

# **Sudden Infant Death Syndrome (SIDS) related to Solar Activity through the Schumann Resonance mechanism.**

**Dr Neil Cherry  
Associate Professor of Environmental Health**

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**Neil.Cherry@xtra.co.nz**

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**Human Sciences Department  
P.O. Box 84  
Lincoln University  
Canterbury, New Zealand**

## **Sudden Infant Death Syndrome (SIDS) related to Solar Activity through the Schumann Resonance mechanism.**

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### **Abstract:**

A significant homeostatic relationship from Geomagnetic Activity (GMA) and Sudden Infant Death Syndrome (SIDS) was found in Ontario, Canada. A similar relationship is plausible elsewhere, but only if the size of the population produces sufficient daily SIDS cases to allow investigation of the daily rates. In Christchurch, New Zealand the population is too small so the monthly SIDS and sunspot relationship was studied to determine if the links between solar activity and SIDS rates, through the Schumann Resonance mechanism, were identifiable. Solar activity through the Schumann Resonance mechanism reduces melatonin in human populations. Reduced melatonin is associated with increased risk of SIDS. Significant correlations were found for adaptive homeostatic “U” and inverse “U” patterns between solar activity and SIDS over prolonged periods of months during 8 years, which included sunspot maximum, sunspot minimum and the transition period.

### **Introduction:**

Sudden Infant Death Syndrome (SIDS-Cot death) was a relatively common factor for infant mortality rates around New Zealand in the 1970s and 80s. The weather and social factors were found to be related to SIDS, including maternal smoking, social deprivation and sleep during winter months, Williams, Mitchell and Taylor (2002) and postnatal depression, Sanderson et al. (2002). These health effects and SIDS are linked to reduced melatonin levels, Sturner et al. (1990), and reduced melatonin from the pineal gland and infants who had died from SIDS had significantly smaller pineal glands, Sparks and Hunsaker (1988) and Sparks et al. (1997). Solar and Geomagnetic Activity (GMA) are causally linked to the reduction of melatonin through the interaction of the Schumann Resonance signal with the human brain, Burch et al. (1999) and Cherry (2002, 2003 a,b,c). Therefore S-GMA is plausibly linked to increased rates of SIDS. O'Connor and Persinger (1999) observed a significant homeostatic relationship between GMA events and SIDS in Ontario Canada. Homeostatic relationships are basic fundamental biological functions. They are typically shown as “U” or inverse U - “∩” patterns, including the core temperature of mammals which have an optimal temperature and health effects become worse above and below the optimal temperature level (“U”). Plant’s temperature related growth rates are maximum at an optimal temperature and lower above and below the optimal temperature (“∩”).

### **SIDS data-set:**

The data-set of the daily incidence of SIDS cases from Christchurch, New Zealand for the period 1979 to 1986 provided 223 cases, ranging from 0 to 8 per month, 24 to 35/year, with only 7 daily events with 2 SIDS/Day and only one with 3 SIDS/Day. The

dominant daily rate is zero. Hence even many of the monthly rates have zero values. The mean monthly incidence rate over the 8-year period is shown in Figure 1.

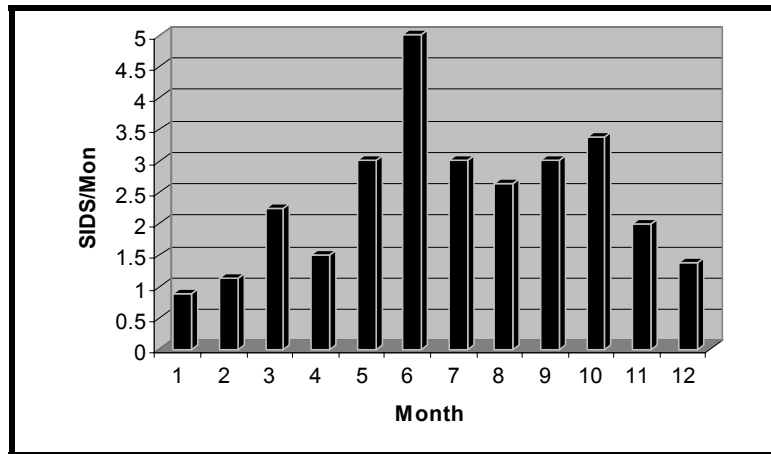


Figure 1: The average monthly SIDS rate in Christchurch, New Zealand over the period, January 1979 to December 1986.

