The Longevity Dividend: Altering the Future Course of Health and Longevity

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Summary

Message 1 **Gompertz saw biology in the life table, and he was right – there is a law of mortality.**

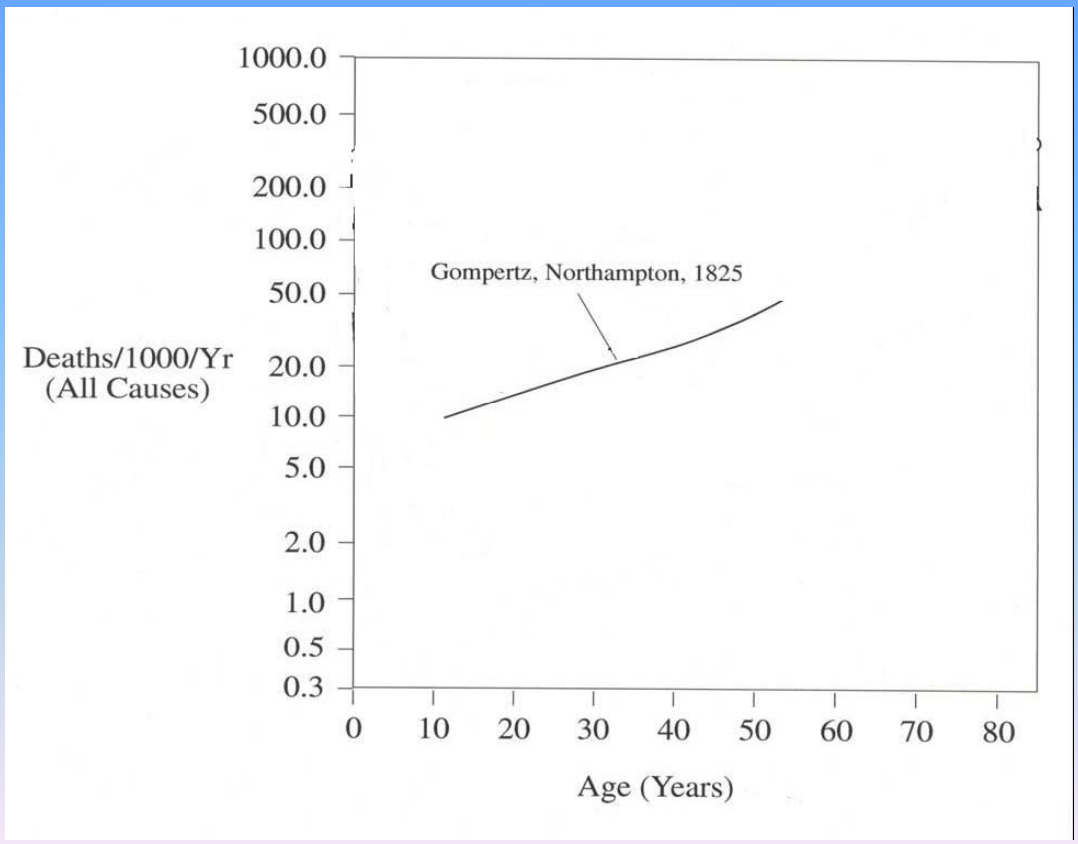
Message 2 **Future trends in mortality and longevity will be driven by biology, not past trends. Linear thinking got us in trouble in the past, and it's still getting us in trouble today.**

Message 3 **A life expectancy of 100 is highly unlikely, but the number of centenarians will rise dramatically.**

Message 4 **Life expectancy is likely to rise rapidly for some, and decline dramatically for others. Education is a longevity trump card.**

Message 5 **If the retirement age was indexed to longevity as originally intended, it would be much higher than it is today. However, raising the retirement age has dramatically different effects on population subgroups.**

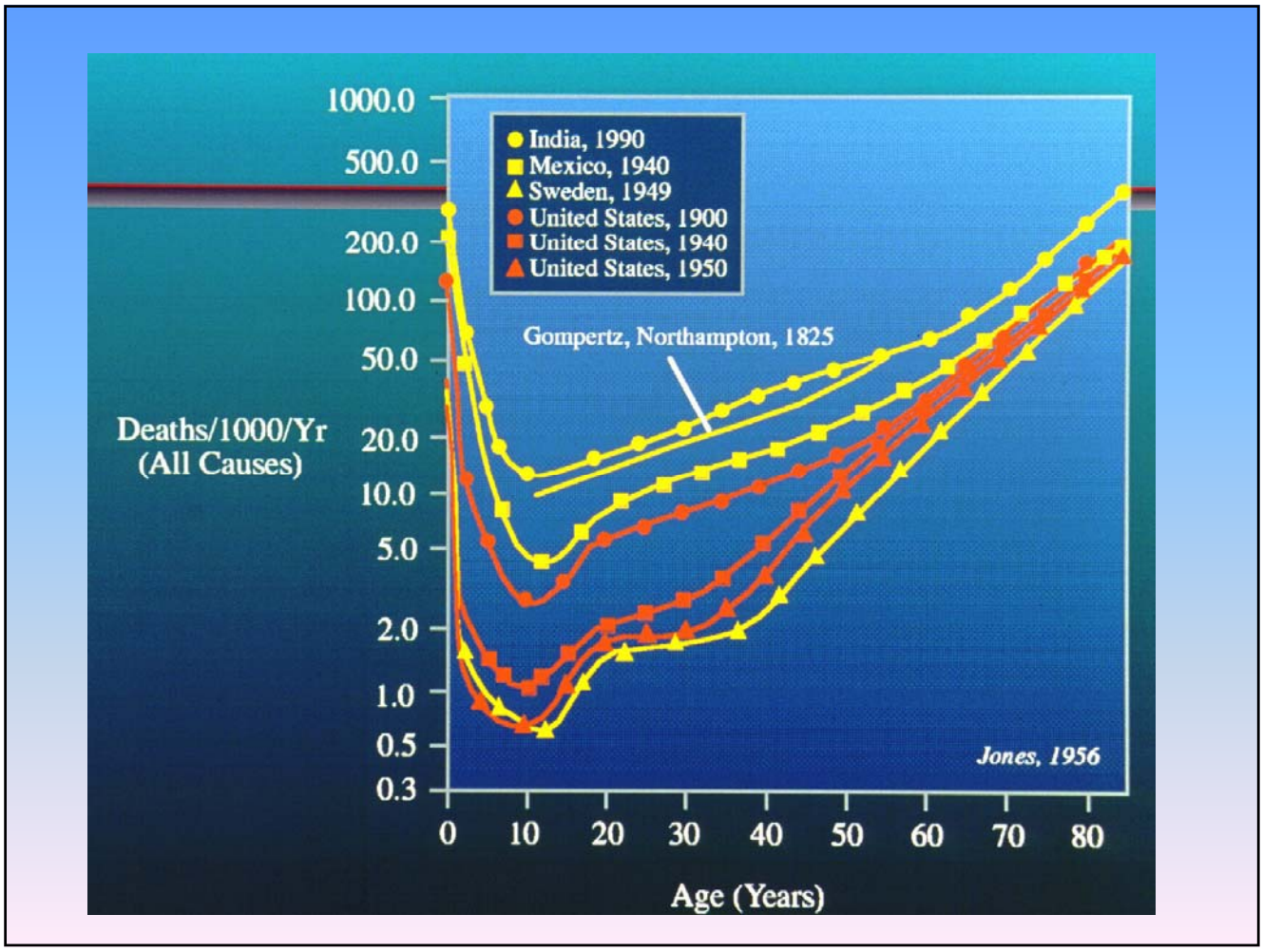
Message 6 **Two forthcoming revolutions in medicine and aging science are about to permanently change the landscape of human longevity in the future.**


1000.0
500.0
200.0
100.0
50.0
20.0
10.0
5.0
2.0
1.0
0.5
0.3
Deaths/1000/Yr
(All Causes)
Gompertz, Northampton, 1825
0
10
20
30
40
50
60
70
80
Age (Years)

separated Gompertz from his fellow actuaries was his attempt to provide a biological explanation for his empirical law—*"and contemplating on this law of mortality, I endeavored to enquire if there could be any physical cause for its existence"* (1825: 333). The work of Gompertz spawned a small but persistent school of thought within the actuarial sciences, exemplified by the work of Makeham (1867), Brownlee (1919), and Greenwood (1928)—all of whom argued that the life table embodied biological principles in addition to its traditional role as a working tool for actuaries. The "law of mortality" was originally developed as an actuarial tool with a focus exclusively on human mortality, but enhanced by modest speculation about the biology of aging.

tion: $\lambda(t)=\alpha\exp(\beta t)$.[1] Perhaps what is more important, Gompertz endeavored to find a physical cause for his law by suggesting that "death may be the consequence of two generally coexisting causes; the one, chance, without previous disposition to death or deterioration; the other, an unspecified force that destroyed the material of organization necessary for life" (Gompertz 1825: 517).

Gompertz (1825) – summarized in Olshansky and Carnes (1997) Ever Since Gompertz


1000.0
500.0
200.0
100.0
50.0
20.0
10.0
5.0
2.0
1.0
0.5
0.3
Deaths/1000/Yr
(All Causes)
India, 1990
Mexico, 1940
Sweden, 1949
United States, 1900
United States, 1940
United States, 1950
Gompertz, Northampton, 1825
Jones, 1956
0
10
20
30
40
50
60
70
80
Age (Years)

The Bridge of Life

The Chances of Death by Karl Pearson (1897)

VI. A Comparison of the Laws of Mortality in Drosophila and in Man

PROFESSOR RAYMOND PEARL

The American Naturalist (1922)

In the first study a rough, purely graphical comparison of the l_x lines of the *Drosophila* and certain human life tables was instituted. This comparison, rough as is was, made apparent at once the fact that there was a fundamental similarity in laws of mortality in these two organisms.

It is my purpose in the present paper to make a more exact comparison of the values of the life table functions in the two cases. It will be seen that the similarity is even closer than was supposed from the rough comparison, and that in fact we are dealing here with qualitatively identical expressions of an obviously fundamental biological law.

VI. A Comparison of the Laws of Mortality in Drosophila and in Man

PROFESSOR RAYMOND PEARL

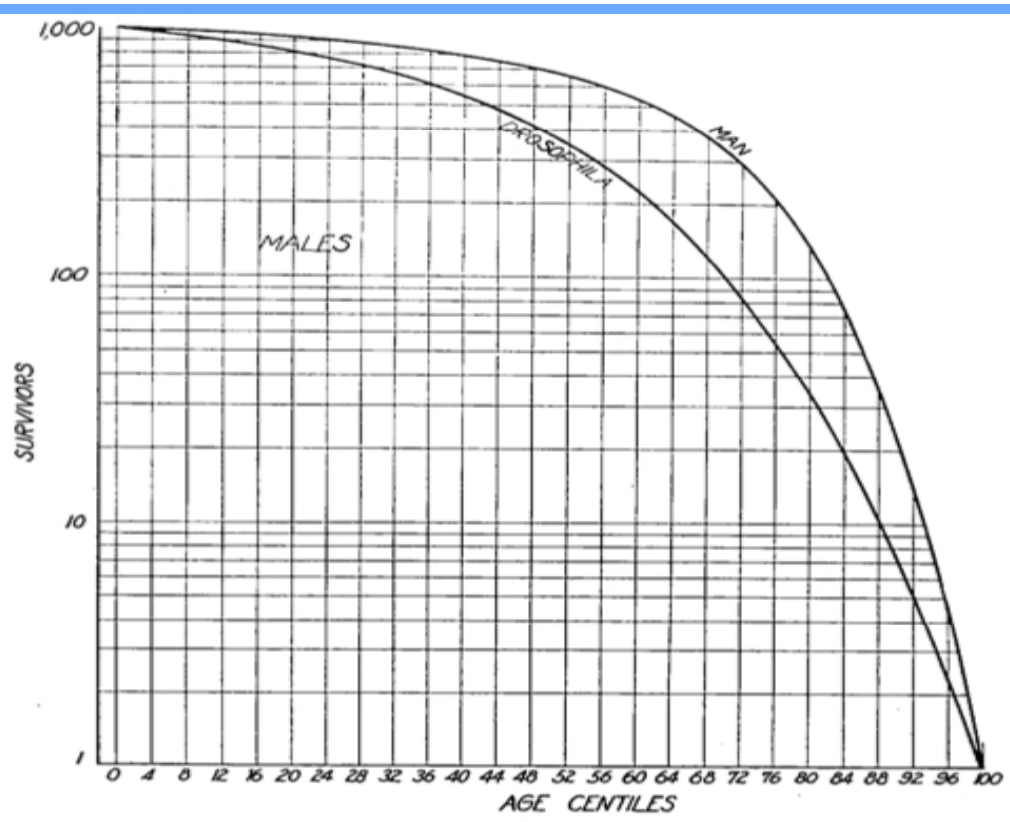
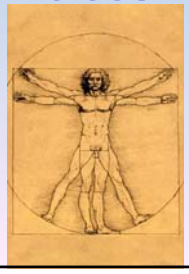


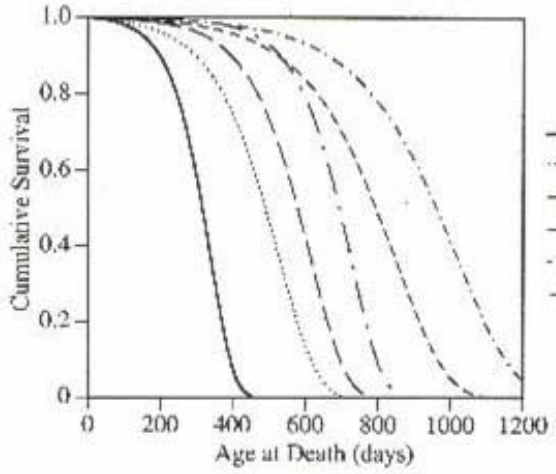
FIG. 1. Comparing the survivorship distributions of *Drosophila* and man (males in both cases) over the equivalent life spans.

Solving the law of mortality required conditions that were difficult to overcome

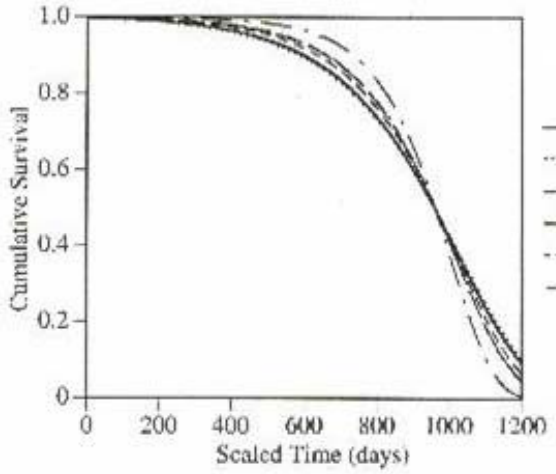
- The ability to reliably measure Intrinsic Mortality
- Access to reliable intrinsic mortality rates for different species
- Scaling Time





