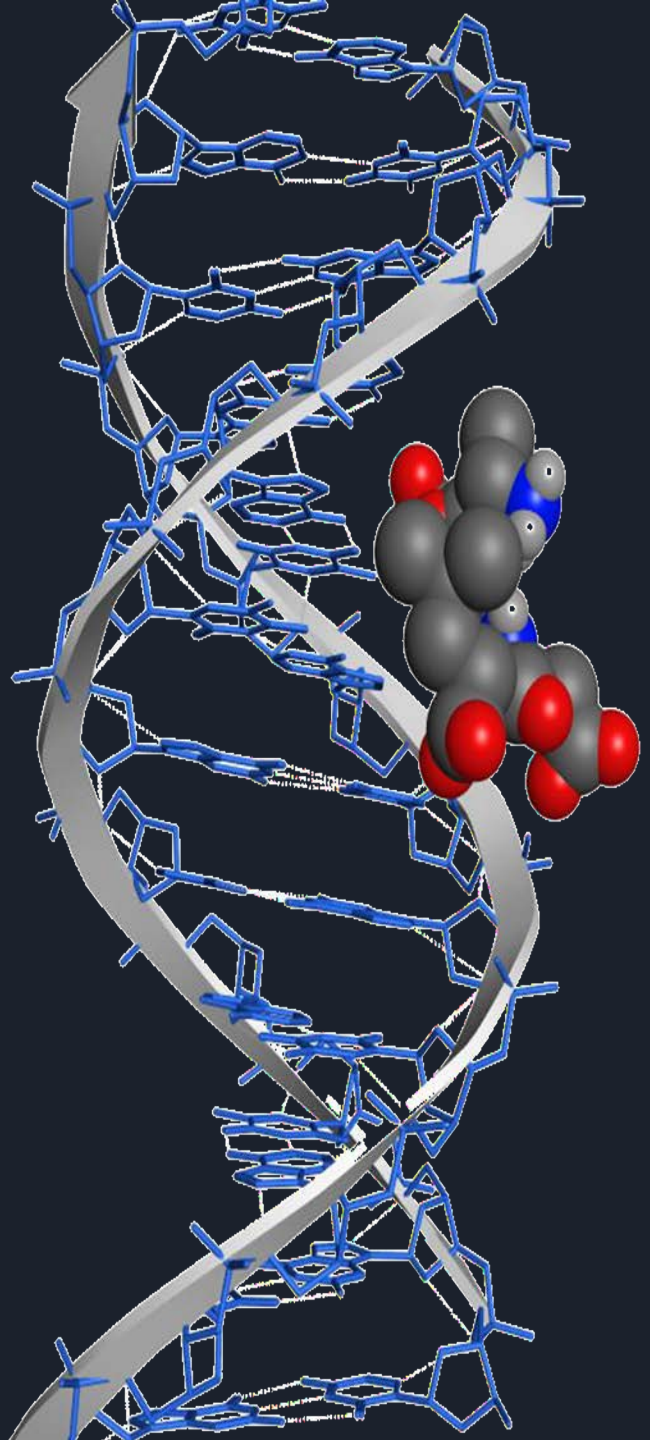


Saint Petersburg Institute of Bioregulation and Gerontology
D'Annunzio University of Chieti–Pescara, Italy

THE MECHANISMS OF PEPTIDE REGULATION OF AGEING

Prof. VLADIMIR KHAVINSON, M.D., Ph.D.

Director of the St. Petersburg Institute of Bioregulation and Gerontology

President of the International Association of Gerontology and Geriatrics - European Region (2011-2015)

Honorary member of the Academic Council of D'Annunzio University of Chieti–Pescara, Italy

http://www.khavinson.info pa@khavinson.info

The Value of Time

> “It’s really clear that the most precious resource we all have is time.”
>
> - Steve Jobs

> “Time is really the only capital that any human being has, and the only thing he can’t afford to lose.”
>
> - Thomas A. Edison

"IN GOD, I TRUST, EVERYONE ELSE HAS TO PRODUCE THE DATA."

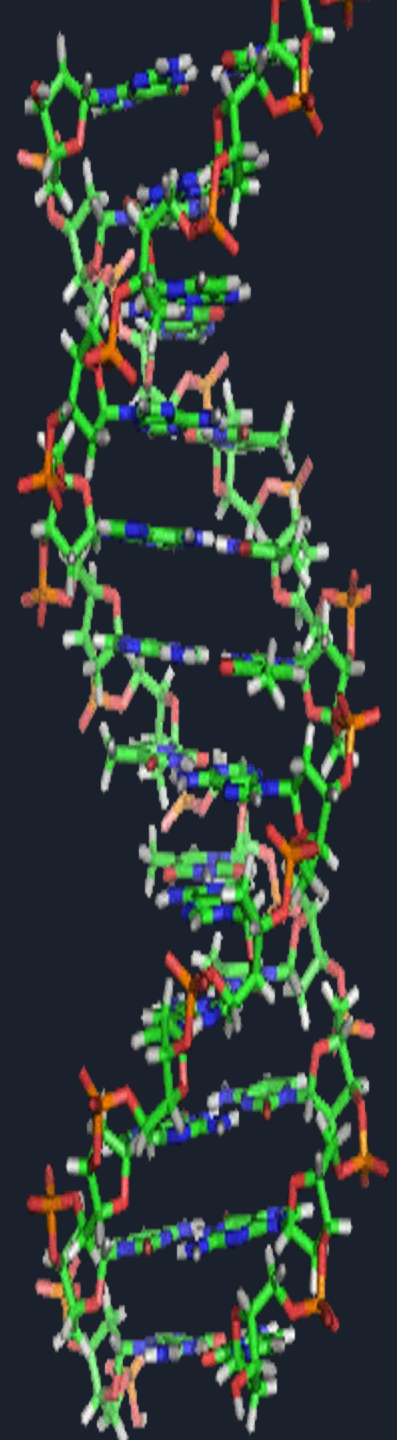
BILL LAWRENCE JD, MS, PH.D

PEPTIDE BIOREGULATOR LONGEVITY PROTOCOL 2017-2024

CELLULAR REPROGRAMMING AND EPIGENETIC REJUVENATION

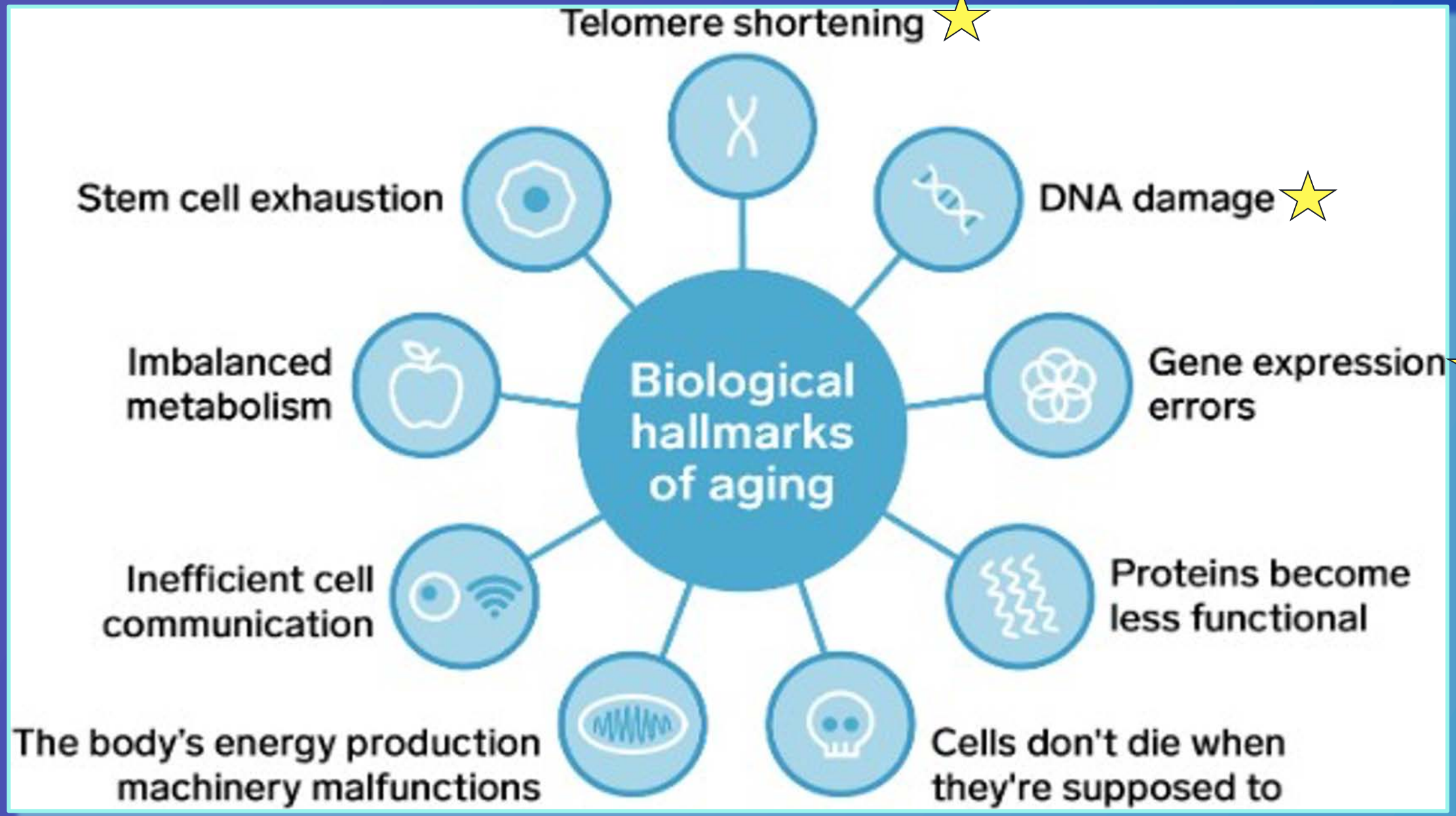
- SCIENTIFICALLY PROVEN
- PUBLISHED CLINICAL HUMAN STUDIES
- AMERICAN LABORATORY DATA CONFIRMS:
  - ORGAN REGENERATION
  - MORTALITY REDUCTION
  - BIOLOGICAL AGE REVERSAL

VLADIMIR KHAVINSON MD, PhD.
BILL LAWRENCE JD, MS, PhD.



Biological Theories of Aging

Cellular Clock Theory	Human cells can divide a maximum of 75-80 times
Free-Radical Theory	Aging occurs due to over-production of unstable oxygen molecules (free radicals)
Mitochondrial Theory	Aging is caused by a decline in mitochondrial function, which leads to increased oxidative damage
Hormonal Stress Theory	Aging in the hormonal system lowers resistance to stress, which increases chance for onset of disease


Telomere shortening
Stem cell exhaustion
DNA damage
Imbalanced metabolism
Biological hallmarks of aging
Gene expression errors
Inefficient cell communication
Proteins become less functional
The body's energy production machinery malfunctions
Cells don't die when they're supposed to

University of Cologne

Published October 8, 2009
N Engl J Med 2009;361:1475-1485
DOI: 10.1056/NEJMra0804615
VOL. 361 NO. 15

"…aging hallmarks are secondary consequences."

"Accumulating DNA damage contributes to systemic aging."

"DNA-damage-driven, gene-length-dependent transcription stress explains most aging hallmarks"

"This includes frailty, neurodegeneration (resembling Alzheimer's disease), hepato-, nephron-, and hematological aging, cardiovascular disease, osteoporosis, etc. —"

Jan Hoeijmakers
Principal Investigator
Global Faculty University of Cologne
Professor of Molecular Genetics at Erasmus University Rotterdam

Innov Aging

2018 Nov 11;2(Suppl 1):592.

PMCID: PMC6228501

DNA DAMAGE: THE MOST STALWART PILLAR OF AGING

L J Niedernhofer [1]

Abstract

Aging has been attributed to stochastic damage to cellular components accumulating over time. By genetically depleting DNA repair mechanisms in mice, we can evaluate the contribution of physiological types of levels of spontaneous DNA damage to the aging process. We found that in mice and people, **reduced DNA repair accelerates the aging process by approximately sixfold**, affecting virtually every organ system. Tissue-specific depletion of DNA repair is sufficient to drive premature onset of age-related disease, including congestive heart failure, adult-onset diabetes, and cognitive impairment.

Ageing Res Rev. 2021 Jul; 68: 101316.

PMID: 33711511

From DNA damage to mutations: all roads lead to aging

Jan Vijg

Abstract

Damage to the repository of genetic information in cells has plagued life since its very beginning 3–4 billion years ago. To cope with this high frequency of damage to the increasingly long DNA molecules that came to encode the growing complexity of cellular functions in cells, DNA repair evolved as one of the earliest genetic traits. Then as now, errors during the repair of DNA damage generated mutations, which provide the substrate for evolution by natural selection. With the emergence of multicellular organisms also the soma became a target of DNA damage and mutations.

Based on an abundance of evidence, DNA damage is now considered as the single most important driver of the degenerative processes that collectively cause aging. Here I will first briefly review the evidence for DNA damage as a cause of aging since the beginning of life.

Nature

volume 592, pages 695–703 (2021)

The central role of DNA damage in the ageing process

Björn Schumacher, Joris Pothof, Jan Vijg & Jan H. J. Hoeijmakers

Abstract

Ageing is a complex, multifaceted process leading to widespread functional decline that affects every organ and tissue, but it remains unknown whether ageing has a unifying causal mechanism or is grounded in multiple sources. Here we synthesize accumulating evidence that DNA damage affects most, if not all, aspects of the ageing phenotype, making it a potentially unifying cause of ageing. Targeting DNA damage and its mechanistic links with the ageing phenotype will provide a logical rationale for developing unified interventions to counteract age-related dysfunction and disease.

Am J Hum Genet. 2019 Aug 1; 105(2): 237–257.

DNA Damage and Associated DNA Repair Defects in Disease and Premature Aging

Vinod Tiwari[1,*] and David M. Wilson, III[1,2,**]

DNA repair is essential for cell vitality, cell survival, and cancer prevention, yet cells' ability to patch up damaged DNA declines with age for reasons not fully understood.

Rejuvenation Res

2009 Jun;12(3):199-208.

Nuclear DNA damage as a direct cause of aging

Benjamin P Best [1]

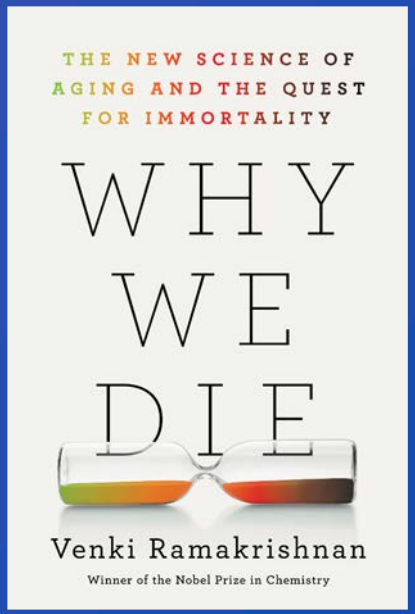
Abstract

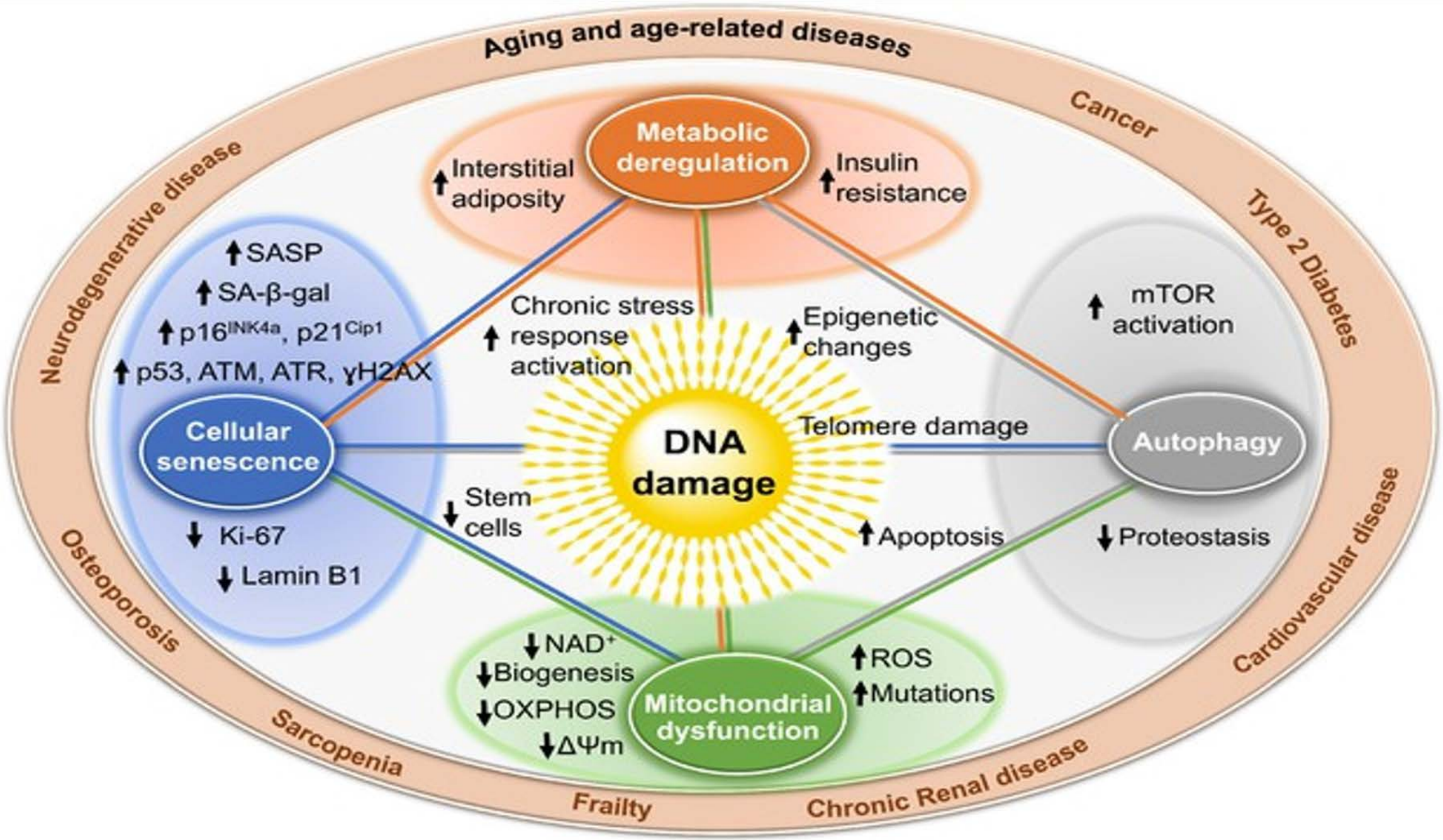
This paper presents evidence that damage to nuclear DNA (nDNA) is a direct cause of aging in addition to the effects of nDNA damage on cancer, apoptosis, and cellular senescence. Many studies show significant nDNA damage with age, associated with declining nDNA repair, and this evidence for the decline of nDNA repair with age is also reviewed. Mammalian lifespans correlate with the effectiveness of nDNA repair. The most severe forms of accelerated aging disease in humans are due to nDNA repair defects, and many of these diseases do not exhibit increased cancer incidence. High rates of cellular senescence and apoptosis due to high rates of nDNA damage are apparently the main cause of the elderly phenotype in these diseases.

Dr. Venki Ramakrishnan
Nobel Prize Chemistry

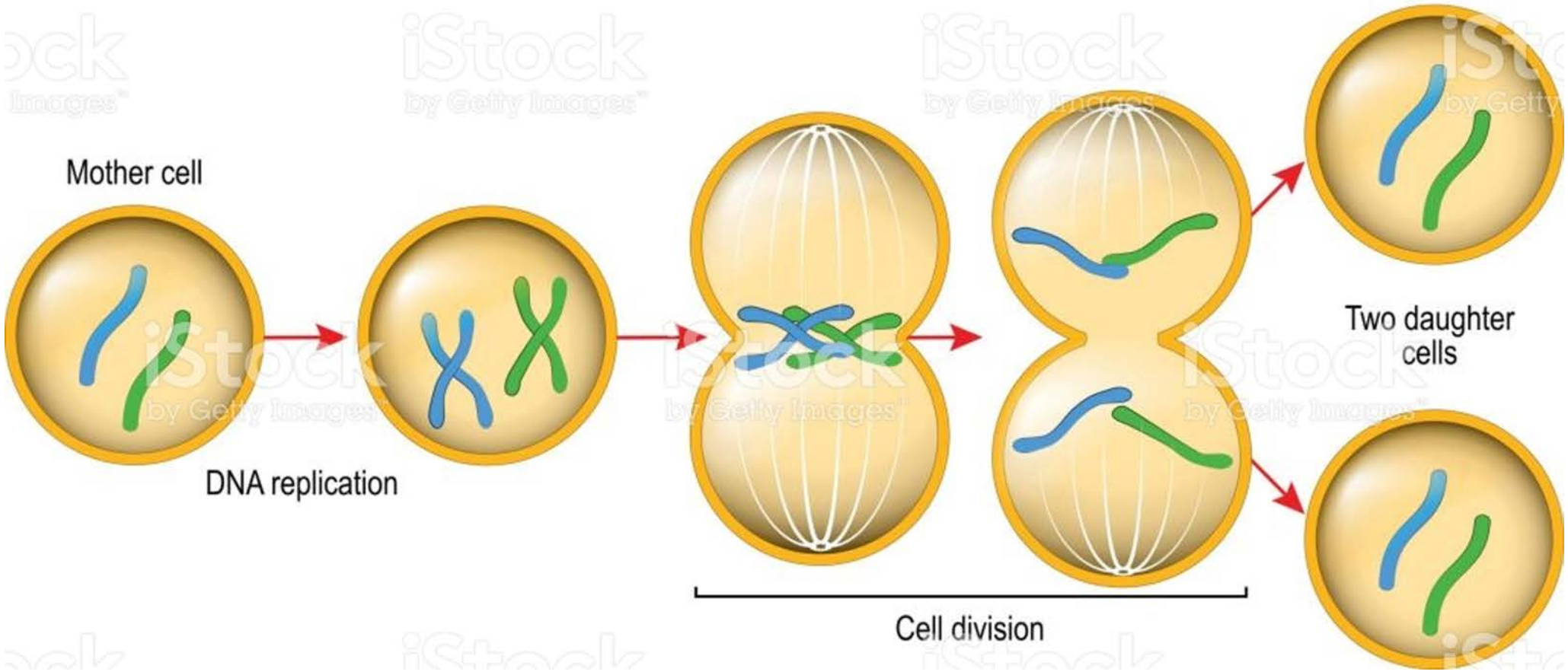
"As we age, the cell's quality control and recycling machinery deteriorates, leading not only to neurodegenerative but also many other diseases of old age.