The annual SIDS rate, Figure 2, shows a slight rising trend and a large inter-annual variation. For this period the sunspot maximum occurred in late 1979 and sunspot minimum in 1986. The highest monthly SIDS rates, over 3.5, were observed in the winters near sunspot maximum, in 1980, and during sunspot minimum in 1986.

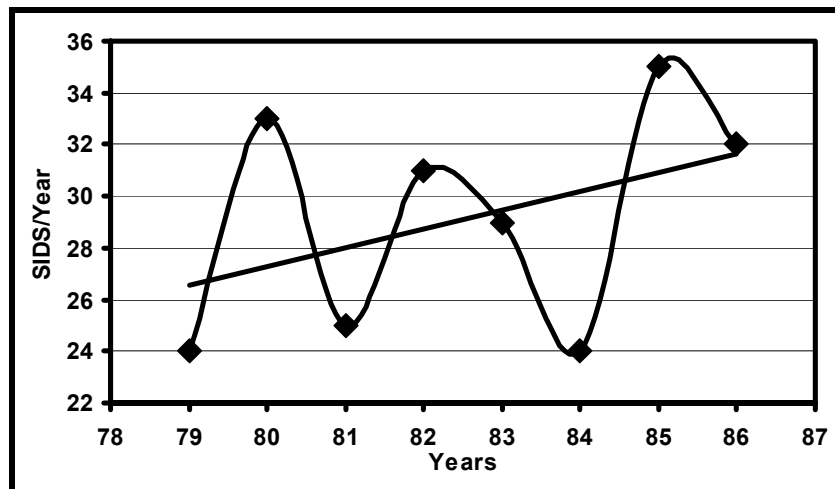


Figure 2: The annual SIDS rates for Christchurch, New Zealand over the period, 1979 to 1986.

Because of the relatively small monthly incidence rate, a 3-month running mean smoothed time-series was used for the SIDS rate and the sunspot number. The SIDS rate was adjusted for the monthly variation, Figure 1 and the trend, Figure 2, in an attempt to remove some of the social and local climate factors. The data was then analysed to test whether there was adaptive homeostatic patterns already found for cardiac illness and suicide rates, Cherry, Larson and Wilson (2003) and Cherry (2003b).

To evaluate the effects of the data smoothing and adjustment, during the transitional declining sunspot period, February 1984 to August 1985, the raw data (whole

numbers) was compared with the monthly sunspot number, Figure 3, and a significant “ $\cap$ ” curve relationship,  $p < 0.005$ , was found.

