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The light of life: Evidence that the sun modulates human lifespan ☆

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Summary This paper describes the effects of radiation, probably ultraviolet radiation (UVR), on the human genome at peaks of solar cycles. This phenomenon was not previously reported because peak cycle lifespan had not been separated from non-peak lifespan. This paper reinforces the findings of others regarding the seasonality of various diseases and that there are factors occurring early *in utero* that increase susceptibility to diseases later in life. The authors use the vital statistics of 320,247 Maine citizens over a 29-year period to show that those born in 3-year peaks of 11-year solar cycles live an average of 1.5 years (CL 1.3–1.7) less than those born in non-peak years. Males are more sensitive than females to this phenomenon, which is statistically demonstrable well into adult life, showing the effect of probable UVR on the early human embryo despite superimposed adult lifetime hazards. The authors also show that changes in seasonal light modulate lifespan differently in males and females and that genome and environment must be tightly interactive early after conception. Published literature supports the hypothesis that UVR suppresses the maternal immune system by producing cytokines in circulating lymphocytes that probably affect the fetal genome.

The intermittent and incompletely predictable solar cycles periodically stress the genomes of all life producing genetic changes which may be harmful or adaptive. The evidence presented in this study indicates that solar cycles, particularly the most irradiant which have occurred over the past 65 years, are fundamental engines of evolution, even underlying natural selection, and we bear their marks even to the end of our lives. Future researchers must further define the pathogenesis of solar radiation on early embryonic development to possibly minimize a predisposition to diseases at their origin.

Background/purpose: This study explores the relationship of season of birth and human lifespan particularly in reference to the intensity of solar radiation that occurs in 11-year cycles.

Methods: The birth years were obtained and lifespan calculated for 320,247 Maine citizens over a 29-year period. Those who were born at 3-year peaks of 11-year solar cycles were separated from those born in non-peak years. Using SAS statistical tools, a randomization technique was used to compare the lifespan between peak and non-peak years to eliminate selection bias, cohort effects, and confounding variables.

Results: Those born in peaks of solar cycles lived an average of 1.5 years (CL 1.3–1.7) less than those born in non-peak years. Males were more sensitive to this phenomenon than females. A similar analysis was performed for month of birth and the pattern of peak to non-peak lifespan difference was nearly identical to the pattern of seasonal variation in light.

☆ Persons born at peaks of 11-year solar cycles lose an average 1.5 years of lifespan.

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Conclusions: Lifespan variation appears to be modulated by seasonal light confirming that genome and environment are closely linked very early after conception. Although the precise pathogenesis is still unknown, the phenomenon must involve radiant energy, probably ultraviolet light, possibly affecting the maternal immune system through the dermis. This study also supports the reliability theory of aging which suggests that events affecting the genome early after conception are important in the expression of adult diseases.

Introduction

There are many articles in the medical literature regarding the effect of sunlight on human disease, much involving malignancy of the integument, but also increasingly about a relationship between month of birth (e.g., seasons) and a variety of developmental, metabolic and malignant diseases [1—10]. Many have argued that this relationship is weak, although the most compelling of these studies involves schizophrenia where there is a 5—8% bias in favor of late winter and early spring birth, a phenomenon unexplained to date [2]. One would expect that if diseases are affected by seasons, lifespan should also be affected. Gavrilov reported a differential lifespan relating to month of birth for 4911 aristocratic women from 19th century Europe. He found that those born in the months of May had longer lives by an average of 3.61 years (CI 2.68—4.54), compared with those born in August [11]. Juckett in a study of 5300 US Congressional Representatives, 2336 House of Commons members and 12,900 University of Cambridge alumni (all males), has suggested that the Sun may also affect lifespan [12,13]. He reported that those born in solar cycle peak years had a 2—3 year shortening of lifespan compared to those born between peaks. More recently, Marzullo reported that the photoperiod, which varies widely over the seasons, coupled with oxidant stress, affects the human embryo early in gestation [14]. The implication in all these studies is that light, in some manner, affects the human genome [15].

