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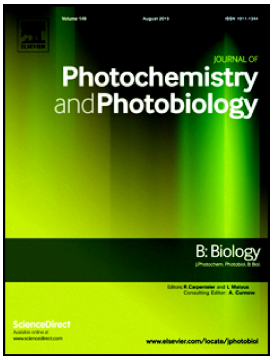

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Solar energy at birth and human lifespan

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ABSTRACT:

Purpose: The purpose of this paper is to examine the role of UVR at birth and its relationship to lifespan and determine whether there are significant differential effects on sex and race. We test if variation in UVR, as determined by solar cycles (long-term variation), is related to survival as measured by age at death.

Methods: The data used 78 million death records from the National Center for Health Statistics (NCHS) from 1979 to 2013 with accidents, suicides, and war casualties deleted resulted in ~63 million records. Records of persons <= 47 years old were also scrubbed because we could not show an effect on lifespan based upon the intensity of solar energy as reflected by sunspot number (SSN). This we hypothesize is due to the protective effect of the hormones associated with growth and reproduction. Also selected were persons afflicted with multiple sclerosis (MS).

Results: Males of all races born with a UVR intensity as estimated by sunspot number (SSN) <= 90 had an average lifespan of 74.4 years, for females of all races, 78.1 years; males born with >90 had an average lifespan of 66.3 years, for females of all races, 70.2 years, resulting in a lifespan decrease of 8.1 years for males and 8.5 years for females. For African-American males born <= 90 SSN, 70.8 years and for >90 SSN, 62.5 years, an 8.3-year decrease; similarly, for African-American females <= 90 SSN, 75.0, for >90 SSN, 65.4 years, a 9.6-year decrease. Higher solar energy *at birth* had an adverse effect on human lifespan. We also found that there were twice as many persons with MS born in >80-90 SSN as in the general population.

Conclusions: There is a statistically significant inverse relationship between exposure to solar energy *at birth* and average human lifespan. Solar energy by some mechanism alters the epigenome at birth, but the effect of higher solar energy becomes apparent after the age of natural selection.

Word count: 321

Key words: ultraviolet radiation, human lifespan, sunspot number, solar energy, multiple sclerosis, epigenetics.

Highlights

- Increased solar energy *at birth* shortens human lifespan an average of eight years.
- Lower solar energy *at birth* favors a longer lifespan.
- The effect of higher solar energy *at birth* becomes more manifest after the age of reproduction.
- Twice as many persons with multiple sclerosis are born in increased solar energy than in the general population.

What is new in this paper?

No one has previously reported, using so large a database, a relationship between exposure to varying solar energy *at birth* and human lifespan. In addition, we discovered a relationship of higher solar energy *at birth* to the autoimmune disease multiple sclerosis.

Does this paper stimulate important questions and new research?

Researchers will be encouraged to test the hypothesis that exposure to solar energy, probably UVR, at birth or during gestation affects the phenotypic expression of the diseases that affect human lifespan.

INTRODUCTION:

Many epidemiological studies have reported that some diseases affecting lifespan occur with higher incidence in persons born in certain months, referred to as seasonality, suggesting an environmental factor is operating during gestation. A seasonal pattern has been observed for schizophrenia, bipolar disorder and some autoimmune diseases like diabetes type 1, regional ileitis, myositis, celiac disease, multiple sclerosis (MS) among others, all, especially MS, likely dependent on genetic, infectious and latitude variations. (1-3)

These results regarding seasonality support the notion that UVR is instrumental in modifying the human genome not only by overt mutation, but also by epigenetic mechanisms. (4, 5) The epigenome is important as it enables an organism to modify its genetic library to match current environmental conditions and therefore to maximize survival.

Our research group has previously reported that human lifespan is associated with solar cycles at birth during which electromagnetic radiation varies in approximately 11-year cycles. We found that those born, and likely conceived, during the three peak years of solar cycles (the MAX) lived, on average, 1.7 years less than those born in the eight non-peak years (the MIN).(6) Others have reported that electromagnetic storm periodicities are mirrored by rhythms in human physiology.(7) The exact mechanism how solar radiation affects the genome and epigenome is the subject of much research.(8) Environmental effects occurring even at conception and early gestation may predispose humans to adult disease.(9-11) In our previously reported research we found that metabolic diseases were suppressed at solar peaks relative to non-peaks, a finding that was opposite of what we found for major mental illness.(12, 13)

The aim of this study is to examine the role of UVR intensity at birth and its relationship to human lifespan and determine if there are significant differential effects on sex and race. We specifically test whether variation in UVR as determined by solar cycles (long-term variation) is related to survival as measured by age at death.

We hypothesize that the high-energy photons in the UVR spectrum are early and major contributors of disease through DNA mutations and variation in solar output modulates these mutations.(14) (15) (16) Sunspots are dark areas on the Sun's surface that indicate magnetic solar storms associated with higher than normal electromagnetic radiation. In this study we used the number of sunspots; e. g, sunspot number (SSN) as a surrogate for the intensity of solar energy.

The rise of epigenetics as a discipline offers evidence that early-life stress affects a variety of infectious, cardio-metabolic and psychiatric diseases later in life which impact the process of ageing itself. (9, 11, 17, 18) DNA methylation, histone acetylation and non-coding RNAs all appear to be involved in determining the extent of adult-onset diseases. (5)

The UVR on Earth's surface is modulated by water vapor, airborne pollutants (ozone, nitrogen and sulfur dioxides, clouds and the Earth's axis so UVR intensity does not necessarily translate directly to intensity of ground UVR. Even though there is only a 0.1-0.2 percent increase in above-atmosphere UVR from solar minima (MIN) to maxima (MAX), our own observatory has shown that UVR intensity at ground level varies 20-40% throughout the sunspot cycle (Cycle 24) where SSN ranged from 0 to 250. (12) UVR that reaches the surface, while very attenuated compared to outer space, is still potent at solar MAX with up to 5,000 joules/m^2-month of UVR (95% is UV-A at 315-400 nm, 5% is UV-B at 280-315 nm) at our ground-level observatory at latitude 44.3 degrees N. Given the mutagenic effects of UVR, small changes in UVR may cause significant changes in the epigenome as reported by Lucock. (19) If these relationships are confirmed, this raises the possibility that manipulation of UVR exposure at birth or perhaps at some points in gestation might be used to modify the phenotypic expression of diseases that affect lifespan. A recent paper by Skjaervo et. al, which supports our work here, was limited by the fact that yearly SSN averages were used and that death records were relatively few (N = 8,662). In this study we increased the resolution of any relationship between UVR exposure at birth and human lifespan by using monthly average SSNs from a sample of ~63 million cases.