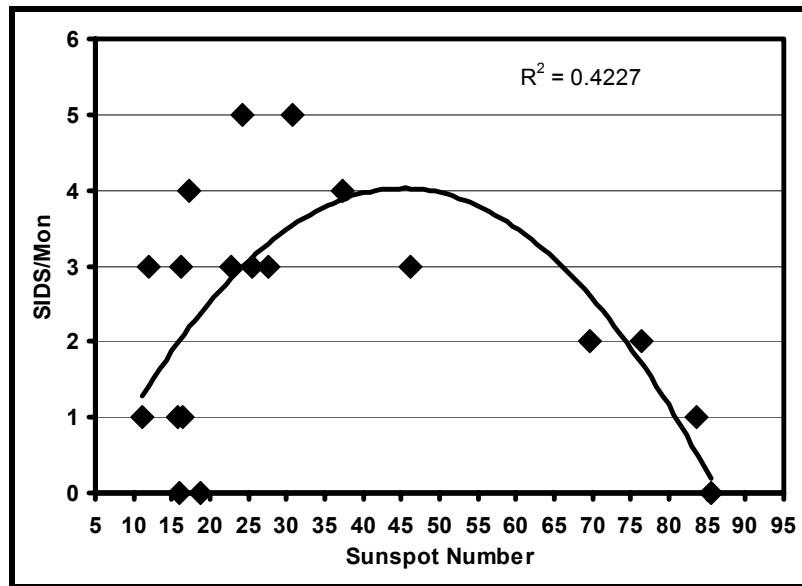


Figure 3: The raw SIDS monthly rates vs Sunspot Number during a transitional sunspot period a few years after sunspot maximum, February 1984 to August 1985. “ $\cap$ ” curve trend  $p < 0.005$ .

The adjusted and smoothed data from the period used in Figure 3 produces the same but slightly smoother and a little more significant,  $p < 0.001$ , “ $\cap$ ” pattern, Figure 4, with the months with zero cases raised and 5 cases reduced, primarily from the monthly seasonal adjustment.

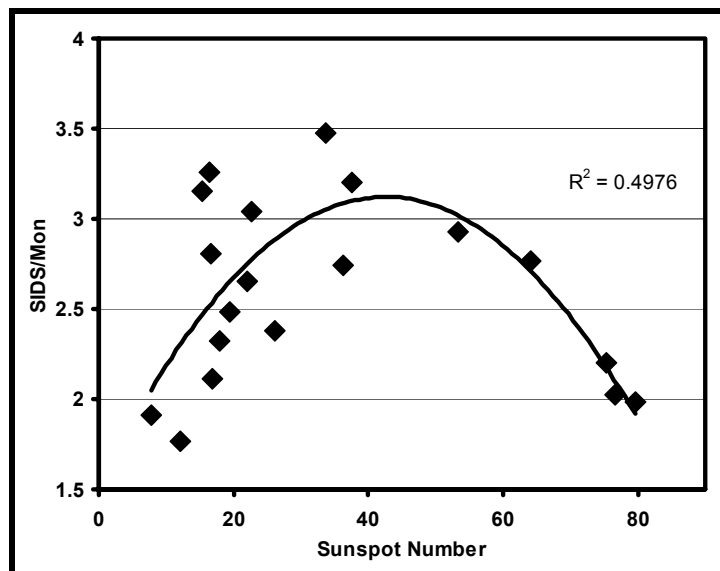


Figure 4: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during a transitional sunspot period, February 1984 to August 1985, “ $\cap$ ” curve trend,  $p < 0.001$ .

Figures 3 and 4 show significant “ $\cap$ ” homeostatic relationships between SIDS and solar activity, with both very low and higher solar activity during this period being associated with much lower SIDS monthly rates. The scatter is quite large but

expected from the small data-set. The rising SIDS trend from right to left is associated with particular periods of decreasing solar activity, which shows an extremely significant “∩” trend,  $p < 0.0000001$ , Figure 5(a). This reveals a very close homeostatic relationship between Solar Activity and SIDS. The prolonged low sunspot falling trend on the left half of Figure 4 is a period of oscillating small sunspot activity that has a lot of scatter, but it still has a significant trend,  $p < 0.02$ , Figure 5(b).