The term ''solar cycle'' refers to the periodic rise and fall of the intensity of solar radiation. Sunspots, dark regions on the surface of the Sun and manifestations of magnetic storms therein, have been counted for centuries and are considered a proxy for the intensity of solar radiation. Observations over many years have shown that sunspot numbers vary on average every 11.1 years (range: ~9—14 years) double-peaking for about three years in each 11-year cycle. Hathaway and others have developed algorithms to predict the intensity of solar cycles [16]. However, the day-to-day accuracy of prediction of solar storms can be predicted accurately only 72% of the time [17]. There is also recent evidence that the Sun has been particularly active over the past 65 years [18].

In this paper we use sunspot numbers as a direct measure of potentially mutagenic radiation to the human genome. Damage to this genome is likely proportional to the incidence of disease and hence ultimately to lifespan. The most damaging wavelength of radiation to DNA is in the ultraviolet range, the most biologically significant being 308 nm (UV-B) [19]. UVR is at least an order of magnitude more damaging to DNA than other ambient ionizing radiations and DNA itself is so linearly sensitive to UVR that it is used in ultraviolet dosimeters [20].

Our own previous reports have hypothesized a limit to human longevity as a result of particularly powerful solar cycles, which we termed ''chaotic solar cycles'' (CSCs), that had a mean annual sunspot (Wolf) number $\geqslant$135. We placed a maximum limit on the reproductive period of species, including humans, which would preferably encompass no more than one chaotic cycle, as two of these powerfully radiant cycles could corrupt the genome of the next generation [21]. Also, in a previous paper based on 11,252 persons, we reported that the human nervous system was particularly sensitive to solar cycle radiation, probably UVR, and we found a twofold increase in depression in those born in solar cycle peak years compared to those born in non-peak years [22]. We acquired enough evidence from the published literature along with our own findings to pursue further the relationship of solar activity to human lifespan [11,15,23].

The data

We obtained Maine Vital Statistics for deaths from 1976 to 2005, a total of 320,247 persons with birth and death dates, some born as early as the year 1870, encompassing 12 solar cycles. Lifespan of each person was calculated from the birth date to the date of death. The average age, standard deviation and cohort size by birth year are plotted and displayed in Figs. 1—3. The average number of annual sunspots per year was also collected from the NOAA Web site and the three peak years (of sunspots) of each of the past twelve cycles was obtained.

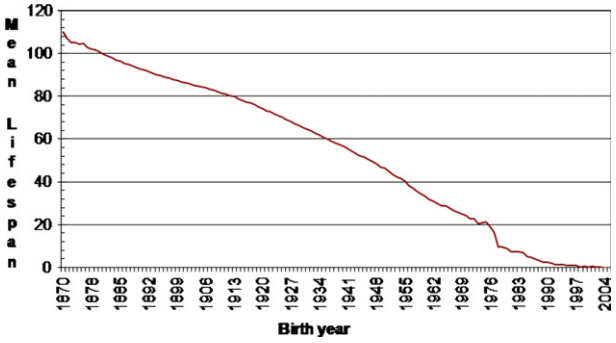

Figure 1 Mean lifespan by birth year for cohort.

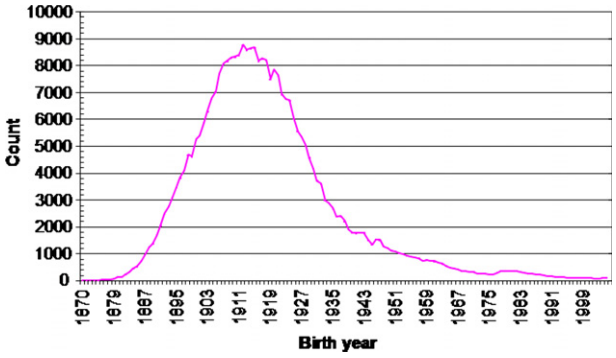

Figure 2 Count by birth year.

