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Hispanic Paradox

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Introduction

The term Hispanic broadly refers to people, nations and cultures that have a historical link to Spain. Many studies shows that Latinos live longer than Caucasians, despite experiencing higher rates of Diabetes and other diseases. Scientists refer to this as the "**Hispanic Paradox**". Hispanics have better survival from cardiovascular disease, which is known to be linked with elevated systemic inflammation. Also Hispanic consume up to four to five fold more beans per head than non-Hispanic. The ULCA team used several biomarkers, including an "epigenetics clock" developed by Horvath in 2013, to track an epigenetic shift linked to aging in the genome, for example, the biological clock measured Latino women's age as 2.4 years. So the last findings strongly suggest that genetics factors linked to ethnicity may influence how quickly a person ages and how long they live. **In my research I will talk about this paradox and its evidence**. **I will also talk about the genes and whether it really influence the age**. **Finally the relation between biological clocks and aging**.

What is this paradox?

1-►BACKGROUND OF THE HISPANIC PARADOX

[1]In the decade between 1990 and 2000, the US Hispanic population growing more than 57. 9 percent, becoming the fastest increasing and largest minority group in the United States. In the year 2000, there were an appreciatory 35.3 million Hispanics, 12.5 percent of the total US population, with Mexican Americans being the largest ethnically apparent subgroup including 20.6 million people. Hispanics who live in the United States have less education, a higher poverty rate and worse access to health care than non-Hispanic whites.

They also represent the ultimate paradigm of healthcare disparities. With the highest rate of uninsured people, the lowest rates of screening for hypercholesterolemia, hypertension, and other CVD risk factors, with the lowest rates of smoking counseling and with the poorest levels of blood pressure control, glycemic control, and other measures of deficient quality of care.

Hispanics had a 24% lower risk of all-cause mortality and lower risks of nine of the leading 15 causes of death in the USA (notably, cancer and heart disease).

[2]About two decades ago, Markides and Coreil published the first article which talks about the evidence of the Hispanic paradox. Markides and Coreil focused on the health of Southwestern Hispanics, mostly Mexican Americans, and [3]more recent studies have examined the history of the paradox with data from Bexar County, Texas dating back to 1935, a 1900 Census microsample on six Texas counties, the 1910 U.S. Census national microsample, and the 1990 linked birth and infant death files. So it was

[1] Resendez, Williams, Haffner, Stern, and Hazuda,2003:1048
Abrafdo-Lanza, Dohrenwend, Ng-Mak, and Blake Turner,1999:1543
[2] Markides and Eschbach,2005:68,69
[3] Palloni and Morenoff:150,151

shown that the Paradox is a phenomenon of relatively recent vintage. After critically reviewing the evidence, Markides and Coreil proposed an epidemiologic paradox: that the health status of Hispanics in the Southwestern United States was more comparable with the health status of non-Hispanic Whites than with that of African Americans despite the fact that socioeconomically, Hispanics were more similar to African Americans than the more advantaged non-Hispanic Whites. This assertion was based on review of evidence on infant mortality, overall life expectancy, mortality from cardiovascular diseases, mortality from certain major cancers, as well as data on functional health.

Markides and Coreil (1986) suggested possible explanations for this epidemiologic paradox, which included certain cultural practices, strong family supports, and selective migration. By the 1990s, the evidence began showing a mortality advantage among Mexican Americans as well as among other Hispanic populations. The epidemiologic paradox now had commonly come to be called the "Hispanic paradox." Franzini, Ribble, and Keddie (2001) published a comprehensive review of the evidence over a 20-year period and concluded that the paradox was apparent in mortality, especially in the older years, but also among infants. They concluded that the causes of the paradox remain largely unknown. They comment on possible problems in vital statistics data and the hypotheses that the paradox may result from either a healthy immigrant effect, that is, disproportionate migration by persons in good health compared with those in poor health, or a "salmon bias," so this theory proposes that Latinos return to their home country to retire or in case of serious illnesses to die in their home. This could bias the mortality rate by lowering it, as foreign deaths will not be tabulated in the US mortality. They conclude that three of these causes may contribute to but do not fully explain the paradox.