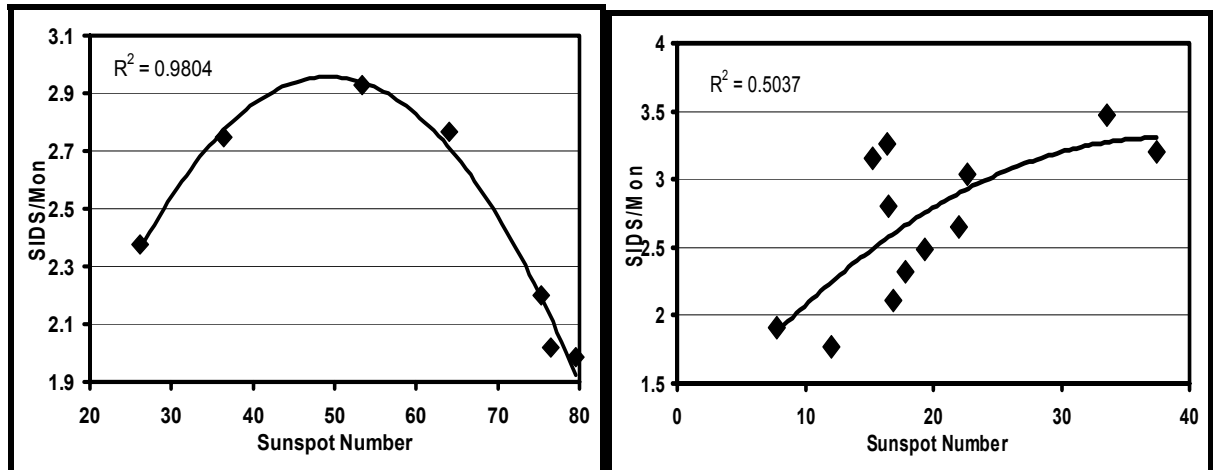


Figure 5: The homeostatic dose-response relationships between SIDS and sunspot numbers for (a) a time series of declining sunspot activity, left, Feb – Aug 1984, “∩” curve trend,  $p < 0.0000001$  and on the right (b) the following period of oscillating low sunspot activity, Sept 1984 to Aug 1985, “∩” curve trend,  $p < 0.02$ .

Figure 6 shows the time series of the smoothed monthly sunspot numbers and SIDS rates for the first four and a half years, showing the maximum sunspot in late 1979 with the declining trend. This shows an inverse relationship to SIDS for very high solar activity, then a positive relationship followed by an inverse relationship. The dose-response for these three periods is given in Figure 7. A later period, associated with moderately high sunspot activity, is shown in Figure 8.

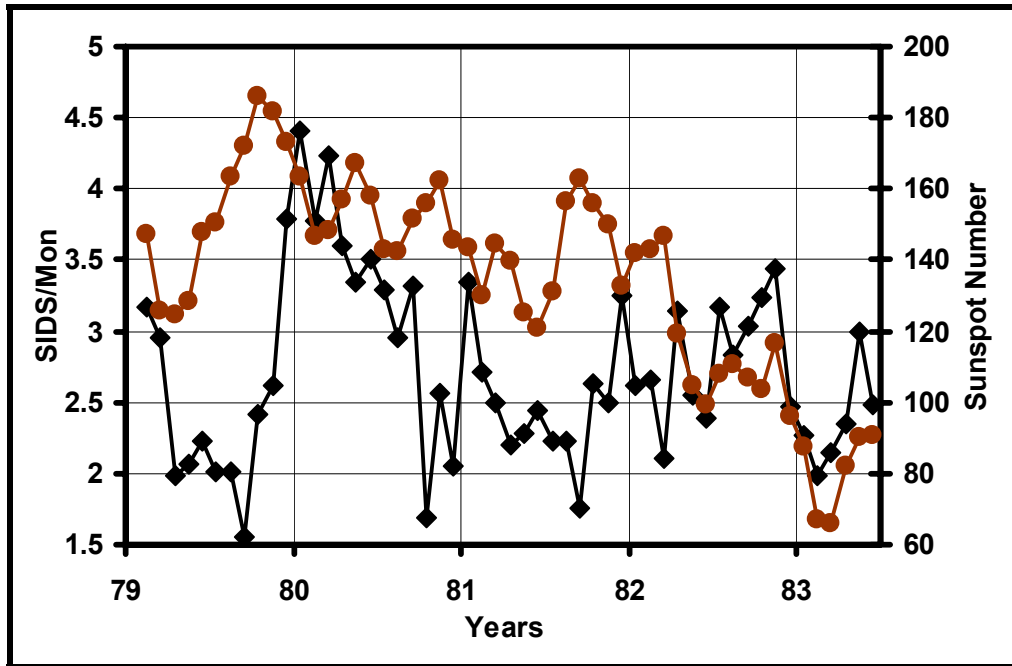


Figure 6: Time series of seasonal and trend adjusted SIDS (black) and sunspot number (red), 3-month smoothed mean rates, for the period from February 1979 to June 1983.