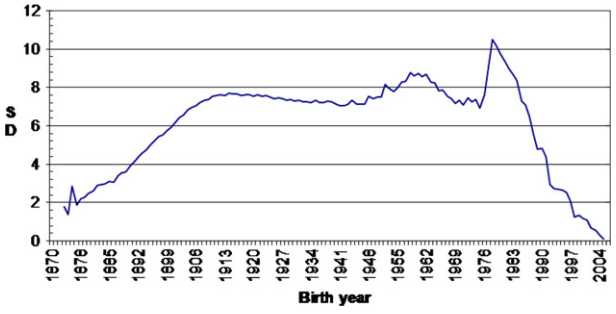

Figure 3 Standard deviation for cohort.

The average annual sunspot number for the past 250 years was found to be 49; for the past 60 years the average is 107.5; for the most powerful cycles (sunspots $\geqslant$135), the average is 154, about three times the 250-year average.

Methodology

The data were analyzed with SAS software. Records with date of birth missing (N = 21,120) or date of death missing (N = 7) were eliminated (total = ~6%). The data were grouped by birth year and the average age by birth year was calculated. To test the hypothesis that there was no difference in lifespan between solar peak years and non-peak years, solar data were defined and grouped as follows: the solar peak year and the year before and the year after the peak were used as the maximum solar period (MAX) (N = 86,811); the years before and after these 3 years were grouped as minimum solar period (MIN) (N = 233,366). We also performed a T-test for sunspot number by peaks and non-peaks; N_{max} = 36, mean = 104.50; N_{min} = 90, mean = 36.99; difference MAX—MIN = 67.51; t-value = 10.03, $p < 0.0001$.

Since sample size was differentially affected by the above grouping method and would adversely influence the overall lifespan distribution for the MAX and MIN groups, we employed the following analysis strategy. Random numbers were assigned to all cases in the data. Data was then sorted by random numbers and then grouped by MAX and MIN. A sample size of 60,000 records for both MAX and MIN groups was selected and the data for both groups randomly assigned to each group. T-tests were used to determine whether the difference between MAX and MIN groups was due to chance variation with a p value of $\geqslant 0.01$, except for June males at $p < 0.22$, March, April and September females at $p < 0.02$, and November males at $p < 0.02$. Data were analyzed for the total sample and by gender. The average annual sunspot number for each birth year was also added to each case and a correlation coefficient between sunspot number and lifespan by gender was calculated.

This study uses the term ''lifespan'' in lieu of ''longevity.'' The latter term implies a biological limit on lifespan intrinsic to a specific organism, while the former term expresses the reality of extrinsic dangers that inevitably shorten the theoretical longevity. In addition, we considered the time of conception to be ten months prior to birth month. Although normal gestation averages 266 days, we believe there is an environmentally-critical time prior to fertilization and uterine implantation which adds a week or two to the usual nine-month gestation, therefore the ten-month offset from birth month.

Results

We calculated the correlation coefficient (CC) that compared sunspot number with age at death. For males (N = 159,361) the CC was −0.31, $p \leqslant 0.001$, for females (N = 159,798) the CC was −0.28, $p \leqslant 0.001$, the latter showing how close females match solar unpredictability with males a slightly poorer match. The greater the sunspot number, the shorter the lifespan.

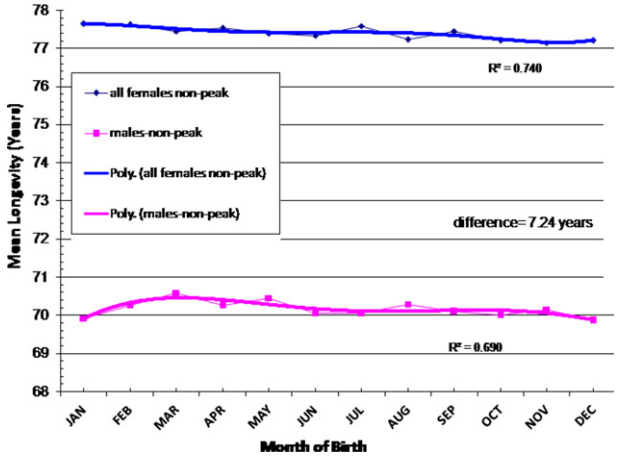

Figure 4 Female and male lifespan by month of birth at non-peaks of solar cycles.

