1. Introduction

Sudden Infant Death Syndrome (SIDS) is the leading cause of postneonatal infant death in the United States with an incidence rate of 0.39/1000 live births [1]. The triple risk model for SIDS posits an infant dies of SIDS when 3 factors converge; an infant with a biological abnormality passes through a critical developmental window (1st year of life), and faces an exogenous stressor (Fig. 1). Our laboratory is specifically interested in abnormalities within the serotonergic system of the brainstem regions critical to respiratory and autonomic regulation [2].

Epidemiological and pathological evidence suggests a role for risk factors that potentially elicit an inflammatory response within the brain (i.e., illness prior to death and hypoxia). To begin to examine the relationship between neuroinflammation and brainstem abnormalities, we first tested the following hypothesis: SIDS, or a subset of SIDS, involves central nervous system inflammation, as identified by increased levels of the cellular immune system marker, neopterin, in the cerebrospinal fluid (CSF).

2. Materials and Methods

3. Results

3.1. SIDS with elevated levels of CSF neopterin

4. Discussion

5. Conclusion

6. Appendix