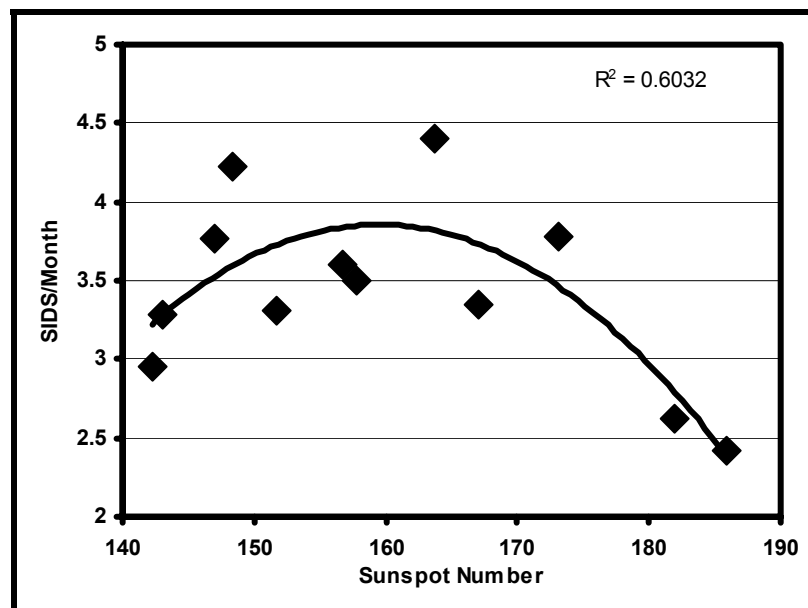


Figure 7: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during a high and declining sunspot period, August 1981 to April 1983, “∩” curve trend,  $p < 0.0002$ .

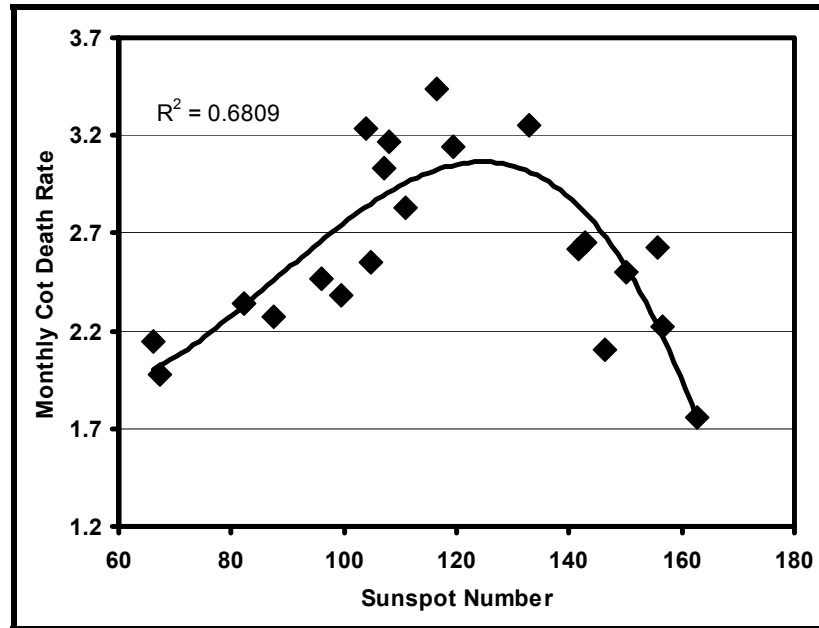


Figure 8: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during a moderately high declining sunspot period, March 1981 to March 1983, “∩” curve trend,  $p < 0.00002$ .