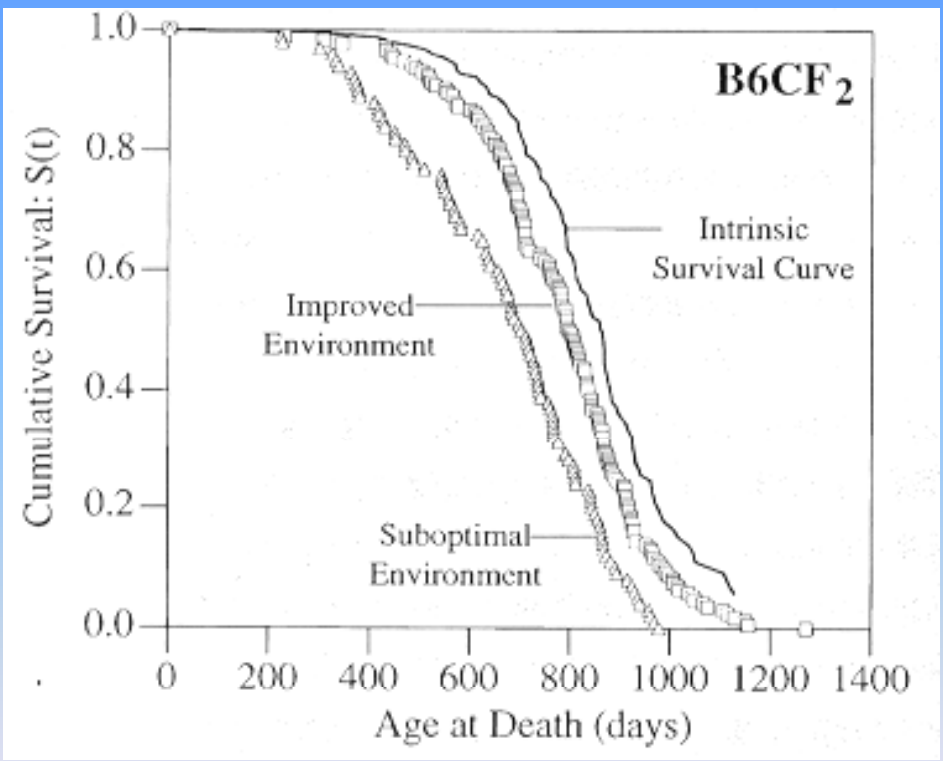


Strain	Median
Ruby	317
MWA	490
C	578
A/J	692
BALB/c	793
B6CF$_1$	961

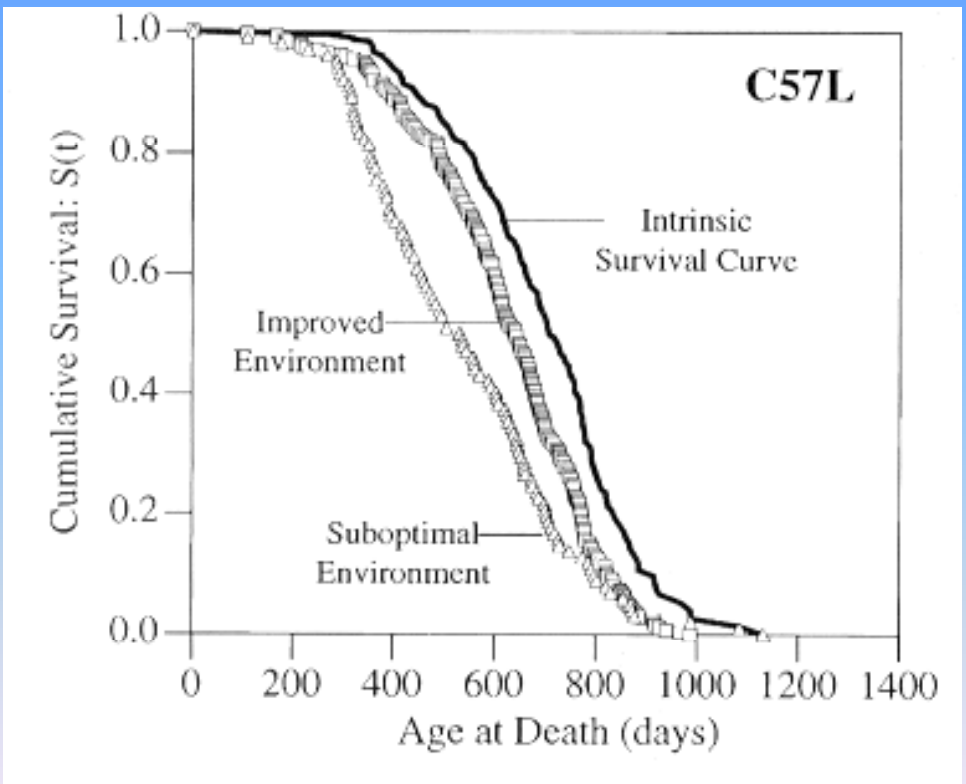


Strain	Median
Ruby	961
MWA	961
C	961
A/J	961
BALB/c	961
B6CF$_1$	961

Demonstration of consistency in the timing of death.

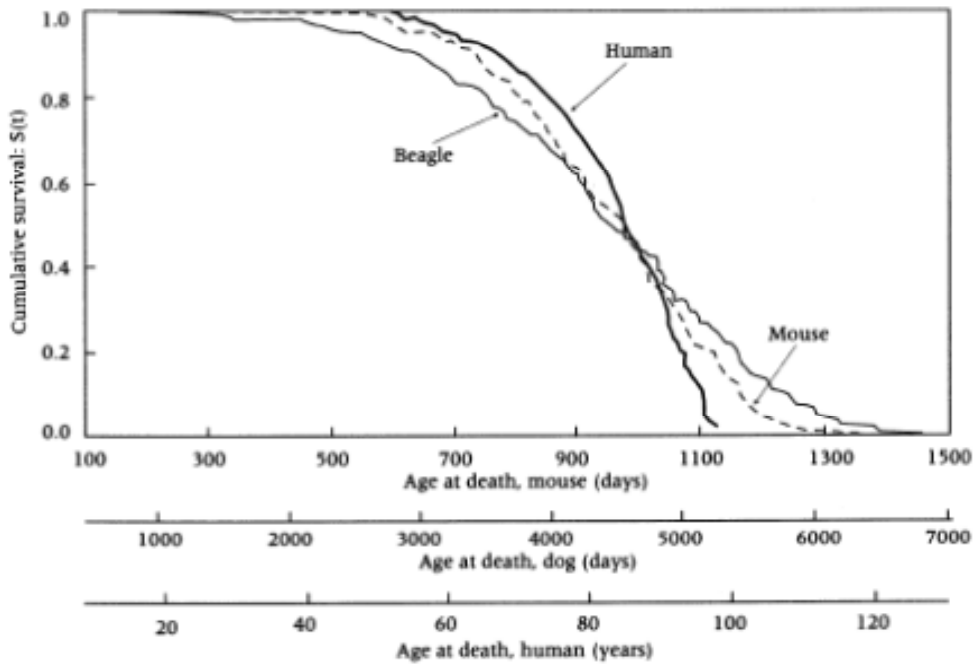


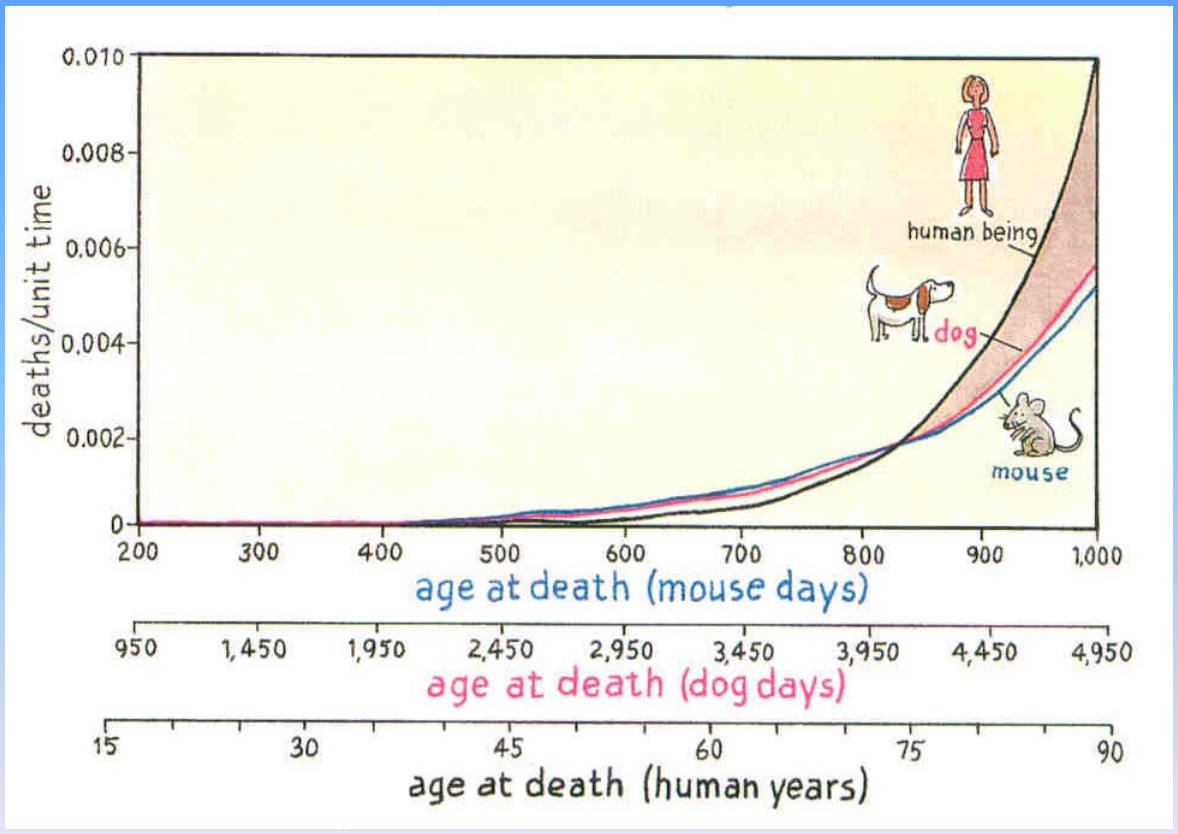
Source: Carnes and Olshansky, 2001



Source: Carnes and Olshansky, 2001

FIGURE 3 Comparison of cumulative survival curves for the mouse, beagle, and human populations plotted on the time scale for the B6CF$_1$ mouse strain. Additional time axes are shown for the beagle and human to demonstrate the effect of scaling





Source: Olshansky, Carnes and Grahn. 1998. American Scientist 86: 52-61

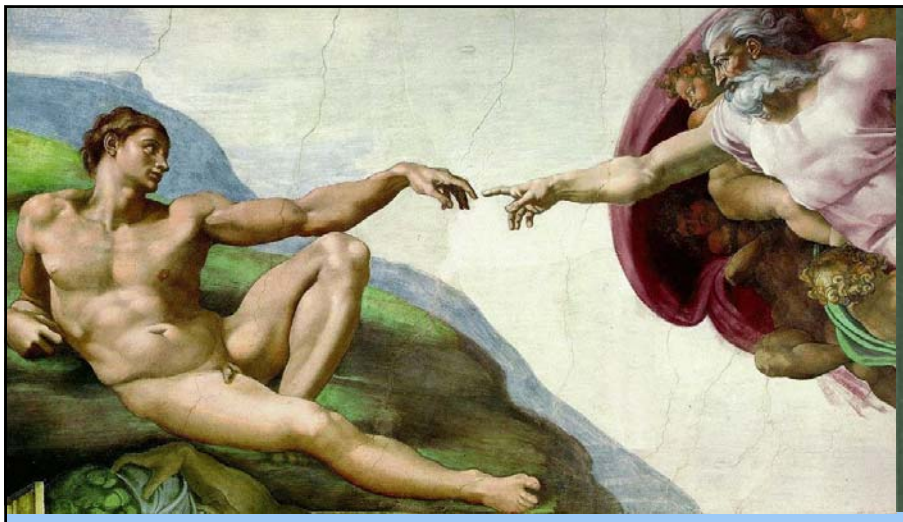
Why Do We Age and Live as Long as We Do?

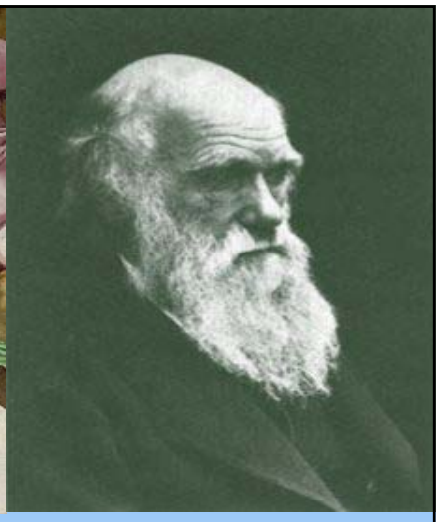
Is There Biology in the Life Table?

"Nothing in biology makes sense except in the light of evolution."

Theodosius Dobzhansky

The American Biology Teacher, March 1973

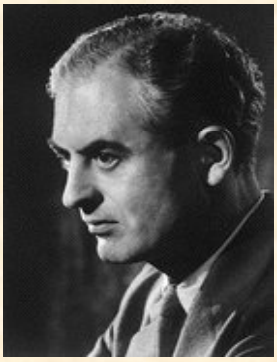




BOTH Michelangelo and Darwin WERE RIGHT

The human body is a miraculous machine that works with near artistic perfection – for a while. Time reveals the "flaws" in a body design that was not intended for long-term use.

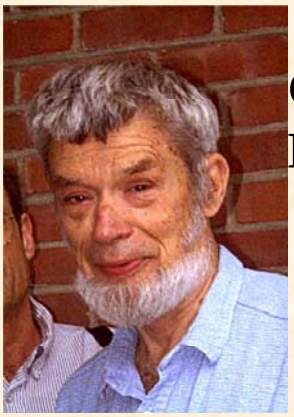
THE EVOLUTIONARY THEORY OF SENESCENCE



SIR PETER MEDAWAR

Mutation Accumulation Genetic Dustbin

Alleles with detrimental affects are "pushed" by natural selection to either side of the reproductive window.



GEORGE WILLIAMS

Antagonistic Pleiotropy

Genes that are harmful late in life are selected if they are favorable early in the lifespan.

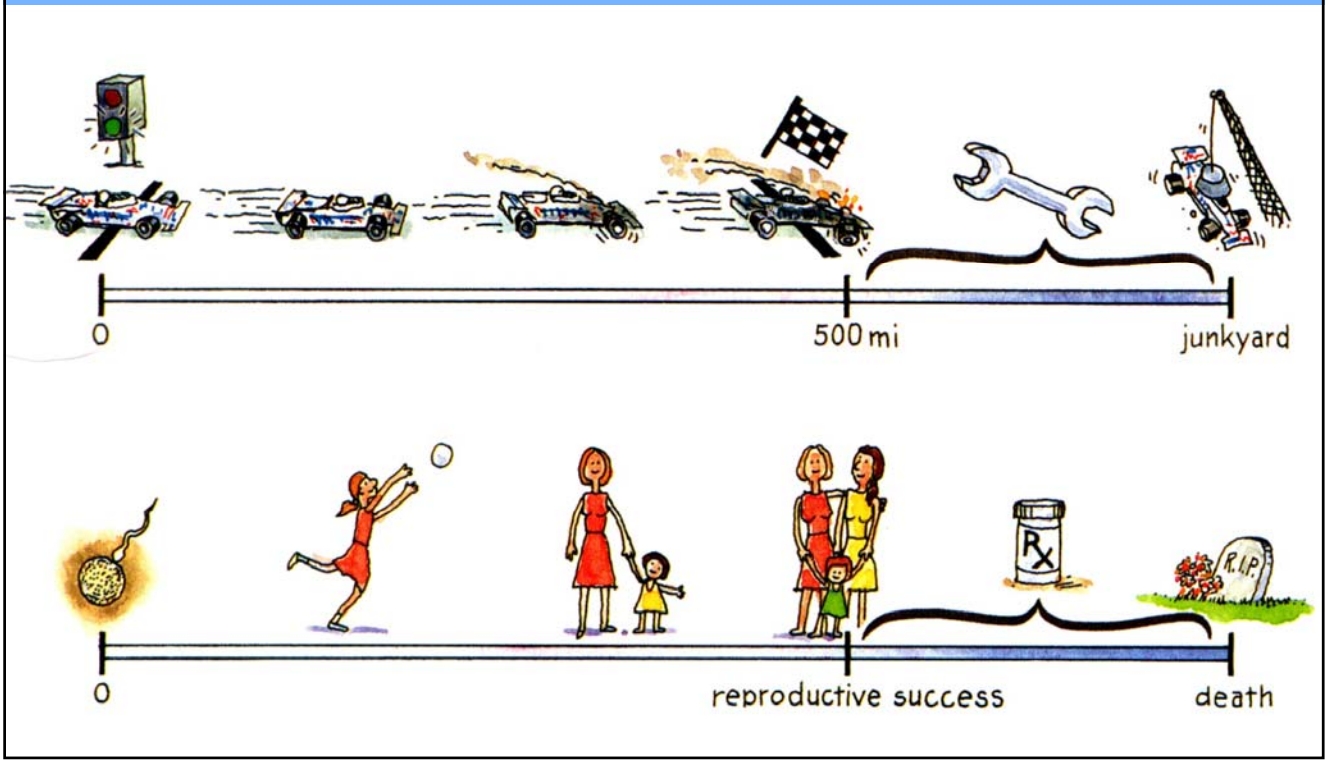
CHARLES DARWIN

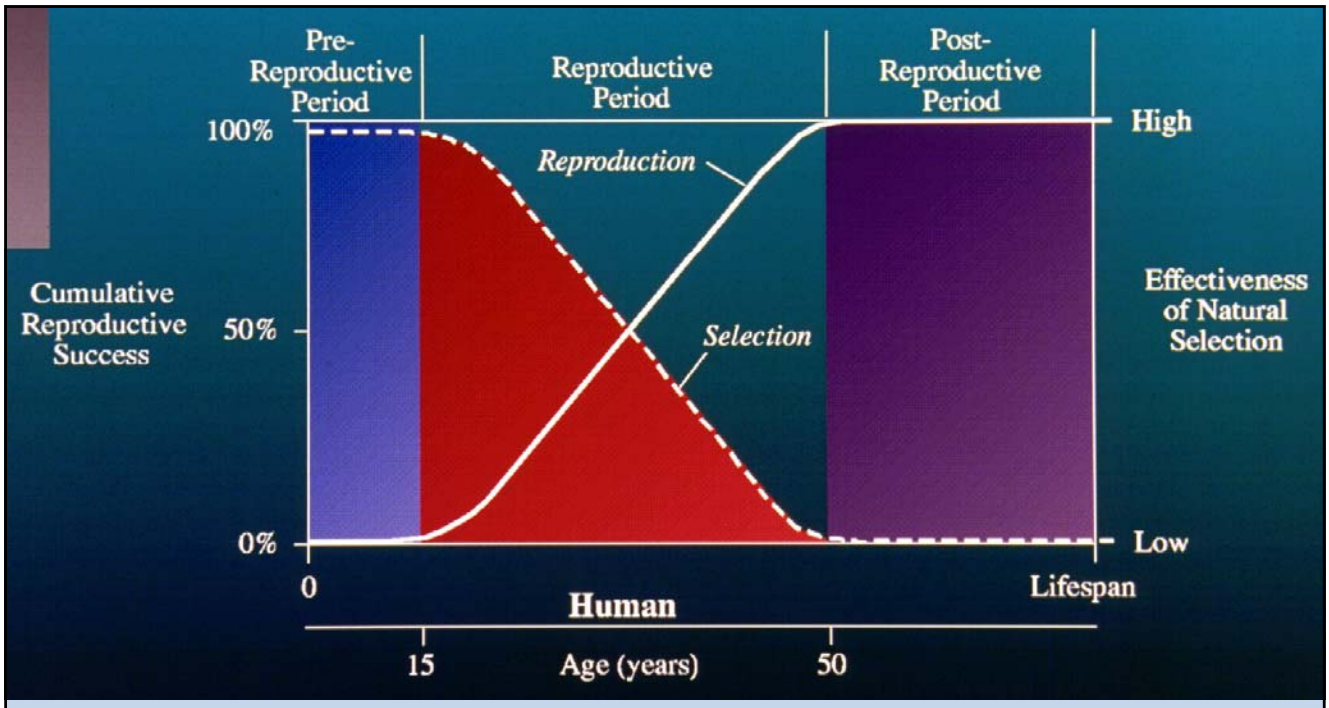
TOM KIRKWOOD

Disposable Soma

In a world with limited physiological resources, selection favors investment in reproduction at the expense of immortality.