METHODS:

Cohort data: We acquired 78,645,528 death records from the United States National Center for Health Statistics (NCHS) 1979 to 2013. We used the following variables from the dataset: year of birth (YOB), month of birth (MOB), sex, year of death (YOD) and race (White, Black, Native-American, and Asian). The dependent variable was lifespan, calculated as the YOB minus the YOD. Records with a lifespan longer than 113 years were designated as outliers and deleted from the analysis. Birth years originally ranged from 1866 to 2013. Table 1 summarizes the original and scrubbed cohort data by sample size, mean age, sex, and race. For this analysis, deaths that occurred by accidents, suicides and war casualties were deleted as well as restricting the cohort to birthdates from 1900 through 2013. Despite major depression related to seasonal changes in sunlight, suicides were deleted as their number was very small relative to the entire dataset. We also discovered that there was essentially no effect of increased solar energy at birth on persons from zero to 47 years old (see Figure 1), during the ages of growth, development and reproduction and these records were removed from the analysis as well. The final dataset was comprised 31,807,486 females and 31,947,344 males (total = 63,7543,830). Multiple sclerosis data (N = 85,202) was derived from the entire dataset by diagnosis code (ICD 10 = G35, ICD 9 = 340) which likely did not cull all cases, but the disease was of sufficient severity to be on death certificates.

Solar data: Solar cycle data as measured by monthly SSN was collected and used as a surrogate for UVR; e. g., the higher the SSN the greater the UVR intensity. The average number of annual sunspots per month and per year was collected from the US Department of Commerce, National Oceanic and Atmospheric Administration (NOAA) web site (http://www.noaa.gov/ or see Data in Brief Appendix A for the SSN file). To examine the influence of solar radiation on

lifespan, sunspot numbers by year and month were matched-merged by year and month with each cohort case's birth year and birth month. Mean SSN for the entire cohort was 47.68 (41.57 median) with a minimum of 0 and maximum of 253 and for the scrubbed data used in this analysis the mean SSN was 43.4 (38.1 median) with a minimum of 0 and a maximum of 253.

Large Data Set Issues:

Others have written about the strengths and cautions associated with large sample sizes. (20) Advantages include the ability to study rare events such as SSN at birth. Large dataset concerns include quality and completeness of the data, sampling bias around internal and external validity, effect size, measurement error, many independent variables and the problem of "*p value"* inflation where large sample sizes generally yield low *p values* and, in turn, diminished effect sizes. Researchers have recommended several strategies to mitigate some of these problems which we used in this study.(20) Since over 63 million records were used in the analysis strategy, we recognized that we are reporting on approximately 81% of the total population (e. g., everyone who died in the United States from 1979 to 2013 with birth years back to 1900, (excluding persons <= 47 years of age). Because of the problem of collinearity with YOB and MOB, we limited the number of independent variables to the three we were studying: SSN, sex (1 = male, 0 = female), and race.

Analysis strategy: Two strategies were used for the analysis. The first was to use regression analysis (SAS 9.3) to test the hypothesis that UVR, as measured by SSN at year of birth (YOB) and month of birth (MOB), affects subsequent age at death; e.g., lifespan. Table 2 displays the

correlation matrix for these variables. The regression (GLM) model tested included the relationship between lifespan, SSN and sex and their respective interactions.

The second strategy was to plot lifespan by SSN to visually assess the relationship between increasing SSN (UVR at the time of birth) and lifespan by sex for all races. Charts were created based on summarizing data by categorizing SSN into intervals of 10 starting with 0-10, 10-20, 20-30, etc. The mean SSN and mean lifespan with respective standard deviations were calculated. Table 3 displays a typical table for White males showing SSN interval, mean and standard deviation for SSN, for mean age by sunspot grouping, and group sample size. The mean lifespan by sex for the White and Black races was plotted by SSN and can be found in Figures 3 and 4. For those who are interested, except for the 90-113 years old cohort (Figure 7), all plots by SSN group by age group; e.g., infancy, early life, puberty and post-menopause are in Appendices B-1 through B-4 and a table of the average lifespan for each of each of these groups is in Appendix B-5 (see Data in Brief).

RESULTS:

Table 2 displays the Pearson correlation matrix indicating that lifespan is inversely correlated with SSN (-0.244, $p<.0001$); e.g., the higher the SSN, the lower the age at death. There are also significant inverse correlations between lifespan with females more affected than males (-.189, $p <= .0001$). Table 4 displays the results of the regression analysis for lifespan as the dependent variable and SSN and sex. Regression coefficients represent the mean change in lifespan for one unit in the predictor variable while holding other predictors in the model constant, therefore isolating the role of one variable from all the others in the model. The main

effects for the full model Lifespan = SSN, and sex for all races were statistically significant (F (*df, 5, 6.38M),* F = 1500291, p<=.0001, with R^2 = .11).

As displayed in Table 4, UVR at birth as measured by SSN for the model including sex and SSN, indicates a statistically significant relationship with lifespan for each racial grouping for R^2 parameter estimate explaining 9% to 10% of the variation in lifespan. The >90 SSN group adversely affects the female sex more than the male sex for all races reported here (see Table 5). Note however that females overall still live longer than males for reasons other than only UVR dose at birth.

Plots of Lifespan:

Figure 2 displays the mean lifespan by SSN groups for the whole data set. The plot shows that SSN group 80-90 is a critical break point for lifespan. ANOVA for SSN <= 90 and those cases >90 show a statistically significant 8-year difference between those born in years with SSN <= 90 versus those being born with SSN >90 (F(df,3,6.38m), F=2,161,778, p<=.0001 with R^2=.09). Male plots have triangle markers, female plots have circle markers. Table 7 gives the percentage of months that have > 90 SSNs. Figure 7 plots those who achieved age 90 or more and who were born exposed to lower UVR (Note the scale on the X-axis). The cut-off for the UVR effects in all Figures, except Figure 7 (90-100 SSN), occurs at the 80-90 SSN group.