Both Figures 7 and 8 show highly significant inverse “U” dose-response relationships between solar activity and monthly SIDS rates, each covering 18 months to 2 years, showing an adaptive change with an initial peak around 160-170 sunspots, and the second period, the peak around the mean sunspot activity of 120-130 sunspots. The smaller sunspot number period is in the last year period of this data shown in Figure 6. Adding the declining lower sunspot period from April 1982 and adding the further declining sunspot period to April 1984 shows a significant “U” dose-response relationship,  $p < 0.0001$ , Figure 9.

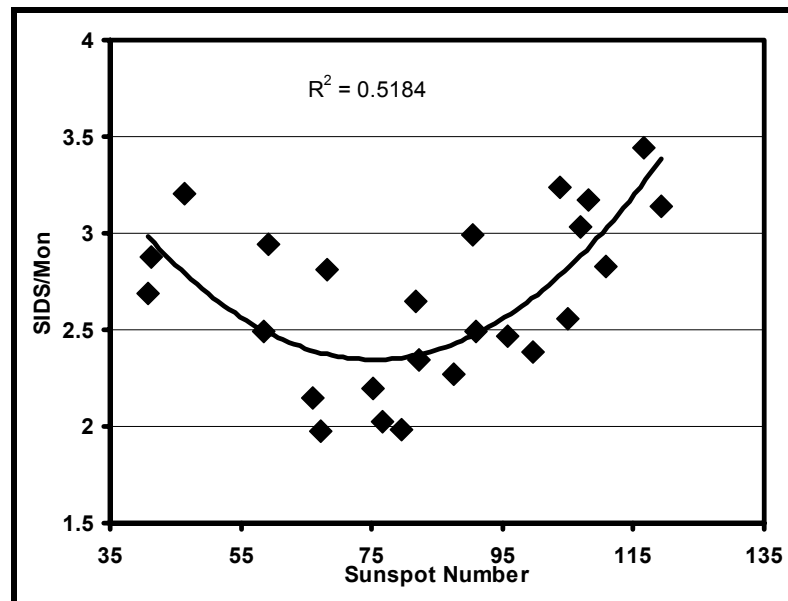


Figure 9: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during a transitional sunspot period, April 1982 to April 1984, “U” curve trend,  $p < 0.0001$ .

Figure 10 shows the time-series rates of SIDS and sunspot number for the final 4 years, 1983 to 1986.

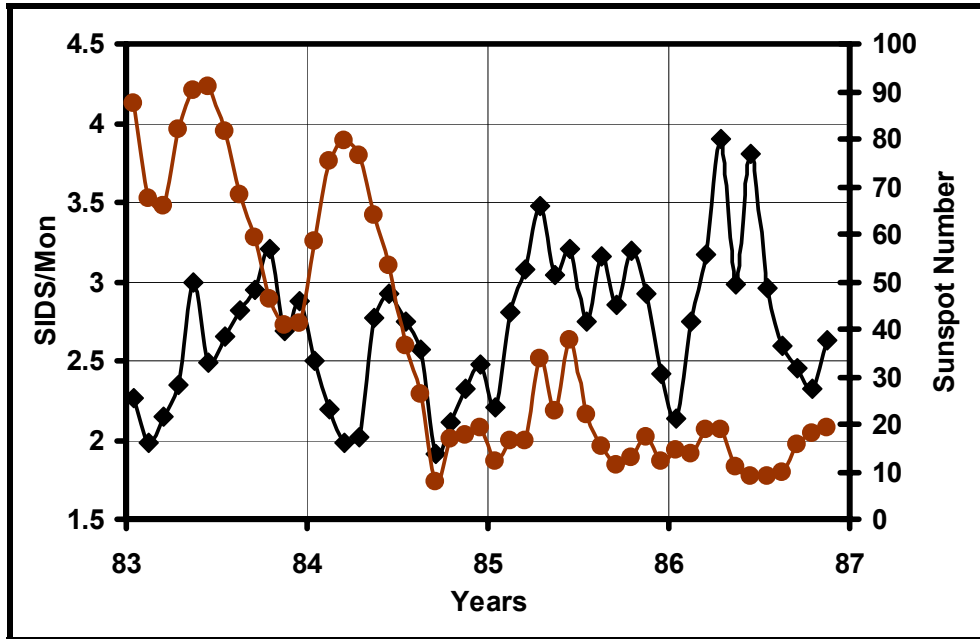


Figure 10: Time series of 3-month smoothed mean, seasonal and trend adjusted rates of SIDS (black) and sunspot number (red), for the period from January 1983 to November 1986.