WHY DO WE LIVE AS LONG AS WE DO?

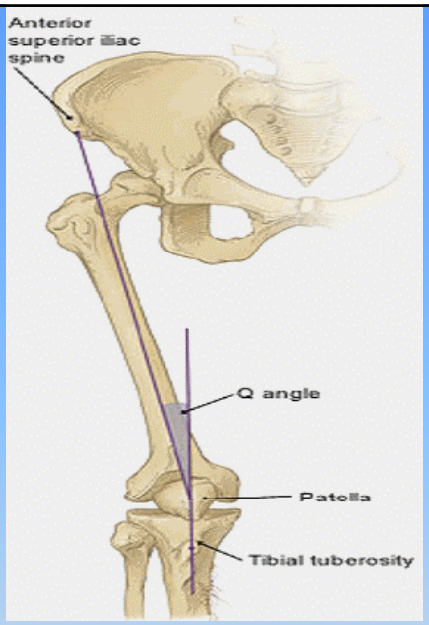




There is a remarkable consistency to the timing of death across species.

Duration of life is calibrated to the onset and length of a species' reproductive window.


77,000 days
Bowhead
Whale
55,000 days
Sea Turtle
29,000 days
Human
26,000 days
Elephant
5,000 days
Dog
1,000 days
Mouse

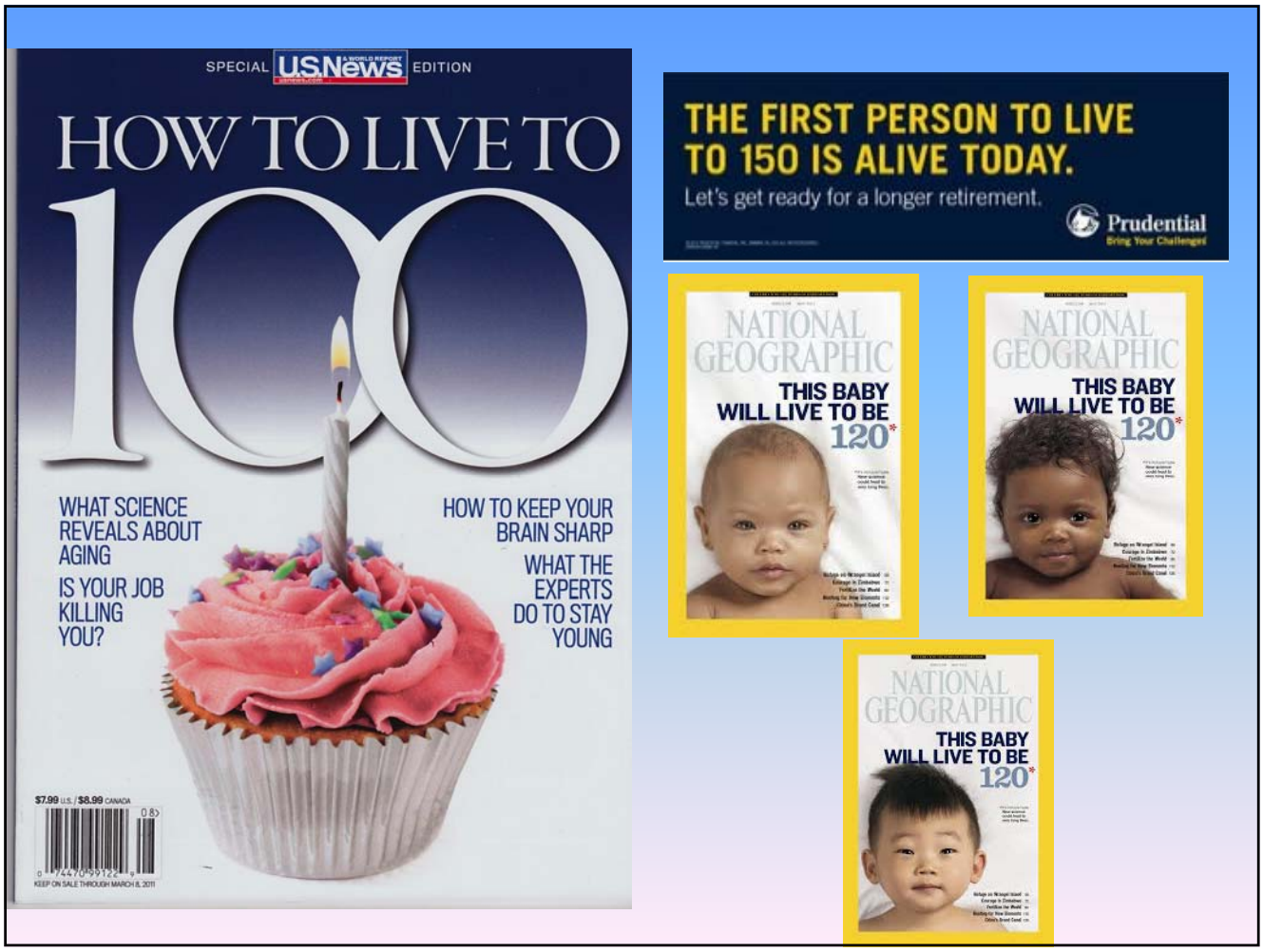


Although there is no genetic program that limits how fast humans are capable of running, there are nevertheless biomechanical constraints on running speed.

Although there is no genetic program that limits the duration of life, there are nevertheless biomechanical constraints on the functioning of body parts that influence how long we live.

Can most live to 100?

Can we really add decades of life to people aged 70+ today faster than we added decades of life to children born in the early 20th century?


SPECIAL U.S.News EDITION
HOW TO LIVE TO 100
WHAT SCIENCE REVEALS ABOUT AGING
IS YOUR JOB KILLING YOU?
HOW TO KEEP YOUR BRAIN SHARP
WHAT THE EXPERTS DO TO STAY YOUNG
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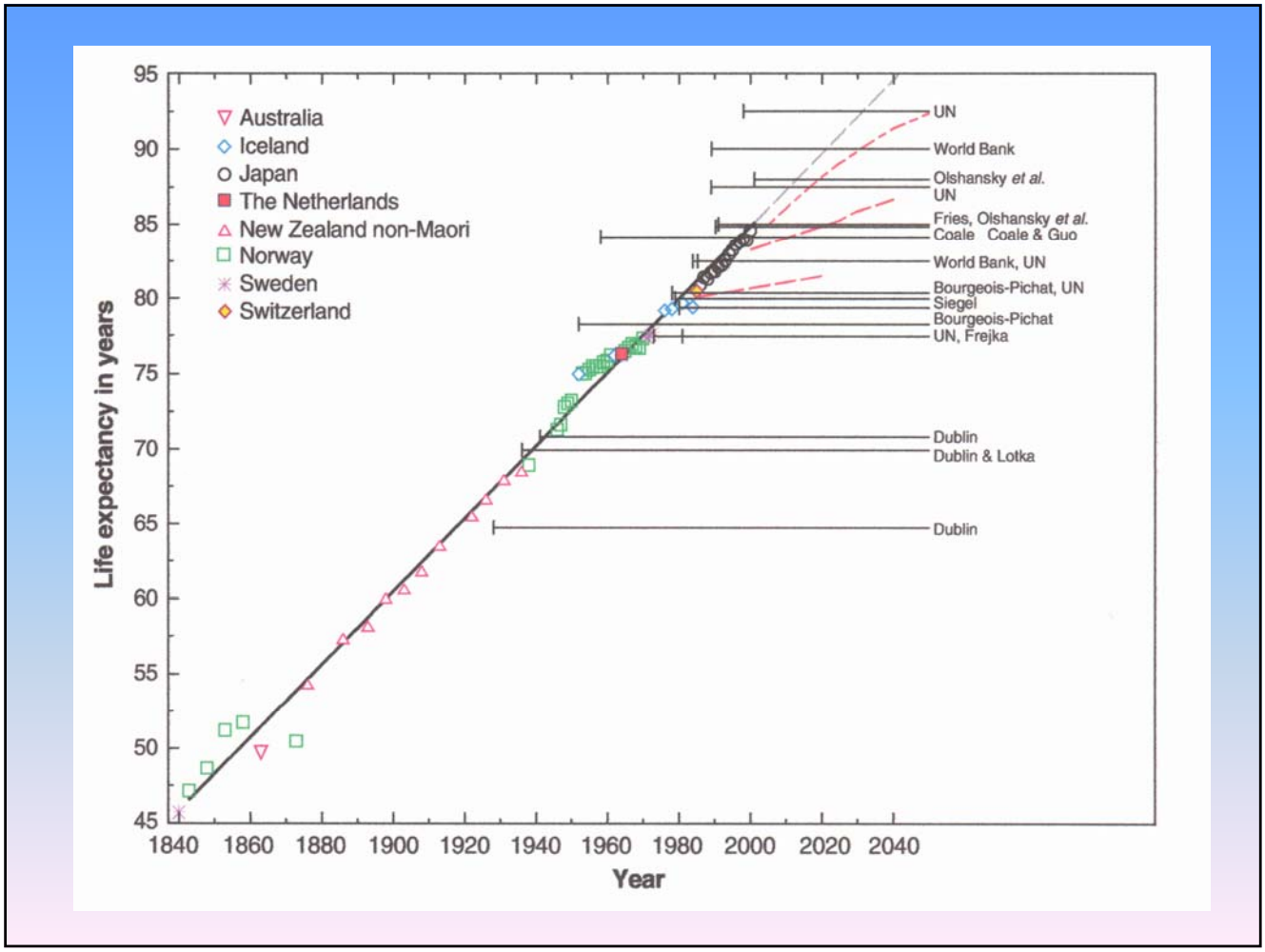
Bring Your Challenges
NATIONAL GEOGRAPHIC
THIS BABY WILL LIVE TO BE 120*
NATIONAL GEOGRAPHIC
THIS BABY WILL LIVE TO BE 120*
NATIONAL GEOGRAPHIC
THIS BABY WILL LIVE TO BE 120*


Australia
Iceland
Japan
The Netherlands
New Zealand non-Maori
Norway
Sweden
Switzerland
UN
World Bank
Olshansky et al.
UN
Fries, Olshansky et al.
Coale Coale & Guo
World Bank, UN
Bourgeois-Pichat, UN
Siegel
Bourgeois-Pichat
UN, Frejka
Dublin
Dublin & Lotka
Dublin
Life expectancy in years
Year
95
90
85
80
75
70
65
60
55
50
45
1840
1860
1880
1900
1920
1940
1960
1980
2000
2020
2040

Does History Repeat Itself?

- Forecasters have a long history of both underestimating and overestimating longevity. Today a new set of errors are being made because they are relying almost exclusively on the extrapolation of past trends into the future. There is definitive evidence that some cohorts in some countries will live shorter lives and others will live longer lives than anticipated from actuarial models.

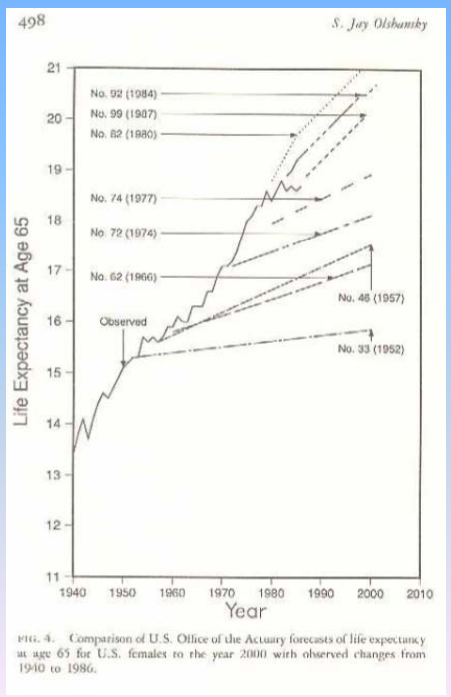


FIG. 4. Comparison of U.S. Office of the Actuary forecasts of life expectancy at age 65 for U.S. females to the year 2000 with observed changes from 1940 to 1986.

