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| **Can the Statistical Behavior of SIDS Tell us About it's Likely Causation?** |
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| **Introduction** The Sudden Infant Death Syndrome (SIDS) is not likely to be explained by universal presence in cases and absence in controls, as otherwise it would have been solved long ago. Indeed any proposed model for SIDS causation must explain the constant mathematical and statistical properties of SIDS. We test whether SIDS is more closely related to a fulminating prodromal acute respiratory infection (ARI) or to fetal-derived prematurity in serotonergic system brain cells leading to a fatal hypoxic-ischemic encephalopathy (HIE).  **Material and Methods** SIDS are characterized by a common lognormal age distribution sparing neonatal infants, by a nominal 50% male excess, and by a common increasing rate with the infants increasing Live Birth Order (LBO) and a seasonal winter maxima, all remaining the same, independent of the change in preferred sleeping positions of the infants, prone or supine. We use U.S. published infant mortality data from wonder.cdc.gov and other countries (Colombia, Argentina, U.K., Europe, Australasia) to make comparisons between the two causes of death (ARI and HIE) to evaluate which most resembles the characteristics of SIDS.  **Results** GENDER: SIDS male excess 50%, ARI male excess 50%, HIE male excess 10%;  AGES: SIDS 90% post-neonatal, ARI 96% post-neonatal, HIE 7% post-neonatal; LIVE BIRTH ORDER: SIDS rate increases with increasing LBO as fit by a probability model for equal numbers of infants at risk at each LBO as  (1 - 0.9^CFM) where CFM (cohabiting family members) equals 2 Parents + number of older siblings =   (LBO + 1).    Here 0.9 is the probability of a CFM *not* carrying a respiratory infection transmissible to the infant and 1 - 0.9^CFM represents the probability that at least one of the CFM *is* a carrier; ARI rate increases with LBO by the identical model; HIE rate decreases from LBO = 1 to LBO = 2, and remains relatively constant until LBO ≥ 6 when it rises; SEASONALITY: SIDS and ARI both peak in winter, HIE does not.  **Conclusions** Our results show that all SIDS are more likely relatable to a fulminating prodromal ARI in an infant who is genetically (X-link recessive) susceptible to acute cerebral anoxia resulting in encephalopathy of respiratory control neurons. The cause of SIDS death by fetal neurological immaturity leading to HIE, commonly called the Triple-Risk Model, is not supported because it explains neither the fixed age-gender distributions of SIDS, nor the increasing SIDS rate with increasing CFM. |
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