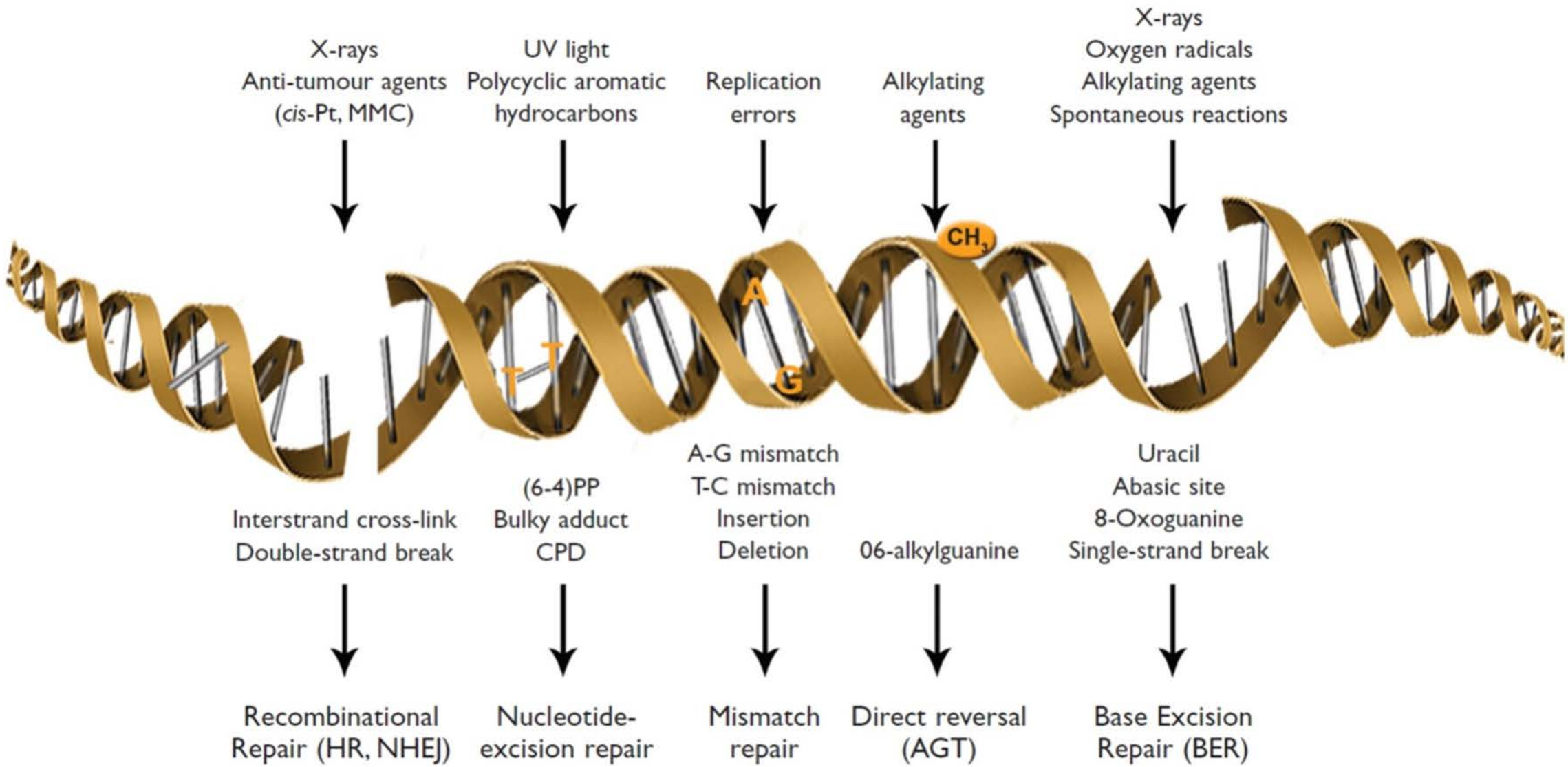
When a sufficient number of cells accumulate defects with age, the symptoms of aging manifest themselves: arthritis, fatigue, susceptibility to infection, decreased cognition, and, more generally, bodies that simply do not work as well as they did in our youth."



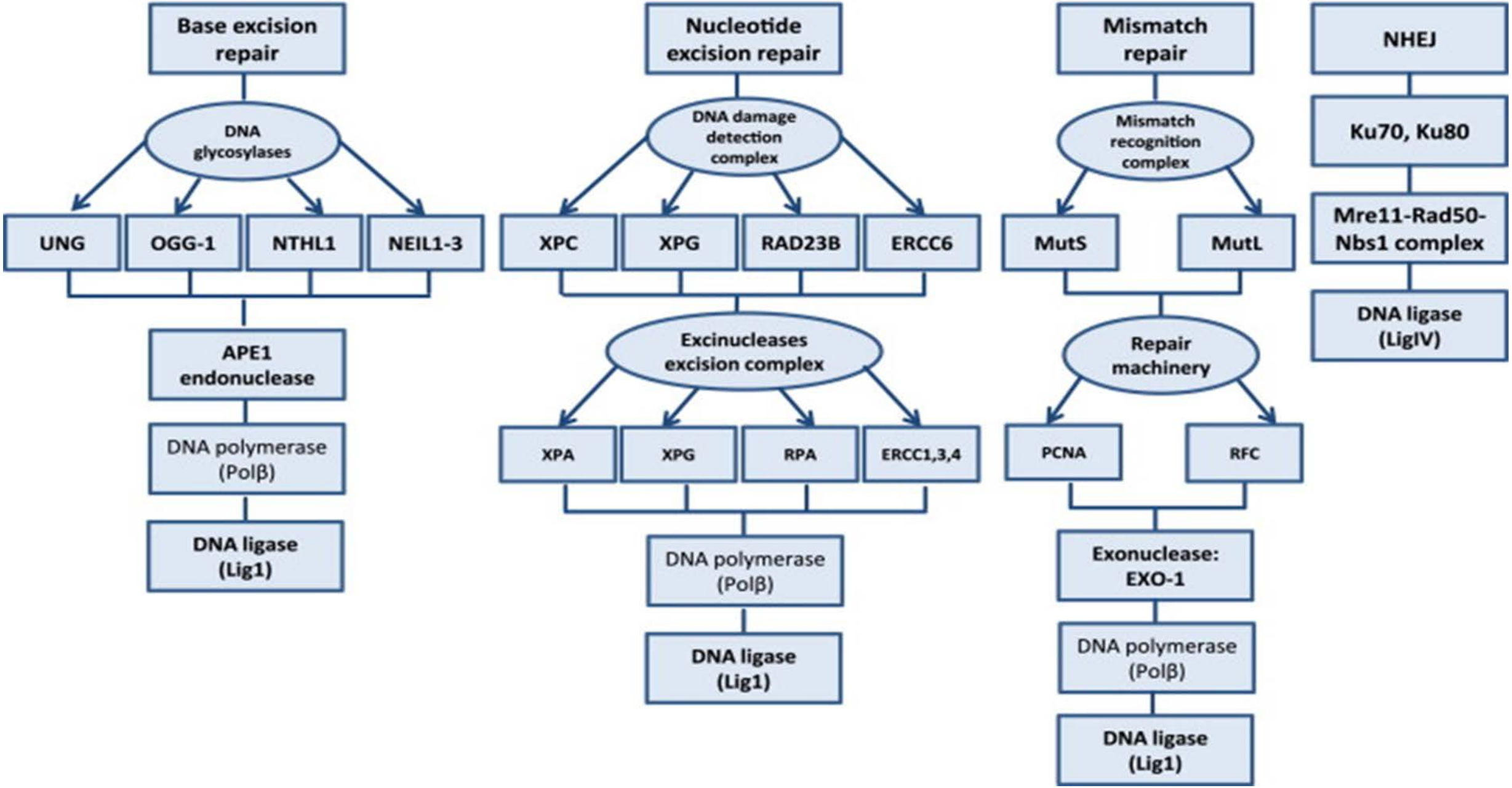

Aging and age-related diseases
Cancer
Type 2 Diabetes
Cardiovascular disease
Chronic Renal disease
Frailty
Sarcopenia
Osteoporosis
Neurodegenerative disease
Metabolic deregulation
Interstitial adiposity
Insulin resistance
SASP
SA-β-gal
p16INK4a, p21Cip1
p53, ATM, ATR, γH2AX
Chronic stress response activation
Epigenetic changes
mTOR activation
Cellular senescence
DNA damage
Telomere damage
Autophagy
Stem cells
Ki-67
Lamin B1
Apoptosis
Proteostasis
NAD+
Biogenesis
OXPHOS
ΔΨm
Mitochondrial dysfunction
ROS
Mutations

MITOSIS

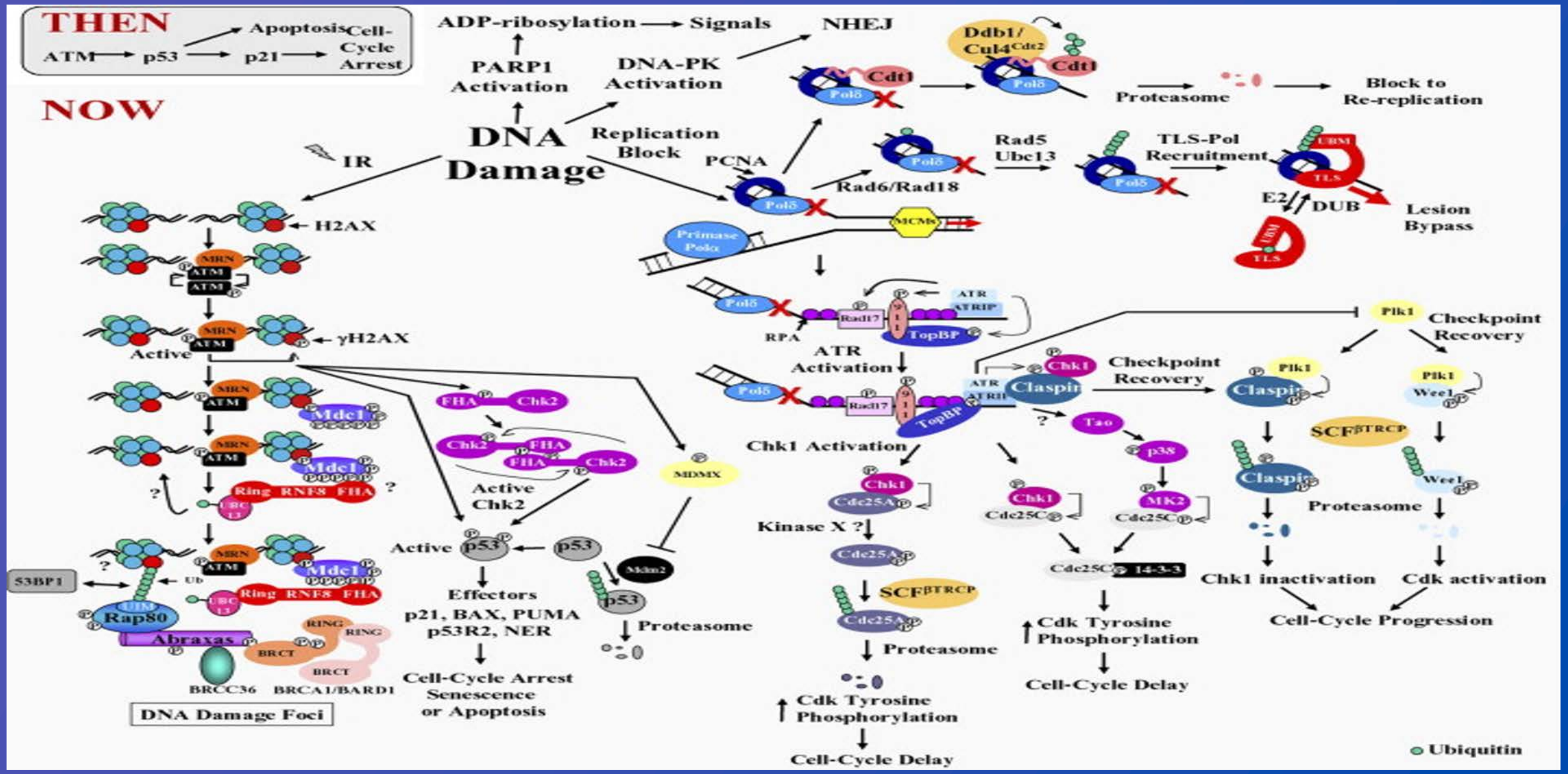

Mother cell
DNA replication
Cell division
Two daughter cells


X-rays
Anti-tumour agents
(*cis*-Pt, MMC)
UV light
Polycyclic aromatic
hydrocarbons
Replication
errors
Alkylating
agents
X-rays
Oxygen radicals
Alkylating agents
Spontaneous reactions
CH3
A
G
T
T
Interstrand cross-link
Double-strand break
(6-4)PP
Bulky adduct
CPD
A-G mismatch
T-C mismatch
Insertion
Deletion
06-alkylguanine
Uracil
Abasic site
8-Oxoguanine
Single-strand break
Recombinational
Repair (HR, NHEJ)
Nucleotide-
excision repair
Mismatch
repair
Direct reversal
(AGT)
Base Excision
Repair (BER)

Major DNA repair systems



DNA REPAIR SYSTEM-WHAT COULD GO WRONG?

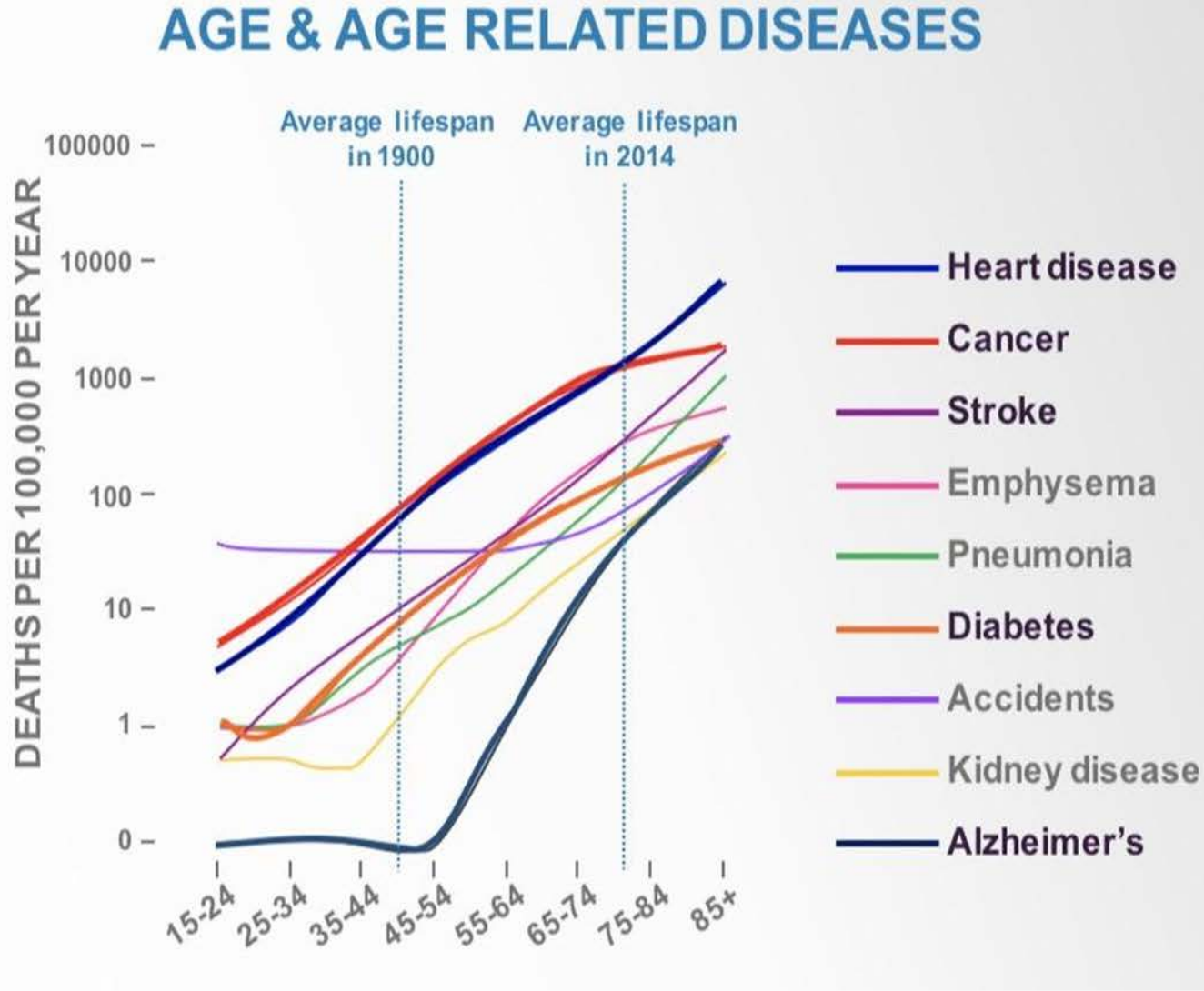


Irvin "Irv" Gordon (USA) clocked his three-millionth mile in his 1966 Volvo 1800S, for a total of 3,039,122 miles.

It was driven the equivalent of nearly 120 complete circumnavigations of the planet or thirteen round trips to the moon!

A Global Epidemic of Age Related Diseases as we Grow Older as a Society

- Nearly 92% of older adults have at least one chronic condition, and 77% have at least two.
- On average, ages 65 to 69 years old take nearly 14 prescriptions, ages 80 to 84 take 18 prescriptions per year.
- Cardiovascular, obesity, diabetes, cancers and respiratory diseases are global epidemics.
- Alzheimer's, diabetes, heart disease and cancer are responsible for 80% of the US Medicare budget.
- Over $30 trillion over the next 20 years
- Metabolic dysfunction is a key factor in most age-related diseases



St. Petersburg Institute of Bioregulation and Gerontology

COLONEL MEDICAL SERVICE
USSR MILITARY CORP
1971-1988

Vladimir Khavinson, M.D., Ph.D.

BIOREGULATOR DIRECTOR S.M. KIROV MEDICAL MILITARY ACADEMY
1988-1993

ASSOCIATE MEMBER OF THE RUSSIAN ACADEMY OF MEDICAL SCIENCES, PROFESSOR, DOCTOR OF MEDICINE

HONORED SCIENTIST AND INVENTOR OF THE RUSSIAN FEDERATION

PRESIDENT OF THE INTERNATIONAL ASSOCIATION OF GERONTOLOGY AND GERIATRICS-EUROPEAN REGION (2011-2015)

USSR COUNCIL OF MINISTERS' AND UKRAINIAN NATIONAL ACADEMY OF SCIENCES PRIZE WINNER

AUTHOR OF 700+ SCIENTIFIC PUBLICATIONS, INCL. 27 MONOGRAPHS

181 DOMESTIC AND INTERNATIONAL PATENTS

INVENTOR OF 6 PHARMACEUTICALS AND 39 PEPTIDE BIOREGULATORS

Cellular Rejuvenation

“Peptide Bioregulators regenerate organs impaired by aging, disease, and trauma.

The peptides developed at the St. Petersburg Institute of Bioregulation And Gerontology constitute a proven biological reprogramming technology, rejuvenating cells to revitalize tissue/organs and entire bodies, ultimately decreasing vulnerability to age-related degeneration and prolonging human life.”