Source: Olshansky, 1988

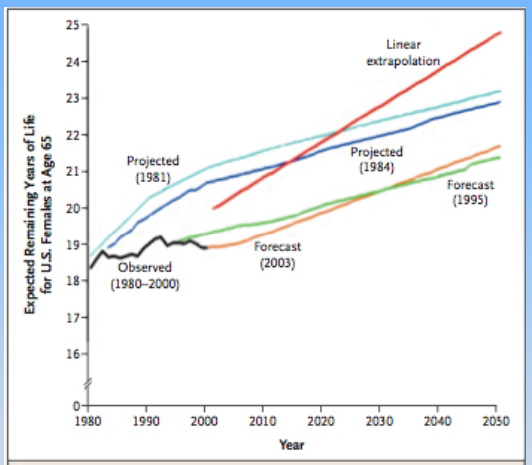
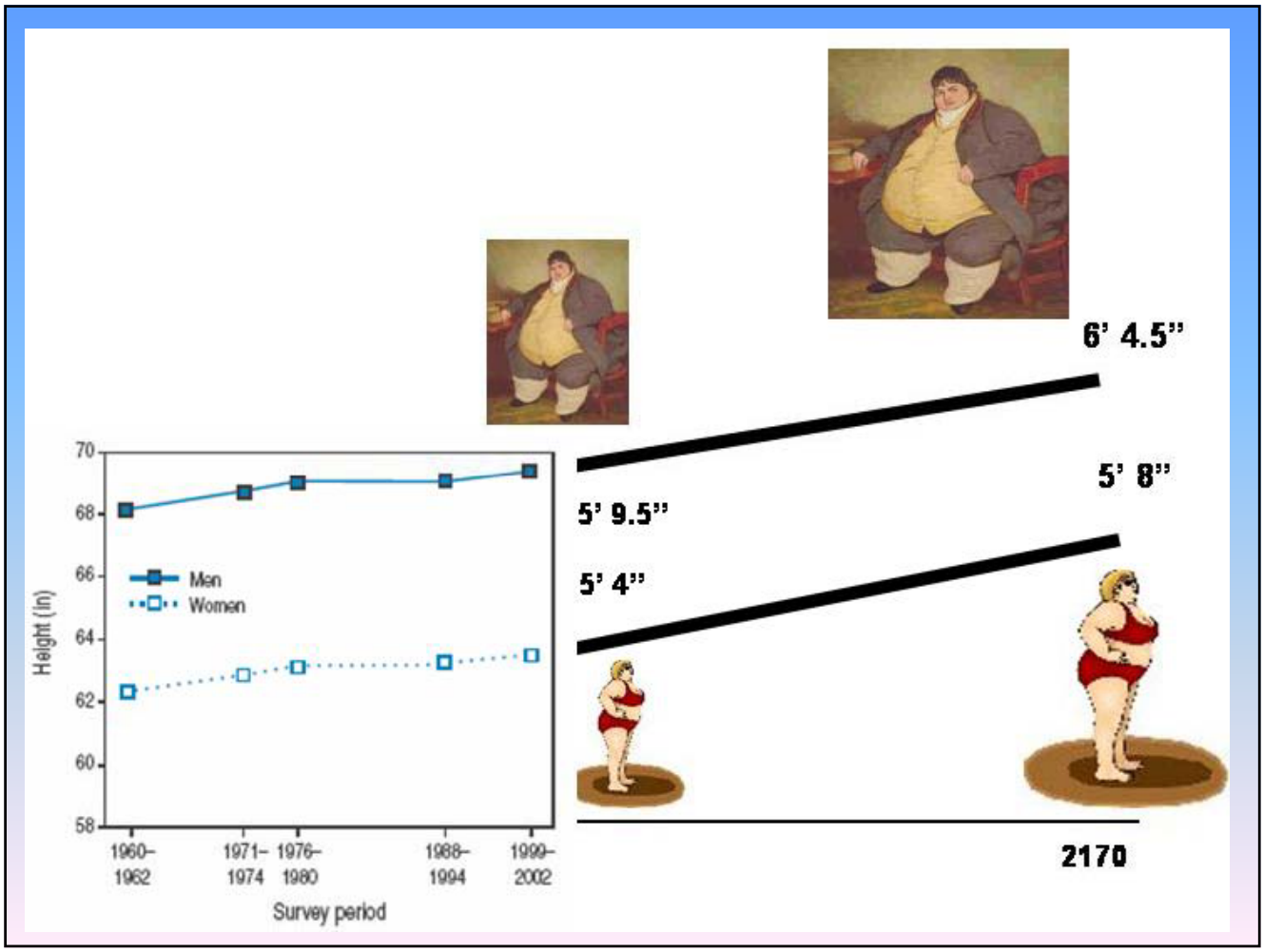
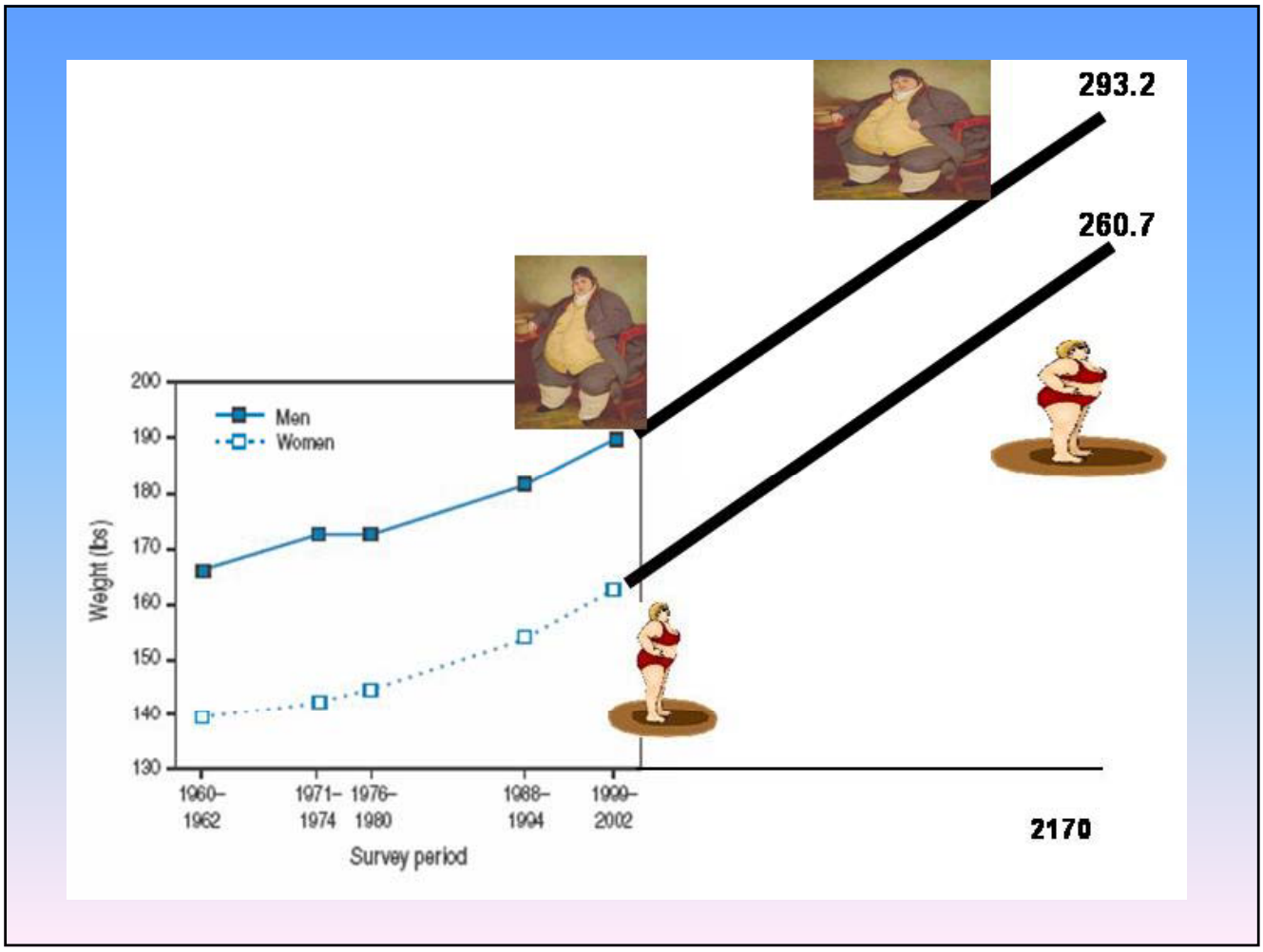


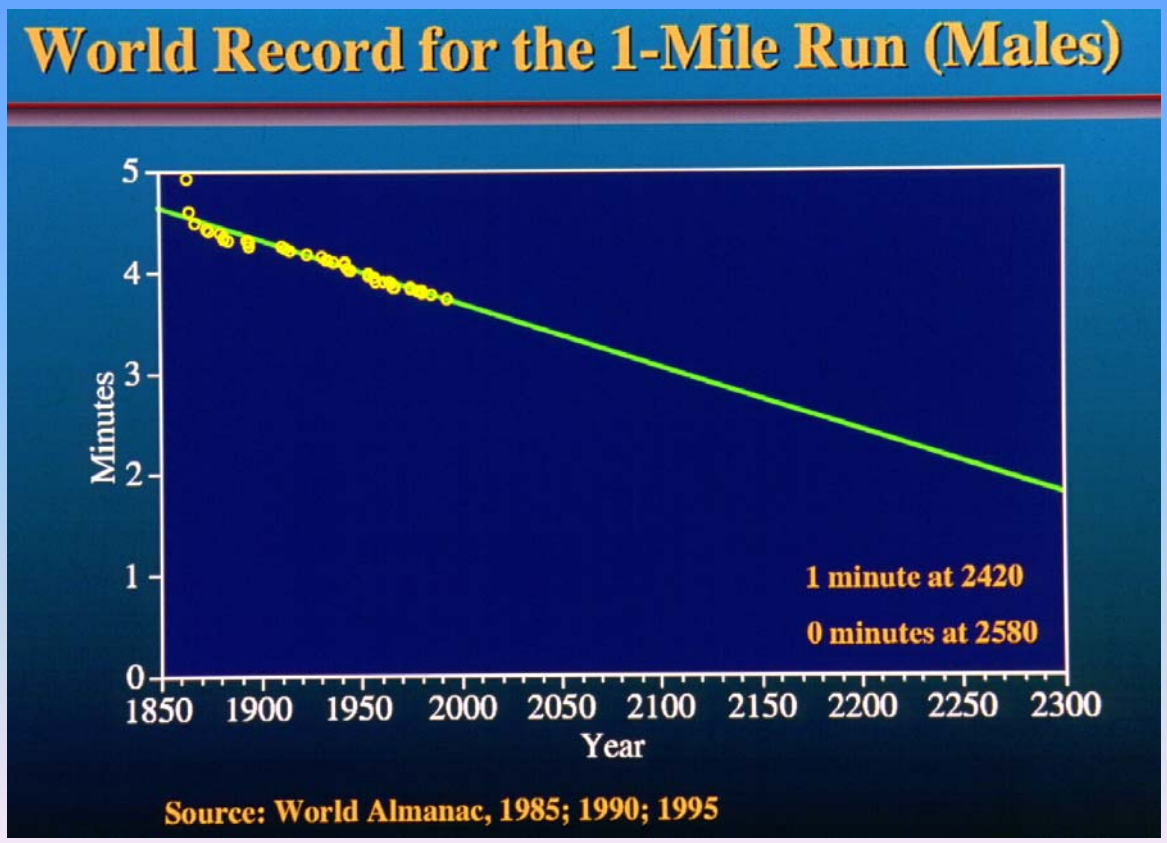
Figure 3. Observed and Projected Life Expectancy at Age 65 for U.S. Females (1980 to 2050).

Shown are observed changes, from 1980 to 2000,[46] in expected remaining years of life at age 65 for females in the United States, projections of the expected remaining years of life at age 65 made by the SSA in actuarial studies published in 1981[46] and 1984,[47] and forecasts based on the SSA's 1995 and 2003 Trustees Reports.[48,49] A forecast of the expected remaining years of life at age 65 for females in the United States, assuming the observed trend from 1940 to 2000 is extrapolated linearly from 2000 to 2050, is shown.

Source: Olshansky et al., 2005


70
68
66
64
62
60
58
Height (in)
Men
Women
1960–1962
1971–1974
1976–1980
1988–1994
1999–2002
Survey period
6' 4.5"
5' 8"
5' 9.5"
5' 4"
2170


293.2
260.7
2170
Men
Women
Weight (lbs)
200
190
180
170
160
150
140
130
1960–1962
1971–1974
1976–1980
1988–1994
1999–2002
Survey period


World Record for the 1-Mile Run (Males)
Minutes
5
4
3
2
1
0
1850
1900
1950
2000
2050
2100
2150
2200
2250
2300
Year
1 minute at 2420
0 minutes at 2580
Source: World Almanac, 1985; 1990; 1995

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doi:10.1093/gerona/gls142

Perspective

Can Human Biology Allow Most of Us to Become Centenarians?

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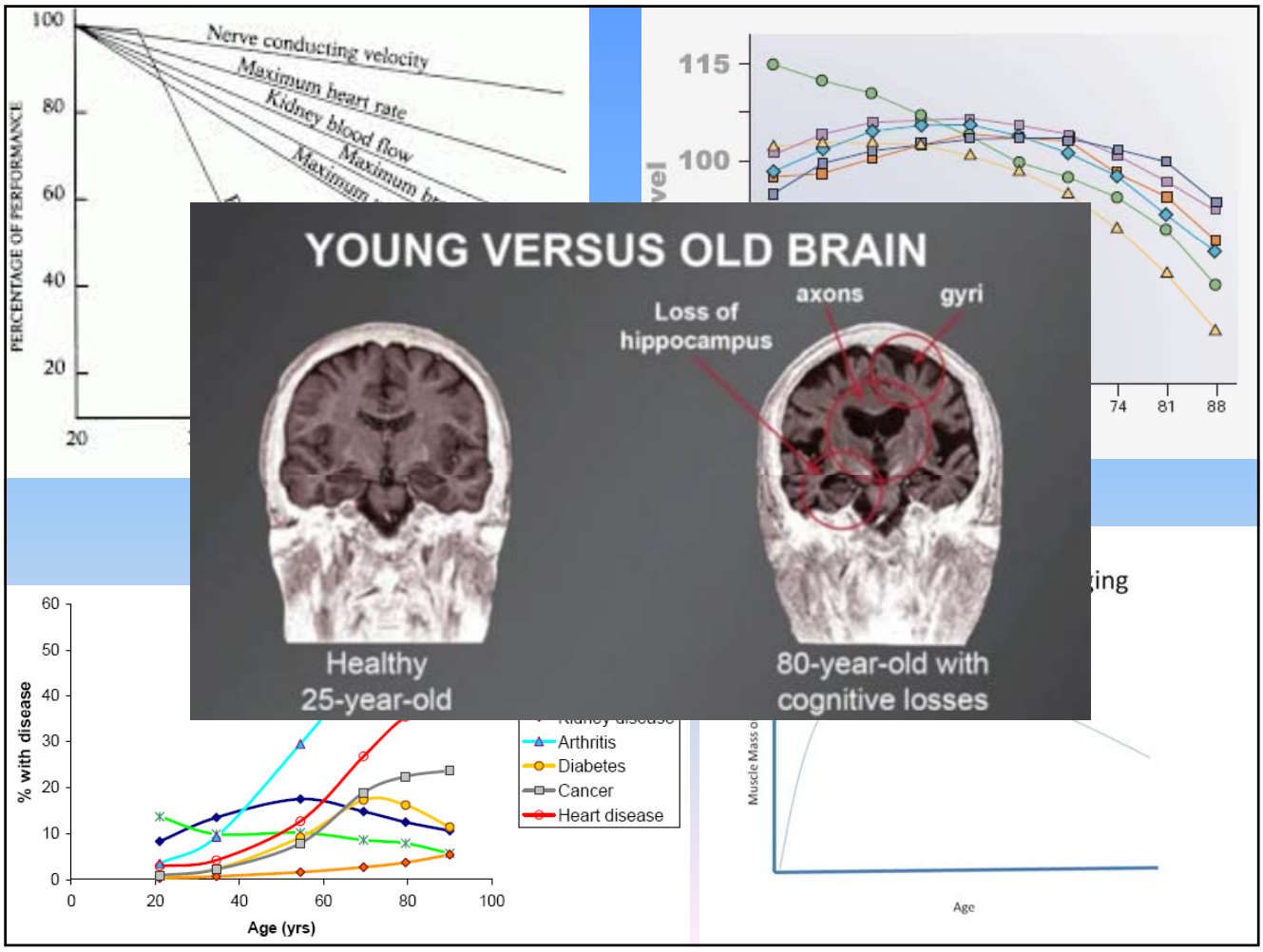
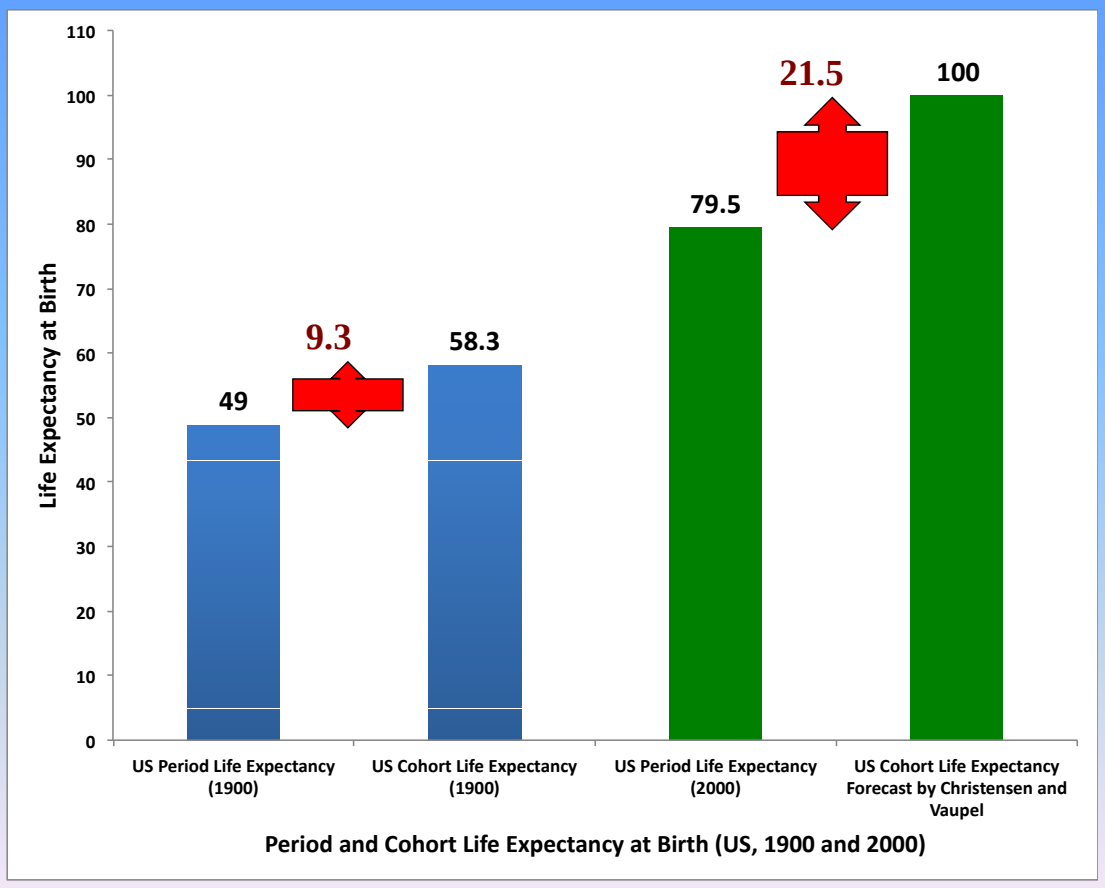
Life span is a topic of great interest in science, medicine and among the general public. How long people live has a profound impact on medical costs, intergenerational interactions, and the solvency of age-based entitlement programs around the world. These challenges are already occurring and the magnitude of their impact is, in part, proportional to the fraction of a population that lives the longest. Some demographic forecasts suggest that most babies born since the year 2000 will survive to their 100th birthday. If these forecasts are correct, then there is reason to fear that the financial solvency of even the most prosperous countries are in jeopardy. We argue here that human biology will preclude survival to age 100 for most people.