For the major drop of solar activity during 1983/84 in Figure 10, the period August 1983 to January 1985, shows an extremely significant “ $\cap$ ” curve pattern,  $p < 0.0000001$ , Figure 11. For the low sunspot activity from August 1984 to July 1985, a positive correlation is visible in Figure 10, producing the significant trend,  $p < 0.01$ , Figure 12. For the very low solar activity under 20 sunspot numbers the SIDS rate for the final year is “U” pattern marginally significant relationship,  $p < 0.05$ , Figure 13.

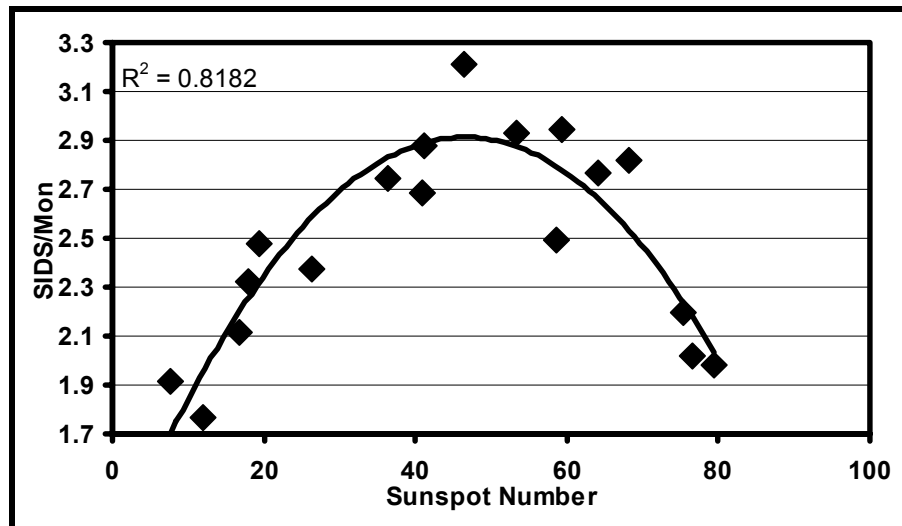


Figure 11: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during a transitional sunspot period, August 1983 to January 1985, “ $\cap$ ” curve trend,  $p < 0.0000001$ .



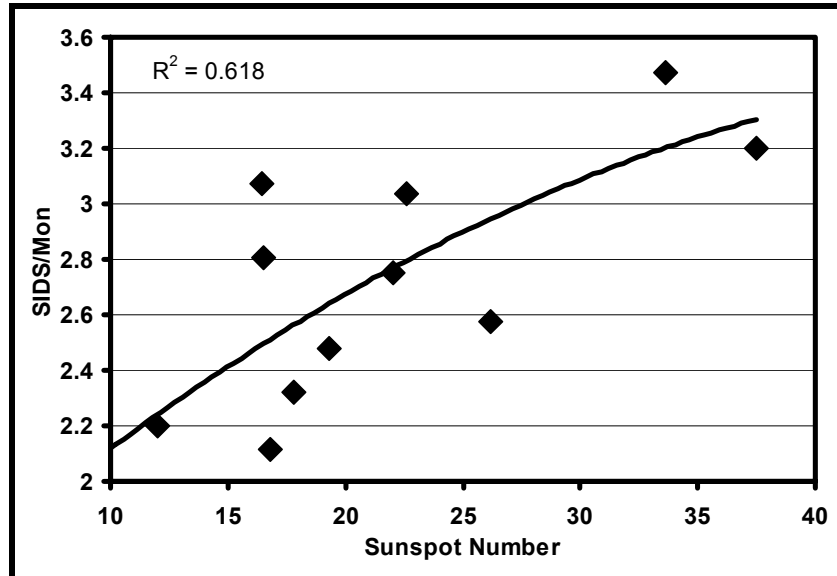


Figure 12: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during the low sunspot, 10-35, period August 1984 to July 1985, trend,  $p < 0.01$ .