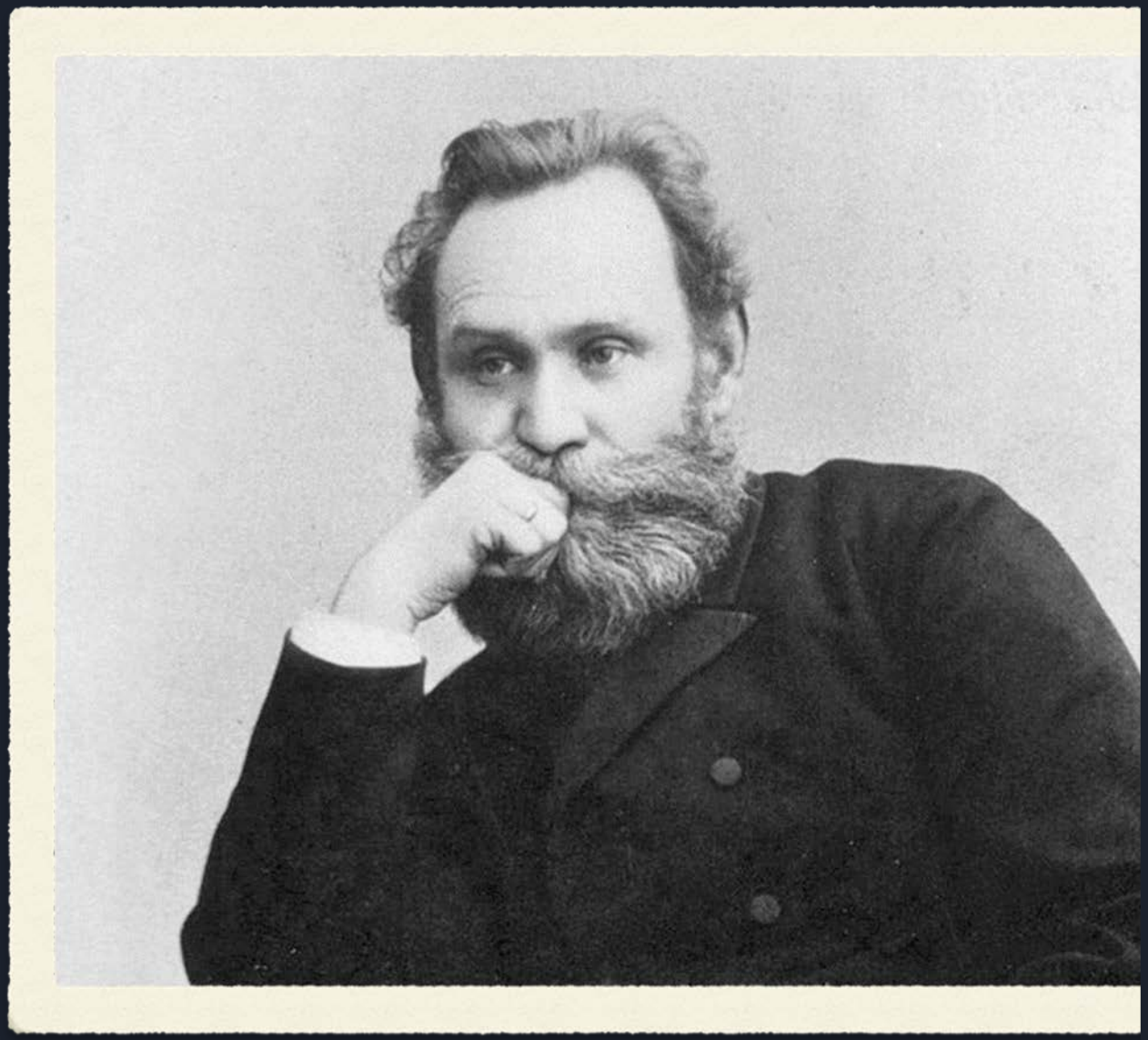
Prof Vladimir Khavinson MD, Ph.D
2003
English Translation from Russian

Professor Vladimir Khavinson, MD, Ph.D.

"It is my forecast that in the next 10-15 years peptide pharmaceuticals will become the most crucial development in the world."

Ivan Pavlov was awarded the 1904 Nobel Prize in Physiology or Medicine for his studies of gastrointestinal physiology.

Prof. Khavinson based Peptide Bioregulator development on Pavlov's discovery of small amino acid chains.



Notable institutions of peptide bioregulator studies:

National Institute on Aging (Baltimore, MD)

Italian National Research Center on Aging (Ancona, Italy)

Institute of Anatomy, Ludwig-Maximilians University of Munich (Munich, Germany)

Prince Felipe Research Center (Valencia, Spain)

University of Antwerp, Dept. of Biomedical Sciences (Antwerp, Belgium)

History of Peptide Bioregulators

Soviet Union Military

Soviet Union Cosmonauts

Russian Peptide Technology was a Military Secret Founded During the Cold War

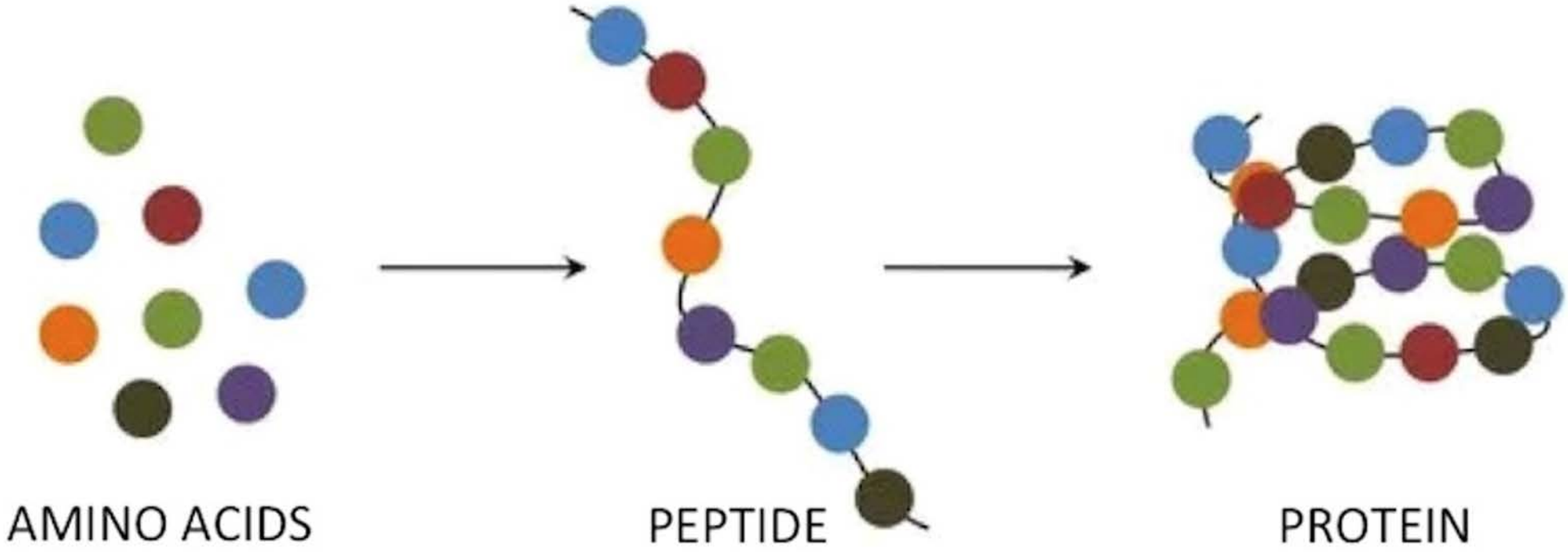
The Russians had to develop solutions for severe medical challenges of the time.

on right in both images is Professor Vladimir Khavinson

Enhancement of vital resource of Russian Olympic team in rhythmic gymnastics

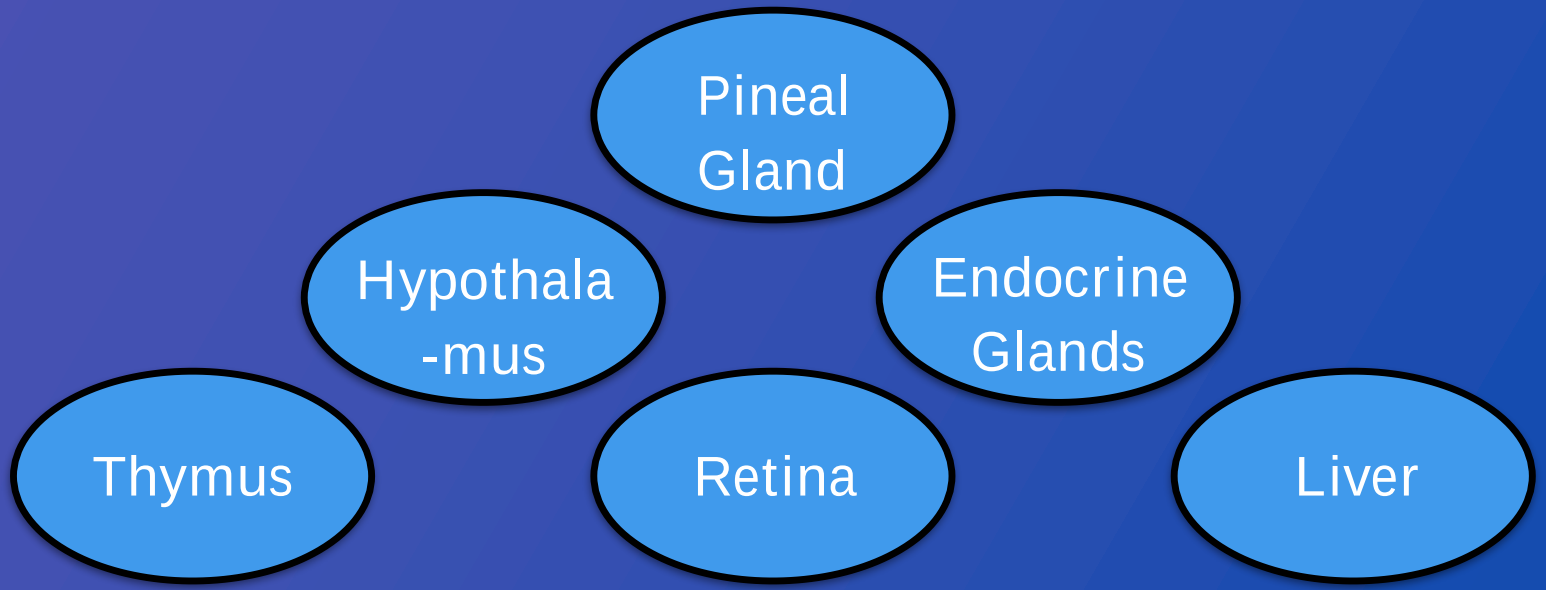
left to right: A. Shumilova (coach), D. Kondakova, A. Zaripova (coach), J. Lukonina, Prof. V.Khavinson, E.Kanaeva, V. Schtelbaums (coach), I. Viner-Usmanova (main coach of team, honored coach of Russia), O. Buyanova (coach), D. Dmitrieva

HOW YOUR BODY USES AMINO ACIDS AS BUILDING BLOCKS



Russian Peptide Bioregulators are Extracted from Whole Food Source

These peptides are isolated, purified, and fractionated low-molecular weight peptides from bovine-sourced organs such as:

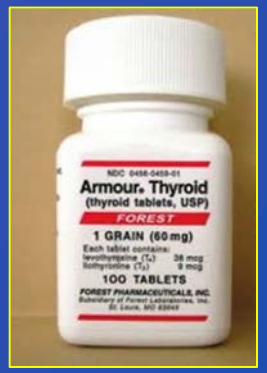


Traditional healing with animals (zootherapy): medieval to present-day Levantine practice

E Lev

Journal of ethnopharmacology, 2003 Elsevier

Animals and products derived from different organs of their bodies have constituted part of the inventory of medicinal substances used in various cultures since ancient times. This article reviews the history of healing with animals in the Levant (the Land of Israel and parts of present-day Syria, Lebanon, and Jordan, defined by the Muslims in the Middle Ages as Bilad al-Sham) throughout history. Intensive research into the phenomenon of zootherapy in the Levant from early medieval to present-day traditional medicine yielded 99 substances of animal origin which were used medicinally during that long period. Fifty-two animal extracts and products were documented as being used from the early Muslim period (10th century) to the late Ottoman period (19th century).



SHORT CHAINS OF AMINO ACIDS

Peptide Bioregulators

- Short chains of 2-4 amino acids-essentially fractions of protein
- Extracted from organs and tissues of animals
- Essential regulators for key cellular processes

Rejuvenation of human organs and systems	Restore the protein-peptide cycle	Telomerase Activation
Activate genes for cellular regeneration	Regulate gene expression and protein synthesis	DNA Methylation

Khavinson V.K. Peptides and ageing. Neuroendocr. Lett. 2002;23:11–144. [PubMed]

Khavinson V.K., Linkova N.S., Tarnovskaya S.I. Short Peptides Regulate Gene Expression. Bull. Exp. Biol. Med. 2016;162:288–292. doi: 10.1007/s10517-016-3596-7. [PubMed]

Anisimov V.N., Khavinson V.K. Peptide bioregulation of aging: Results and prospects. Biogerontology. 2010;11:139–149. doi: 10.1007/s10522-009-9249-8. [PubMed]

PEPTIDE BIOREGULATORS REGULATE GENE EXPRESSION

Short peptides (di-, tri-, and tetrapeptides) are signaling molecules capable of interacting with DNA and histone proteins, acting as regulatory factors.

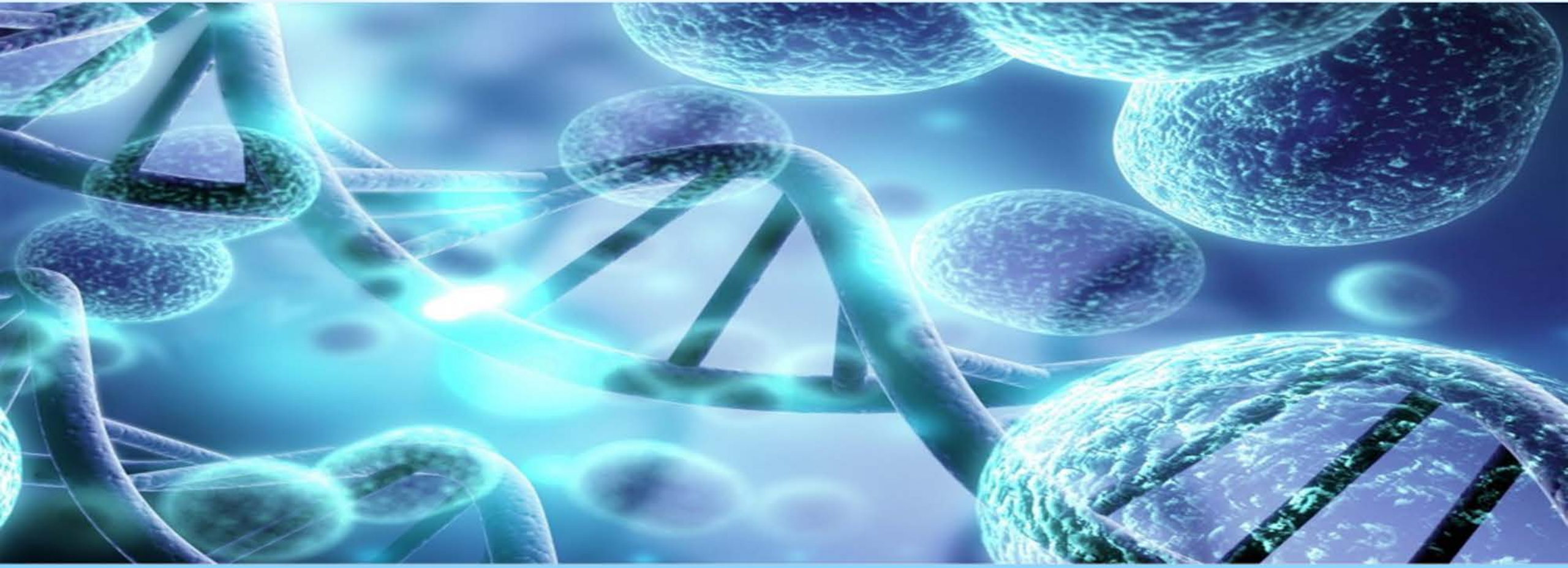
Clinical studies confirm short peptides **regulate functional activity, proliferation, apoptosis, and differentiation** of human, animal, and plant cells.

22 NATURAL PEPTIDE BIOREGULATOR EXTRACTS

Bioregulators	Source of the peptides
Bonomarlot®	Bone marrow
Bonothyrk®	Parathyroid gland
Cerluten®	Brain
Chelohart®	Heart
Chitomur®	Bladder
Endoluten®	Pineal gland
Epitide®	Pineal gland
Glandokort®	Adrenal gland
Gotratix®	Muscle
Libidon®	Prostate
Pielotax®	Kidney

Bioregulators	Source of the peptides
Stamakort®	Stomach
Sigumir®	Cartilage
Suprefort®	Pancreas gland
Svetinorm®	Liver
Taxorest®	Respiratory System
Testoluten®	Testis
Thyreogen®	Thyroid gland
Ventfort®	Vessels
Visoluten®	Eyes
Vladonix®	Thymus
Zhenoluten®	Ovaries

PEPTIDE BIOREGULATORS



BIOLOGICAL AGE REVERSAL

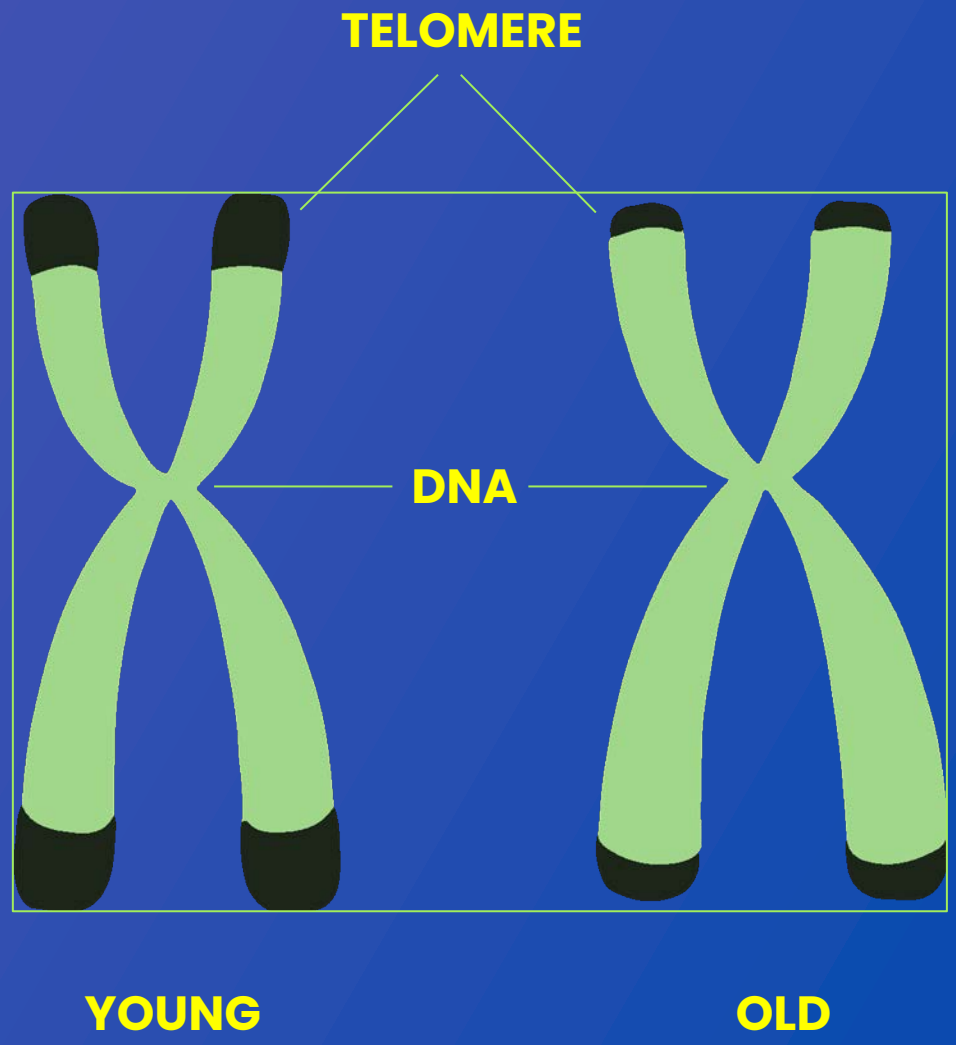
ST. PETERSBURG INSTITUTE OF BIOREGULATION AND GERONTOLOGY

Telomerase Activation & DNA Methylation Longevity Interventions

Telomeres and Aging

“Every time our cells divide, our telomeres get a little shorter, and every time they shorten, our cells age.”

William Andrews, Ph.D



Longevity Relevance of Telomeres

"Telomerase, slows the rate at which telomeres degrade, and research indicates people with longer telomeres have less risk of developing the common illnesses of aging."