No!


YOUNG VERSUS OLD BRAIN
Loss of hippocampus
axons
gyri
Healthy 25-year-old
80-year-old with cognitive losses
PERCENTAGE OF PERFORMANCE
Nerve conducting velocity
Maximum heart rate
Kidney blood flow
% with disease
Age (yrs)
Arthritis
Diabetes
Cancer
Heart disease
Age



Source: US Social Security Administration and Christensen et al., 2009

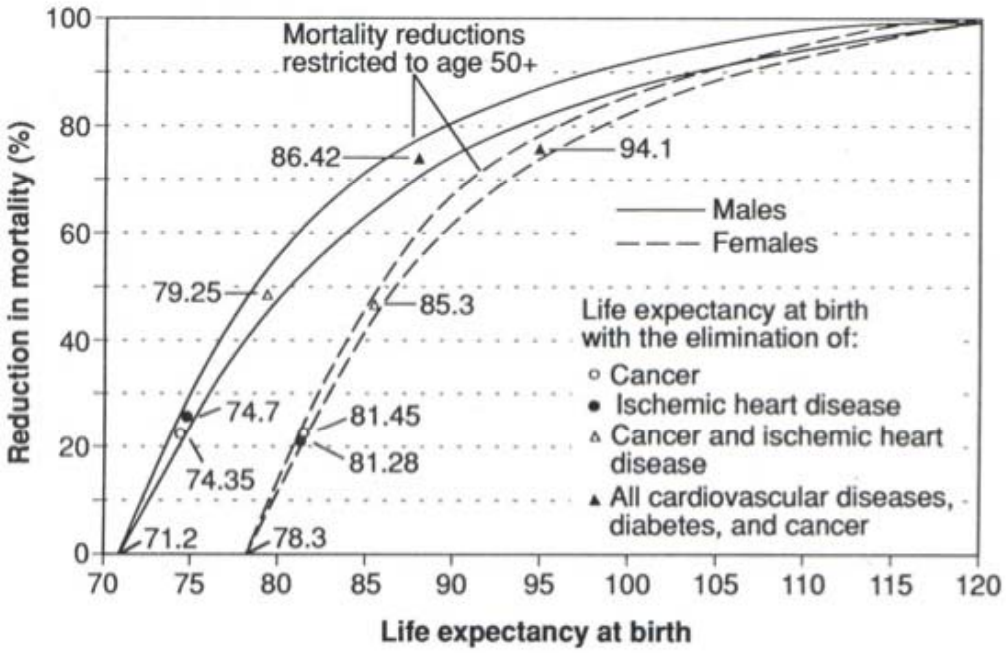
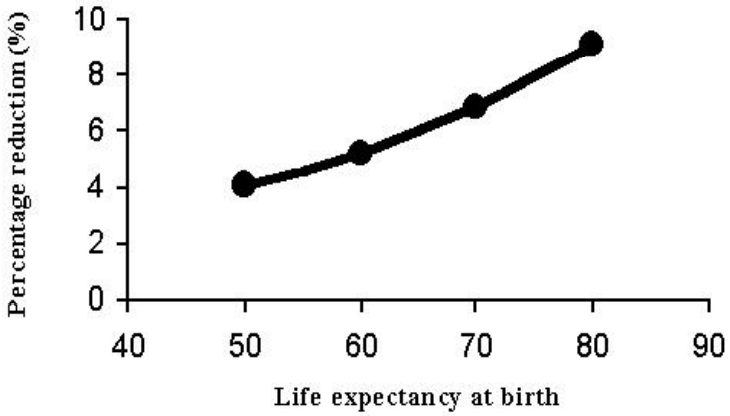
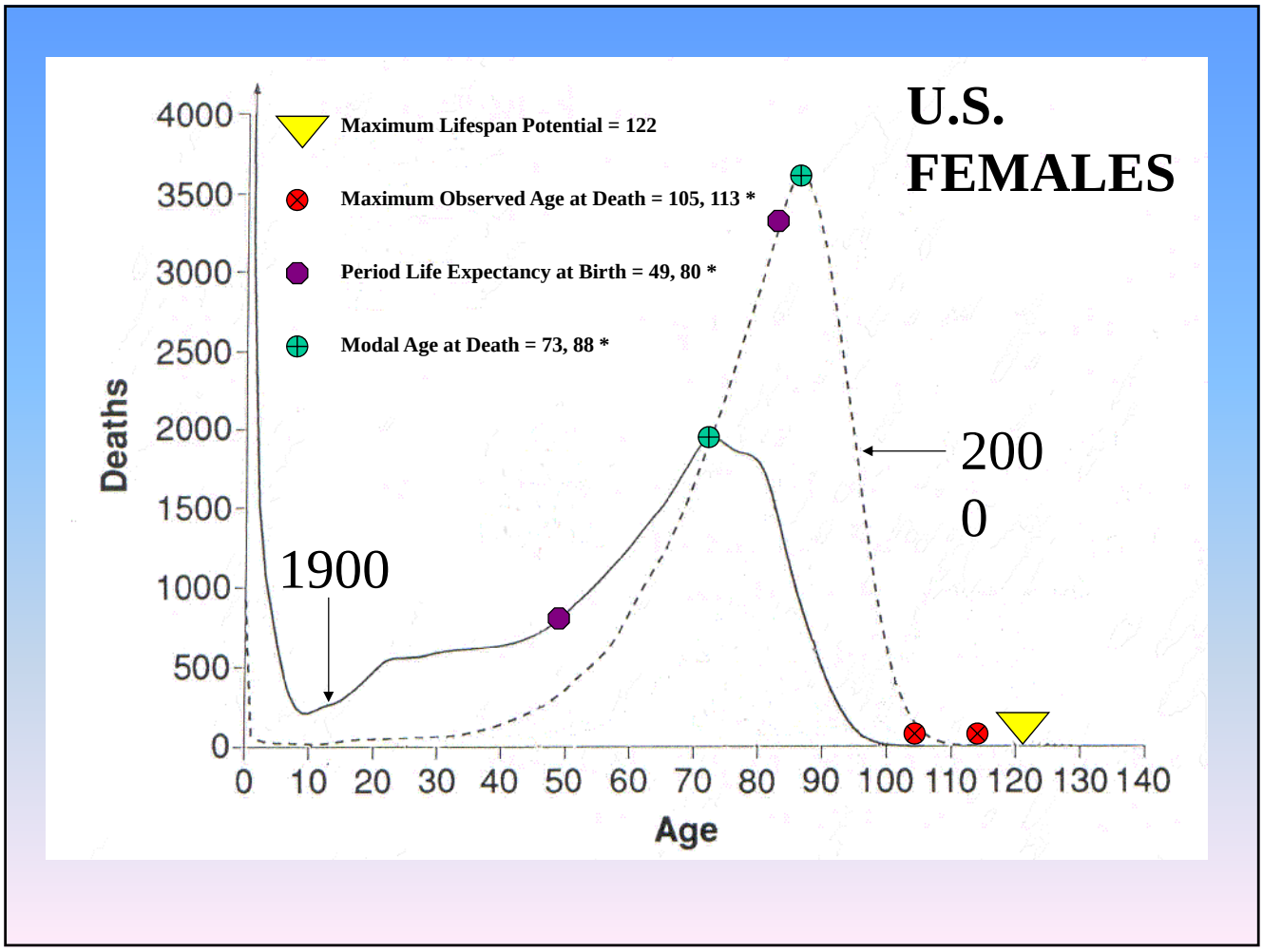


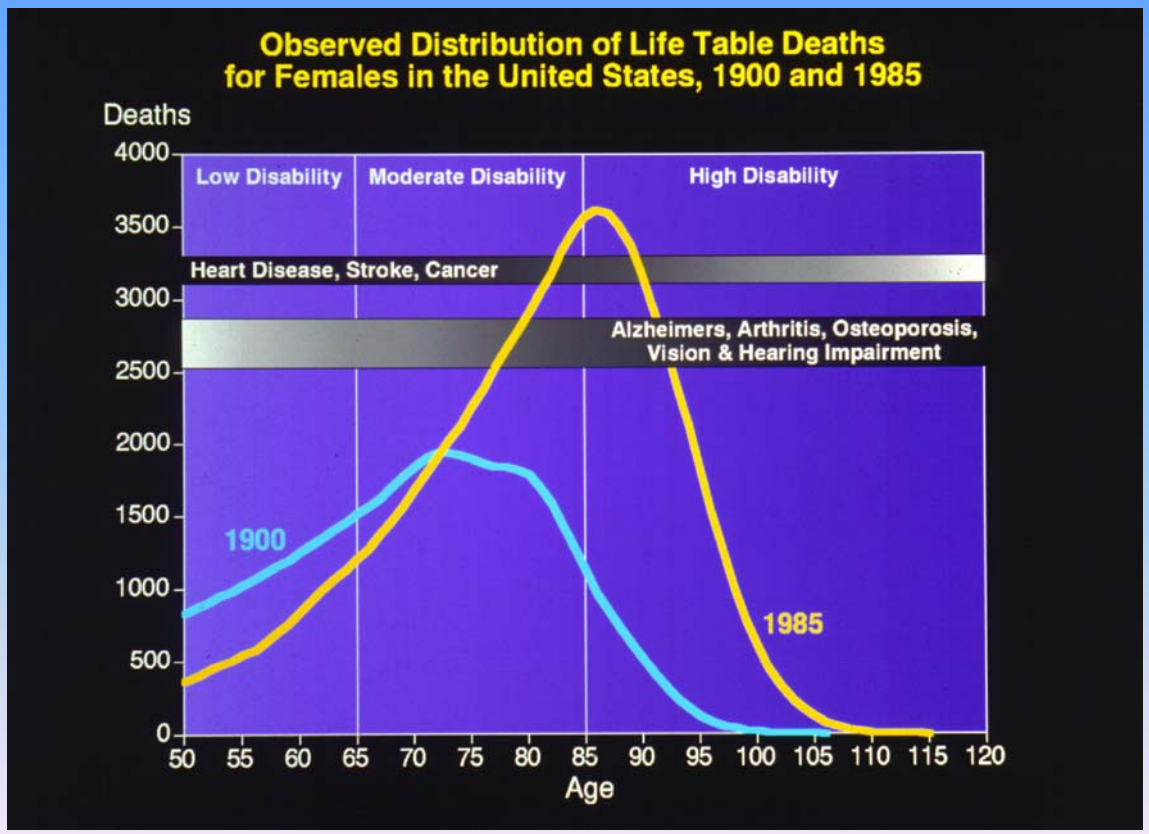
Fig. 2. Percentage of reduction in the conditional probability of death for the United States (from 1985 levels) required to produce a life expectancy at birth from 80 to 120 years.

Olshansky et al., 1990. *Science*.



Olshansky et al., 2001. *Science*.


U.S.
FEMALES
Maximum Lifespan Potential = 122
Maximum Observed Age at Death = 105, 113 *
Period Life Expectancy at Birth = 49, 80 *
Modal Age at Death = 73, 88 *
1900
2000
Deaths
Age
4000
3500
3000
2500
2000
1500
1000
500
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140

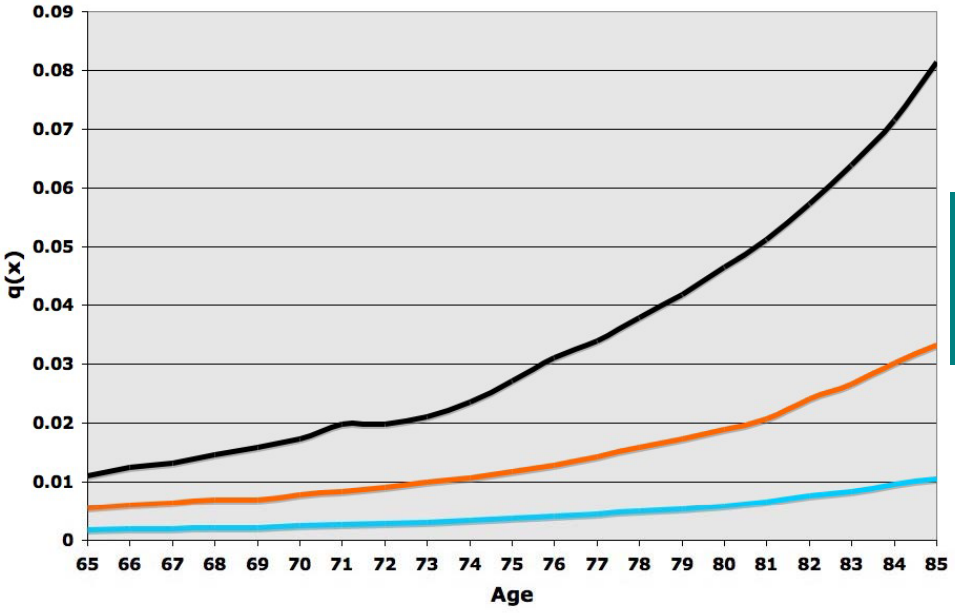

Observed Distribution of Life Table Deaths
for Females in the United States, 1900 and 1985
Deaths
4000
3500
3000
2500
2000
1500
1000
500
0
Low Disability
Moderate Disability
High Disability
Heart Disease, Stroke, Cancer
Alzheimers, Arthritis, Osteoporosis,
Vision & Hearing Impairment
1900
1985
50 55 60 65 70 75 80 85 90 95 100 105 110 115 120
Age

There is no demographic, actuarial, or biological justification for concluding that most (or even half of the population) can live to 100