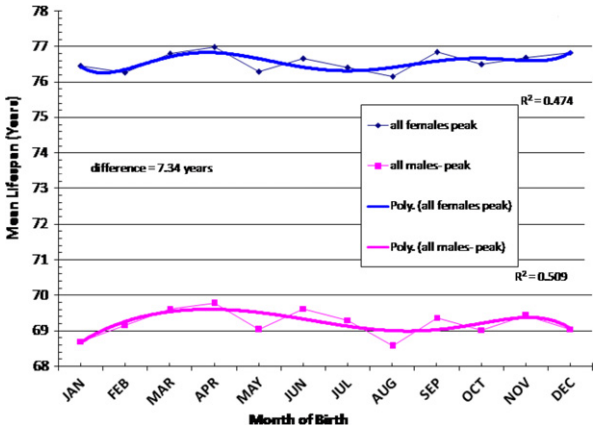

Figure 5 Female and male lifespan by month of birth at peaks of solar cycles.

The mean lifespan of persons born in peak solar cycle years was compared with those born in non-peak years. The mean difference of peak minus non-peak is −1.52 years (CL −1.73 to −1.32, $p < 0.0001$, SE = 0.10) [MAX = 60,000; mean = 72.54, MIN = 60,000; mean = 74.07, $p \leqslant 0.0001$]. That is, persons lived an average 1.5 years less if they were born in the peak years of solar radiation. We compared the lifespan of those born in non-peak and peak years by month of birth in Figs. 4 and 5. It is apparent that most of the variation in lifespan from month to month exists in the peak years, but both graphs show a consistent difference in the lifespan of males and females of ~7 years, favoring females. The correlation coefficient between males and females at peaks of cycles is 0.72 (p = <0.0001). Table 1 summarizes the mean difference by month of birth between MAX and MIN cycle years and the difference between MAX−MIN for males minus MAX−MIN for females in the last column of the table. Mean MAX−MIN for the months are statistically significantly different, $p \leqslant 0.01$, with five exceptions mentioned in Methods. The absolute value of MAX−MIN is an indication of the sensitivity to the effects of solar radiation of the persons born in that month. The last column of Table 1 is plotted in Fig. 6. The graph represents the total effect of solar radiation peaks between both genders, thereby for the entire population. Note that females appear more sensitive to solar peaks if conception occurred in rapidly increasing or decreasing light as approaching or regressing from the seasonal equinoxes, whereas the males appear more affected by extremes of light as seen in both solstices. Using integral calculus, we calculate the area of the three peaks most affecting females above the X-axis (3884 units), and the area of the two peaks below the X-axis (3027 units) most affecting males and find their ratio (3884/3027) to be 1.28:1.00 (males:females). The males comprise 56% (of the total risk, 3884/

Table 1 MAX−MIN by month of conception/birth and gender

Month of conception	Month of birth	MAX−MIN male	MAX−MIN female	Column 3 minus column 4
APR	JAN	−1.497	−1.467	−0.030
MAY	FEB	−1.659	−2.185	0.526
JUN	MAR	−2.060	−0.880	−1.180
JUL	APR	−1.327	−0.950	−0.377
AUG	MAY	−1.718	−1.209	−0.509
SEP	JUN	−0.536	−1.147	0.611
OCT	JUL	−1.366	−1.847	0.481
NOV	AUG	−2.163	−1.788	−0.375
DEC	SEP	−1.236	−0.996	−0.240
JAN	OCT	−1.796	−1.284	−0.512
FEB	NOV	−1.070	−1.700	0.630
MAR	DEC	−1.248	−1.065	−0.183
Average		−1.473	−1.377	