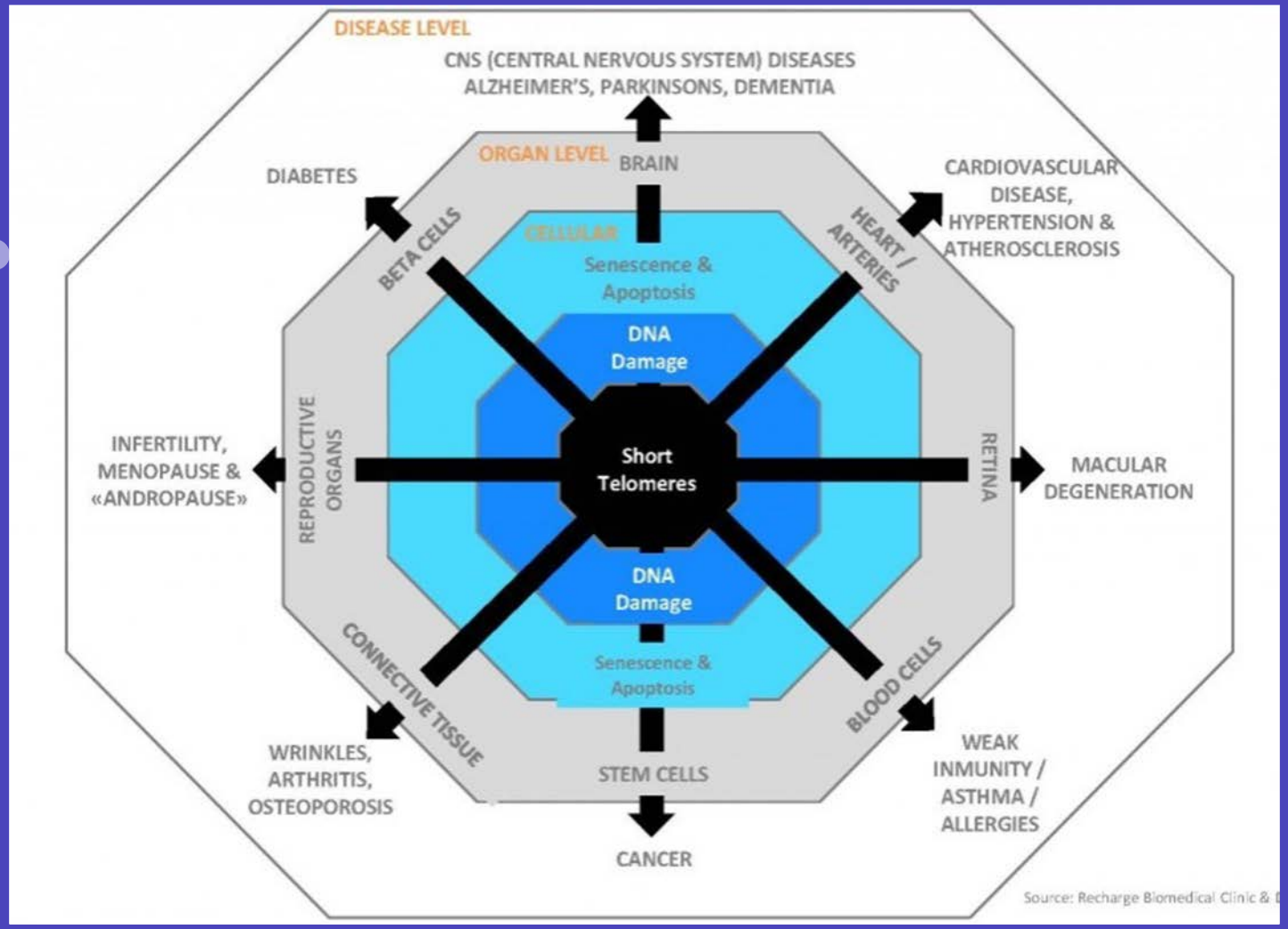
– Elizabeth Blackburn, Ph.D.
2009 Nobel Laurate

"Telomerase Activation is the single most promising approach to reversing the effects of aging."

– Michael Fossel, MD, Ph.D.
The Telomerase Revolution

Telomerase is a primary indicator of the following:

- Aging
- Mortality
- Chronic Disease



"

Shortened Telomeres Play a Central Role in the Development of Age-Related Diseases

Telomeres and Disease

Fraternal twins with the shortest telomeres had **three times greater risk of death** than their co-twins with the longest telomere measurements

Short telomeres are associated with increased risk for:

- **CANCER**
- **DIABETES**
- **HEART DISEASE**
- **ACCELERATED AGING**

Telomere shortening in human diseases Chiou Mee Kong, Xiao Wen Lee and Xueying Wang Department of Biochemistry, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Johansson, S et al. Telomere length predicts survival independent of genetic influences (2007) *Aging Cell*, 2007.

Short Telomeres Increase Risk of Severe COVID-19

Abstract

Telomeres are non-coding DNA sequences that protect chromosome ends and shorten with age. Short telomere length (TL) is associated with chronic diseases and immunosenescence. The main risk factor for mortality of coronavirus disease 2019 (COVID-19) is older age, but outcome is very heterogeneous among individuals of the same age group. Therefore, we hypothesized that TL influences COVID-19-related outcomes. In a prospective study, we measured TL by Flow-FISH in 70 hospitalized COVID-19 patients and compared TL distribution with our reference cohort of 491 healthy volunteers. We also correlated TL with baseline clinical and biological parameters. We stained autopsy lung tissue from six non-survivor COVID-19 patients to detect senescence-associated β-galactosidase activity, a marker of cellular aging. We found a significantly higher proportion of patients with short telomeres (<10th percentile) in the COVID-19 patients as compared to the reference cohort (P<0.001). Short telomeres were associated with a higher risk of critical disease, defined as admission to intensive care unit (ICU) or death without ICU. TL was negatively correlated with C-reactive protein and neutrophil-to-lymphocyte ratio. Finally, lung tissue from patients with very short telomeres exhibit signs of senescence in structural and immune cells. Our results suggest that TL influences the severity of the disease.

•Antoine Froidure 1 2, Manon Mahieu 3, Delphine Hoton 2 4, Pierre-François Laterre 2 5, Jean Cyr Yombi 2 6, Sandra Koenig 1, Benoit Ghaye 2 7, Jean-Philippe Defour 3 8, Anabelle Decottignies 3

BIOGERONTOLOGY

Epithalon Peptide Induces Telomerase Activity and Telomere Elongation in Human Somatic Cells

V. Kh. Khavinson, I. E. Bondarev, and A. A. Butyugov

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 135, No. 6, pp. 692-695, June, 2003
Original article submitted April 24, 2003

Addition of Epithalon peptide in telomerase-negative human fetal fibroblast culture induced expression of the catalytical subunit, enzymatic activity of telomerase, and telomere elongation, which can be due to reactivation of telomerase gene in somatic cells and indicates the possibility of prolonging life span of a cell population and of the whole organism.

Key Words: *peptide; Epithalon; telomerase; telomeres; fibroblasts*

✦124 Participants-Primarily Physicians

✦Various Dosages (low, medium, high)

*No other lifestyle changes or interventions prescribed

Primary Bioregulators Used

- ❖Arterial System (Ventfort)
- ❖CNS (Cerluten)
- ❖Cartilage (Sigumir)
- ❖Liver (Syetinorm)
- ❖Pancreas (Suprefort)
- ❖Pineal Gland (Endoluten)
- ❖Thymus (Vladonix)

https://www.antiaging-systems.com/288-peptides

SpectraCell Laboratories
Science + Health + Solutions

6030 North Course Drive, Houston, TX 77072
1(800) 227-5227 | support@spectracell.com | www.spectracell.com

PATIENT

NAME: Adams, [redacted]
AGE: 71
DOB: 12/17/1949
GENDER: Male
PATIENT ID: 21-013-00076

SPECIMEN

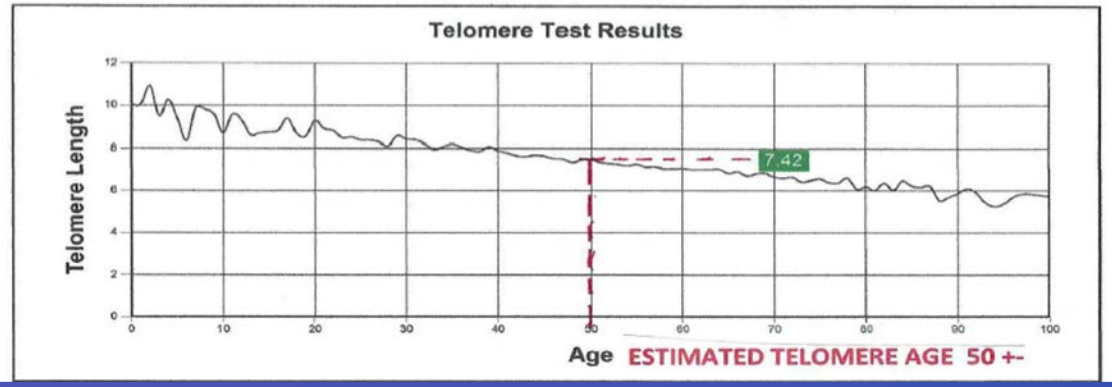
ACCESSION ID: 2101130106
DATE COLLECTED: 01/12/2021
ORDER ID: 1105-0000000078C-210113
DATE RECEIVED: 01/13/2021
DATE REPORTED: 01/26/2021

PROVIDER

ACCOUNT ID: 78C
CLIENT NAME: SpectraCell DTC Account
ADDRESS: DTC Account

Telomere

Tests	Results	Units
Telomere Interpretation	7.42	Kb
Telomere Percentage	72.00	%



2021 Telomere Age **50**

SpectraCell Laboratories
Science + Health + Solutions

6030 North Course Drive, Houston, TX 77072
1(800) 227-5227 | support@spectracell.com | www.spectracell.com

PATIENT

NAME: Adams, [redacted]
AGE: 72
DOB: 12/17/1949
GENDER: Male
PATIENT ID: 22-188-00079

SPECIMEN

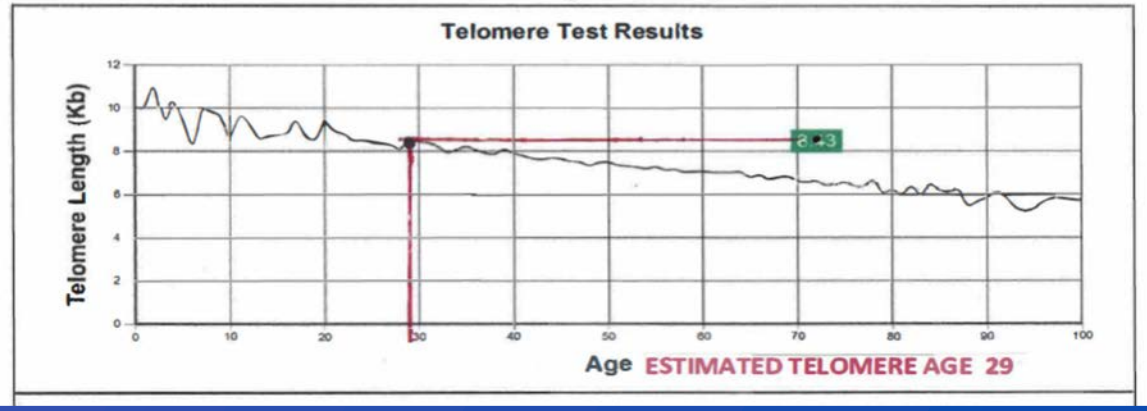
ACCESSION ID: 2207070096
DATE COLLECTED: 07/05/2022
ORDER ID: 1094-0007434073-3-220707
DATE RECEIVED: 07/07/2022
DATE REPORTED: 08/05/2022

PROVIDER

ACCOUNT ID: 74340733
CLIENT NAME: Lawrence Bill
ADDRESS: 3560 Piedmont Rd NE Apt 206 Atlanta, GA 30305

Telomere

Tests	Results	Units
Telomere Length (Average)	8.43	Kb
Telomere Percentile (Relative to others in the same age group)	92.00	%



2022 Telomere Age **29**

SpectraCell Laboratories
Science + Health + Solutions

6030 North Course Drive, Houston, TX 77072
1(800) 227-5227 | support@spectracell.com | www.spectracell.com

PATIENT

NAME: [redacted], Connie
AGE: 77
DOB: 1/23/1944
GENDER: Female
PATIENT ID: 21-147-00141

SPECIMEN

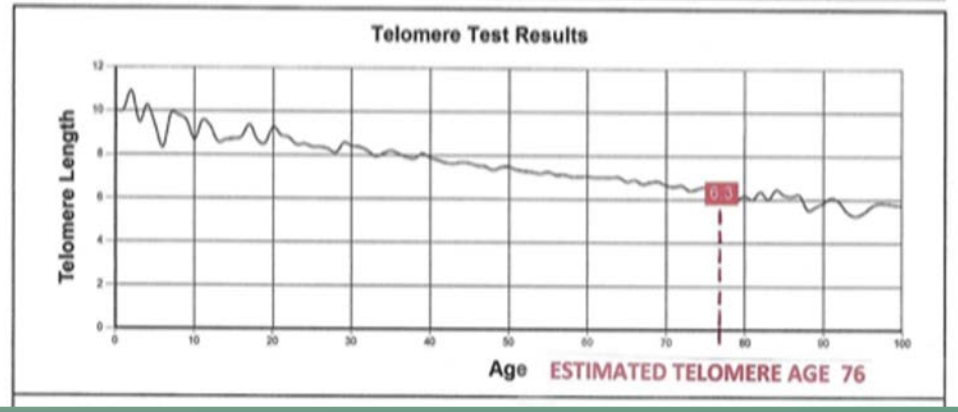
ACCESSION ID: 2105270178
DATE COLLECTED: 05/26/2021
ORDER ID: 1177-00074340733-210527
DATE RECEIVED: 05/27/2021
DATE REPORTED: 06/28/2021

PROVIDER

ACCOUNT ID: 74340733
CLIENT NAME: Lawrence Bill
ADDRESS: 3560 Piedmont Rd NE Apt 206 Atlanta, GA 30305

Telomere

Tests	Results	Units
Telomere Interpretation	6.30	Kb
Telomere Percentage	47.00	%



2021

Telomere Age

76

SpectraCell Laboratories
Science + Health + Solutions

6030 North Course Drive, Houston, TX 77072
1(800) 227-5227 | support@spectracell.com | www.spectracell.com

PATIENT

NAME: [redacted], Connie
AGE: 79
DOB: 1/23/1944
GENDER: Female
PATIENT ID: 21-147-00141

SPECIMEN

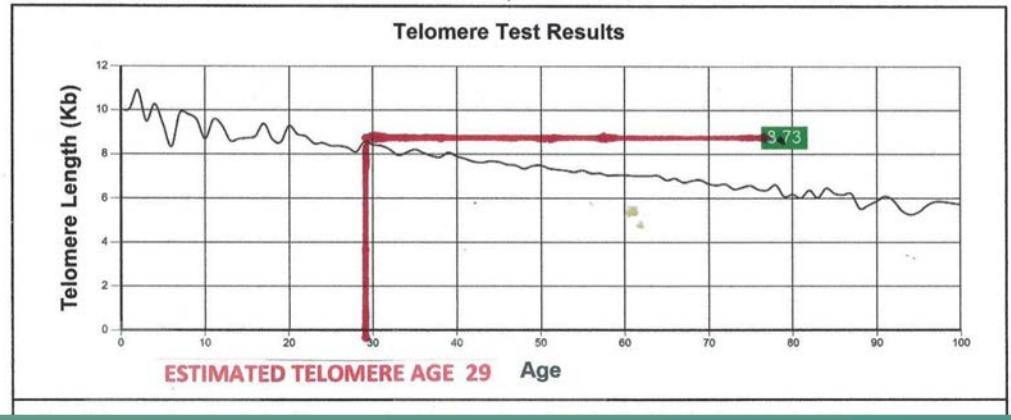
ACCESSION ID: 2212290048
DATE COLLECTED: 12/28/2022
ORDER ID: 1048-00074340733-221229
DATE RECEIVED: 12/29/2022
DATE REPORTED: 01/23/2023

PROVIDER

ACCOUNT ID: 74340733
CLIENT NAME: Lawrence Bill
ADDRESS: 3560 Piedmont Rd NE Apt 206 Atlanta, GA 30305

Telomere

Tests	Results	Units
Telomere Length (Average)	8.73	Kb
Telomere Percentile (Relative to others in the same age group)	96.00	%



2023

Telomere Age

29

Torah Bright

Olympic gold and Silver Winner

32

Chronological Age

56

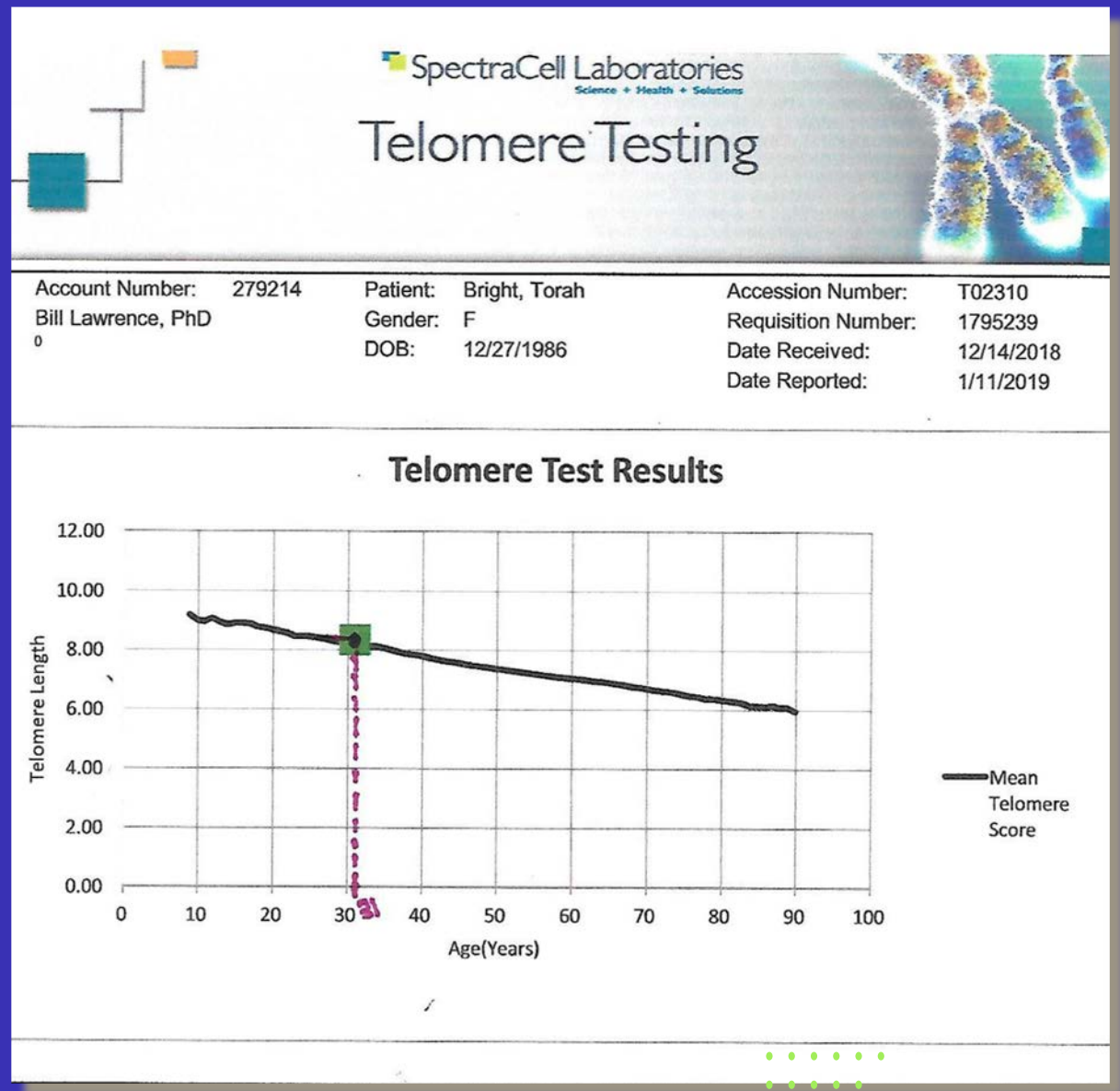
Telomere age at baseline

TELOMERE ACTIVATION PROTOCOL

Torah Bright

Post Peptide intervention estimated telomere age equivalent to:

31 yr old



TELOMERASE ACTIVATION PROTOCOL (TAP) CLINICAL STUDY

KHAVINSON PEPTIDE BIOREGULATORS

RESULTS

2017- 2022

CELLULAR AGE BASED ON TELOMERE LENGTH (Kb)