The data reported shows that there is a deleterious effect of SSN on lifespan when SSNs are >90 for those above reproductive age; e.g., >47 years old and this has a differential effect based on race (see Table 6). By examining the intensity of UVR on approximately 63 million individuals at the time of their MOB and YOB we find an average loss of ~8 years in lifespan between those

born at low levels of radiation, <= SSN, versus those born at high levels of radiation, >90 SSN. Using categorical 80-90 SSN as in the Figures, which included those with longer lifespans, yielded a ~13-year difference between <= 80-90 SSN and >80-90 SSN. The Norwegian study previously referenced reported a 5.2-year shorter lifespan in those born in solar MAX, roughly 3 years out of an 11-year cycle.(21)

The effect of UVR on the incidence of multiple sclerosis (MS):

The plots of lifespan for MS also break at 80-90 SSN and at >80-90 SSN the curves decrease nearly linearly. Table 5 summarizes only the >80-90 SSN groups for the general population and for MS by race and sex. The table shows the slope of curves in Figures 5 and 6, the percent N >80-90 SSN of the total N, the average age between the groups <= 80-90 SSN and the >80-90 SSN, and the last column gives the age difference in years between the two groups for White and Black males and females. The White race has a greater slope indicating more sensitivity to UVR. Due to relatively small Ns we did not plot Asians or Native-Americans for this paper. Note in Table 5 that the difference in lifespan between White females and MS White females at >80-90 SNN is (13.7 – 8.0 =) 5.7 years with approximately twice the percent of MS females (27% MS vs 14% general population) born exposed to high solar energy at birth. Although the relationship of UVR to the incidence of MS has been extensively reported in the past, the we do not believe this relationship with solar energy *at birth* has been reported previously.(22)

DISCUSSION:

Mechanisms involving UVR damage to DNA and its epigenome:

This paper shows that solar energy, as represented by variation in UVR intensity (SSN) at birth has an inverse relationship to lifespan losing an average of 8 years (>90 SSN) and 13 years (>80-90 SSN) affecting females more than males for all races studied.

Increasing age accumulates more mutations, both of DNA (nuclear and especially mitochondrial) and the epigenome, which affect longevity.(23) DNA has transcriptional and posttranslational repair mechanisms involving nucleotide excision repair to protect the genome from UVR, but less is known about how methylation or hypo-methylation of histones are affected by non-ionizing radiation like UVR. (24) It is known that UV-A photons (340-400 nm) counteracts demethylation of genes and may even cause hyper-methylation which suppresses immunity. (25) Moreover, UV-A does not damage DNA as much as UV-B.

UVR effects must involve the integument of mother or infant in some way. It has been recently shown that T-lymphocytes have intrinsic photosensitivity and blue light can enhance their motility, so the immune system is directly affected by light on the skin. (26) North American and Mediterranean population research supports a role for sunlight exposure in epigenetic processes. (27) Recent work from Lucock et al. has shown that UVR exposure is negatively related to red blood cell folate levels.(19) This is important because deficiency of this vitamin (B-9) predisposes to more uracil being incorporated into DNA, a process that increases DNA fragility and also may decrease histone methylation affecting the epigenome. In addition, UV-A degrades folate into products that generate reactive oxygen species which may further damage the epigenome.(28, 29) Beckett et al. recently reported that the degree of methylation of the vitamin D receptor (VDR) acts as a molecular adaptation to light exposure. (30) The presumption here is that events in the skin, modulated by UVR, can affect the immune system.

In this study we found that Blacks were less sensitive to changes in SSN group (see Figure 5) based upon a lesser slope compared to Whites. Epidermal melanin protects the integument from UV-B while not so protective in a high UV-A environment. In fair skin UV-A may be a detriment by producing more reactive oxygen species and genotoxicity. (31) There is a paradoxical higher mortality from skin and other selected cancers in dark-skinned persons possibly because the suppression of the immune system by UV-A and at higher latitude Blacks may not obtain enough UV-B to produce protective vitamin D.(31-33) Mechanisms for adaptation to different UVR environments may be related to folate polymorphisms. (34)

We hypothesize that the epigenome, if exposed to higher UVR at birth; e. g., higher SSN groups, might be guided into earlier and more abundant reproduction therefore sparing longer exposure to mutation (from ionizing and non-ionizing radiation), and active DNA repair mechanisms would be very active during times of development and reproduction. Exposure to higher UVR at birth could *down-regulate* putative UVR receptors, and later in life more UVR (primarily UV-A) would be required to suppress inflammation, autoimmune disorders, and subsequent cardiovascular or malignant disease. While the relationship of inflammation and malignancy has been known for some time, a recent paper reaffirmed the suspected association between autoimmune disease and cardiovascular risk. (35) Another paper regarding MS, a prevalent autoimmune disease of the nervous system, describes low UVR in the first trimester of pregnancy as being associated with 50% more MS in offspring.(10) This paper reinforces a receptor hypothesis and is graphically illustrated in Figure 8.

Similarly, the epigenome exposed to low UVR at birth *up-regulates* UVR receptors so that later in life less UVR is needed to suppress inflammation, autoimmune, cardiovascular and malignant

disease. With low UVR at birth the epigenome would theoretically detect a safer environment lessening the need for early production of multiple progeny, favoring autophagy for long-term survival of the soma. Autophagy is the process of removing damaged, diseased, or cancerous cells, thereby allowing healthy ageing. (36, 37) Supporting this assertion is a recent Italian paper that found beclin-1, a key regulator of autophagy, to be more active in healthy centenarians.(37) As Figure 7 shows, an increased sensitivity to lower levels of UVR occurs *at birth* in those attaining their 10^{th} decade of life.

The hormones of reproduction appear to blunt the effects of UVR at birth until after menopause (~age 47) in females and likely sooner in males.

Note that an average 8.5-year loss of lifespan for >90 SSN applies to 11% of our data (see Table 7). Persons whose birth is associated with SSN >90 should be aware of diseases associated with inflammation and the immune system and screen for them proactively. Perhaps in the future it may be possible to mitigate autoimmune diseases by manipulating light in gestation or at birth. This task lies ahead for other researchers.

CONCLUSIONS:

- Solar energy during gestation and *at birth* plays a deterministic, disproportionate role in human lifespan. While varying UVR appears to effect growth, development and reproduction, its effect on mortality becomes evident after menopause; e.g., > age 47.
- Solar energy, probably UVR, by some mechanism in concert with folate or other photoactive molecules, affects the epigenome, which we postulate programs growth and development; where lower UVR *at birth* favors autophagy and a longer lifespan through repair of the soma; higher UVR *at birth* favors the germ line, earlier

reproduction, a more active immune system which may induce autoimmune diseases that shorten lifespan.