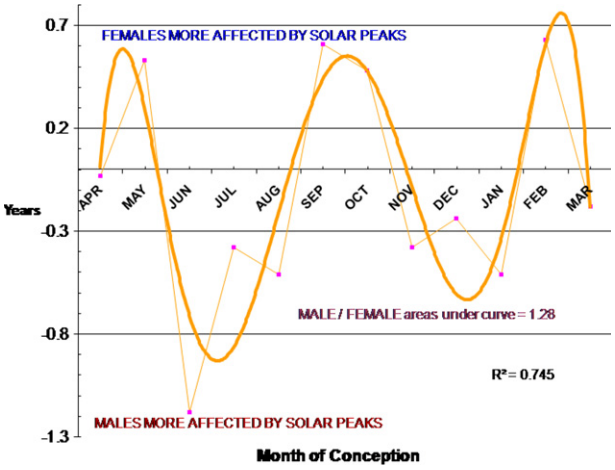

Figure 6 MAX—MIN for males *MINUS* MAX—MIN for females by month of conception.

6911 = 0.56) for the whole year, while females the remaining 44%. This supports findings in previous studies that males are subject to 28% more disease than females [24,25].

Fig. 7 shows the absolute difference between day and night (in h) by month of the year. Note the close similarity between Figs. 6 and 7. The correlation coefficient between the two curves is 0.71, with a possible maximum of 0.72 given the uncertainty imposed on biological systems by a variable Sun. Using integral calculus, the difference in areas under the trend lines of Figs. 6 and 7 is 1424 units, which is (1424/1617 = 0.88), an effective ~90% similarity between the curves. We believe this is compelling evidence that human lifespan is modulated by seasonal variation in light particularly driven by peaks of solar cycles.

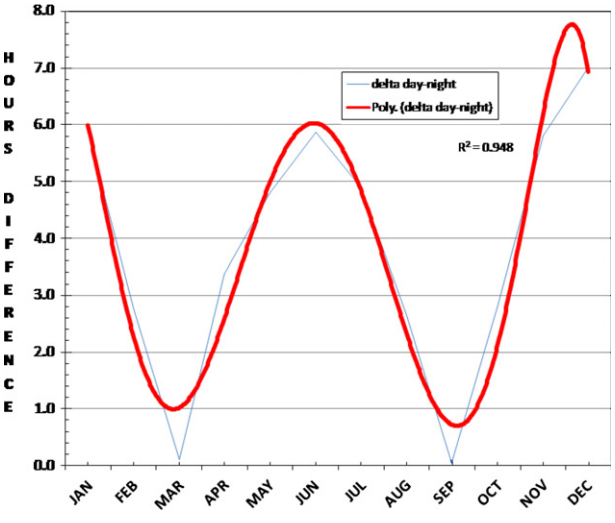

Figure 7 Absolute difference (in h) between day and night duration at 44° latitude.

Discussion

This paper describes the effects of radiation, probably UVR, on the human genome at peaks of solar cycles. This phenomenon was not previously reported because peak cycle lifespan had not been separated from the non-peak lifespan. This paper reinforces the findings of others regarding the seasonality of various diseases and that there are factors occurring early *in utero* that increase susceptibility to diseases later in life [15]. Our findings also support the reliability theory of aging championed by Gavrilov that postulates the effects of intrinsic defects at the onset of conception that are expressed over an individual's lifetime [23,26]. More defects mean more disease, especially after the period of natural selection. An analysis of the Gompertz (exponential) law of aging also embraces sensitivity to initial conditions as a modulator of lifespan, although the majority of the aging process described by the Gompertz law is related to extrinsic hazards to the organism [27]. The Weibull (power) law of aging explains the final failure of redundant systems and better describes the lifespan curve of the very old (>90 years old to super-centenarians). The human lifespan comprises features of both these laws of aging. It has been widely accepted that the genes for advanced age are not affected by the natural selection (reproductive) process, but rather by chance. We have as yet unpublished evidence that the very old do not show a difference in lifespan between peak and non-peak solar cycles. This implies that they possessed a resistance to solar radiation at the onset of life, either because of a certain intron (epigenetic) suite, or increased DNA-repair, anti-free radical mechanisms.

Our report not only reinforces the presence of intrinsic defects at the onset of life, but also evokes the probability that there is a selection process at conception which affects lifespan, as possibly 50% of all pregnancies end in spontaneous abortion, so it is especially difficult to survive the vicissitudes of life at its onset. The fact that we can readily detect the effects of peak solar cycles after a lifespan of over seven decades speaks to their importance in the origin of adult disease.