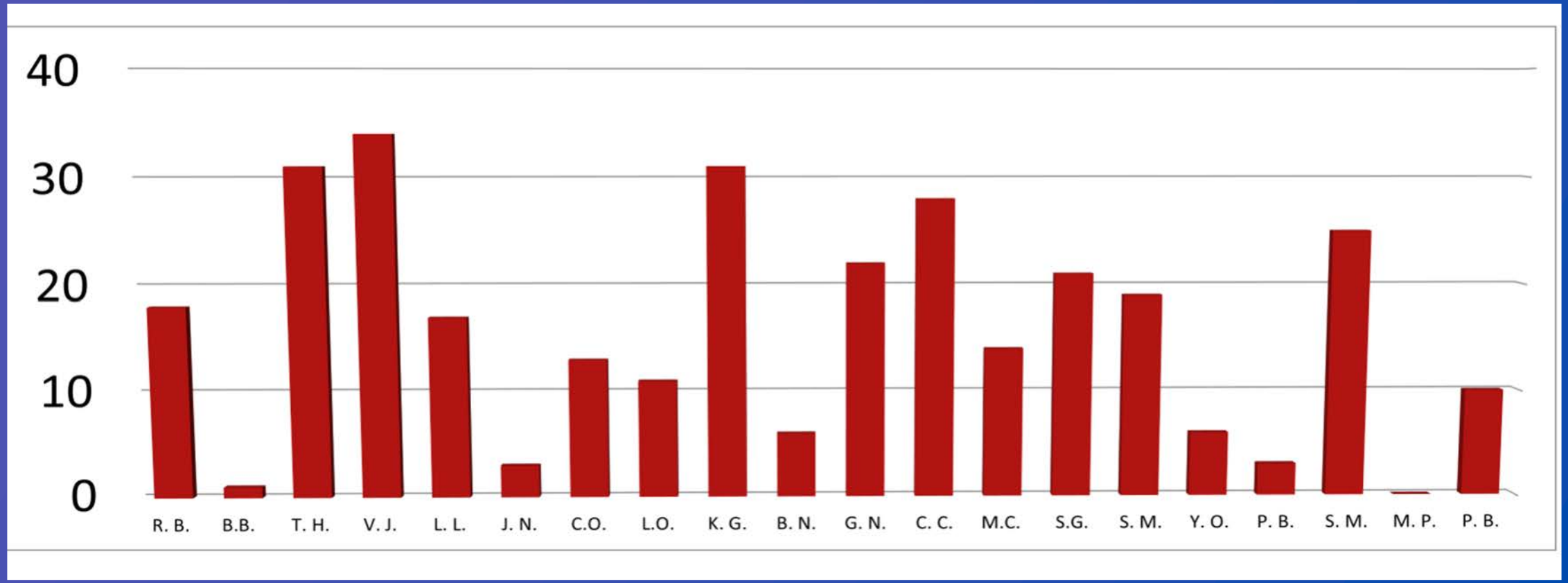
		BASELINE TEST				SECONDARY TESTING							RESULTS		
SUBJECT	TEST DATE	BASELINE AGE	TELOMERE AGE	TELOMERE PERCENTILE	Kb	TEST DATE	SECONDARY AGE	TELOMERE AGE	TELOMERE PERCENTILE	Kb	DOSAGE PROTOCOL	DURATION (MONTHS)	TELO AGE REDUCTION (years)	kb Gain	Kb Loss
R. BISHOP	2017 Jan	57	80*	18	5.65	22-Sep	63	49	88	7.13	Low	68	31	1.48	
B. BISHOP	2017 Jan	58	80*	13	5.88	22-Sep	64	62	49	6.89	Low	68	18	1.01	
T. Humphrey	2017 July	69	75	8	5.98	2019-Jan	71	44	79	7.71	High	18	31	1.73	
V. Jones	2016 June	60	62	49	6.84	18-Dec	62	33	83	8.13	High	30	29	1.29	
L. Lawrence	2017 Feb	69	80*	1	6.34	18-Aug	70	63	8	7.27	Low	18	19	0.92	
J. Nyboer	2016 Aug	73	50	94	8.32	17-Nov	74	47	99	8.33	Low	14	3	0.01	
C. Olson	2017 Feb	76	80*	3	5.4	18-Dec	78	67	68	6.9	Medium	22	13	1.5	
L. Olson	2017 Feb	75	80*	8	6.22	18-Dec	76	69	66	6.86	Medium	22	11	0.64	
K. Guthrie	2018 Feb	71	76	1	5.94	18-Sep	72	45	79	7.57	Medium	15	31	1.63	
B. Nottingham	2018 Jan	68	74	1	5.77	18-Nov	69	68	56	6.86	High	11	6	1.09	
G. Nottingham	2017 June	64	72	18	6.04	18-Nov	66	50	69	7.45	High	17	22	1.41	
C. Cleveland	2021 May	77	76	47	6.3	22-Dec	79	29	96	8.73	Medium	19	47	2.43	
M. Carter	2018 Jan	55	68	14	7.9	18-Jul	56	54	55	8.6	High	8	14	0.7	
S. Gronemeyer	2019 Dec	73	80	31	6.02	21-Jan	74	50	75	7.51	High	13	26	1.49	
B. Lawrence	2017 Jan	71	72	50	6.4	18-Aug	72	44	82	7.77	Med	20	28	1.3	
Y. Oh	2016 June	81	84	53	5.96	18-Sep	83	73	64	6.57	High	27	11	0.61	
M. Poling	2017 Jan	68	80+	7	6.07	18-Dec	69	57	65	7.12	High	23	23	1.05	
L. Hirsch	2017 Jan	67	80+	5	6.03	18-Dec	68	32	89	8.26	Medium	23	36	2.23	
P. Bradfield	2018 April	74	77	56	6.63	18-Oct	75	75	58	6.72	Low	7	2	0.37	
T. Bright	2018 Feb	31	56	6	7.35	18-Dec	32	31	55	8.36	High	11	25	1.01	
M. Pompa	2017 Sept	49	35	57	7.83	21-Jan	53	8	99	10.2	Medium	28	27	2.38	
D. Pompa	2017 Sept	51	56	43	6.52	21-Jan	55	44	63	7.61	Medium	28	12	1.09	

NOTES:

* Telomere "age" of 80 is maximum reported as limited data available above age eighty

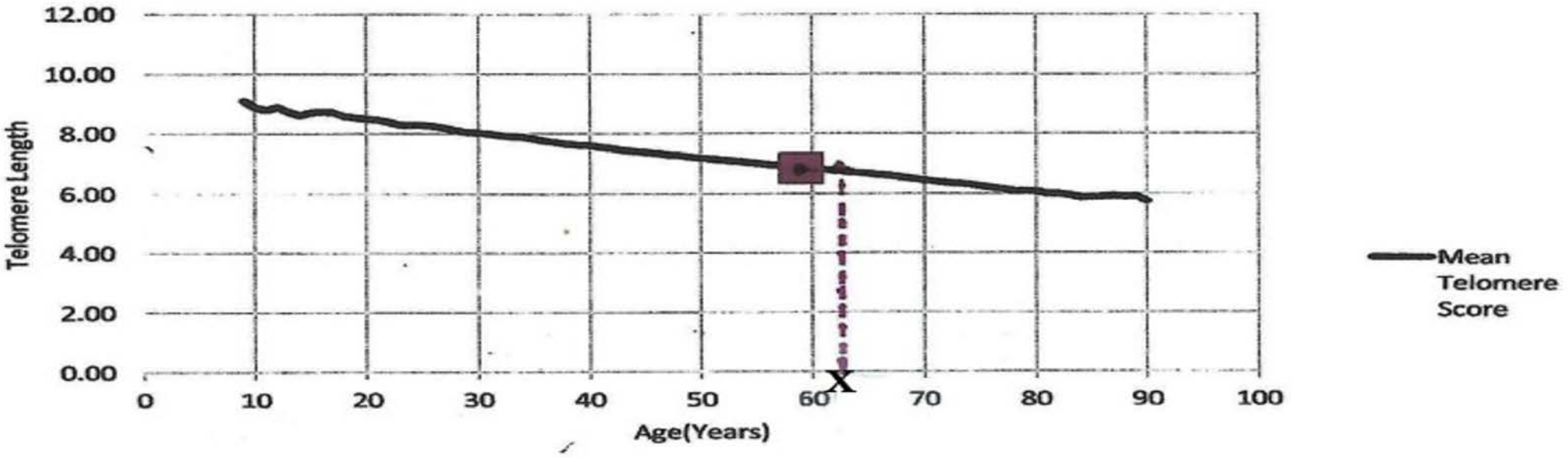
Dosage indication of Low, Medium, High refers to number of Khavinson peptides taken monthly

Reduction in Telomere “Age”



Account Number:	279214	Patient:	Jones, Ves	Accession Number:	Q01666
Bill Lawrence, PhD		Gender:	F	Requisition Number:	1290041
508 Main St.		DOB:	6/25/1956	Date Received:	6/10/2016
Unit #3348				Date Reported:	6/29/2016
Atlanta, GA 30324					
United States					

Telomere Test Results



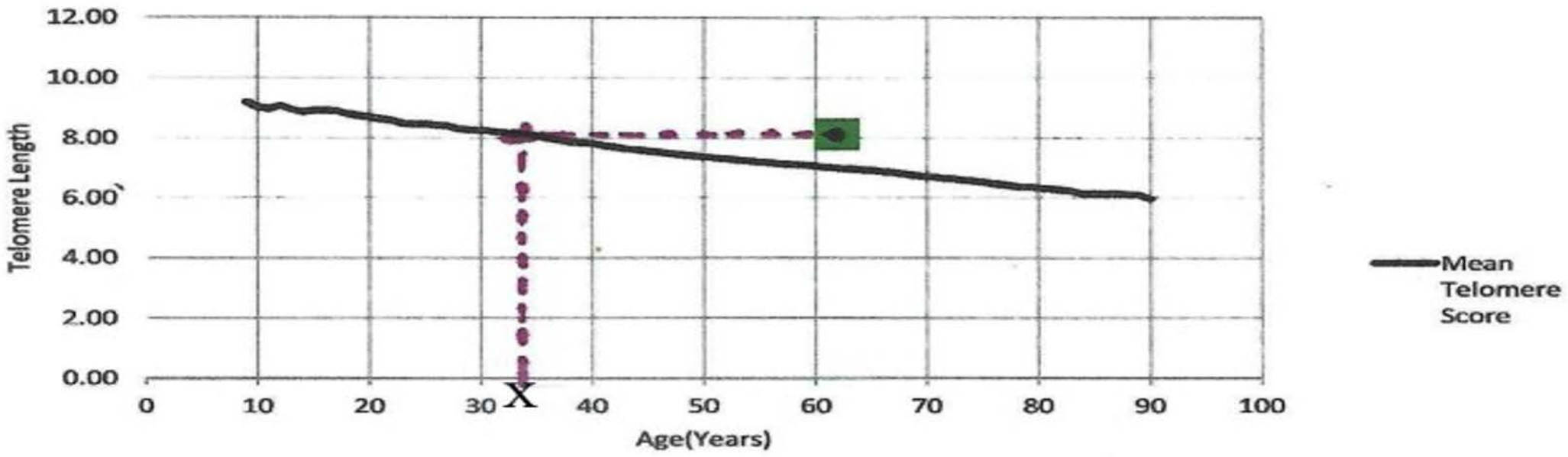
CHRONOLOGICAL AGE 60 **TELOMERE AGE 63**

Patient Telomere Score: **6.84**

Percentile Relative to Patient Age and Population: **49%**

Account Number:	279214	Patient:	Jones, Ves	Accession Number:	S95443
Bill Lawrence, PhD		Gender:	F	Requisition Number:	1788168
o		DOB:	6/25/1956	Date Received:	11/27/2018
				Date Reported:	12/20/2018

Telomere Test Results

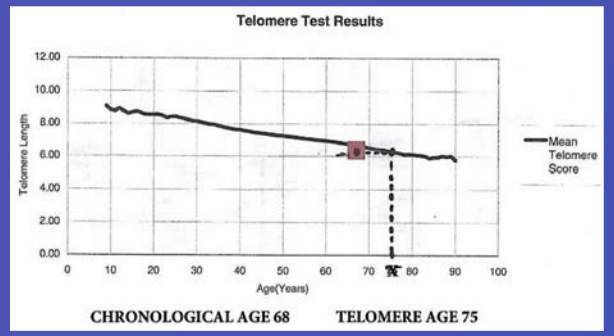


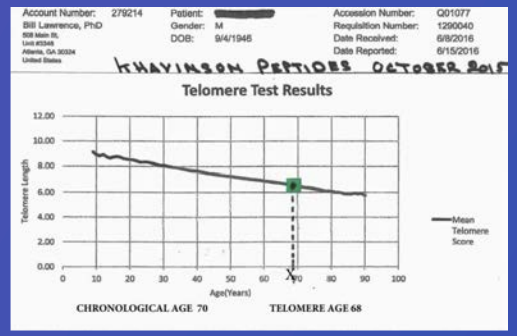
CHRONOLOGICAL AGE 62 TELOMERE AGE 33

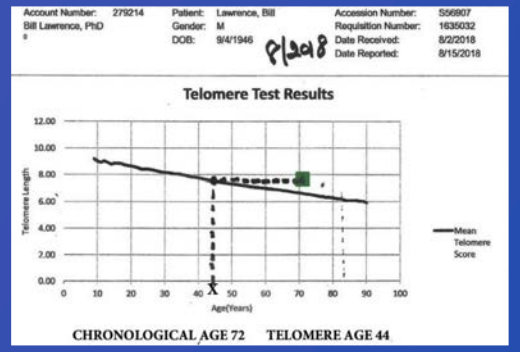
Patient Telomere Score: **8.13**

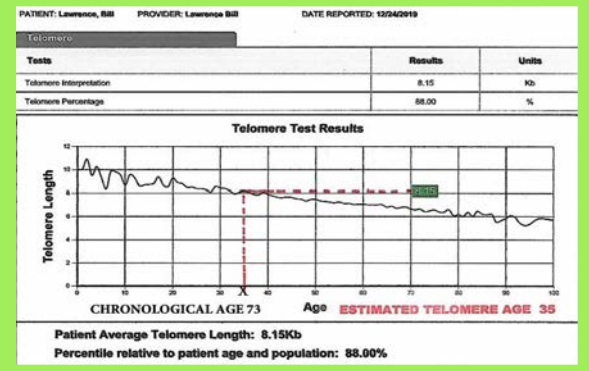
Percentile Relative to Patient Age and Population: **83%**

Bill Lawrence
Previous Results









2014

68 Chronological Age

75 Telomere Age

6.38Kb Telomere Length

2016

70 Chronological Age

68 Telomere Age

6.54Kb Telomere Length

2018

72 Chronological Age

44 Telomere Age

7.70Kb Telomere Length

2019

73 Chronological Age

35 Telomere Age

8.15Kb Telomere Length

Bill Lawrence

Results 2023

Chronological Age

76

Telomere Age

23

Telomere Length

8.65Kb

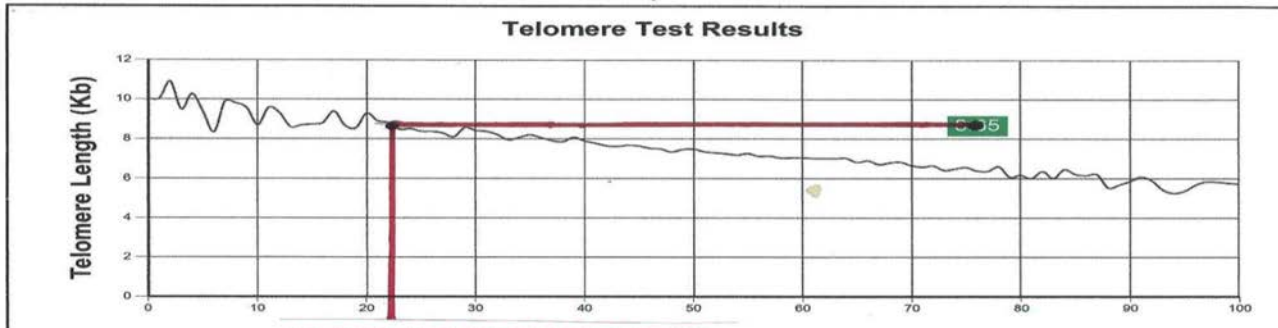
SpectraCell Laboratories
Science + Health + Solutions

6030 North Course Drive, Houston, TX 77072
1(800) 227-5227 | support@spectracell.com | www.spectracell.com

PATIENT		SPECIMEN		PROVIDER	
NAME Lawrence, Bill	AGE 76	ACCESSION ID 2306080114	DATE COLLECTED 06/07/2023	ACCOUNT ID 74340733	CLIENT NAME Lawrence Bill
DOB 9/4/1946	GENDER Male	ORDER ID 1112-00074340733-230608	DATE RECEIVED 06/08/2023	ADDRESS 3560 Piedmont Rd NE Apt 206 Atlanta, GA 30305	
PATIENT ID 19-119-00002			DATE REPORTED 06/16/2023		

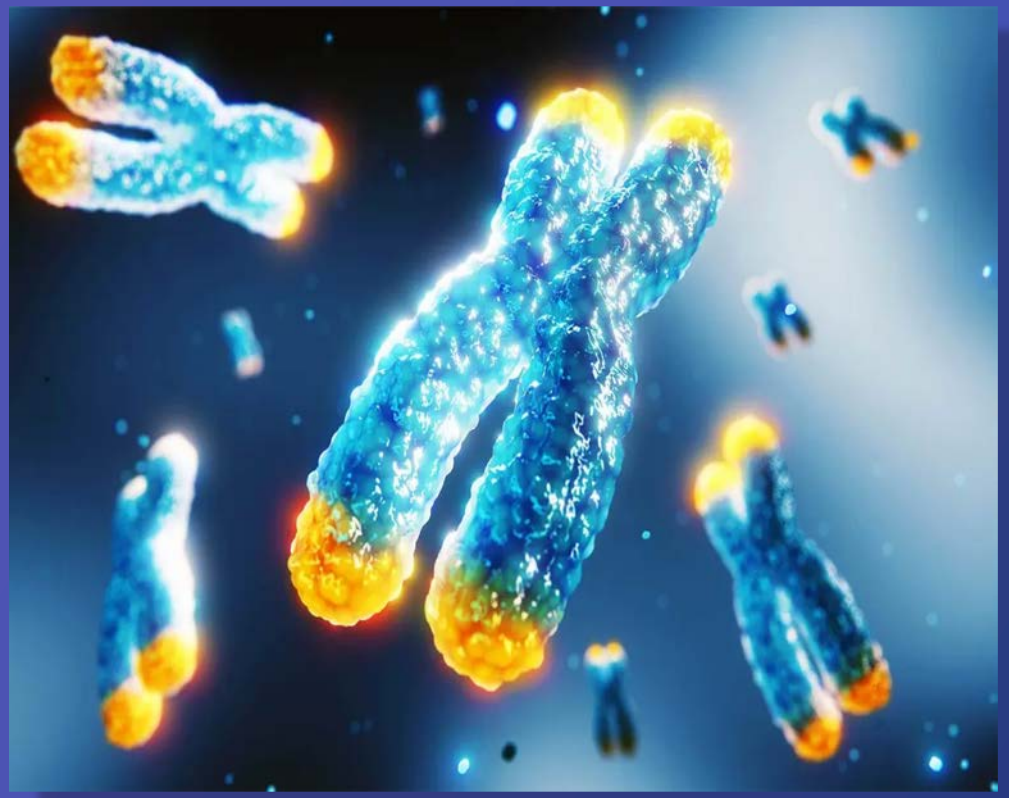
Telomere

Tests	Results	Units
Telomere Length (Average)	8.65	Kb
Telomere Percentile (Relative to others in the same age group)	95.00	%



Your Telomere Result is derived by measuring telomeres in nucleated white blood cells and calculating the average telomere length of these cells, which are obtained from whole blood via venipuncture.

The line on the graph represents tens of thousands of telomere results obtained at SpectraCell Laboratories over the course of a decade, and indicates what the average telomere length is for people in different age groups. The higher the telomere score, the "younger" the cells. If your telomere result is below the reference line, indicated by a red box, then your telomeres are shorter than others in the same age group. If your telomere result is above the reference line, indicated by a green box, then your telomeres are longer than others in your age group.



The average decrease in cellular or “biological age” was 21.62 years over the three-year period

Epigenetic Methylation Study

Bill Lawrence
JD, MS, Ph.D

VLADIMIR KHAVINSON, MD. PhD.