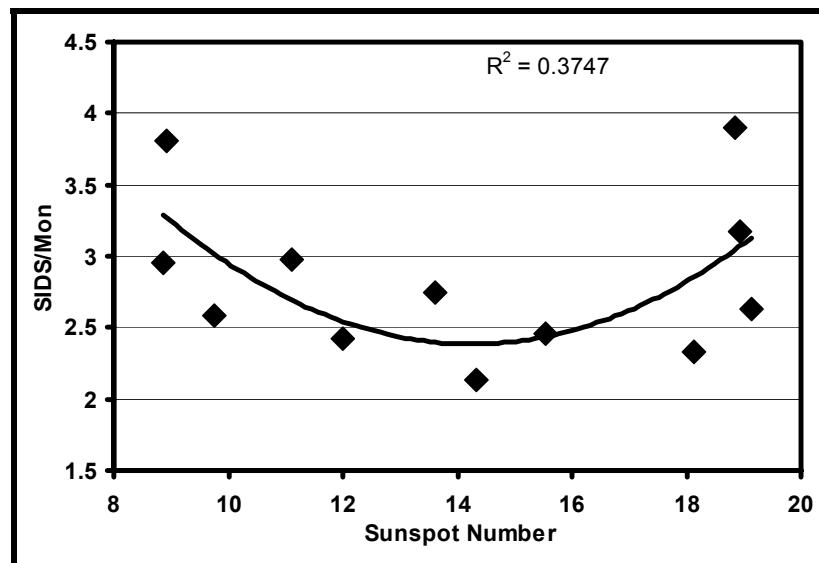


Figure 13: The 3-month running mean, seasonal and trend removed SIDS monthly rate and sunspot number during the very low sunspot period, December 1985 to November 1986, "U" curve trend,  $p < 0.05$ .

During the low sunspot activity around sunspot minimum during 1986 a significant "U" shaped homeostatic relationship exists between the monthly SIDS and sunspot number rates.

### Conclusions:

Sudden Infant Death Syndrome is shown by this analysis to have significant homeostatic patterns that are adapted year by year over the solar cycle from sunspot maximum to sunspot minimum. Since babies are conceived and born at particular stages of the solar cycle it is reasonable that they will be more quickly and highly

adaptive than adults. The results occur even though the data-set is quite small. The dose-response “U” and “∩” curves show considerable scatter but have clear, consistent and significant patterns.

Of course, it is not the sunspots on the surface of the sun that directly cause human health effects, such as SIDS. So, how do spots on the sun cause Sudden Infant Death Syndrome on earth? The sunspot number is a measure of solar activity that produces ionizing particles, alpha and beta particles, and electromagnetic radiation of X-Rays, microwaves and UV radiation. The X-Rays ionize the lowest layer of the Ionosphere, the D-Region, Brekke (1997). The D-Region has an extremely significant diurnal solar cycle, solar flare and 11-year sunspot cycle variations, Craig (1965). The height of a particular electron density in the D-Region modulates the intensity of the global Schumann Resonance signals, which have a strong diurnal cycle and are closely related to solar storms and sunspot activity, Cherry (2002, 2003a). Human brains, including infants, detect and respond to the Schumann Resonance signals, with one reaction that reduces the pineal gland output of melatonin, Cherry (2002, 2003a,b,c). This causes human health effects including SIDS as this study confirms. The analysis also confirms that the long-term changes over the solar cycle are adaptively homeostatic.

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