2-►EXPLANATION FOR THE HISPANIC PARADOX

[4]The most frequently offered explanations for the Paradox in the literature are:

- ***the under registration of infant deaths in the target population***: it has been renounced outright, but probably too quickly and hastily.[5] Some assessments are found of some data sets where the authors assert that errors of under-registration could not reasonably be large enough to account for the observed patterns of infant mortality differences.[6] Admittedly the claim for that particular data set may be coercing but as a general explanation does not have the power to close the case once and for all. The important point is that this alternative explanation has not yet been excluded and may undermine the case for a paradox.

[4] Palloni and Morenoff:151,152,153
5 Preston,1996:193–209.
6 Forbes and Frisbie, 1991: 639–660.

- ***differences in the maternal risk profile***: These risks may reinforce the paradox, not explain it because if Hispanic women do have a risk profile conducive to higher infant mortality, and that is why it is not a really explanation. Indeed, as argued above, standardizing for conditions thought to be related to infant mortality may either exaggerate, abate, or leave invariant the contrasts between groups, and where changes are observed they are usually inconsequential.
- ***differences in maternal behaviors***: it is the most widely accepted explanation. As Gutmann and colleagues[7] argue: "Perhaps the most promising explanation to emerge is the finding that women of Mexican-origin are characterized by a more healthful lifestyle that buffers or offsets disadvantaged sociodemographic conditions. For example, Mexican-origin women smoke less, consume less alcohol, and have more nutritious diets than do non-Hispanic white women." This argument holds that the target group's advantage over non-Hispanic whites and blacks is rooted in maternal attributes that are (1) strongly related to conditions affecting neonatal not post neonatal mortality and (2) acquired in social and normative contexts that are very different from those they encounter in the U.S.
- ***effects of social networks****:* it has a very similar logic: mothers of Mexican-origins retain active linkages to and membership in strong social networks that help cushion and offset the potentially deleterious influence of stressors.[8] As was the case with behaviors, networks are assumed to be rooted in the context of the country of origin and are expected to weaken with length of stay in the U.S.
- ***the healthy migrant hypothesis that immigrants represent a selectively healthy group that are not representative of the wider target population***: Two hypotheses are part of the explanation based on migration effects: the "healthy-migrant effect" and the "salmon-bias effect." If the observed difference between Hispanic and non-Hispanic mortality is a result of migration effects, one cannot conclude that there are characteristics—genetic, socially produced, or culturally acquired—conferred upon individuals by virtue of their membership in the group, that translate into health advantages and lower mortality. One cannot do so because the observed difference between migrants and nonmigrants is net of measured characteristics, which almost certainly will not include all those that are relevant to both

7 Gutmann, Frisibie, Deturk and Blanchard, 1998
[8] Sherraden and Barrera, 1994.

migration decision making and to health and mortality. On average, migrants are healthier than those who do not migrate and may be healthier than the average individual in the receiving population.

3-►RECENT EVIDENCE FOR THE HISPANIC PARADOX

[9]Studies based on many data sources, including national and state vital statistics; local surveys; and, most significantly, national linked data files, such as the National Longitudinal Mortality Study (NLMS)1 and the NHIS-MCD, have found support and evidence for the Hispanic mortality paradox. Most studies have showed that after pertinent demographic and socioeconomic characteristics are controlled, the Hispanic population as a whole fares better in adult all-cause mortality than do non-Hispanic whites. Using the NLMS, Sorlie et al. (1993) there is a study which used multivariate hazard models estimated for adults aged 25 and older and controlled for demographic and socioeconomic characteristics, found that both Hispanic men and Hispanic women have significantly lower all-cause mortality than do non-Hispanic whites. Two other studies based on the NLMS, but not exclusively focused on the Hispanic population, reported the same results (Singh and Siahpush 2001, 2002). One of these studies found that U.S.-born Hispanics have lower all-cause mortality than do U.S.-born non-Hispanic whites and that foreign-born Hispanics have lower all-cause mortality than do foreign-born non-Hispanic whites. Studies that have disaggregated the Hispanic population by national subgroup have reported varying support for the Hispanic mortality paradox.

