Pediatr Res. 45, 166-72. 2. Kinney, HC, et al., 2015. *Acta Neuropathologica*, *129*, 65–80. 3. Sun, W, et al., 2014. Cell Death Dis. 23,5. | |  |  |  |  | | --- | --- | | **CONTACT** | | | Name: | **Arunnjah** | | Lastname: | **Vivekanandarajah** | | E-mail: | **aviv2617@uni.sydney.edu.au** | | Country: | **Australia** | | Institution | **University of Sydney** | | Cellphone: | **+61449874303** | | City: | **Sydney** | |