- Those who develop MS are twice as likely to be born exposed to higher solar energy than the general population. This appears to be caused by MS persons having about half the sensitivity to UVR; e. g., half the slope than the general population.
- This study supports the notion that epigenetic responsiveness to UVR is an ancient adaptation conserved in most, if not all surface organisms, including humans.

List of Abbreviations:

SSN- Sunspot Number

UVR- ultraviolet radiation

MAX- maximum radiation in a solar cycle (~ 3 years duration)

MIN- minimum radiation in a solar cycles (~ 8 years duration)

YOB- year of birth

MOB- month of birth

NCHS- National Center for Health Statistics

NOAA- National Oceanic and Atmospheric Administration

MS- multiple sclerosis.

Limitations of the study: This is a retrospective, statistical study. A given individual may not necessarily develop autoimmunity or have a shortened lifespan if born under high SSN conditions. Interactions between the genome and epigenome vary and the former may overwhelm the latter (as in those <= 47 years old) in the phenotypic expression of disease. We did not plot the full SSN groups for Asians or Native-Americans as they had essentially the same 80-90 SSN cut off.

Advantages of the study: A large database allows knowing actual birth and death dates, not relying on life expectancy tables. The quality and size of data from the National Center of Health Statistics are unparalleled.

Competing Interests Statement: The authors certify that there are no competing interests. This research did not receive any grants from funding agencies in the public, commercial, or not-for-profit sectors.

FUTURE WORK:

We hope to test our hypothesis that UVR affects human lifespan by encouraging other researchers to use UVR, or the absence of same, at birth or during gestation in a prospective study using mice for example, to see if they can affect the expression of adult disease.

DECLARATIONS:

Ethics approval and consent to participate: Only de-identified data was obtained from the NCHS. No specific human or animal was used for this study.

Consent for publication: Both authors (GED and WEL) give consent for publication.

Availability of data and material: NCHS data are publicly available upon request. NOAA data (see Data in Brief) are available on website http://www.noaa.gov. Any data used in the Figures are available upon request.

Competing interests: The authors are not aware of any competing interests.

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Authors' contributions: Both authors were involved writing the manuscript. WEL did the statistical work in the statistical package SAS. GED was responsible for the Figures in the study.

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Table 1: Summary statistics for the cohort: Total sample compared to scrubbed data.

	N (%) original	Mean age (SD) original	N (%) scrubbed	Mean age (SD) scrubbed
Cohort Total	78,645,146 (100%)	71.59 (19.37)	63,755,906	75.58 (11.80)
Males (all races)	39,955,284 (50.80%)	67.91 (19.71)	31,947,344	73.35 (11.42)
Females (all races)	38,689,862 (49.20%)	74.40 (18.24)	31,807,486	77.82 (11.75)
Caucasian	67,823,316 (86.24%)	72.91 (18.43)	55,682,064	76.12 (11.60)
African-American	9,365,576 (11.91%)	62.99 (22.74)	6,957,010	71.58 (12.26)
Native-American	372,509 (0.47%)	58.50 (23.80)	247,251	70.55 (12.34)
Asian-American	1,083,745 (1.38%)	68.01 (21.70)	808,879	74.86 (12.34)

Table 2: Pearson Correlation Coefficients among the primary variables in the study

	Lifespan 1	**Sex 2**	**SSN 3**
1	1	-.189	-.244
2		1	.038

All correlations p<=.0001, (n=63,755,204)

Table 3: Sunspot Groupings Summary table for White Males

SSN range	Group Mean Lifespan years (sd)	SSN Mean (sd)	Group N
1-10	73.3 (16.5)	4.4 (3.0)	7,814,573
10-20	68.1 (19.7)	14.6 (3.1)	4,827,588
20-30	68.1 (18.8)	24.9 (2.8)	3,884,140
30-40	71.4 (18.1)	35.1 (3.0)	3,652,358
40-50	71.1 (19.3)	45.0 (2.8)	2,648,136
50-60	70.7 (17.8)	54.9 (2.8)	3,725,590
60-70	70.7 (16.7)	65.1 (3.1)	3,183,168
70-80	71.9 (16.6)	74.5 (3.0)	2,394,287
80-90	65.3 (19.7)	84.8 (2.8)	1,823,541
90-100	61.7 (21.7)	95.1 (2.6)	1,243,476
100-110	60.7 (20.7)	105.4 (3.0)	1,097,409
110-120	58.7 (20.2)	115.4 (2.9)	948,612
120-130	53.7 (21.4)	125.8 (3.0)	867,541
130-140	45.2 (22.0)	134.5 (2.8)	415,326
140-150	44.5 (19.4)	145.2 (2.6)	371,359
150-160	48.9 (23.8)	156.1 (22.2)	340,493
160-170	43.9 (19.0)	165.9 (2.3)	362,391
170-180	36.4 (18.4)	174.3 (2.1)	181,874
180-190	44.9 (15.2)	186.3 (2.9)	199,792
			39,981,654
plotted data	*plotted data*		*total*

Table 4: Parameter Estimates for the Regression Equation for Lifespan by Sex and SSN for N=63,755,204

Variable	R^2	Parameter Estimate (SE)	T value	P value	n
White	.09				55,682,064
Intercept		81.5 (.003)	25250.6	<.0001	
Sex	.05	-4.5 (.006)	983.3	<.0001	
SSN	.04	-.07 (.0001)	--1264.5	<.0001	
Sex*SSN	.09	-.004 (.00008)	-48.3	<.0001	
Black	.11				6,957,010
Intercept		77.7 (.021)	8173.0.	<.0001	
Sex	.07	-4.6 (.013)	341.1	<.0001	
SSN	.03	-0.09 (.0003)	-553.6	<.0001	
Sex*SSN	.10	-.01 (.0002)	-58.8	<.0001	
Native American	.10				247,251
Intercept		76.5 (.05)	1473.8	<.0001	
Sex	.08	-3.6 (.07)	-50.3	<.0001	
SSN	.01	-.08(.0001)	-107.9	<.0001	
Sex*SSN	.10	-.01 (.001)	9.1	<.0001	
Asian	.09				868,879
Intercept		80.2 (.06)	2807.4	<.0001	
Sex	.07	-2.5 (.04)	-63.9	<.0001	
SSN	.01	-.08 (.0005)	-183.6	<.0001	
Sex*SSN	.09	-.002 (.0006)	3.2	<.001	