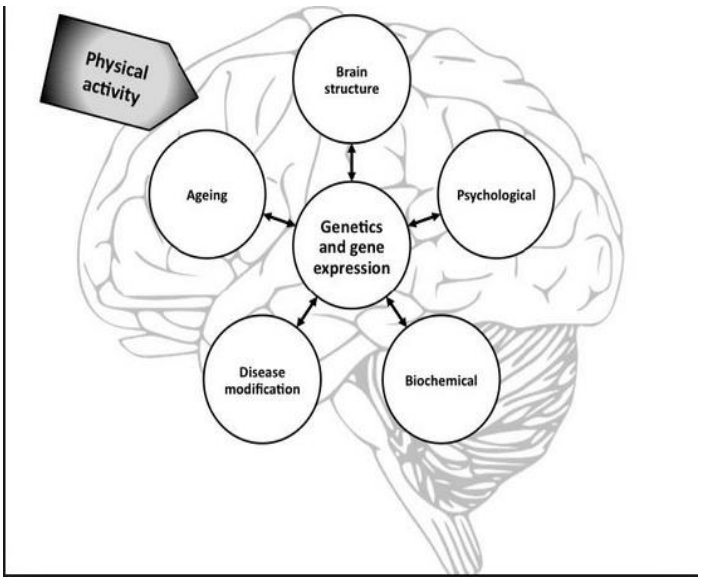
[9] Markides and Eschbach,2005:70-72
Palloni And Arias,2004:386,387

s Aging in our Genes?

1-►HOW IS THE AGE AFFECTED BY THE GENES

Aging is a complex, challenging phenomenon that requires multiple, interdisciplinary approaches to unravel its puzzles, it is also a genetic program. There are many indications that aging is a genetic program in most higher organisms including humans so:

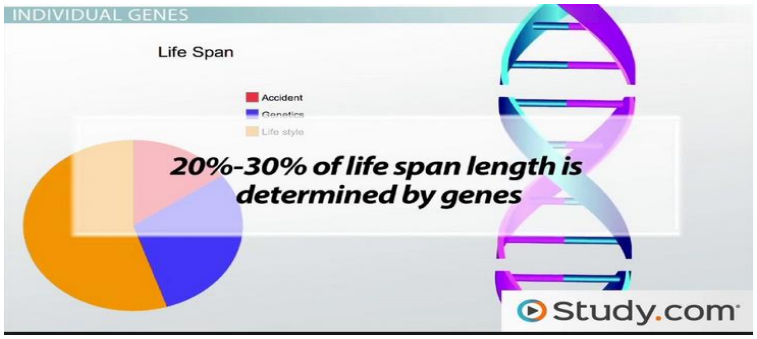
- Many of the genes that regulate aging have been conserved since the dawn of eukaryotic life[10]. All other such highly conserved genes have been protected by natural selection because they form an essential core to life processes. Natural selection has evidently treated aging as a core life process.
- Hormesis: The fact that life span can be readily extended under genetic control when conditions are most harsh and challenging (e.g. starvation) indicates that the body is "holding back" on life span at times when the environment is more favorable[11].
- Breeding animals for longevity does not necessarily impair fertility as is demanded by popular theories based on pleiotropy or trade-offs[12]. In fact, many single-gene mutations are known to extend life (especially in worms), for which no major cost has yet been identified .
- One-celled eukaryotes are subject to two modes of programmed death: apoptosis and replicative senescence [13]. This fact in itself vitiates the classical theoretical contention that it is impossible for programmed death to evolve, requiring as it would an



[10] Guarente and Kenyon,2000:255-262.
[11] Forbes,2000 :12-24.
Masoro,2007 :1-17
[12] Leroi, Chippindale and Rose,1994:1244-1257.
[13] Clark,2004: 7-20.

implausible triumph of group selection over individual selection. Both processes and their function have been conserved, so that they continue to play a role in the aging of higher organisms, including humans.