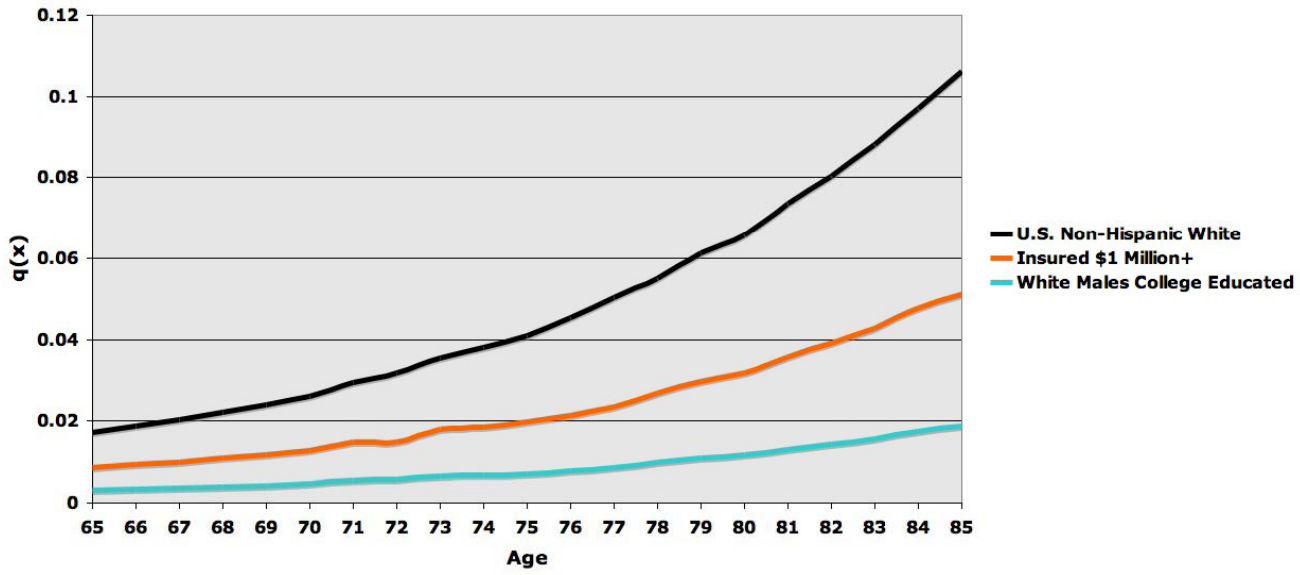
Education

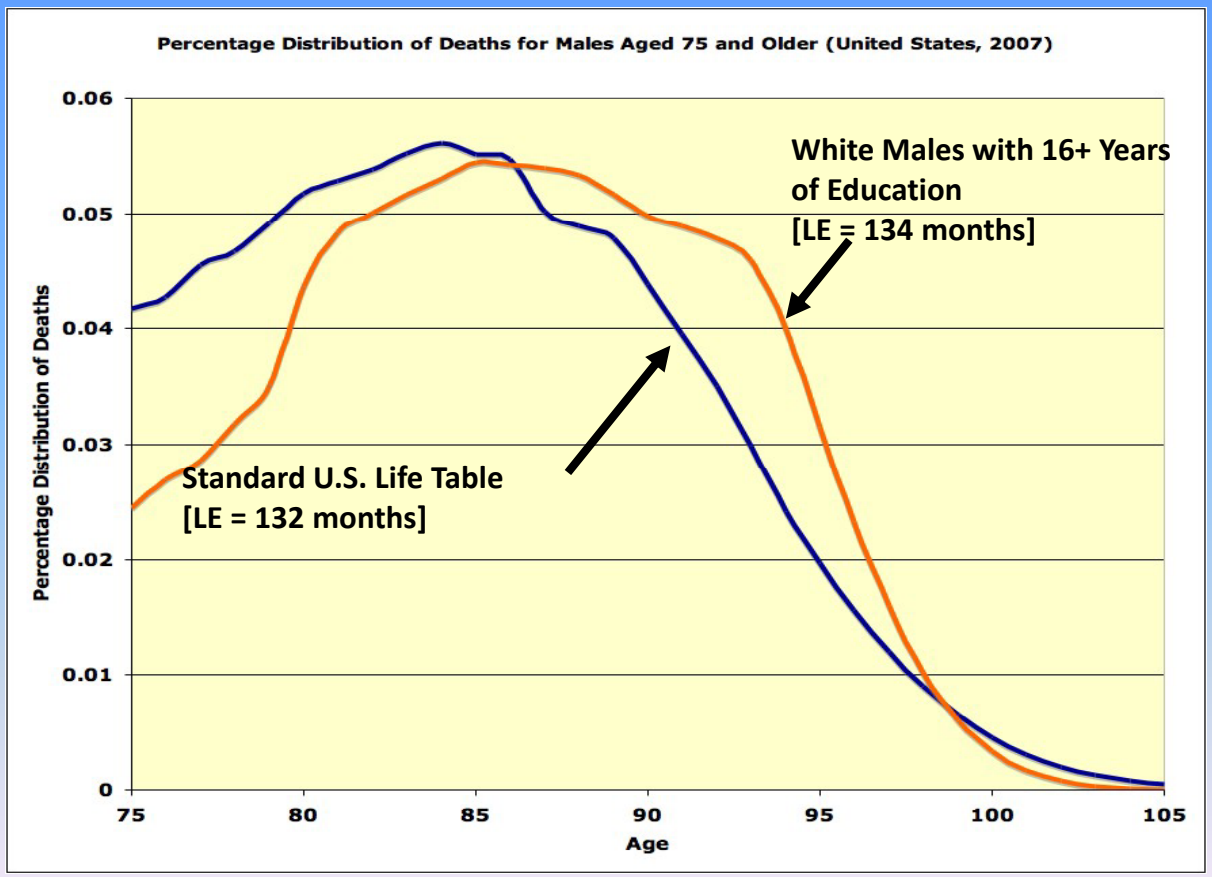
The Longevity Trump Card

Conditional Probability of Death [q(x)] for Females in the U.S. (U.S. Non-Hispanic White, Insured with $1 Million+ Policies, and Whites with College Education (2005)



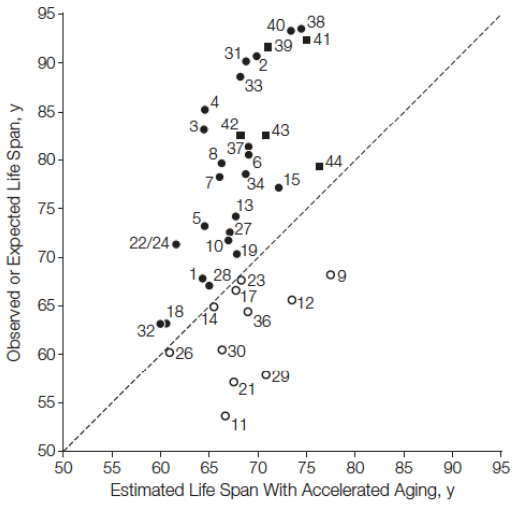
Conditional Probability of Death [q(x)] for Males in the U.S. (U.S. Non-Hispanic White, Insured with $1 Million+ Policies, and Whites with College Education (2005)





The average duration of life will be only 2 months greater, but the distribution of death by age will be dramatically different.

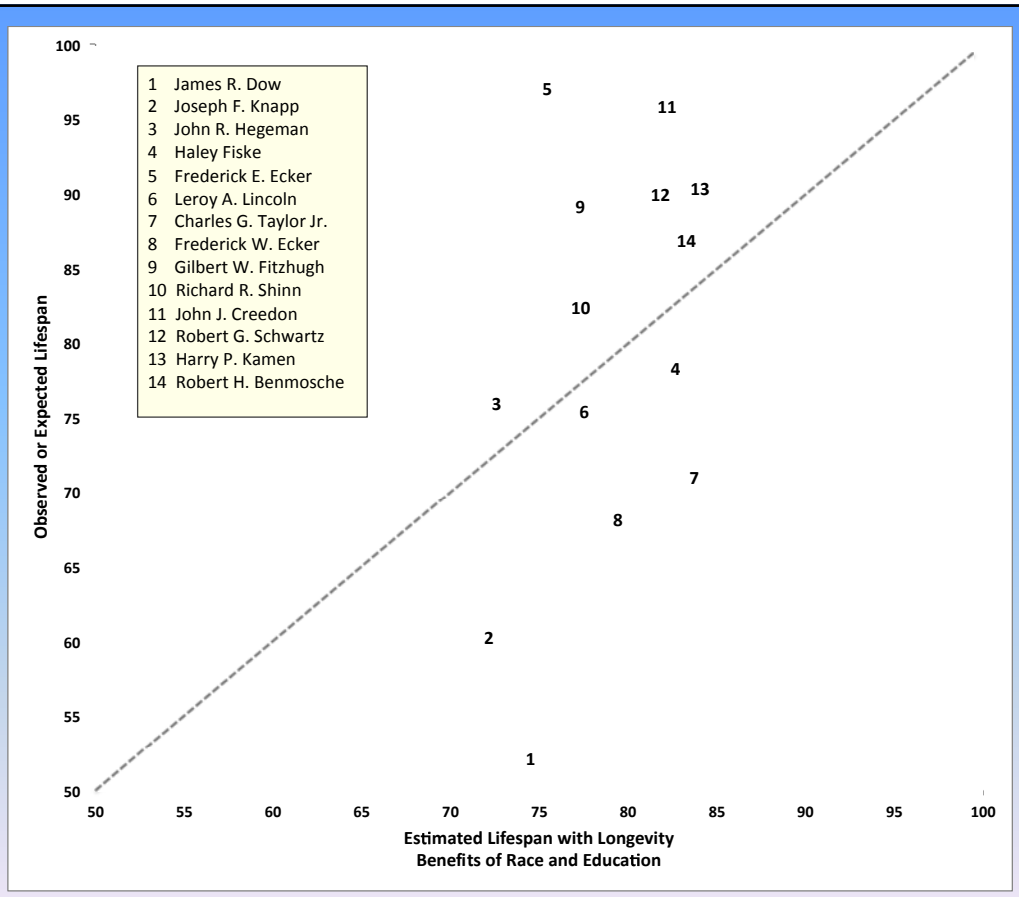
Figure. Estimated and Observed Life Span of US Presidents Who Died From Natural Causes



		Age at inauguration, y			Age at inauguration, y
1	George Washington	57.2	23	Benjamin Harrison	55.5
2	John Adams	61.3	25	William McKinley	54.1
3	Thomas Jefferson	57.9	26	Theodore Roosevelt	42.9
4	James Madison	58.0	27	William Howard Taft	51.5
5	James Monroe	58.9	28	Woodrow Wilson	56.2
6	John Quincy Adams	57.7	29	Warren G. Harding	55.3
7	Andrew Jackson	62.0	30	Calvin Coolidge	51.1
8	Martin Van Buren	54.2	31	Herbert Hoover	54.6
9	William Henry Harrison	68.1	32	Franklin D. Roosevelt	51.1
10	John Tyler	51.0	33	Harry S Truman	60.9
11	James K. Polk	49.3	34	Dwight D. Eisenhower	62.3
12	Zachary Taylor	64.3	35	John F. Kennedy	43.7
13	Millard Fillmore	50.5	36	Lyndon B. Johnson	55.2
14	Franklin Pierce	48.3	37	Richard Nixon	56.0
15	James Buchanan	65.9	38	Gerald Ford	61.1
16	Abraham Lincoln	52.1	**39**	**Jimmy Carter**	**52.3**
17	Andrew Johnson	56.3	40	Ronald Reagan	70.0
18	Ulysses S. Grant	46.9	**41**	**George H. W. Bush**	**64.6**
19	Rutherford B. Hayes	54.4	**42**	**Bill Clinton**	**46.4**
20	James A. Garfield	49.3	**43**	**George W. Bush**	**54.5**
21	Chester A. Arthur	52.0	**44**	**Barack Obama**	**47.5**
22/24	Grover Cleveland	48.0			

The diagonal line represents an exact match between estimated life span with accelerated aging and observed or expected life span. Presidents who appear above the line lived longer than their estimated life span while those who appear below the line died before their estimated life span. Expected life spans of living presidents are based on their current ages. Presidents who did not die of natural causes (Lincoln, Garfield, McKinley, and Kennedy, indicated by shading) and living presidents (Carter, G. H. Bush, Clinton, G. W. Bush, and Obama, indicated by bold) were included in estimates of mean age at inauguration and estimated mean life span at age of inauguration with accelerated aging. These presidents were excluded from analyses involving observed survival because they are either still alive or did not die from natural causes.

Source: Olshansky, S.J. 2011. Aging of US Presidents. *JAMA* 36(21): 2328-29



Source: Olshansky, SJ. 2012. Longevity of CEOs of MetLife.

A Possible Decline in Life Expectancy in the United States in the 21st Century?

S. Jay Olshansky, Ph.D.
University of Illinois at Chicago

Douglas J. Passaro, M.D.
University of Illinois at Chicago

Ronald C. Hershow, M.D.
University of Illinois at Chicago

Jennifer Layden, MPH
University of Illinois at Chicago

Bruce A. Carnes, Ph.D.
University of Oklahoma

Jacob Brody, M.D.
University of Illinois at Chicago

Leonard Hayflick, Ph.D.
University of California at San Francisco

Robert N. Butler, M.D.
International Longevity Center

David B. Allison, Ph.D.
University of Alabama at Birmingham

David S. Ludwig, M.D., Ph.D.
Children's Hospital, Boston

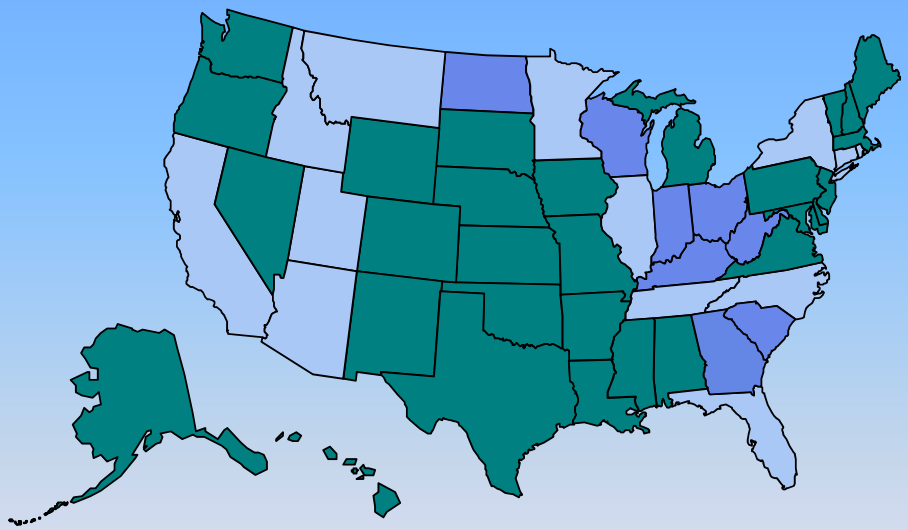
New England Journal of Medicine
2005 352:1103-1110.

Funding: NIH/NIA; NIDDK; IGPA

Obesity Trends* Among U.S. Adults

1985

(*BMI ≥30, or ~ 30 lbs overweight for 5’ 4” person)