[For variable “sex”, if sign is negative, males are more affected; sign generally has more significance than absolute value]

Table 5: Summary of > 80-90 SSN Group (see Figures) for slope and %N by race for general population and for those with multiple sclerosis(MS)

Race/Sex	Slope >80-90 SSN	% N >80-90 SSN	N	Average age < 80-90 minus >80-90 SSN	Delta age (years)
ALL males >47	-1.72	17%	31,947,344	74.4 – 62.7	11.7
ALL females >47	-2.14	14%	31,807,486	78.6 – 64.8	13.8
MS males >47	-1.02	31%	27,057	62.7 – 54.6	8.1
MS females >47	-1.11	31%	49,297	64.2 – 55.0	9.2
White females >47	-2.20	14%	27,766,478	79.1 – 65.3	13.7
MS White females	-1.07	27%	41,675	67.2 – 59.2	8.0
Black females >47	-1.74	17%	3,450,292	74.9 – 62.1	12.8
MS Black females	-0.79	33%	3,902	62.7 – 57.0	5.7
Asian females >47	-1.99	18%	403,637	77.6 – 63.8	15.8
White males >47	-1.77	16%	27,781,053	74.8 – 63.1	11.7
MS White males	-1.00	28%	22,539	65.9 – 58.7	7.2
Black males >47	-1.37	20%	3,473,172	70.8 – 60.3	10.5
MS Black males	-0.72	35%	1,754	61.5 – 56.9	4.6
Asian males >47	-1.74	19%	461,516	68.6 – 55.5	13.3

Table 6: Average lifespan and SSN for ALL, White and Black races

Scrubbed data

	Lifespan (avg SSN) n	Lifespan (avg SSN) n	Lifespan (avg SSN) n	Lifespan (avg SSN) n	Lifespan (avg SSN) n	Lifespan (avg SSN) n
	White male	White female	Black male	Black female	ALL male	ALL female
> 90 SSN	67.0 (128) 3,337,866	71.0 (123) 2,755,837	62.5 (132) 543,390	65.4 (130) 453,377	66.3 (128) 3,972,213	70.2 (124) 3,282,032
< = 90 SSN	74.8 (35) 24,525,001	79.2 (34) 25,063,360	70.8 (36) 2,948,722	75.0 (35) 3,011,521	74.4 (35) 27,975,131	78.7 (35) 28,525,828
delta age (years)	7.8 P<=.0001	8.1 P<=.0001	8.2 P<=.0001	9.6 P<=.0001	8.1 P<=.0001	8.5 P<=.0001

Table 7: Frequency of Months <= 90 SSN versus >90 SSN (scrubbed data)

SSN Group	Frequency	Percent	Cumulative Frequency	Cumulative Percent
GT90	7,254,245	11.4	10,986,263	**11.4**
LE90	56,500,959	88.6	63,755,204	100.00

P<=.0001

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Figure 1: Male and Female lifespan, all races, by SSN Group, <=47 years old

Figure 2: Male and Female lifespan by SSN group at birth (all cases, all races)

Figure 3: Female lifespan for Whites and Blacks >47 years old by SSN Group

Figure 4: Male lifespan for Whites and Blacks >47 years old by SSN Group

Figure 5: Lifespan of White and Black Males and Females >80-90 SSN

Figure 6: Lifespan of White and Black Males and Females with Multiple Sclerosis >80-90 SSN

Figure 7: Male and Female lifespan for ages 90-115 by SSN Group (all races)

Figure 8: The receptor hypothesis using multiple sclerosis as an example

Figure 1: Male and Female lifespan, all races, by SSN Group, <=47 years old

N_{male}= 4,309,245; N_{female}= 2,411,750

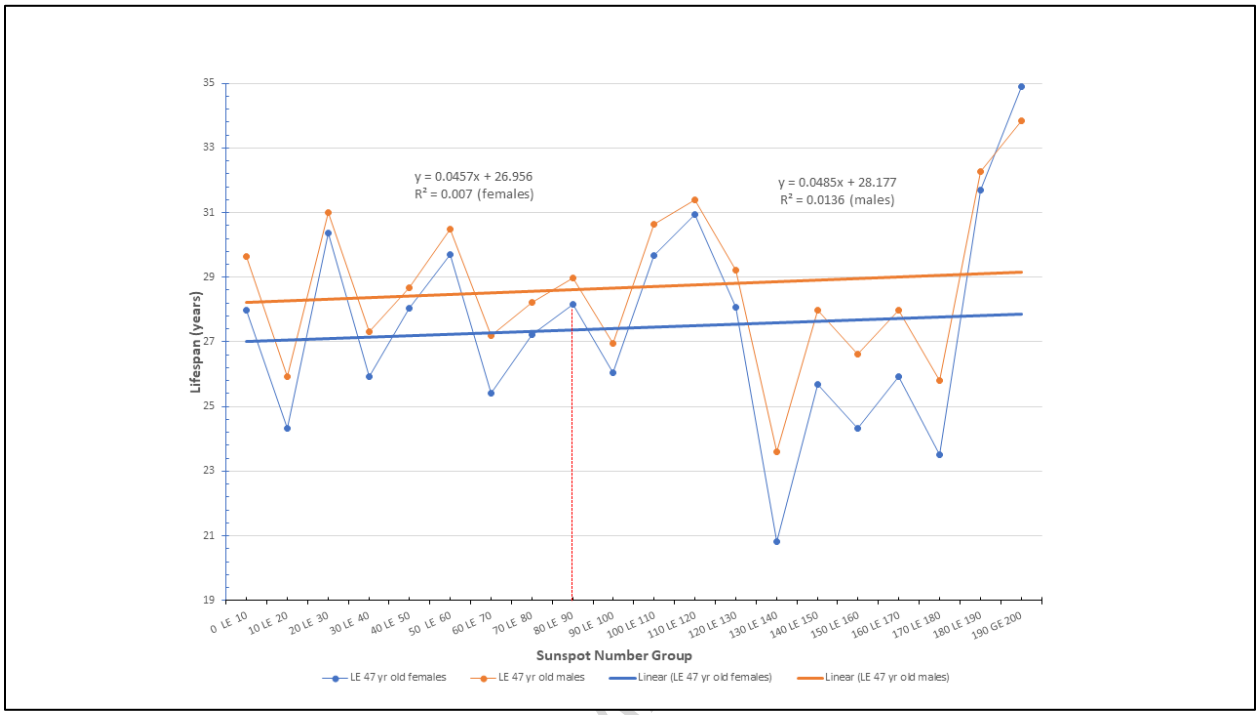

Figure 2: Male and Female lifespan by SSN group at birth (all cases, all races)