The pathogenesis of solar radiation on the human embryo is speculative. We do know that many, if not all diseases, are mediated in some way by inflammation and the immune system. Lymphocytes circulate through the reticuloendothelial system including the gut and integument. It is possible that maternal lymphocytes sustain irradiation through the dermis and produce cytokines that

eventually mediate changes in a developing ovum or conceptus [28]. Studies in plants have shown that UVR (B) reduces genome stability, and that instability may engender both harmful and occasionally useful mutations [29]. Also, the motility and fertility of mammalian and fish sperm are negatively affected by blue (360 nm) and UV (294 nm) light [30].

Again referring to the findings of Gavrilov in 19th century females, we can now explain why the months of May and December had the greater lifespan as, referring to our Fig. 6, the month of May corresponds to conception in August, which favors females over males; the month of December, with conception in March, is similar. Conversely, Gavrilov's shortest lifespan month was February, corresponding to May conception, which was most adverse to females in Fig. 6. One might expect if both genders were taken together, that the longest lifespan would be found closest to the *X*-axis in Fig. 6. Indeed, Gavrilova reported that life expectancy at age 80 was greatest in individuals born in January, corresponding to April conception in Fig. 6 [31].

The early human embryo is gender-biased by UVR in that females are apparently adversely affected by rapidly variable radiation while male embryos are adversely affected by extremes — too little or too much. Further genetic studies may eventually explain why that selection process may result in more risk-tolerant behavior in males in contrast to more conservative, risk-adverse behavior in females who must nurture, and are the most responsible for, the next generation.

The intermittent and incompletely predictable solar cycles periodically stress the genomes of all life producing genetic changes which may be harmful or adaptive. We believe that solar cycles, particularly the most irradiant which have occurred over the past 65 years, are fundamental engines of evolution, even underlying natural selection, and we bear their marks even to the end of our lives. It was Darwin's view in referring to natural selection that there is ''grandeur in this view of life.'' We believe that the findings reported in this paper further expand this grandeur to include the extraterrestrial influence of a variable Sun on biological evolution.

Conclusions

- Peaks of solar cycles, through some as yet unknown mechanism early in gestation, decrease the human lifespan approximately 1.5 years.
- The intrinsic uncertainty (28%) of solar storms is reflected in the human genome and in the differential lifespan of human males and females.
- The increased radiance of the Sun, especially over the past 65 years and based upon sunspots numbers approximately three times the 250-year average, may account for an alleged increased incidence of mental and neurodevelopmental abnormalities.
- Human lifespan is affected by yearly variation in solar cycle intensity and also by monthly (seasonal) variations, confirming that the genome and the environment are interactive at the very beginnings of life.
- Our findings suggest that manipulating light intensity or variation at the time of conception may have a greater effect on the future expression of disease, hence lifespan, than many interventions later in life.

Study strengths

The study uses readily available vital statistics data requiring only date of birth, date of death and gender. The data represents an entire spectrum of ages in a population. The population studied is relatively homogenous, approximately 96% Caucasian, which is not typical of other states. Also, data on sunspot numbers are well known and the methods of their calculation well accepted. Despite the inevitable existence of other variables that effect human lifespan, e.g., maternal nutritional status, smoking habits and other diseases, this study avoids these confounding variables by using randomization.

Study weaknesses

The study only uses data from the State of Maine at latitude $\sim 44^{\circ}$, encompassing 29 years. Approximately 6% of the acquired vital statistics data were missing some part of the data and could not be included in the analysis.

Future work

Our work should encourage others to investigate the dynamics of the maternal immune system and its relationship to neurodevelopment in early embryos. Studies of UVR intensity as well as its variation may be crucial to the discovery of the pathogenesis of lifespan variation due to solar cycles. This pathogenesis, once understood, may open new pathways for manipulating the human genome to minimize the expression of disease in

adult life. Epidemiologically, we must also look for a relation between an apparent increase in mental disorders and mutagenic solar radiation.

Acknowledgements

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