If a program for aging were designed by a human engineer, it would be based on a central (flexible) timekeeping mechanism; but this is not necessarily the system that natural selection has bequeathed us. In particular, there is long-term group selection in favor of aging, but strong short-term individual selection against it. In order to shield affirmative aging mechanisms from dismantlement at the hand of individual selection, it is likely that evolution has embedded them below the surface, and deployed redundant time-keeping [14]. A single master clock would be easily hijacked by individual selection. We might expect to find several interdependent and redundant clocks for aging.



Scientists have identified unique genetic signatures strongly associated with a long and healthy life, findings that could help to further the understanding of how certain genes may offer protection from common age-related diseases like cancer, dementia and cardiovascular disease. And one day the data might lead to the development of genetic tests to predict whether a person can expect to live into old age as well as guide intervention efforts to prevent age-related illness.

A person's life span is thought to be largely determined by the combined effects of genetics and environmental factors. Twin studies, however, suggest genetics only account for approximately 20 to 30 percent of an individual's chance of surviving to age 85[15].

Lifestyle choices, like diet, exercise and smoking habits, surely play a big role in determining the period of time that one will live, also how well one ages. Studies show that Seventh-Day Adventists, whose church encourages behaviors that promote healthy aging, have a well-documented average life span of 88 years, approximately eight years longer than the average U.S. citizen. For the most part, Adventists exercise regularly, are vegetarian, and don't smoke or drink alcohol.

[14] Williams,1957:398-411.
[15] www.springernature.com/us

Genetic factors can contribute to the degree of longevity in at least two important ways: An individual may inherit certain genetic variations that cause to him or her a disease that decreases longevity; other gene variants may confer disease resistance, thereby increasing it.

To better understand the genetic components of longevity, the researchers analyzed the DNA of more than 800 subjects between the ages of 95 to 119 and compared it with DNA from random controls. The genome-wide survey identified specific genetic variations, or SNPs (single-nucleotide polymorphisms), that were associated with the longevity group.

Next, the researchers developed a genetic model including 150 SNPs in order to compute the predisposition of an individual toward EL. Their model successfully predicted exceptional longevity in a different sample of centenarians (individuals that live to age 100) with 77 percent accuracy. This demonstrates that EL is strongly associated with complex combinations of genetic variants.

The researchers found that, based on subjects' genetic profiles, the centenarians could be further divided into 19 subgroups, some of which were associated with delayed onset of age-related diseases such as dementia, hypertension and cardiovascular disease. These signatures represent different genetic paths to age 100 and beyond, Sebastiani said in the press conference.

"Centenarians are indeed a model of aging well," Perls said. Previous work has shown that 90 percent of centenarians are disability-free at the age of 93. In industrialized nations approximately one out of every 6,000 people lives beyond the age of 100. Super centenarians, or individuals that are older than 110, are even rarer—only one in seven million fall into this category.

Surprisingly, the researchers found that approximately 15 percent of control subjects also had the genetic signature associated with longevity. This suggests that many more people have the genetic potential to survive into old age than previously thought. "We know a lot about the human genome, but we also know that there is a lot that remains to be discovered," Sebastiani said. "Genetics is fundamental in EL, but it's not the only thing. So there may be other factors like environment or other lifestyles that may help people live longer and healthier lives," he added.

The researchers conclude that EL may result from an enrichment of longevity-associated gene variants that may counteract the effects of having a disease-associated gene.