N_{male}= 39,981,654; N_{female}= 38,841,687

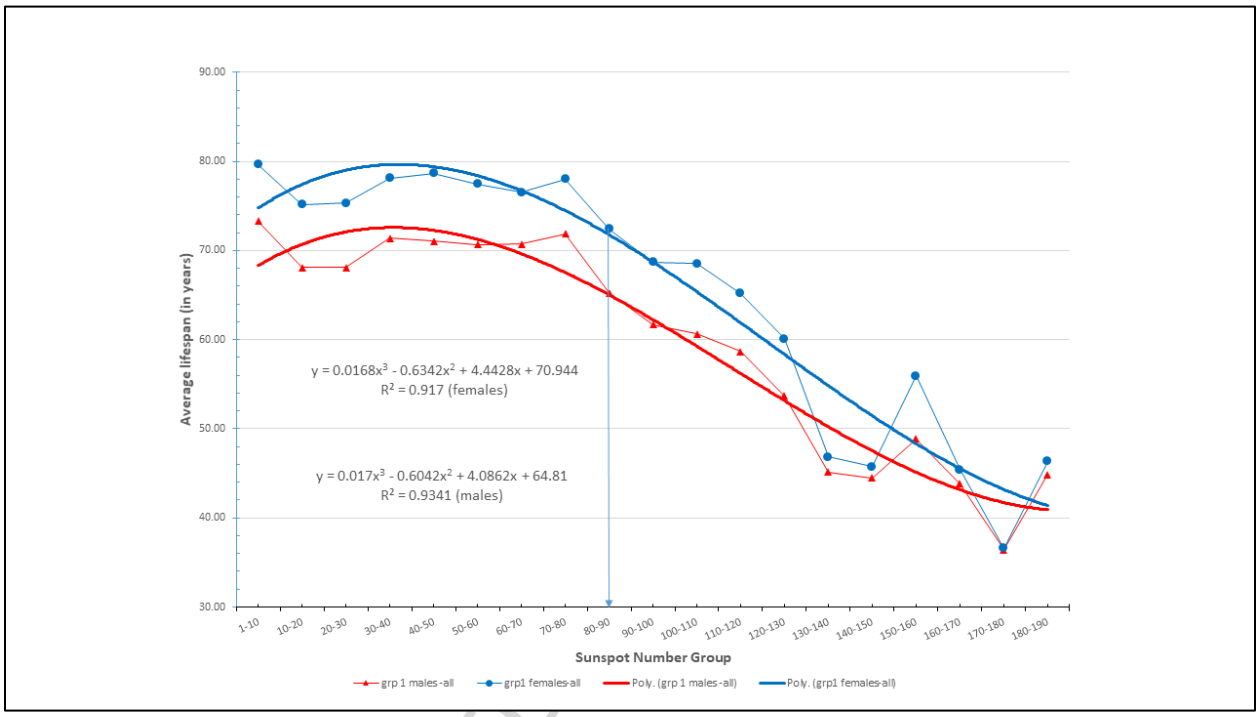

Figure 3: Female lifespan for Whites and Blacks >47 years old by SSN Group

$N_{female\ white}$= 27,766,478; $N_{female\ black}$= 3,450,292

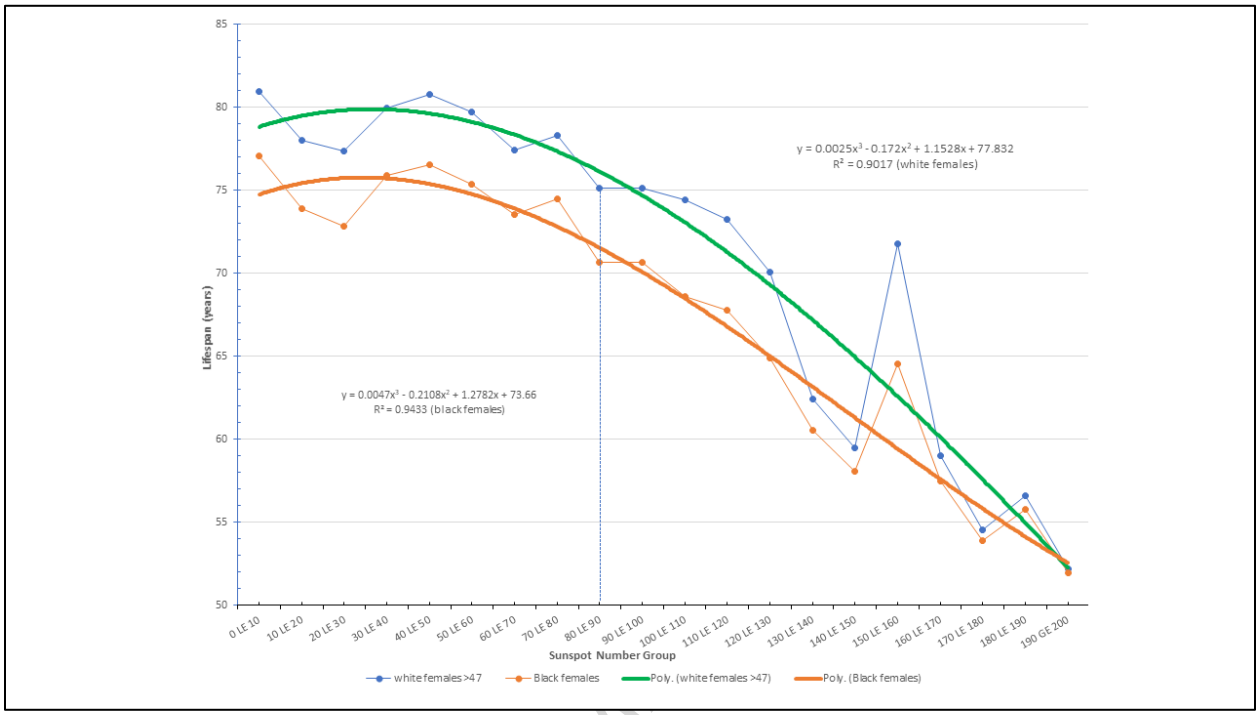

Figure 4: Male lifespan for Whites and Blacks >47 years old by SSN Group

$N_{male\ white}$= 27,781,053; $N_{male\ black}$= 3,473,172