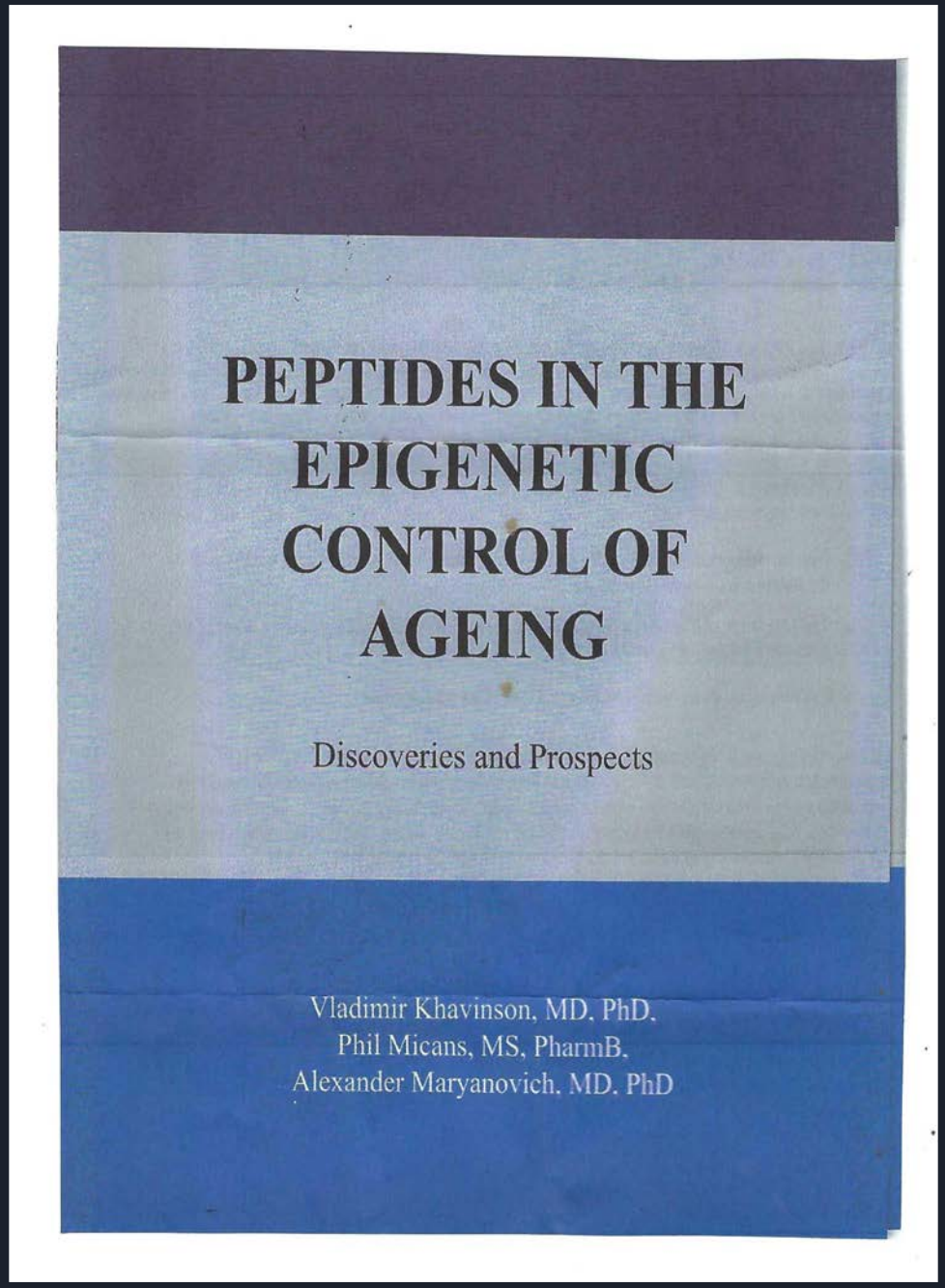
PHIL MICANS, MS. PharmB.

St. Petersburg Institute of Bioregulation and Gerontology

President (2011-2015) International Association of Gerontology and Geriatrics For Europe

THE SCIENCE OF EPIGENETIC AGE MODIFICATION

PUBLISHED 2017



Epigenetic Aspects of Peptide-Mediated Regulation of Aging

V. Kh. Khavinson[a, b], A. Yu. Solov'ev[b], D. V. Zhilinskii[b], L. K. Shataeva[b], and B. F. Vanyushin[c, d]

[a] *Pavlov Institute of Physiology of the Russian Academy of Sciences, nab. Makarova 6, St. Petersburg, 199034 Russia*

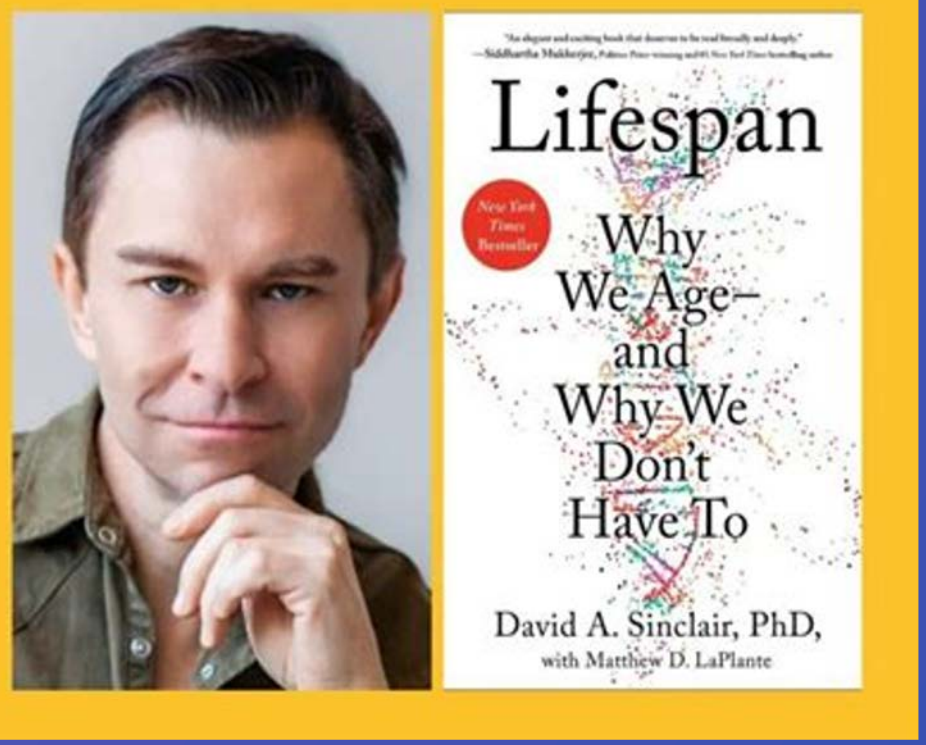
[b] *St. Petersburg Institute of Bioregulation and Gerontology, pr. Dinamo 3, St. Petersburg, 197110 Russia*

[c] *Belozerskii Research Institute of Physico-Chemical Biology*

[d] *Moscow State University, Moscow, 119991 Russia*

Abstract—Endogenous peptides in the cyto- and nucleoplasm are formed upon the specific proteasomal degradation of nuclear proteins. These peptides are formed by short blocks of amino-acid residues with charged side groups and therefore a high local concentration of electrostatic charge of either sign is characteristic of them. These peptides are capable of complementary binding to certain short nucleotide sequences in DNA strands. This binding can cause a significant weakening of the interstrand bonds in the double helix of DNA and therefore stimulate the splitting of strands, which is necessary for gene transcription and replication. Aging is always accompanied by a decrease in the degree of genome methylation. The age-related decrease of the degree of methylation of nucleotide repeat sequences in the genome promotes the site-specific binding of short peptides to DNA, which hinders the hydrolysis of non-methylated DNA fragments by endonucleases. The available experimental data on the peculiarities of binding to methylated DNA are indicative of the involvement of short peptides in the epigenetic regulation of aging processes.

"These peptides are capable of complementary binding to certain short nucleotide sequences in DNA strands...The available experimental data on the peculiarities of binding to methylated DNA are indicative of the involvement of short peptides in the epigenetic regulation of aging processes" (2012).



"I have little doubt that cellular reprogramming is the next frontier in aging research. One day, it might be possible to reprogram cells via pills that stimulate the activity of the OSK factors or the TETs. This may be simpler than it sounds. Natural molecules stimulate the TET enzymes…"

-David Sinclair, PhD.

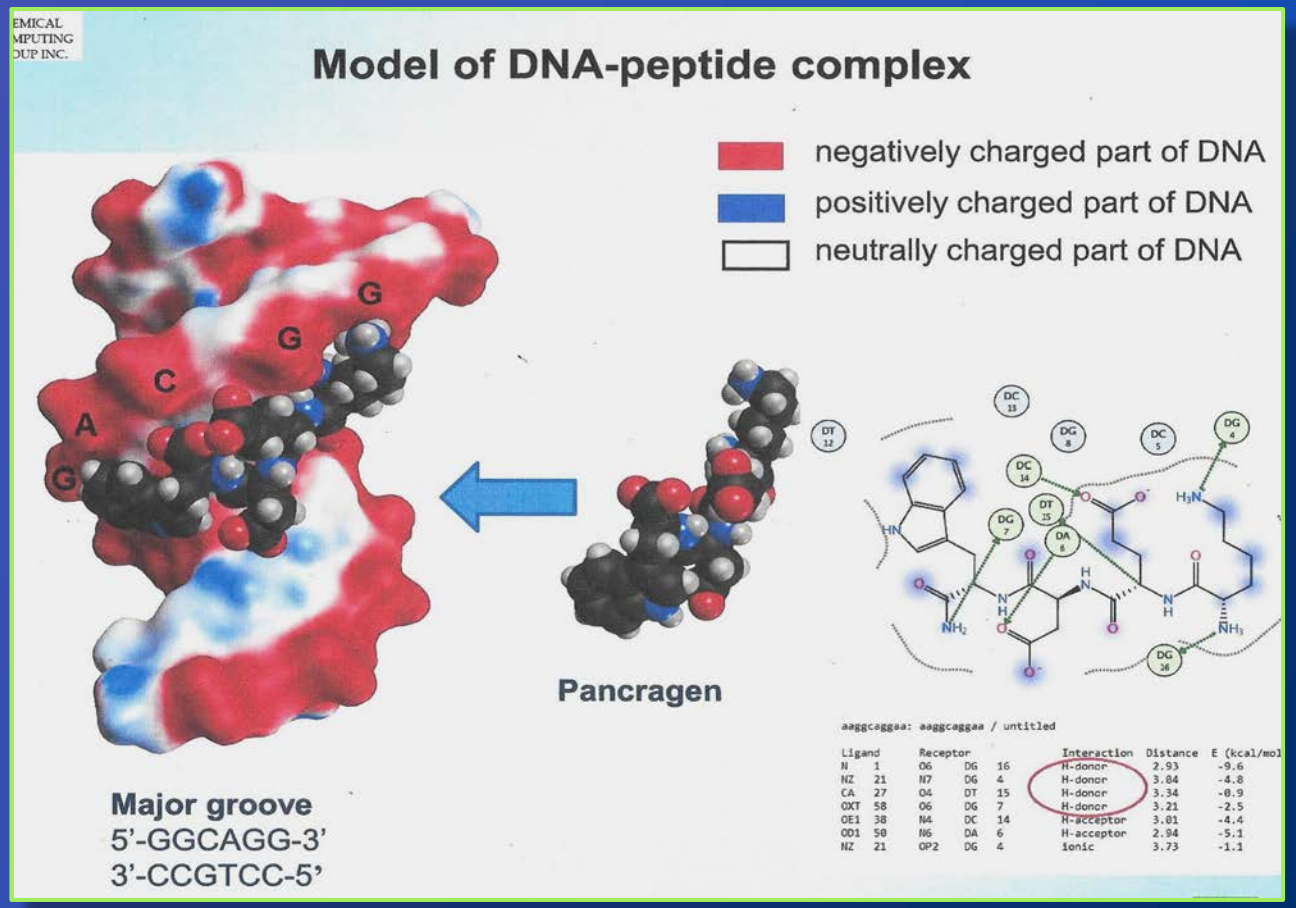
Molecules. 2021 Nov

Peptide Regulation of Gene Expression: A Systematic Review

Vladimir Khatskelevich Khavinson,1,2 Irina Grigor'evna Popovich,1 Natalia Sergeevna Linkova,1,* Ekaterina Sergeevna Mironova,1 and Anastasiia Romanovna Ilina1

Abstract

Peptides are characterized by their wide range of biological activity: they regulate functions of the endocrine, nervous, and immune systems. The mechanism of such action of peptides involves their ability to regulate gene expression and protein synthesis in plants, microorganisms, insects, birds, rodents, primates, and humans. Short peptides, consisting of 2–7 amino acid residues, can penetrate into the nuclei and nucleoli of cells and interact with the nucleosome, the histone proteins, and both single- and double-stranded DNA. DNA–peptide interactions, including sequence recognition in gene promoters, are important for template-directed synthetic reactions, replication, transcription, and reparation. Peptides can regulate the status of DNA methylation, which is an epigenetic mechanism for the activation or repression of genes.



Biochemistry (Mosc). 2015 Mar;80(3):310-22. doi: 10.1134/S0006297915030062.

Epigenetic mechanisms of peptidergic regulation of gene expression during aging of human cells.

Ashapkin VV[1], Linkova NS, Khavinson VKh, Vanyushin BF.

Belozersky Institute of Physico-Chemical Biology, Lomonosov Moscow State University, Moscow, 119992, Russia. ashapkin@genebee.msu.ru.

Khavinson, V.K., Solov'ev, A.Y., Zhilinskii, D.V. *et al.* Epigenetic aspects of peptide-mediated regulation of aging. *Adv Gerontol* **2**, 277–286 (2012).

Epigenetic Aspects of Peptide Mediated Regulation of Aging

a Pavlov Institute of Physiology of the Russian Academy of Sciences, nab. Makarova 6, St. Petersburg, 199034 Russia

b St. Petersburg Institute of Bioregulation and Gerontology, pr. Dinamo 3, St. Petersburg, 197110 Russia

c Belozerskii Research Institute of Physico_Chemical Biology

d Moscow State University, Moscow, 119991 Russia

Short Peptides Regulate Gene Expression. Bull. Exp. Biol. Med. 2016;

Peptides were found to participate in gene-expression regulation and protein synthesis, according to the peptide cascade model, modulating multiple physiological functions of the organism [22,23,24,25]. These processes are aimed at preventing DNA damage, or its eventual suppression, and stimulating its repair, with the aim of restoring cellular homeostasis.

22. Khavinson V.K., Lin'kova N.S., Tarnovskaya S.I. Short Peptides Regulate Gene Expression. Bull. Exp. Biol. Med. 2016;162:288–292. doi: 10.1007/s10517-016-3596-7. [PubMed] [CrossRef] [Google Scholar]

23. Ashapkin V., Khavinson V., Shilovsky G., Linkova N., Vanuyshin B. Gene expression in human mesenchymal stem cell aging cultures: Modulation by short peptides. Mol. Biol. Rep. 2020;47:4323–4329. doi: 10.1007/s11033-020-05506-3. [PubMed] [CrossRef] [Google Scholar]

24. Khavinson V., Diomede F., Mironova E., Linkova N., Trofimova S., Trubiani O., Caputi S., Sinjari B. AEDG Peptide (Epitalon) Stimulates Gene Expression and Protein Synthesis during Neurogenesis: Possible Epigenetic Mechanism. Molecules. 2020;25:609. doi: 10.3390/molecules25030609. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

25. Ashapkin V.V., Linkova N.S., Khavinson V.K., Vanyushin B.F. Epigenetic mechanisms of peptidergic regulation of gene expression during aging of human cells. Biochemistry. 2015;80:310–322. doi: 10.1134/S0006297915030062. [PubMed] [CrossRef] [Google Scholar]

What is Epigenetics?

Every cell in your body has the exact same DNA sequence.

So how do your cells act so differently? It is due to what DNA expression is turned on and turn off.

Lifestyle and environment influence gene expression

Diet, stress, exercise, sleep, climate, nutraceuticals, peptides, etc.

The frequency or timing of instructions generates proteins

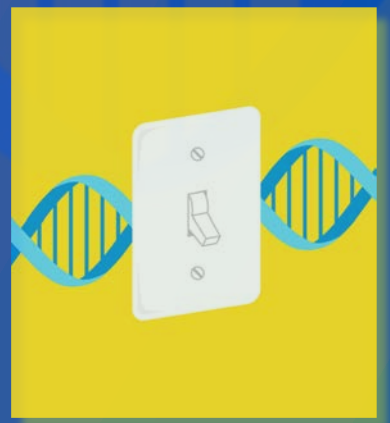
Epigenetic changes turn genes “on” and “off”

Epigenetic changes are reversible

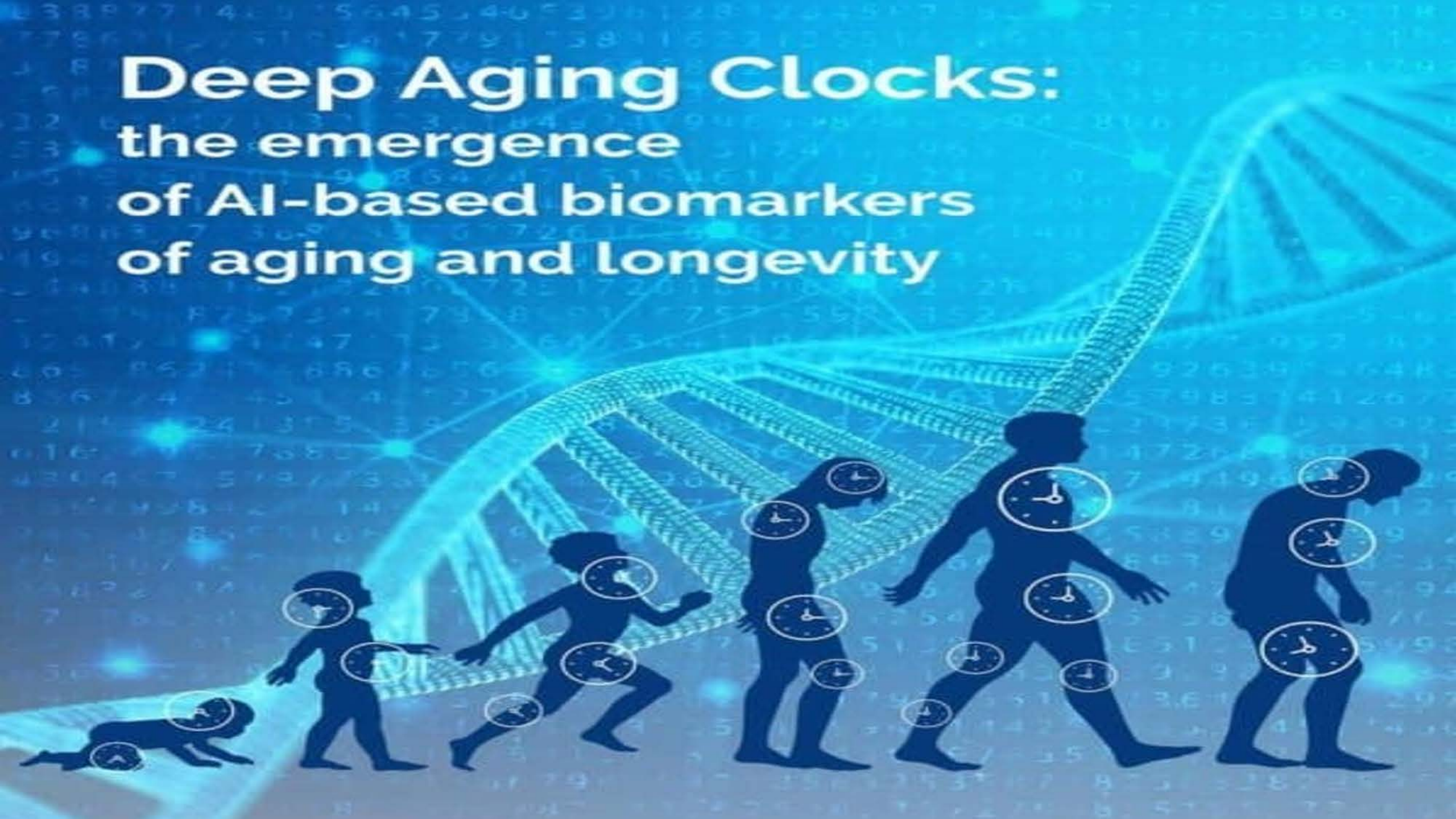
Epigenetics do not alter DNA sequence but how DNA is “read”

Proteins influence or change gene expression

Gene status affects biological age



Deep Aging Clocks:
the emergence of AI-based biomarkers of aging and longevity



Dr. Steven Horvath

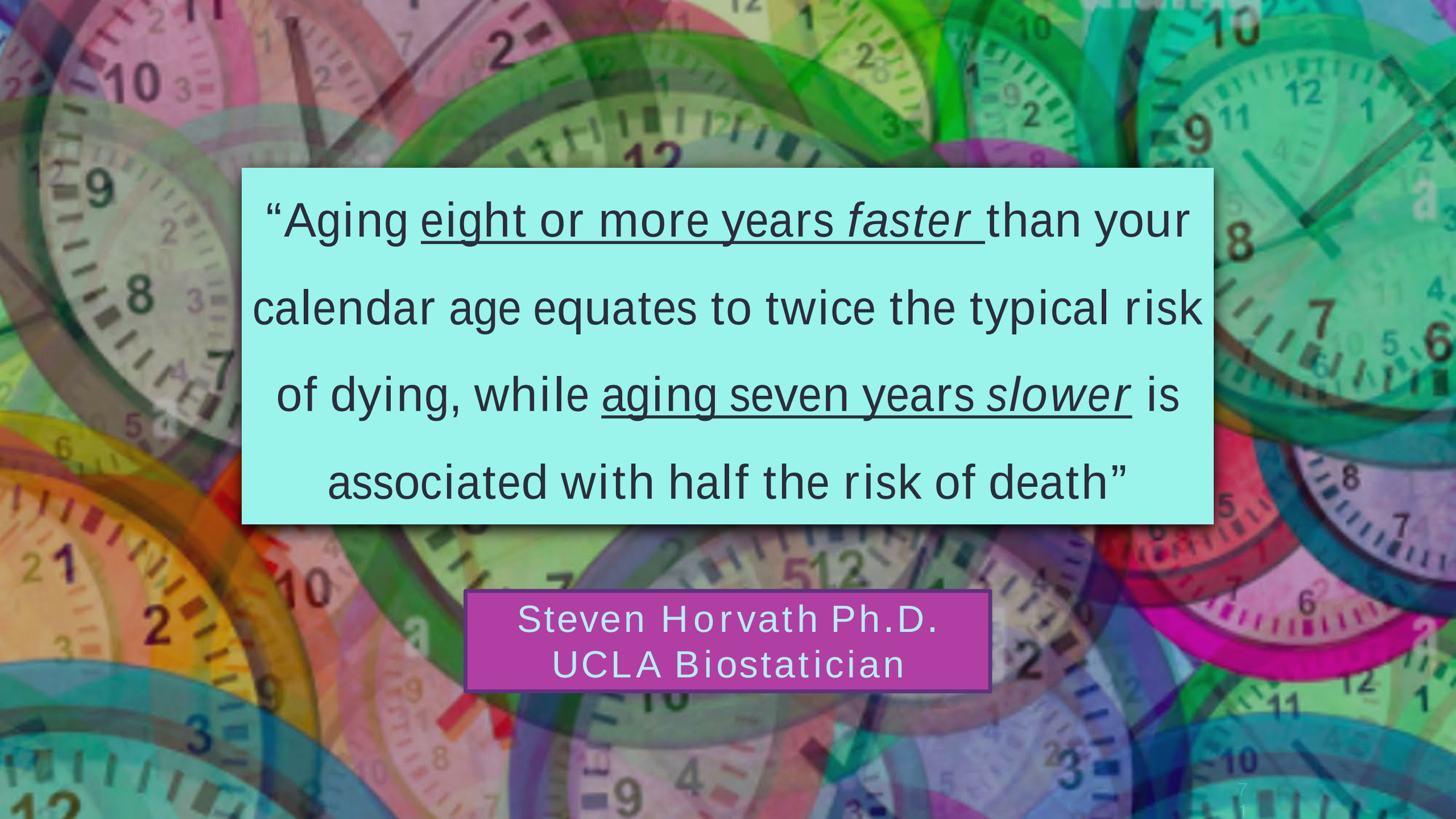
UCLA geneticist Steve Horvath led a team of 65 scientists in seven countries to record age-related changes to human DNA, calculate the biological age of blood and estimate a person's lifespan. A higher biological age — regardless of chronological age — consistently predicted an earlier death.