2-►AGING CLOCKS

[16]A clock has both a motor and a dial. It not only measures the passage of time, but also stores reference information about what time it is now. Biological systems also measure the passage of time and keep a record of the current age. We know this because even if the metabolism is transiently disrupted, the body remembers how old it is and restores to a homeostatic condition that reflects its current age. Yeast cells can be frozen, and remember their age when thawed. Starfish are a dramatic example: a starfish can be dismembered and regenerated from a piece of itself, and the individual that regrows has a remaining life span that depends upon the age of the original animal from which it was cloned. In a clock, the motor and escapement or pendulum are more complicated than the clock dial, and we might expect the same thing in biology. Even though the information that is processed to determine the rate of aging is diverse and complex, dependent through neuroendocrine processing on multiple internal and environmental factors, nevertheless we might reasonably hope that the clock dial itself is considerably simpler. The worst case would be if the body only subjects itself to damage and dysregulation with age. The information about age could conceivably be stored globally as damaged proteins and mutated DNA and wrinkled skin and decalcified bones, etc. This is a possibility that has been raised by Aubrey de Grey (personal communication), and it would not bode well for anti-aging science. In this scenario, it may be that aging is programmed, but the nature of the program offers no opportunities for simple, upstream interventions that slow aging or set back the clock. We are left with the challenge of engineering a repair for diverse forms of damage, just as if aging had been a disordered accumulation of damage.

But there are several good indications that this pessimistic scenario is not the case, and in fact there is a relatively simple, localized "clock dial", with hands that we might hope to reset to an earlier age.

• Aging responds to signals (e.g. mTOR, SIRT1, FOXO, IGF1) in a powerful way that would not be possible if there was only the state of damage to mark the passage of time.

• Some interventions are known that successfully roll back the clock in model animals. Adult carrion beetles [17]can be manipulated with starvation to regress back to a larval state, and they can cycle multiple times through their life history. *Turritopsis* has been

[16] J. J. Mitteldorf,2013:1049
[17] Beck, and Bharadwaj,1972:1210-1211

known to do this in the wild [18]. Lab mice have been rejuvenated with telomerase [19]and with blood factors.[20]

• Finally, though we may not know the mechanism (besides state of damage) by which the body stores information about age, we can be confident that there is one, because it is absolutely essential for development. The body needs to know when to grow, when to stop growing, when to begin expressing sex hormones, triggering the onset of fertility. It is not reasonable to think that these events are triggered by a state of accumulated damage or by telomere length. Whatever clock mechanism is responsible for the timing of development and puberty is capable in theory of continuing its operation for the purpose of timing senescence. Almost all the aging interventions that have been explored experimentally to date manipulate the clock *mechanism* but not the *dial*, affecting the rate of aging but not directly the present age. The exception appears to be in telomerase activation .[21]This constitutes evidence that replicative senescence is one clock affecting human aging, and telomere length is its clock dial. Another possible exception, less well understood, was found for drosophila flies placed on caloric restriction (CR) in mid-life. Promptly upon application of CR, their mortality rate drops to a rate characteristic of an earlier age .[22]

3-►BIOLOGICAL CLOCK

Circadian biological clocks have been widely studied and are partially understood. There is evidence for an annual clock that contributes, along with environmental cues, to patterns of migration and hibernation; but mechanisms have not been identified. And the timing of growth, reproductive maturity, and senescence remain still more mysterious. The mammalian circadian rhythm is known to be regulated from the suprachiasmatic nucleus, a small structure in the middle of the brain[23]. A chemical mechanism (based on peroxiredoxins) has recently been discovered that might underlie all circadian clocks . The cycle is responsive to light and dark, and the intrinsic period is close enough to 24 h that circadian timing becomes entrained with diurnal cycles. It has been proposed that seasonal cycles in mammals are mediated through a response to light in the pineal gland. The mechanism is thought to be independent of the circadian clock[24]. Again, there is an intrinsic cycle time that is modulated by temperature and duration of daylight. In female mammals, onset of puberty is controlled by a single chemical signal: gonadotropin-releasing hormone (GnRH). But the timing of this trigger is controlled in turn by a complex calculation, based in neural as well as