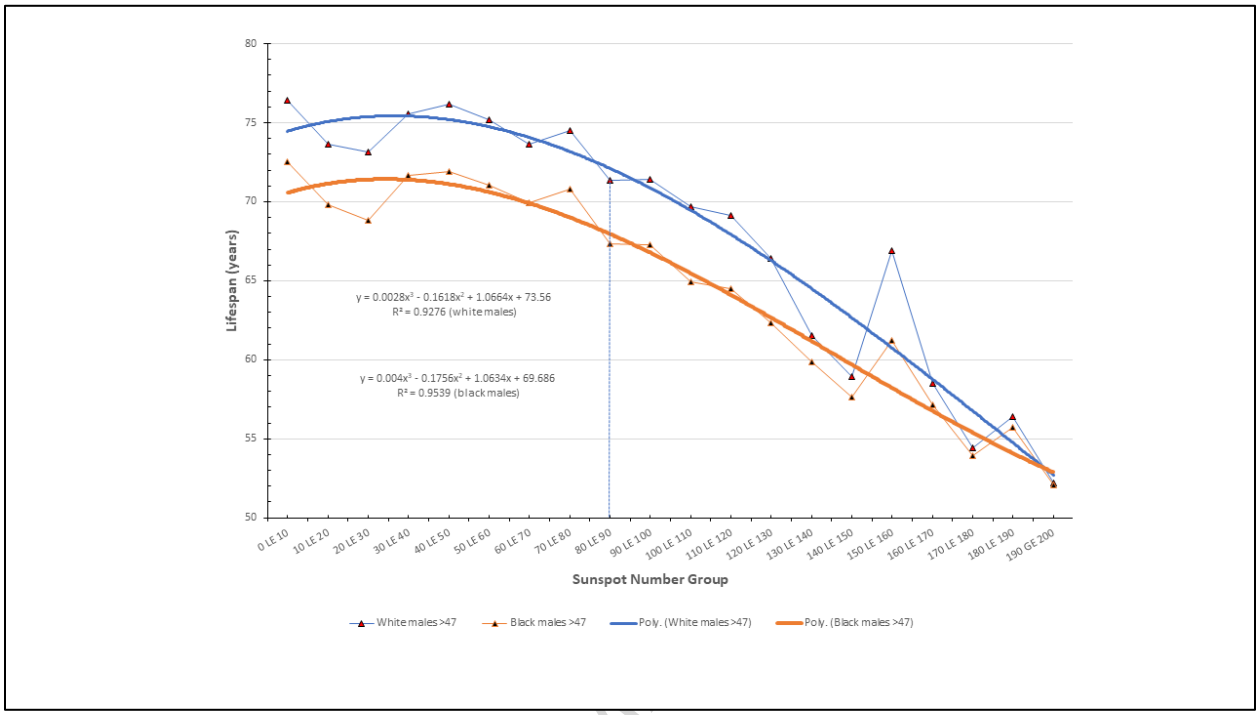

Figure 5: Lifespan of White and Black Males and Females >80-90 SSN

$N_{\text{White males >80-90 SSN}} = 4{,}472{,}948$; $N_{\text{Black males >80-90 SSN}} = 690{,}329$

$N_{\text{White females}} = 3{,}790{,}553$; $N_{\text{Black females >80-90 SSN}} = 591{,}383$

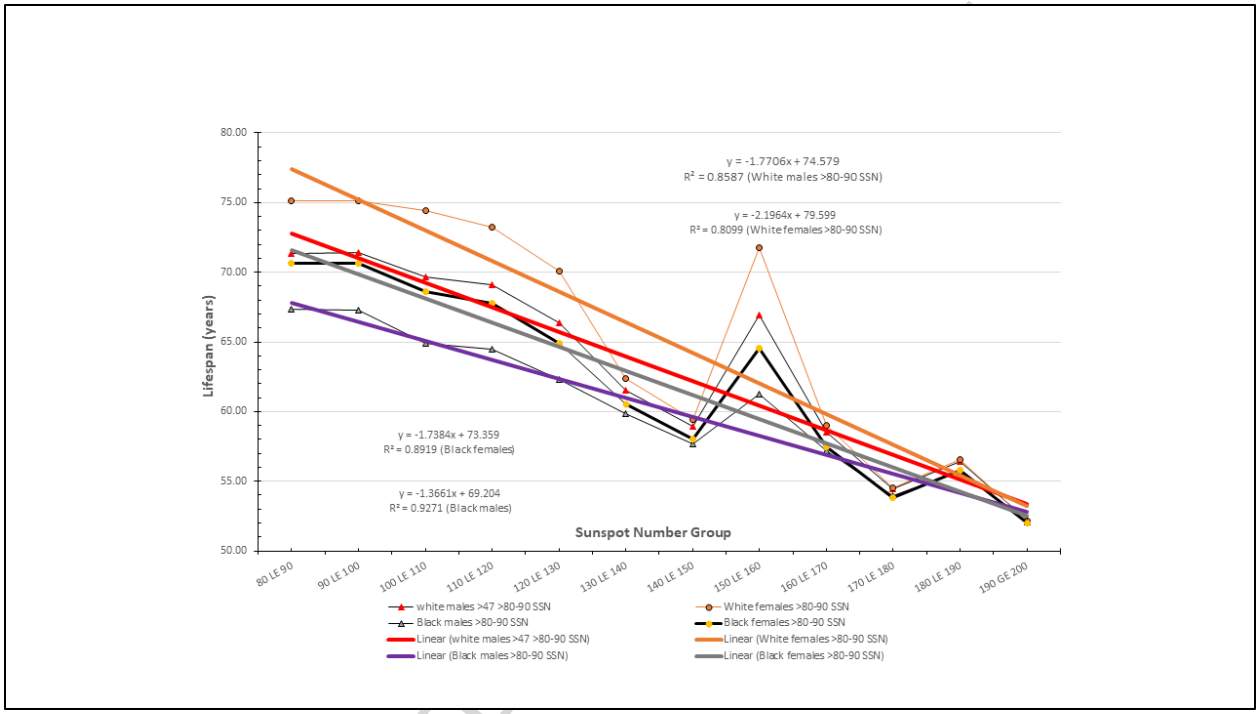

Figure 6: Lifespan of White and Black Males and Females with Multiple Sclerosis >80-90 SSN