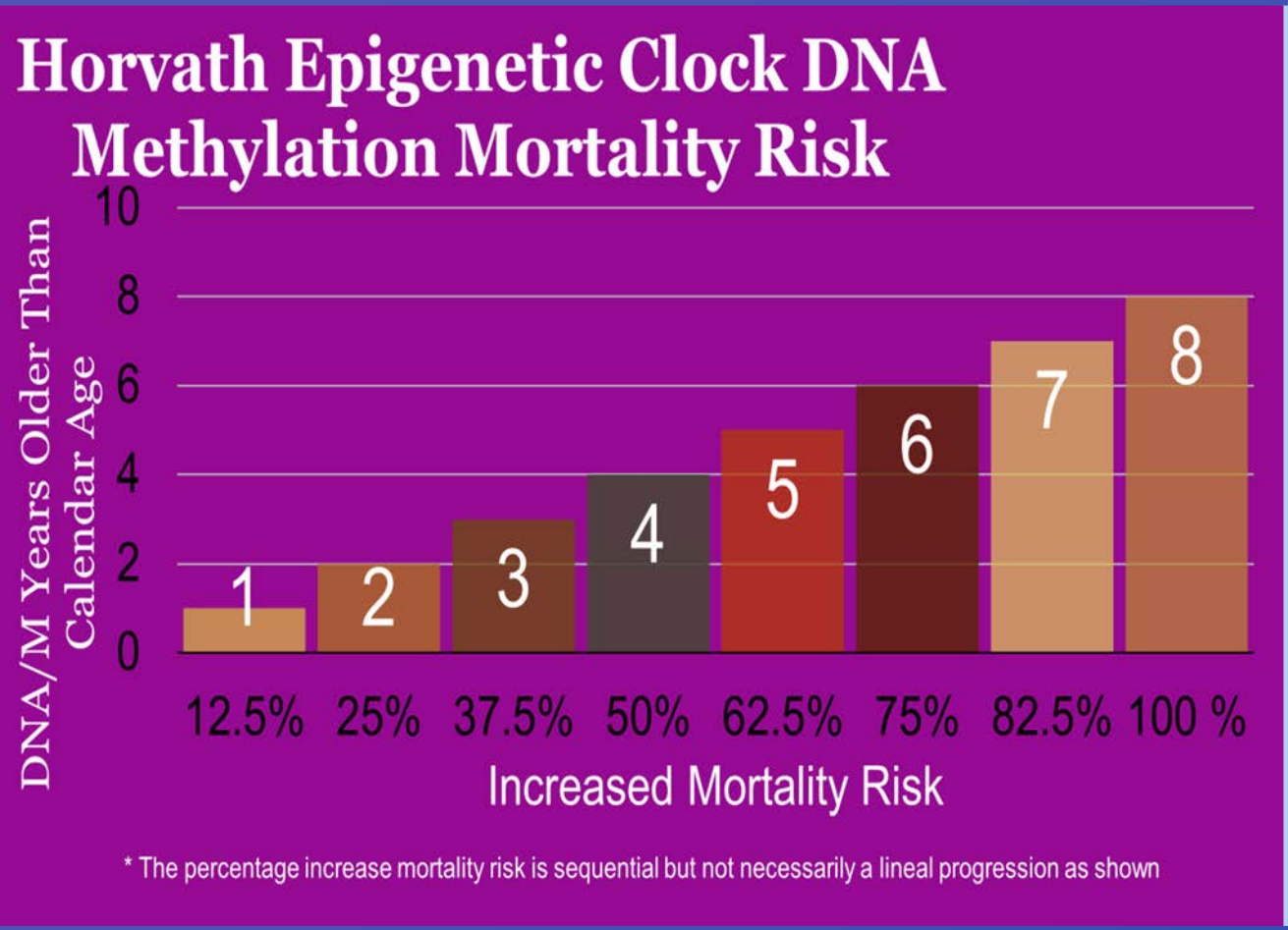
Each one-year increase in epigenetic age above C age=

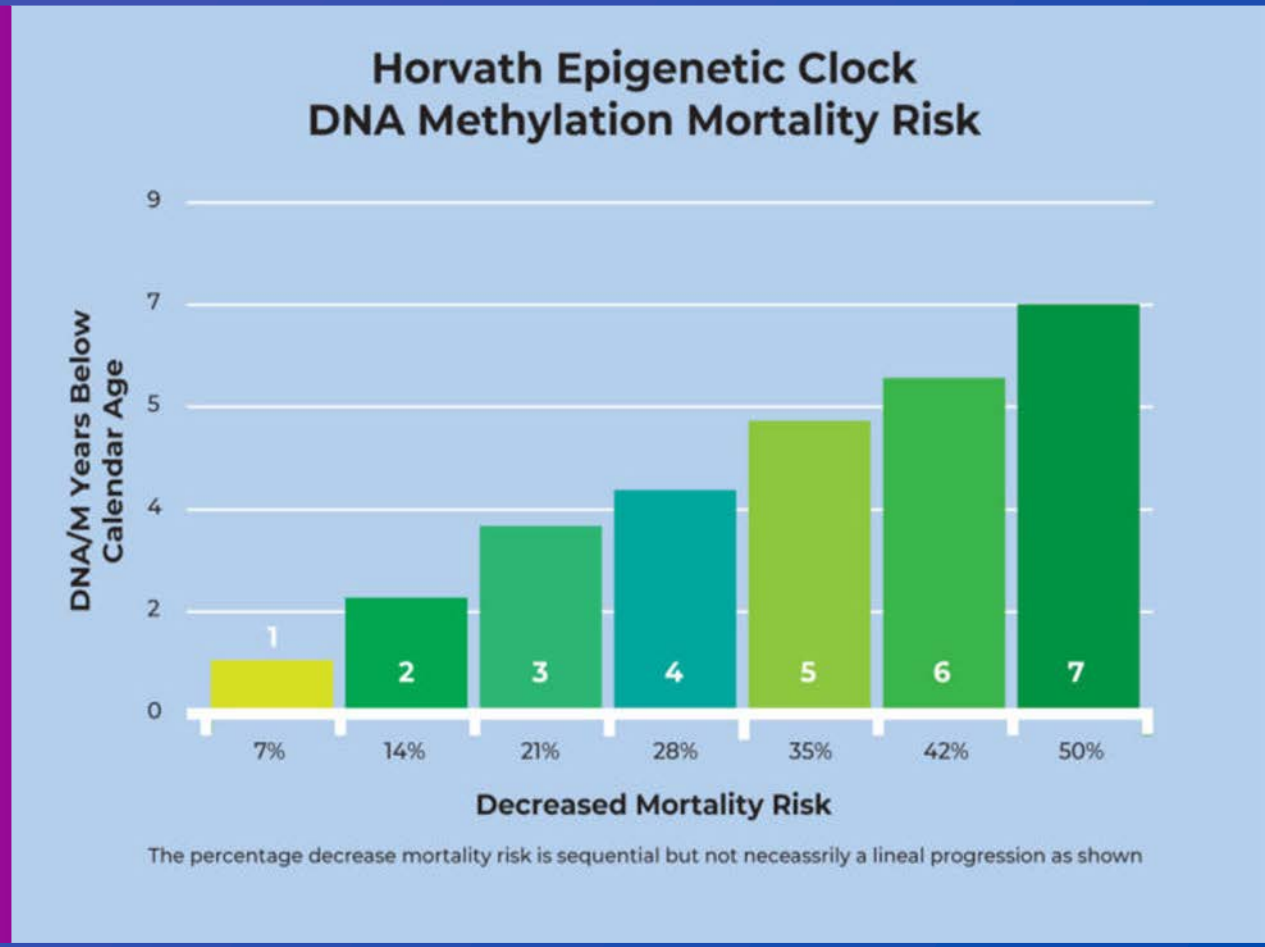
6% increased risk of developing cancer within 3 years

and

17% increased risk of dying of cancer in the next 5 years.


"Aging eight or more years *faster* than your calendar age equates to twice the typical risk of dying, while aging seven years *slower* is associated with half the risk of death"
Steven Horvath Ph.D.
UCLA Biostatician





7yrs above "C" age=82.5% increased mortality risk

7yrs below "C" age=50% decreased mortality risk

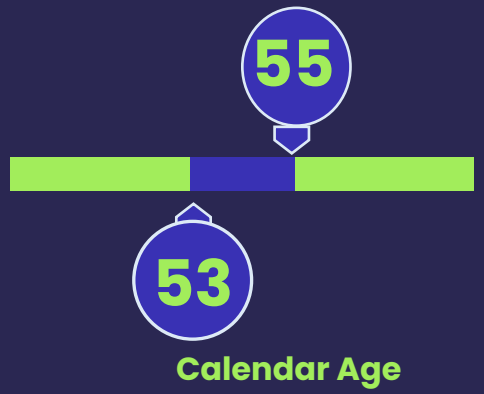
Epigenetic Age Reversal: Daniel

September 2019

Initial Test Report Date

Current Test Results

DNAge®



Calendar Age

DNA Index

Your DNAge index* shows you are at 16 percentile of your age.
It means you are younger than 16% of people at your age.

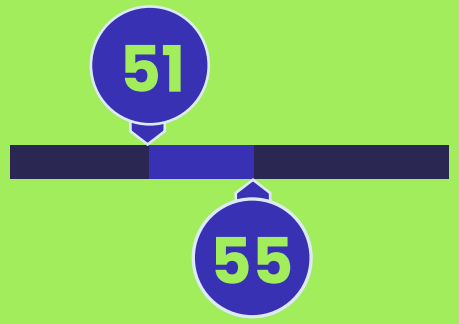
25% increase all-cause mortality risk

February 2021

Final Test Report Date

Current Test Results

DNAge®

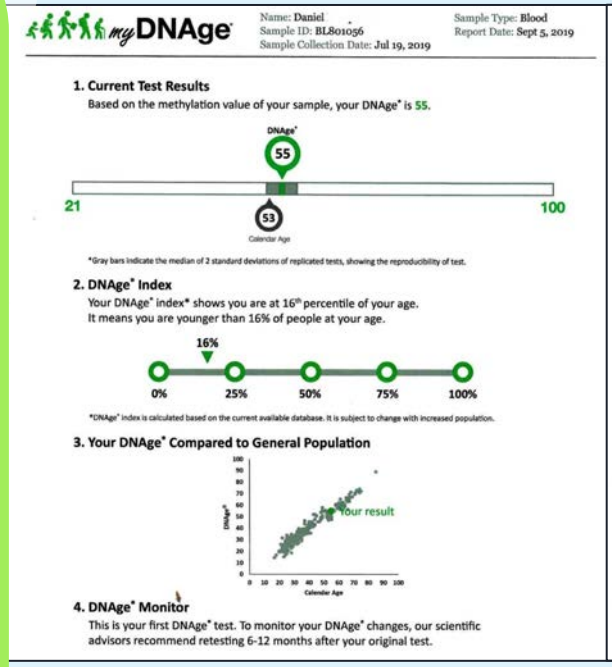


Calendar Age

DNA Index

Your DNAge index* shows you are at 86th percentile of your age. It means you are younger than 86% of people at your age.

28% decreased all-cause mortality risk



myDNAge

Name: Daniel
Sample ID: BL80156
Sample Collection Date: Jul 19, 2019
Sample Type: Blood
Report Date: Sept 5, 2019

1. Current Test Results

Based on the methylation value of your sample, your DNAge* is 55.

DNAge* 55 | 21 | 53 | 100 | Calendar Age

*Gray bars indicate the median of 2 standard deviations of replicated tests, showing the reproducibility of test.

2. DNAge* Index

Your DNAge* index* shows you are at 16th percentile of your age.
It means you are younger than 16% of people at your age.

16% | 0% | 25% | 50% | 75% | 100%

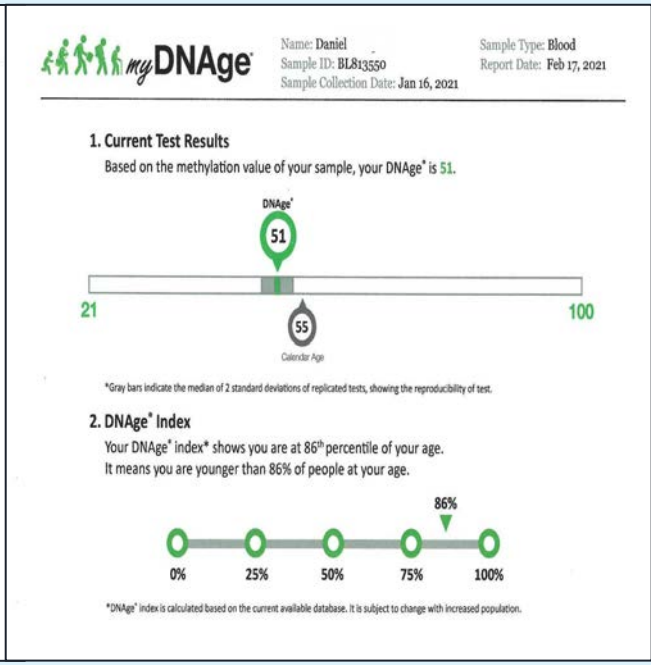
DNAge index is calculated based on the current available database. It is subject to change with increased population.

3. Your DNAge* Compared to General Population

DNAge* | Your result | Calendar Age

4. DNAge* Monitor

This is your first DNAge* test. To monitor your DNAge* changes, our scientific advisors recommend retesting 6-12 months after your original test.



myDNAge

Name: Daniel
Sample ID: BL813550
Sample Collection Date: Jan 16, 2021
Sample Type: Blood
Report Date: Feb 17, 2021

1. Current Test Results

Based on the methylation value of your sample, your DNAge* is 51.

DNAge* 51 | 21 | 55 | 100 | Calendar Age

*Gray bars indicate the median of 2 standard deviations of replicated tests, showing the reproducibility of test.

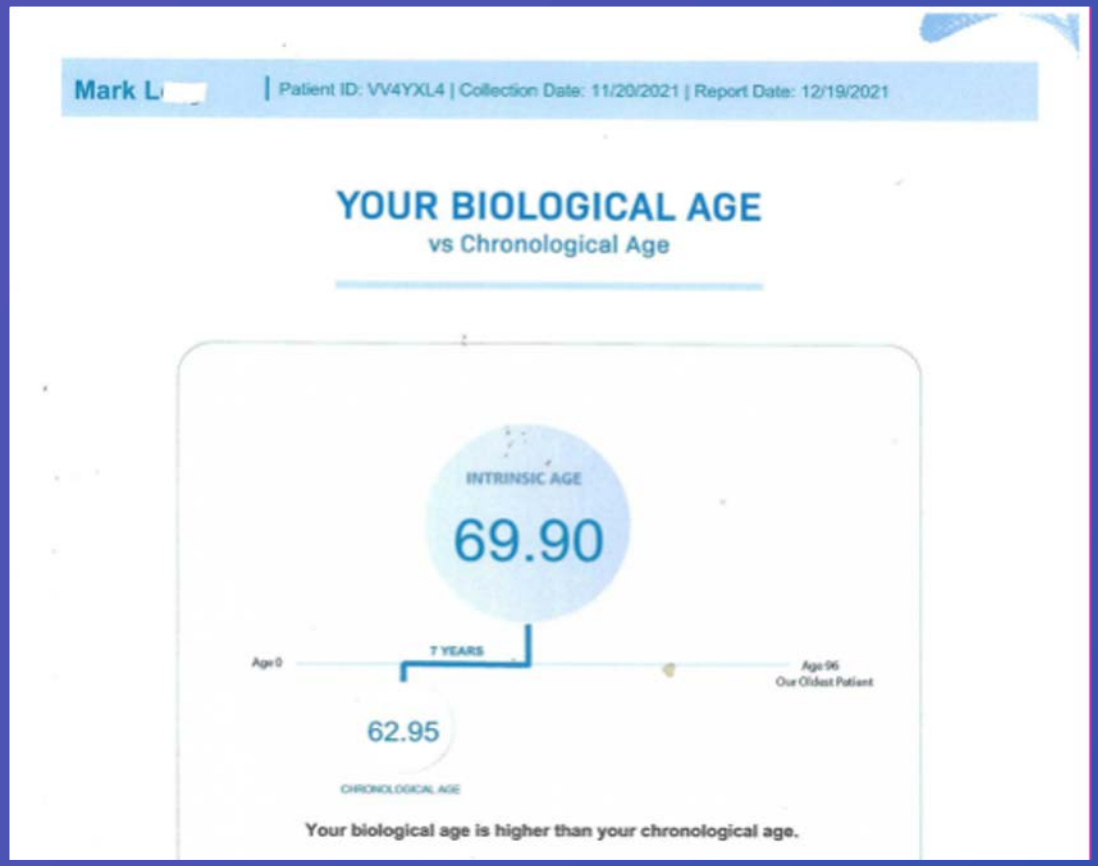
2. DNAge* Index

Your DNAge* index* shows you are at 86th percentile of your age.
It means you are younger than 86% of people at your age.

86% | 0% | 25% | 50% | 75% | 100%

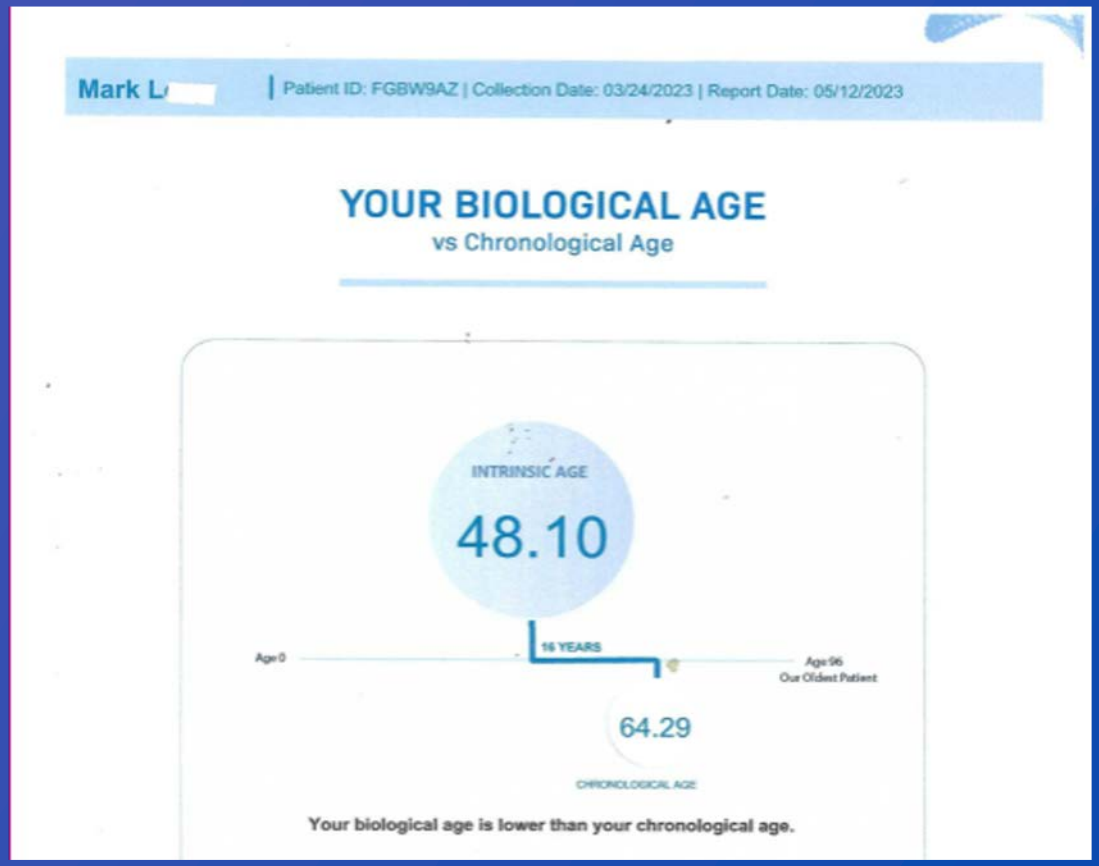
DNAge index is calculated based on the current available database. It is subject to change with increased population.