18 Piraino, Boero, Aeschbach, Schmid, ,1996:302-312

19 Bernardes de Jesus, Vera, Schneeberger, Tejera, Ayuso, Bosch, and Blasco, 2012:691-704

20 Katcher, 2013: 1061-1070

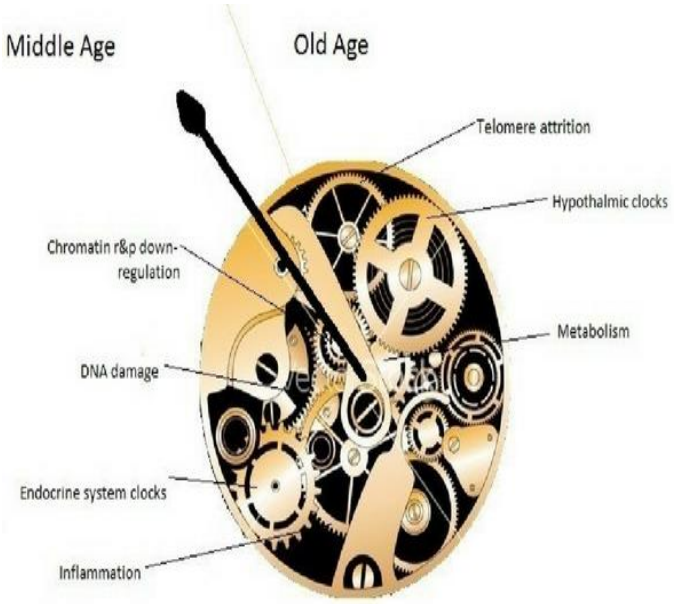
21 Bernardes de Jesus, Schneeberger, Vera, Tejera, Harley, and Blasco,2011:604-621

22 Mair, Goymer, Pletcher, and Partridge,2003:1731-1733

23 Klein, Moore, and Reppert,1991

24 Danks, 2005: 609-619.

hormonal mechanisms. "The anatomical development of the GnRH secretory system occurs relatively early in life, and that the synthetic capacity is present well before puberty in that GnRH mRNA expression reaches adult levels"[25]. Timing responds to olfactory cues, stress, fat reserve, activity, season of the year, and other stimuli. The workings of this clock remain mysterious. Aging responds to these same cues, and perhaps to others. There is reason to believe that the aging clock mechanism is at least as complex as the developmental clock. Though aging is programmed, it may not be programmed in a simple way. This accounts for the challenge that aging has posed for research and medical intervention. The fact that aging progresses under genetic controlsuggests a promising approach to anti-aging interventions, and yet the complexity of the timing mechanism has slowed the pace of progress. Although relationships between the circadian clock and the aging clock have been documented, these are not such as to suggest that the aging clock depends directly on a count of circadian cycles. For example, dysregulation of the circadian clock *in either direction* leads to accelerated aging in flies[26]. The Neuroendocrine Theory of Aging was proposed by Vladimir Dilman in 1954 [32]. Homeostatic control of hormone secretions is supported by the hypothalamus, and different hormonal levels are maintained as appropriate for different stages of growth and development. Dilman's hypothesis was that the trajectory of changes in the hypothalamus has a kind of momentum ("hyperadaptosi") that carries forward after maturity and results in "dysregulation" that characterizes the aging phenotype. The Neuroendocrine Theory is an early precedent for the Epigenetic Theory described below. "The life span, as one of the cyclic body functions regulated by "biological clocks", would undergo a continuum of sequential stages driven by nervous and endocrine signals"[27]. But Dilman did not frame this theory within the context of an adaptive program shaped by natural selection, and therefore the concept of a biological clock sits uncomfortably within its narrative. The Immunologic Theory