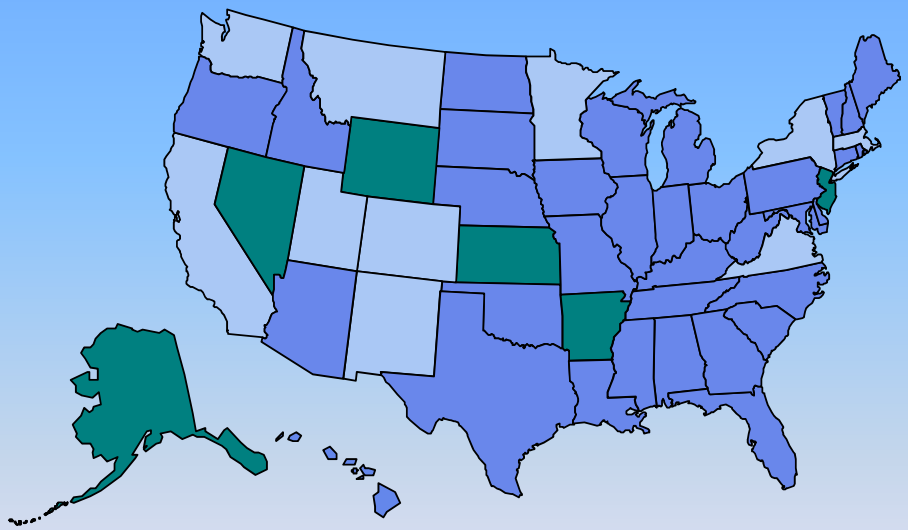


No Data | <10% | 10%–14% | 15%–19% | 20%–24% | ≥25%

Source: CDC

Obesity Trends Among U.S. Adults

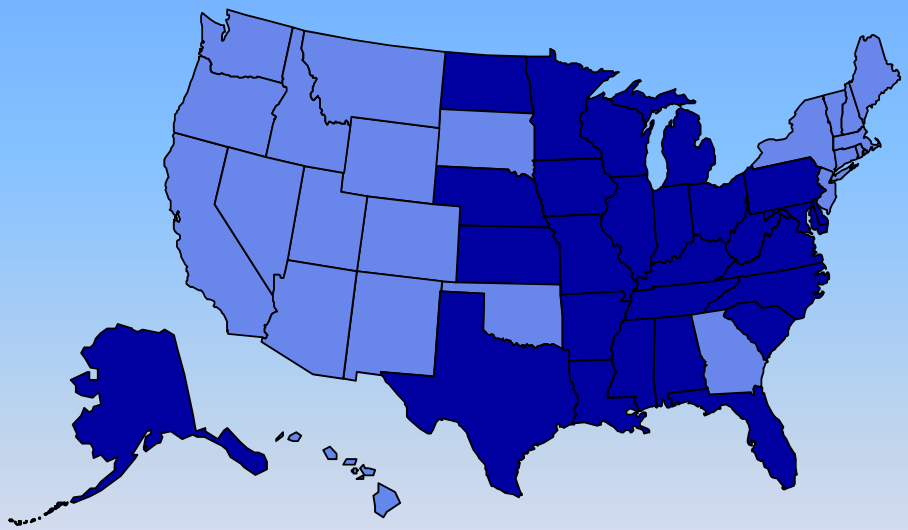
1990



No Data <10% 10%–14% 15%–19% 20%–24% ≥25%

Obesity Trends Among U.S. Adults

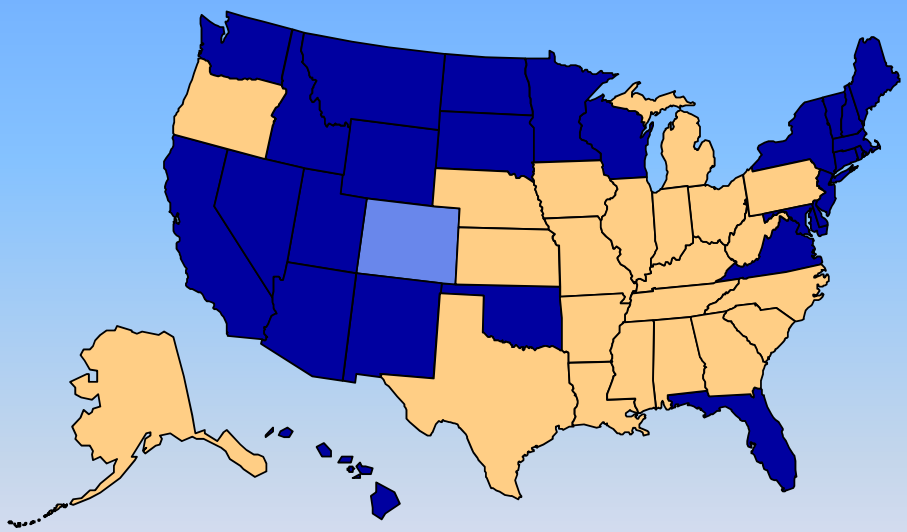
1995



No Data | <10% | 10%–14% | 15%–19% | 20%–24% | ≥25%

Obesity Trends Among U.S. Adults

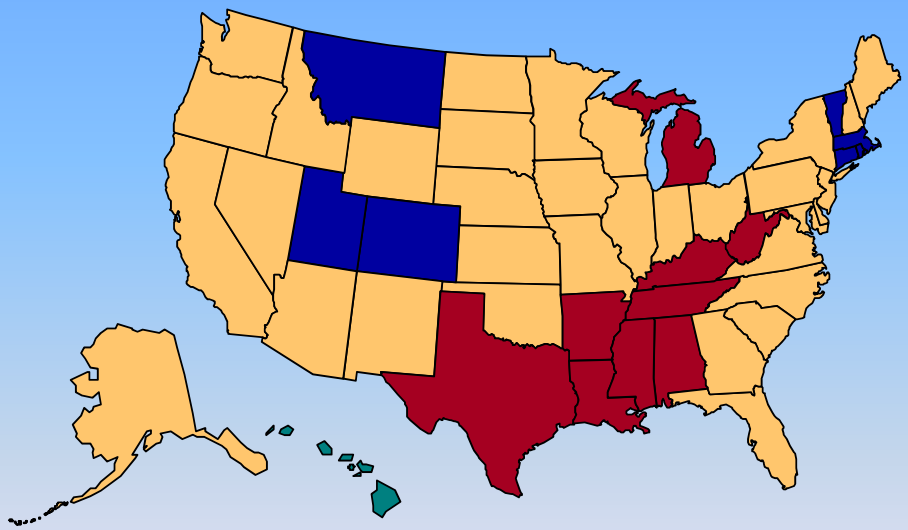
2000



No Data <10% 10%–14% 15%–19% 20%–24% ≥25%

Obesity Trends Among U.S. Adults

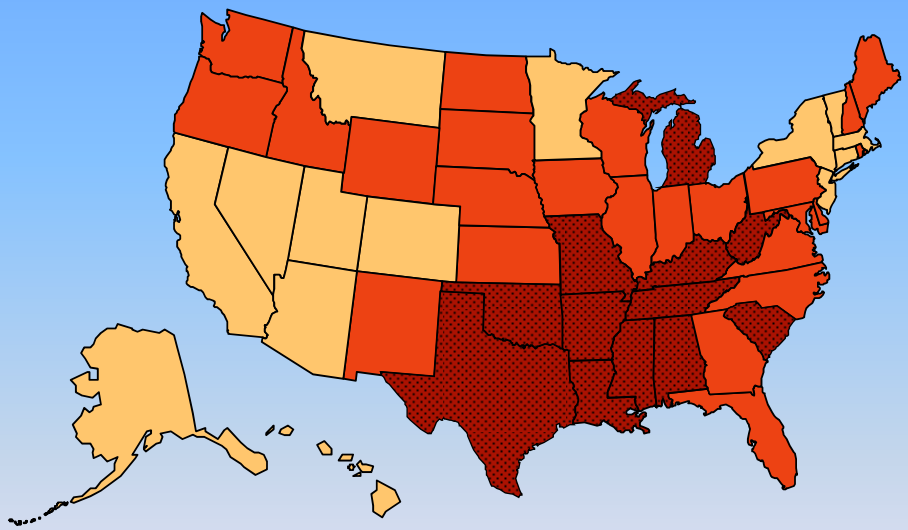
2004



No Data | <10% | 10%–14% | 15%–19% | 20%–24% | ≥25%

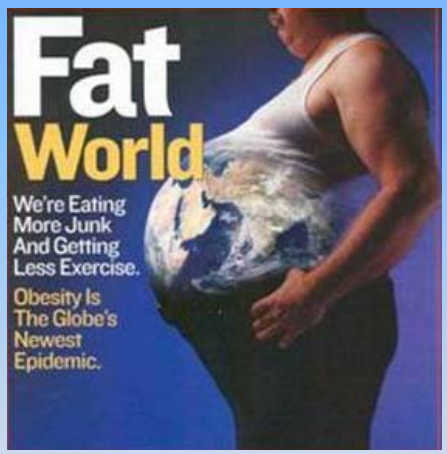
Obesity Trends Among U.S. Adults

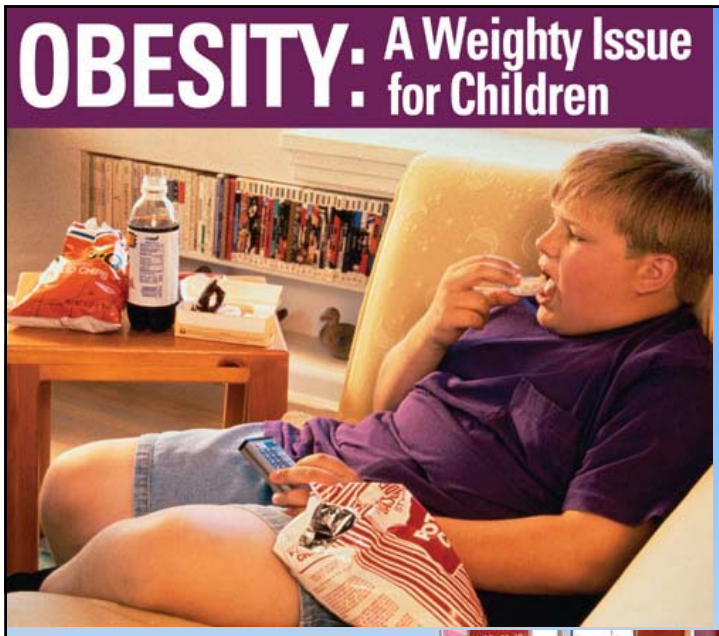
2010



No Data | <10% | 10%–14% | 15%–19% | 20%–24% | 25%–29% | ≥30%

Obesity is a global pandemic




OBESITY: A Weighty Issue for Children


LIES ABOUT SOCIAL SECURITY BY ALLAN SLOAN
WATER ON MARS
New Hints of Life
'ME, MYSELF & IRENE'
The Wild Men of Comedy
Fat for Life?
Six Million Kids Are Seriously Overweight. What Families Can Do.
By Geoffrey Cowley & Sharon Begley


www.StrangeCosmos.com

By Eric N. Reither, S. Jay Olshansky, and Yang Yang

New Forecasting Methodology Indicates More Disease And Earlier Mortality Ahead For Today's Younger Americans

DOI: 10.1377/hlthaff.2011.0092
HEALTH AFFAIRS 30,
NO. 8 (2011): –

ABSTRACT **Traditional methods of projecting population health statistics, such as estimating future death rates, can give inaccurate results and lead to inferior or even poor policy decisions. A new "three-dimensional" method of forecasting vital health statistics is more accurate because it takes into account the delayed effects of the health risks being accumulated by today's younger generations. Applying this forecasting technique to the US obesity epidemic suggests that future death rates and health care expenditures could be far worse than currently anticipated. We suggest that public policy makers adopt this more robust forecasting tool and redouble efforts to develop and implement effective obesity-related prevention programs and interventions.**

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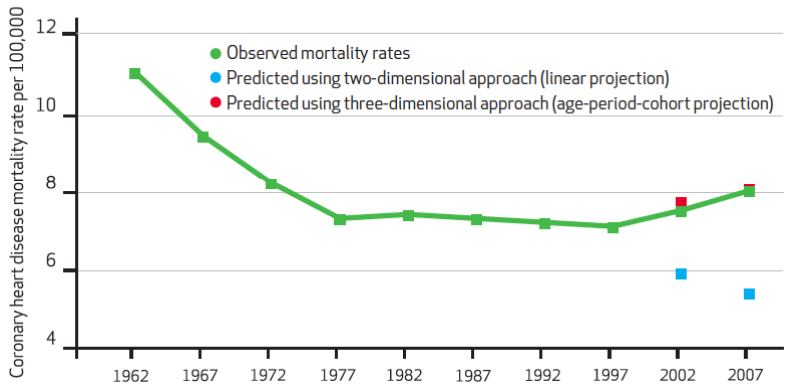
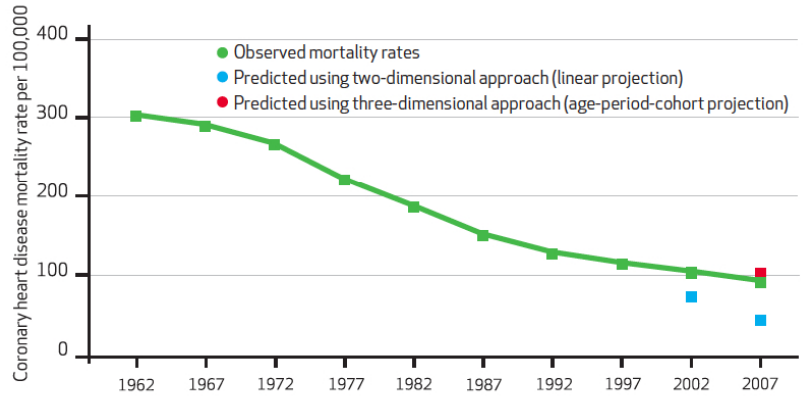
S. Jay Olshansky is a professor in the School of Public Health at the University of Illinois, in Chicago.

Yang Yang is an associate professor in the Department of Sociology and the Lineberger Comprehensive Cancer Center at the University of North Carolina, , in Chapel Hill.

Two- Versus Three-Dimensional Projections For Coronary Heart Disease Mortality Among US Males Ages 25–29, 1962–2007



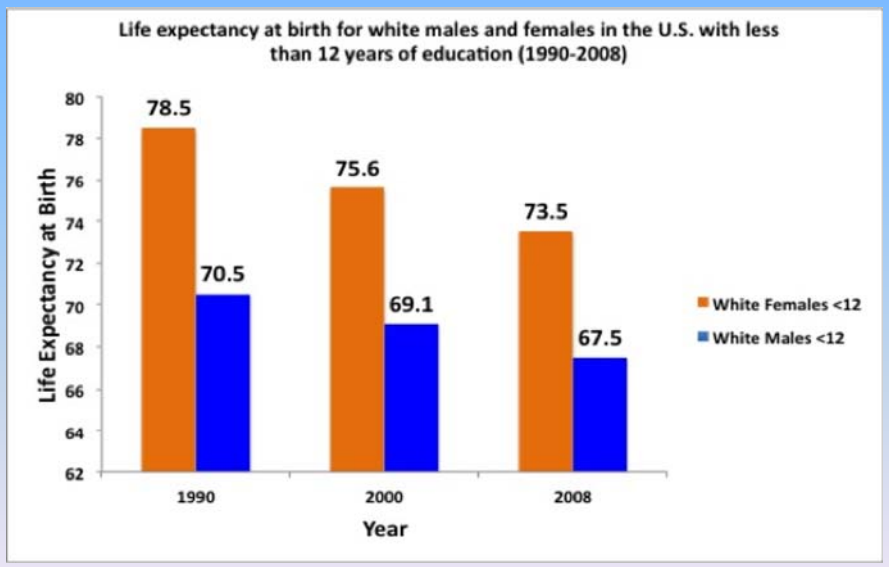
Two- Versus Three-Dimensional Projections For Coronary Heart Disease Mortality Among US Males Ages 45–49, 1962–2007



DISPARITIES

By S. Jay Olshansky, Toni Antonucci, Lisa Berkman, Robert H. Binstock, Axel Boersch-Supan, John T. Cacioppo, Bruce A. Carnes, Laura L. Carstensen, Linda P. Fried, Dana P. Goldman, James Jackson, Martin Kohli, John Rother, Yuhui Zheng, and John Rowe

Differences In Life Expectancy Due To Race And Educational Differences Are Widening, And Many May Not Catch Up



Olshansky SJ et al. 2012. *Health Affairs*.

Resetting Social Security: What is Fair?

Olshansky et al., 2014
MacArthur Research Network on an Aging Society [in preparation]

"When it is realized that too large a proportion of the population would probably be left idle with a retirement age of 65, the general feeling will undoubtedly be that a constant retirement age should be banished, or that it should be left as a balancing item" (p.8)

Williamson and Myers, 1937

"we can therefore expect a considerable shift in the retirement age. Advancing it five years would make a lot of difference. There may be times when it could be reduced" (p.8)

Williamson and Myers, 1937

"Similarly, in the future when there are a great many persons over 65, most of the able-bodied individuals will and should continue working to age 70 or 75 if their services seem needed" (p.18)

Myers, 1938

Source: Olshansky et al., 2014. Resetting Social Security: What is Fair? MacArthur Foundation Research Network on an Aging Society. (in preparation)

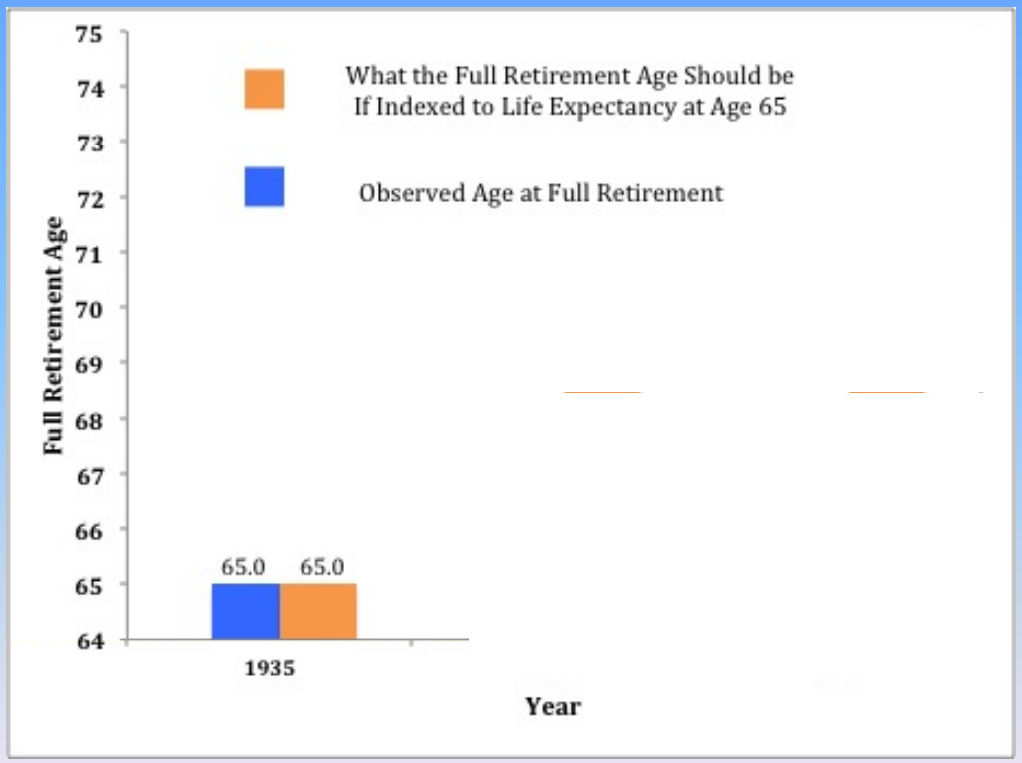
Table 1. Life expectancy at birth [$e_{(0)}$], age 65 [$e_{(65)}$], and conditional survival from age 25 to age 65 [$S_{(25-65)}$], by sex (U.S., 1935, 1983, 2010)

	$e_{(0)}$			$e_{(65)}$			$S_{(25-65)}$		
	M	F	T	M	F	T	M	F	T
1935	59.0	63.0	60.9	12.0	13.4	12.7	59.8	67.7	63.5
1983	71.0	78.1	74.7	14.4	18.6	16.7	74.8	85.5	80.9
2010	76.4	81.2	78.9	17.9	20.5	19.4	82.3	89.1	85.7

Source: HMD, 2013. http://www.mortality.org/ (data accesses 4/19/13)