$N_{\text{MS Black males >80-90}} = 1{,}754$; $N_{\text{MS Black females >80-90}} = 3{,}902$

$N_{\text{MS White males >80-90}} = 22{,}539$; $N_{\text{MS White females}} = 41{,}675$

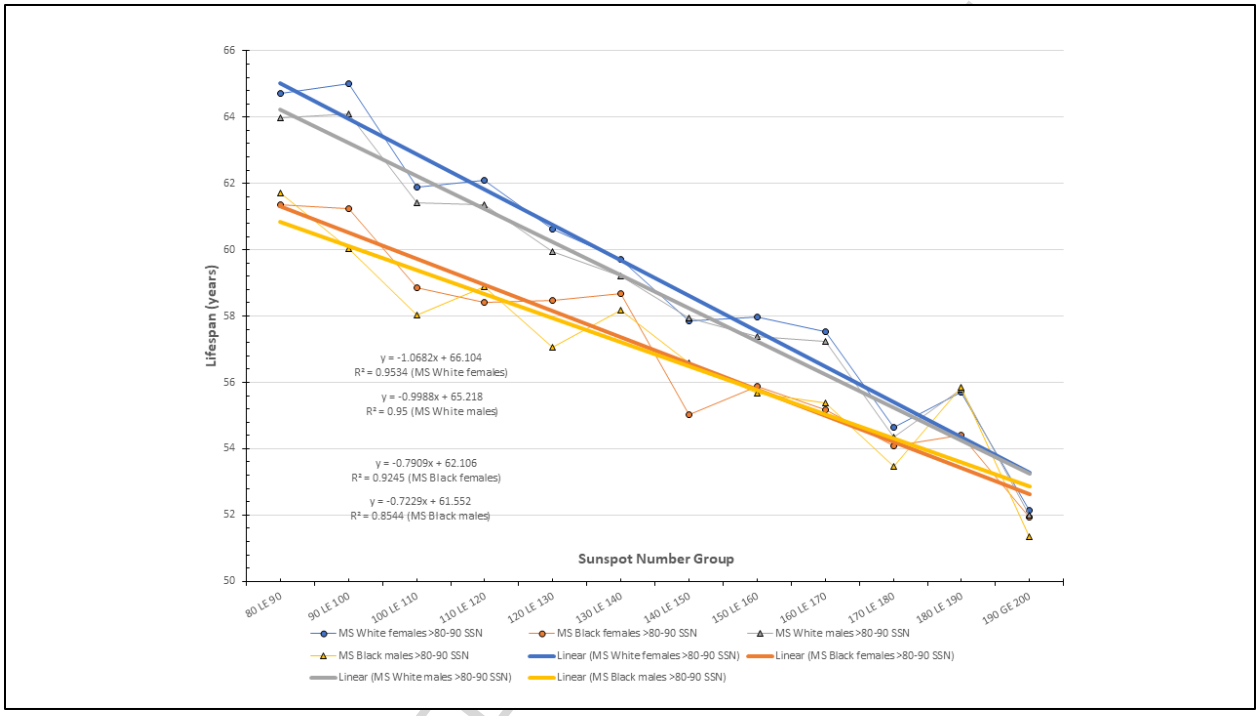

Figure 7: Male & Female lifespan for ages 90-115 by SSN Group (all races)

N_{male}= 2,947,489; N_{female}= 7,124,041

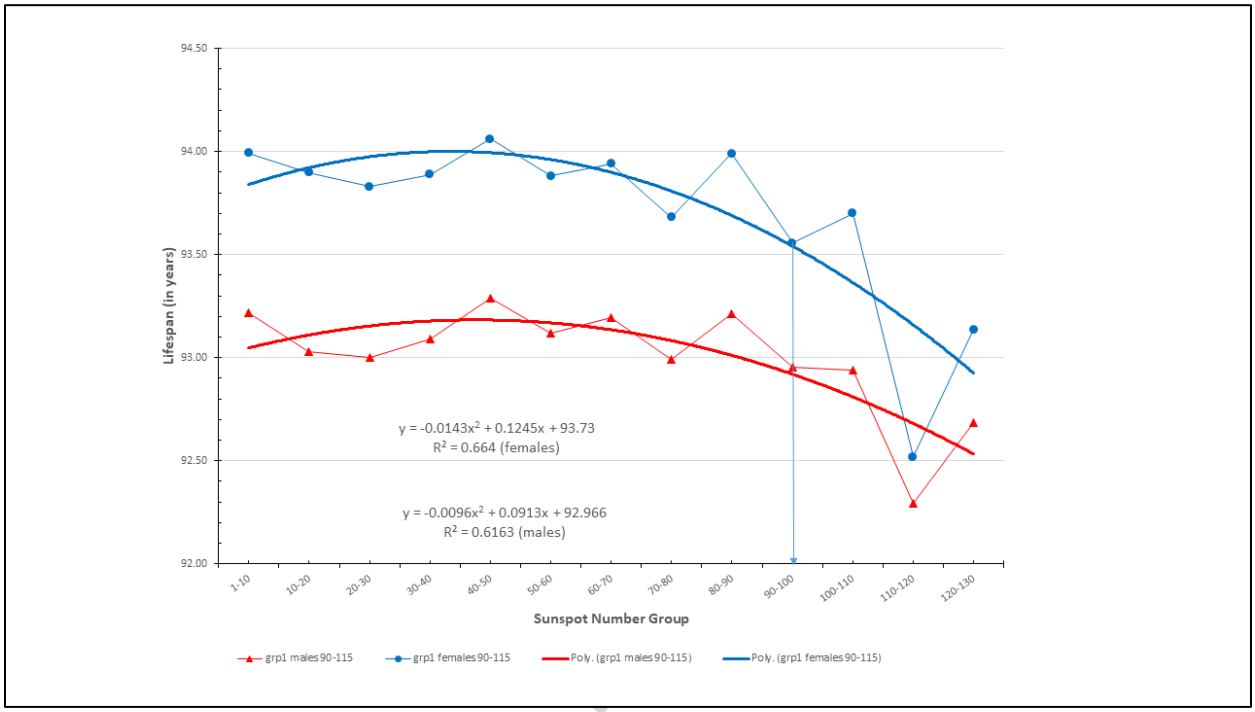

Figure 8: The receptor hypothesis using the autoimmune disease multiple sclerosis