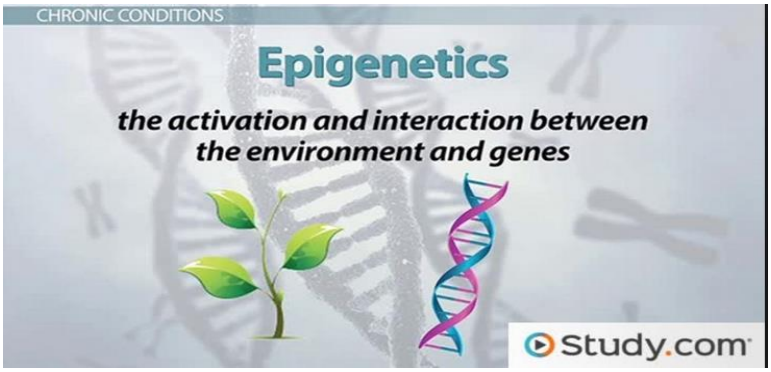


25 Ebling, 2005: 675-683.
26 Kumar, Mohan, and Sharma,2005: 641-653.
27 Weinert and Timiras, 2003: 1706-1716.

was proposed by Roy Walford in 1962.[28]The proliferation of immune cells in the blood constitutes a kind of clock, which becomes dysfunctional as the number of cells multiply. The body's cells are mutating as the number of different immune memory cells is multiplying. Chance coincidences result in the immune system attacking self with increasing frequency over time. Walford noted how many diseases of old age have a relationship to autoimmunity, but never connected this to programmed aging. He saw thymic involution as an independent cause of immune failure, and perhaps another aging clock. The hypothesis that cellular senescence represents a primary aging clock was promoted by West, culminating in a popular book published 2003[29].

4-►EPIGENETIC CLOCK HYPOTHESIS

In the fall of 2012, an article appeared by Adiv Johnson and a diverse team of scientists from the US and Europe pulling together evidence that the methylation state of the genome is related to the body's age, and proposing methylation as an appropriate target for anti-aging research. I would extend their proposal to argue that, if we believe there is an aging clock, the methylation state of the genome (especially in stem cells, because of their persistence) is logically the first place to look for its "clock dial". Seeking a system of global signals that affects the metabolic state of the entire body, we would look as far upstream as possible. Upstream takes us to gene expression. Further up, there may be signals that affect gene expression globally, but these, too, are products of genes, and hence they can be regarded as part of a self-modifying program for gene expression. If there is not in evidence another, separate clock which feeds down to affect gene expression, then it is logical to assume that this self modifying program functions as a clock in its own right. We know that gene expression changes with age, and that this has the potential to affect all aspects of the metabolism and the aging phenotype. If there is an aging clock, then its output must be transduced so as to affect gene transcription. Merging the clock into the transcription state of the genome would be the most economical implementation of a clock mechanism, obviating the need for a separate record of the age state of the body. Gene transcription is affected by transient signaling, and also by more persistent epigenetic markers. The most important of these persistent



28 Walford, 1964: 195-197.
29 West, 2003

markers is the genome's methylation pattern. The "methylome" contains information that is both programmable and persistent. Cytosine (the "C" in ACGT) is one of the four nucleic acid residues that form chromosomal DNA. Within the DNA molecule, cytosine can accept a methyl group to form 5-methylcytosine, and this suppresses transcription locally in genes where methylation has occurred [30]. Methylation patterns tend to be copied along with DNA replication, and they can even last through several generations as a form of epigenetic inheritance [31]. An epigenetic clock has the potential to regulate

growth, development and sexual maturity, as well as aging. If no other clock has been discovered that controls the timing both of development and aging, then our default hypothesis ought to be that the epigenetic state of the genome is its own clock. The methylation state of the genome is also self-modifying in the sense that transcription of methyl transferases and related enzymes creates the mechanism for feeding back upon the methylation state. This feedback implies the basis for a clock mechanism. Genes that are transcribed today create the metabolic environment that cascades into signals that reconfigure the methylation state and program the genes that will be transcribed tomorrow. The above constitutes a general, theoretical argument for epigenetic state as an aging clock. There are also specific experimental results that point in this direction.