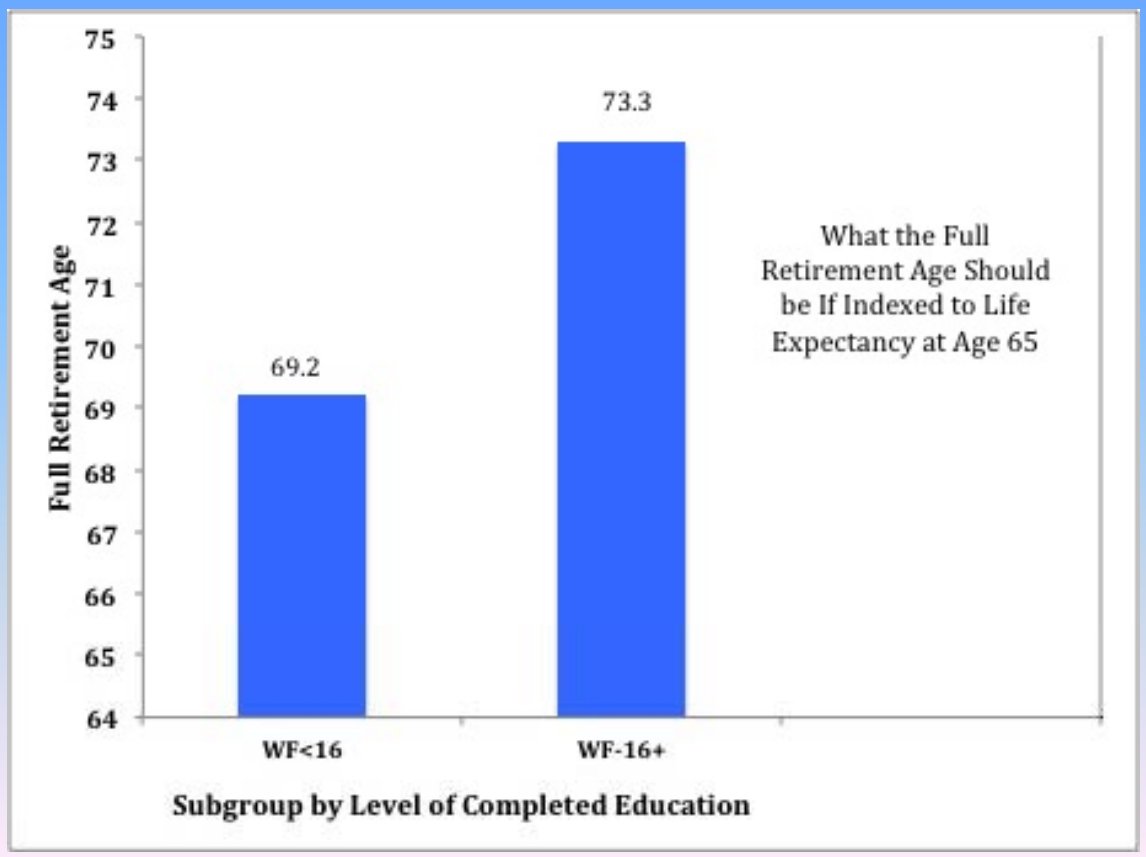
Olshansky et al., 2014. Resetting Social Security: What is Fair? MacArthur Foundation Research Network on an Aging Society. (in preparation)

Figure 1. Observed and hypothetical full retirement ages indexed to life expectancy at age 65 (U.S., 1935, 1983, 2010)



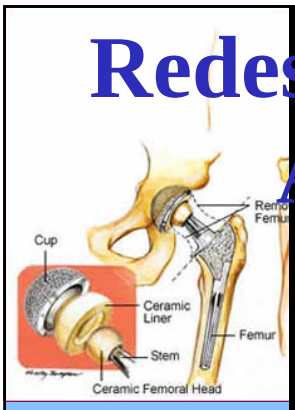
Olshansky et al., 2014. Resetting Social Security: What's Fair? MacArthur Foundation Research Network on an Aging Society. (in preparation)

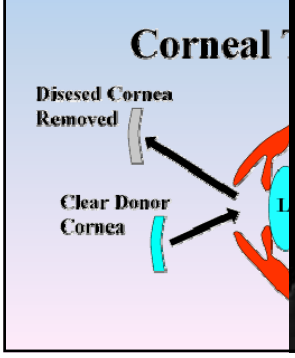
Figure 2. Hypothetical full retirement ages indexed to life expectancy at age 65 for White Females by Level of Completed Education, 2010



The Next Health and Longevity Revolution is Forthcoming

Redesign / Replace Body Parts Already in Existence





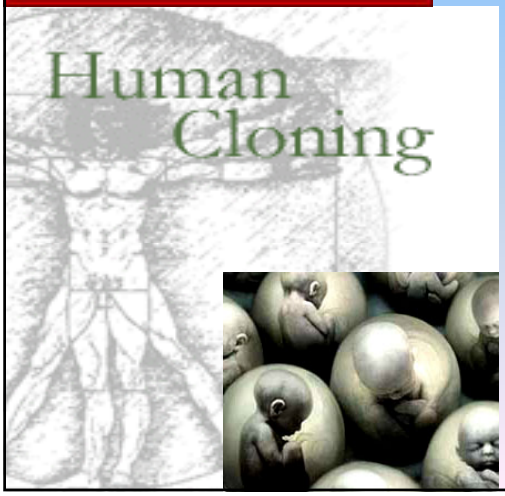
BRAIN TRANSPLANT

ONE ORGAN WE WILL HAVE A DIFFICULT TIME "FIXING"

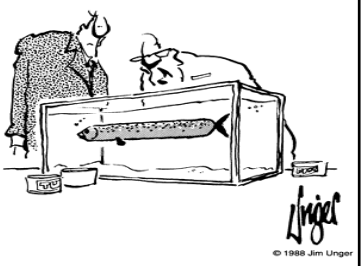
Modifying the Underlying Biology of Humans

Sex and trait selection

Germ line modification



Therapeutic cloning



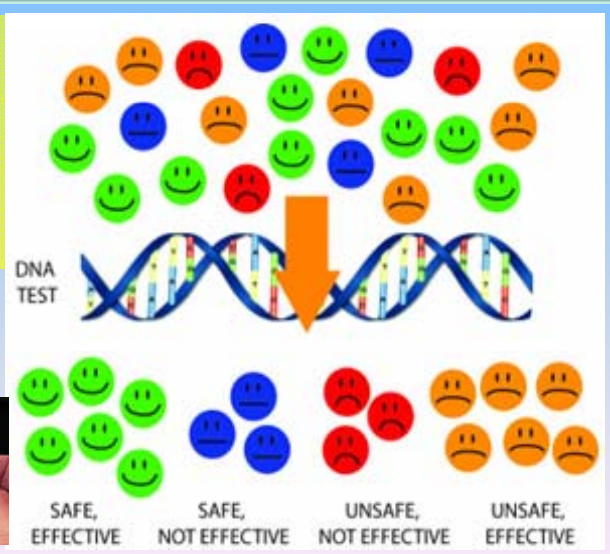
Genetic engineering to treat or eliminate diseases

Exciting Advances in Biomedical Technology

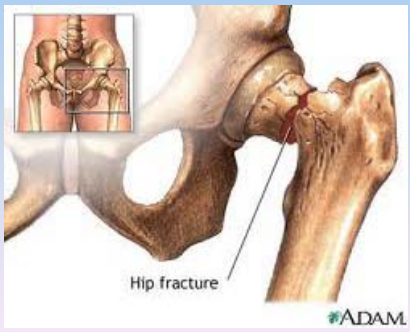
Personalized Medicine

Person 1: 5.5 mg. of medicine X

Person 2: 6.2 mg. of medicine X



Reducing the risk of fatal diseases by treating them as if they are independent of each other may extend the period of old age.




TheScientist
MAGAZINE OF THE LIFE SCIENCES
fight
AGING
JAY OLSHANSKY & COLLEAGUES HAVE A PLAN
Daniel Perry
Richard A. Miller
Robert N. Butler
THE TOP 50 PLACES TO POSTDOC
A TOWN BOUNCES BACK AFTER BIG PHARMA LEAVES
MARC VIDAL CALLS FOR A $100 MILLION INTERACTOME PROJECT
A JOURNAL EDITOR SHOWS HOW HE AVOIDS IMAGE FRAUD
PLUS:
WHAT MEDICAL TOURISM MEANS FOR BIOTECHS


In pursuit of the
LONGEVITY DIVIDEND
What should we be doing to prepare for the unprecedented aging of humanity?
S. JAY OLSHANSKY, DANIEL PERRY, RICHARD A. MILLER, ROBERT N. BUTLER
Imagine an intervention, such as a pill, that could significantly reduce your risk of cancer. Imagine an intervention that could reduce your risk of stroke, or dementia, or arthritis. Now, imagine an intervention that does all these things, and at the same time reduces your risk of everything else undesirable about growing older: including heart disease, diabetes, Alzheimer and Parkinson disease, hip fractures, osteoporosis, sensory impairments, and sexual dysfunction. Such a pill may sound like fantasy, but aging interventions already do this in animal models. And many scientists believe that such an intervention is a realistically achievable goal for people. People already place a high value on both quality and length of life, which is why children are immunized against infectious diseases. In the same spirit, we suggest that a concerted effort to slow aging begin immediately – because it will save and extend lives, improve health, and create wealth.

July, 2008

Robert N Butler president, International Longevity Center, New York, USA

Richard A Miller professor, University of Michigan, Ann Arbor, MI, USA

Daniel Perry executive director, Alliance for Aging Research, Washington, DC, USA

Bruce A Carnes professor, University of Oklahoma, Oklahoma City, OK, USA

T Franklin Williams professor emeritus, University of Rochester School of Medicine and Dentistry, Rochester, NY, USA

Christine Cassel president, American Board of Internal Medicine, Philadelphia, PA, USA

Jacob Brody professor, University of Illinois at Chicago, 1603 West Taylor Street, Chicago, IL 60612, USA

Marie A Bernard professor, University of Oklahoma, Oklahoma City, OK, USA

Linda Partridge director , Institute of Healthy Ageing, University College London, London

Thomas Kirkwood director, Institute for Ageing and Health, Newcastle University, Newcastle

George M Martin scientific director, American Federation for Aging Research, Seattle, WA, USA

S Jay Olshansky professor, University of Illinois at Chicago, 1603 West Taylor Street, Chicago, IL 60612, USA

New model of health promotion and disease prevention for the 21st century

Our susceptibility to disease increases as we grow older. **Robert Butler and colleagues** argue that interventions to slow down ageing could therefore have much greater benefit than those targeted at individual disease

LDI Leading Organizations / Research Advisory Committee

afar american federation for aging research

THE GERONTOLOGICAL SOCIETY OF AMERICA

Alliance FOR AGING RESEARCH

GLENN FOUNDATION FOR MEDICAL RESEARCH

NATIONAL INSTITUTE ON AGING
National Institutes of Health

AGE American Aging Association

Affiliated Research Institutions and Universities

The BUCK INSTITUTE FOR RESEARCH ON AGING

MAYO CLINIC

UIC University of Illinois at Chicago

EINSTEIN Albert Einstein College of Medicine

UNIVERSITY OF WASHINGTON 1861

USC UNIVERSITY OF SOUTHERN CALIFORNIA 1880

VE RI TAS HARVARD

The Private Sector

The New York Times

BITS

Tech Titans Form Biotechnology Company

BY CLAIRE CAIN MILLER AND ANDREW POLLACK
SEPTEMBER 19, 2013

Silicon Valley has an obsession with immortality, and not just as science fiction. Many people here say they believe that the day when technology makes it possible to live forever is just around the corner.

On Wednesday, some of the tech world's most formidable players announced an effort to get closer to that point, with a new biotechnology company to fight the aging process and the diseases that accompany it.

The company, Calico, was conceived and backed by Google, whose co-founder and chief executive, Larry Page, portrayed it as one of the company's long-shot projects, like self-driving cars. Arthur D. Levinson, 63, the former chief executive of Genentech and the chairman of Apple, agreed to be the chief executive and is also an investor.

Larry Page, Google's co-founder and chief executive, and Arthur D. Levinson, a former chief executive of Genentech and the chairman of Apple. (Left: John G. Mabanglo/European Pressphoto Agency; Right: Roche)

panies since stepping down as chief executive of Genentech in 2009, after the company's acquisition by Roche.

Dr. Levinson said that at first Calico would be "more of an institute certainly than a pharmaceutical company," focusing on basic research aimed at picking apart the biological mechanisms behind aging.

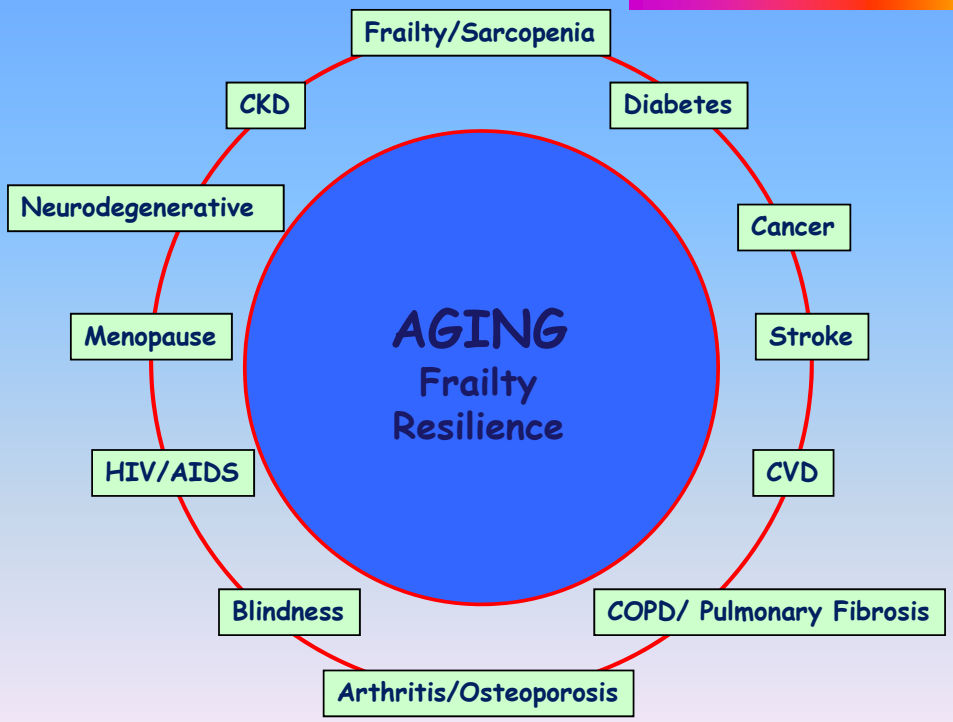
is Calico's only employee for now, would not say when, or even if, Calico hoped to develop a drug to fight aging.

An anti-aging drug has been a long-sought goal, both by some consumers and by companies, as well as by various hucksters. Rather than treat each particular disease, retarding aging could potentially pre-

Source: ***New York Times*****, September 16, 2013.**

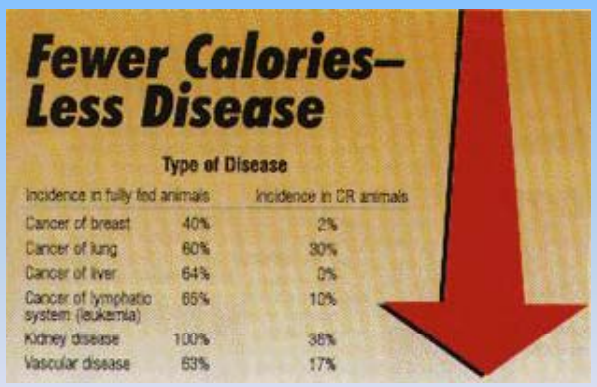

HUMAN LONGEVITY, INC.
ABOUT
mission & people
SCIENCE & TECHNOLOGY
advanced products
PARTNERS
strategic collaborations
MEDIA
resources & news
CAREERS
we're hiring
Aging is the single biggest risk factor for virtually every significant human disease…
…our goal is to extend and enhance the healthy, high-performance lifespan and change the face of aging. For the first time, the power of human genomics, informatics, next generation DNA sequencing technologies, and stem cell advances are being harnessed in one company, Human Longevity Inc., with the leading pioneers in these fields. Our goal is to solve the diseases of aging by changing the way medicine is practiced.
It's not just a long life we're striving for, but one which is worth living.
Human Longevity Inc. (HLI) Launched to Promote Healthy Aging Using Advances in Genomics and Stem Cell Therapies
@JCVenter on CBS Morning Show http://t.co/RPaInhQpGm #genomics
[2 days ago]

AGING BIOLOGY IS AT THE CORE OF CHRONIC DISEASES



Do We Need to Know in Advance Which Scientific Pathways to the Longevity Dividend Will Work?

Genetics of long-lived people



Caloric restriction

Compounds with properties that appear to slow aging

LDI Primary Goals

1. **Accelerate research on aging**

1. **Accelerate translation of research into therapeutic interventions**

- **Extend healthy life**
- **Reduce health care costs**
- **Reduce the burden of disease**
- **Reduce gap between life expectancy and healthy life expectancy**


REMEMBER THE TWENTY EXTRA YEARS YOU ADDED TO YOUR LIFE THROUGH CLEAN, HEALTHY LIVING? - WELL, THESE ARE THEM.

300 Word Video