• Gene expression profiles change substantially with age. There is reason to believe that an individual with youthful gene expression is functionally a youthful individual [32].

• Methylation has about the right degree of persistence.We know that methylation contains epigenetic information that is passed on in a soft way when DNA is replicated.

• In general, methylation decreases with age (though there are characteristic regions that become hypermethylated). Hypomethylation has been associated with "frailty" and markers of biological age.[33]

• Fruit flies with a copy of the methyl transferase *dnmt2* in their genome lived 58% longer than control flies. Conversely, flies engineered to be +/– for *dnmt2* had life spans 25% shorter .[34]

• Similar experiments with mice yield more nuanced results. Early, studies that have not been reproduced reported that methylation was actually higher in *dnmt1* +/– mice than in +/+ controls [35]. More recently, neural deficiencies and low bone densities, increasing with age, have been reported associated with engineered *dnmt1* deficiencies .

[30] Johnson, Akman, Calimport, Wuttke,Stolzing, and de Magalhres, 2012: 483-494.

[31] Jablonka and Raz,2009: 131-176.

[32] Johnson, Akman, Calimport, Wuttke, Stolzing and de Magalhres,2012: 483-494.

[33] Bellizzi, D'Aquila, Montesanto,Corsonello, Mari, Mazzei, Lattanzio, and Passarino,2012:169-179.

[34] Lin,Tang, Reddy,and Shen,2005:861-864.

[35] Yung, Ray, Eisenbraun, Deng, Attwood, Eisenbraun, Johnson, Miller, Hanash and Richardson, 2001 :268-276.

• In monozygotic twins, methylation patterns are similar when young, but diverge over time. This suggests a stochastic component that may account for diverse dysregulations associated with aging. Age is determined almost certainly by the detailed pattern of methylation and other epigenetic markers, not simply the crude quantity of methylation. And yet there is evidence that senescing cells are characterized by progressive demethylation, so that chromosomes in younger cells tend to be more methylated than in older cells [36]. The possibility that demethylation may be an aging clock was first proposed by Bowles based on the fact that "aging is accompanied by DNA demethylation" . In fact, the animal genome loses practically all 5-methylcytosines during its life, the rate of the loss being inversely proportional to maximal lifespan of the species . The same occurs in cell cultures, again the rate being inversely proportional to the cell lifespan (Hayflick limit)

Drugs targeted to sirtuins [37] have found some earlier success in extending life span by indiscriminate silencing of gene expression. The mechanism of sirtuins is mediated via histone de-acetylation rather than methylation,

but the ease with which simple silencing of genes could extend life span is suggestive. Also, protein-restricted and methionine-restricted diets retard aging [38], presumably by dialing down expression of many genes indiscriminately.

[36] Wilson, and Jones,19831055.

[37] Kelly,2010 :245-263.

[38] Zimmerman,Malloy,Krajcik,2003:47.

Conclusion and Suggestions

We found that there are several reasons that cause Latinos to live longer, so they face a 24% lower risk of death than other racial group. According to Horvath, the UCLA research points to an epigenetics explanation for Latinos' longer life spans. Epigenetics the study of changes of the DNA molecule that influence which genes are active but don't alter the DNA sequence itself. So someway genetic factors are associated with increased longevity, because an individual may inherit certain genetic variations that predisposed him to disease that decreases longevity, other gene variation may confer disease resistance thereby increasing it. According to many researches, the biological clock measured Latino women's age as 2.4 years younger than non-Latino women of the same age after menopause, so that clock 's rate speeds up or slows down depending on a person's age. New discoveries proves that scientists can rewind the body's biological clock and restore it to zero. I think there will be many researches that make us understand how certain genes may offer protection from common age-related diseases like cancer, dementia and cardiovascular disease. One day this might lead to the development of genetic tests to predict whether a person can expect to live into old age as well as guide intervention efforts to prevent age-related illness, and know how to slow the aging process for everyone.

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