2021 Baseline Results



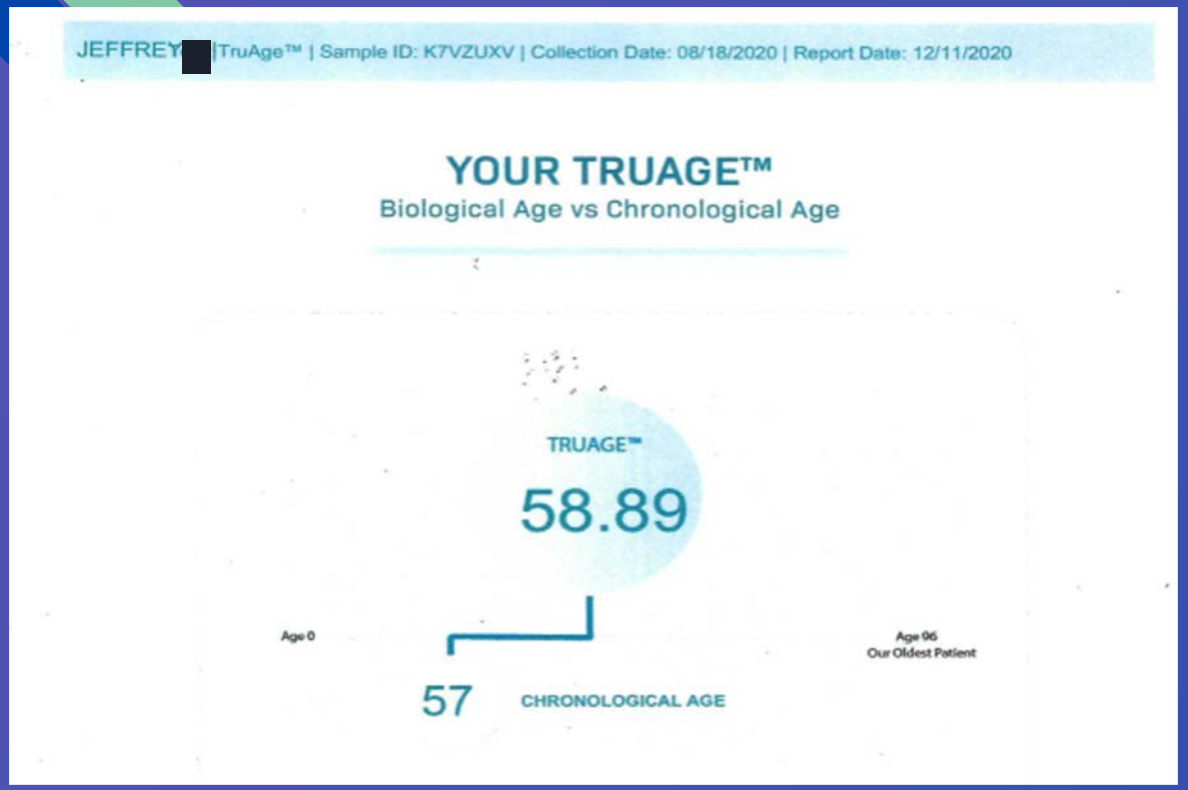
7 yrs older biologically=82.5%
INCREASED all-cause mortality risk

2023 Results



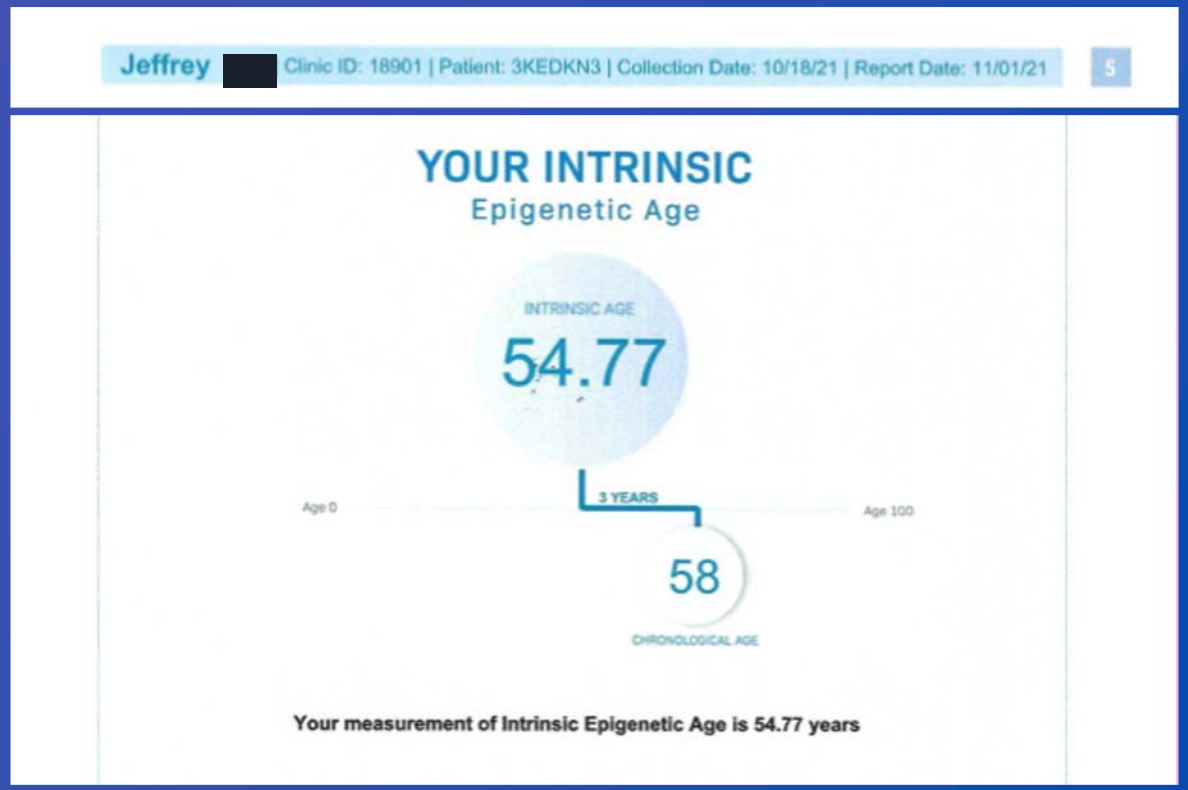
16 yrs younger biologically=114%
DECREASED all-cause mortality risk

2020 Baseline Results



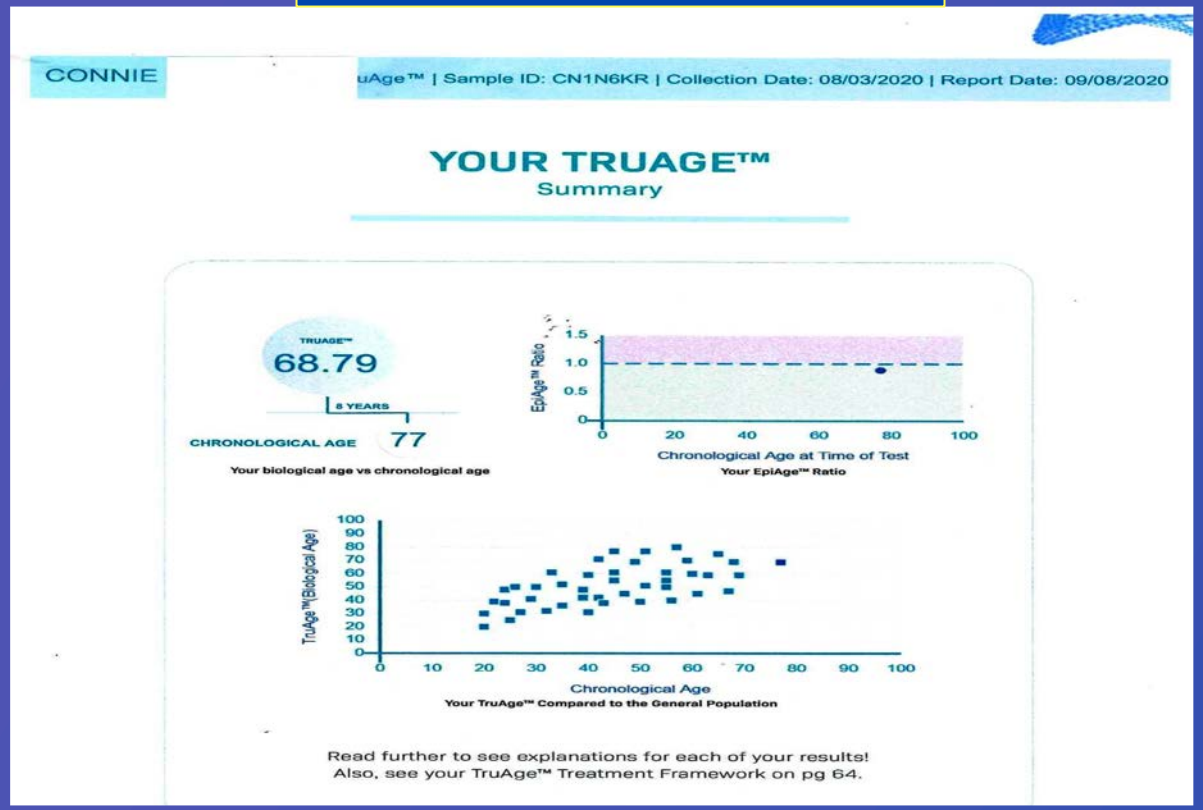
2 yrs above "C" age=25% INCREASED all-cause mortality risk

2021 Results



3 yrs below "C" age=21% DECREASED all-cause mortality risk

2020 Baseline Results


CONNIE

uAge™ | Sample ID: CN1N6KR | Collection Date: 08/03/2020 | Report Date: 09/08/2020

YOUR TRUAGE™
Summary

TRUAGE™ 68.79
8 YEARS
CHRONOLOGICAL AGE 77
Your biological age vs chronological age

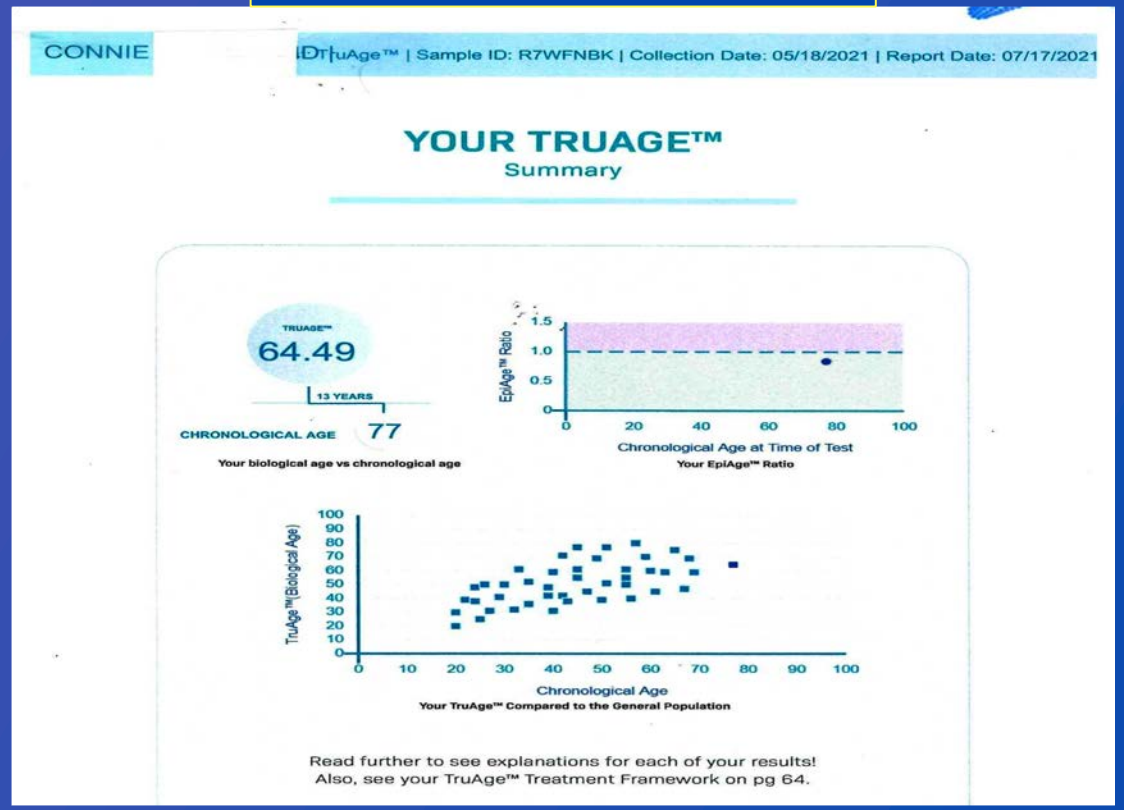
EpiAge™ Ratio
Chronological Age at Time of Test
Your EpiAge™ Ratio

TruAge™ (Biological Age)
Chronological Age
Your TruAge™ Compared to the General Population

Read further to see explanations for each of your results!
Also, see your TruAge™ Treatment Framework on pg 64.

8 yrs below "C" age=55% DECREASED all-cause mortality risk

2021 Results


CONNIE

IDTruAge™ | Sample ID: R7WFNBK | Collection Date: 05/18/2021 | Report Date: 07/17/2021

YOUR TRUAGE™
Summary

TRUAGE™ 64.49
13 YEARS
CHRONOLOGICAL AGE 77
Your biological age vs chronological age

EpiAge™ Ratio
Chronological Age at Time of Test
Your EpiAge™ Ratio

TruAge™ (Biological Age)
Chronological Age
Your TruAge™ Compared to the General Population

Read further to see explanations for each of your results!
Also, see your TruAge™ Treatment Framework on pg 64.

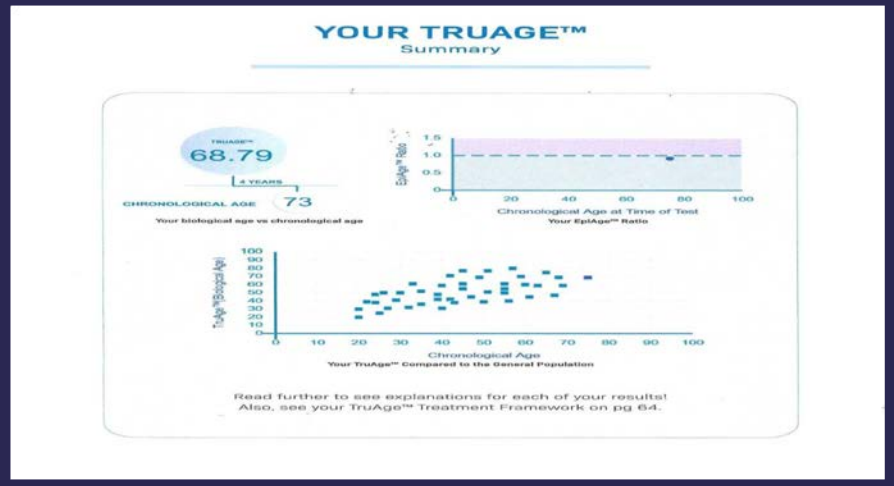
12 yrs below "C" age=85% DECREASED all-cause mortality risk

YOUR EXTRINSIC
Epigenetic Age

Parameters	Reference Range	Percentage Values (%)
Bcell	20% to 40%	1.89%
CD4T		24.16%
CD8T		3.32%
NK		4.63%
Lymphocyte Total		34.00%
Neutrophils	40% to 60%	60.82%
Monocytes	2% to 8%	6.23%
Eosinophils	1% to 4%	0.00%
CD4T/CD8T Cell Ratio	1 to 4	7.27

Extrinsic Age= Epigenetic Age of the Immune System

Bill Lawrence Results



Report Date
09/08/2020

68.8
TRUAGE™

73
Chronological Age

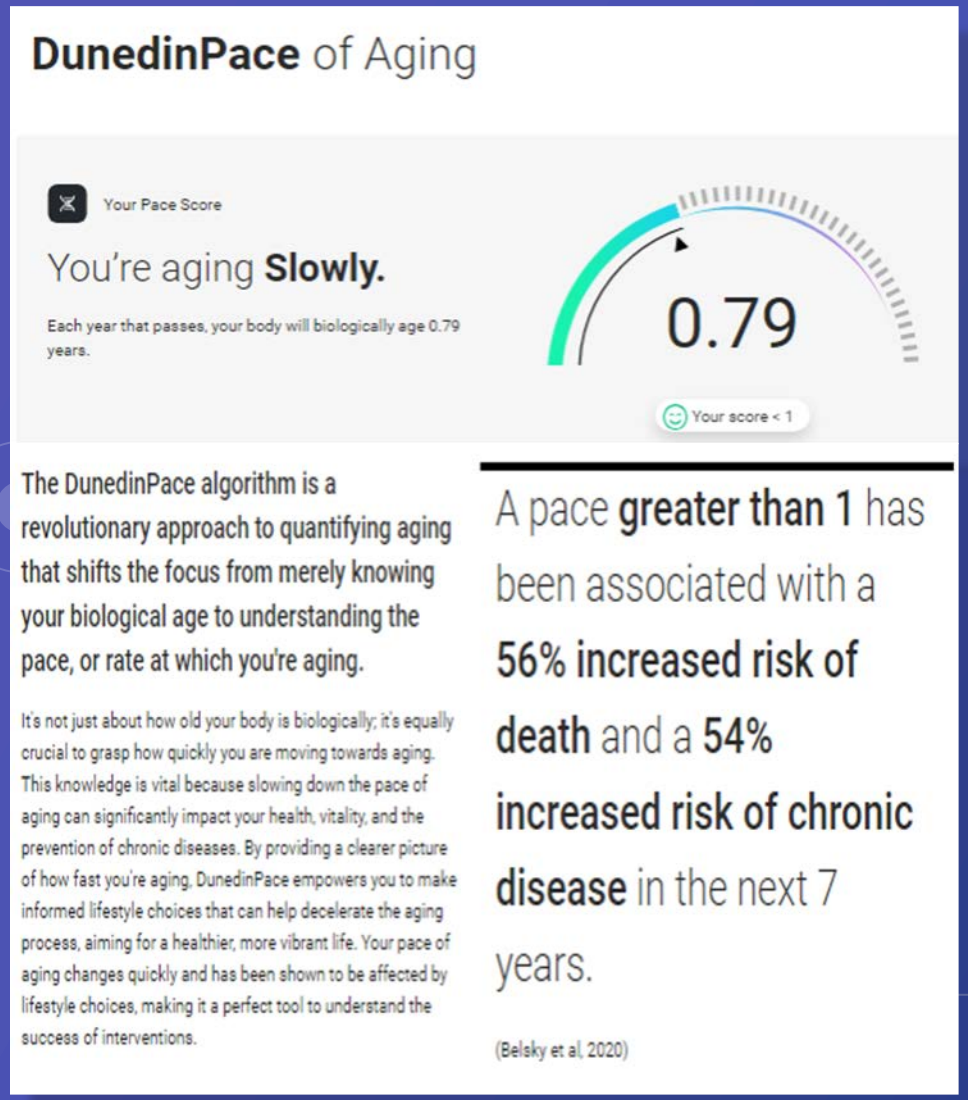
FOUR YEARS LESS THAN "C" AGE
*28% REDUCED RISK OF ALL CAUSE MORTALITY

Report Date
05/07/2022

64.9
TRUAGE™

75
Chronological Age

TEN YEARS LESS THAN "C" AGE
*70% REDUCED RISK OF ALL CAUSE MORTALITY



DUNEDIN PACE VALUE

TruDiagnostic™ Results

Pace of aging compares biological aging to 1 standard calendar year. For example, if your results are '*0.8*', that means your body is currently aging 0.8 years for each year that passes.

PEPTIDE LONGEVITY PROGRAM
LANCE XXXXX
EPIGENETIC AGE
COMPARATIVE RESULTS
2023-2024

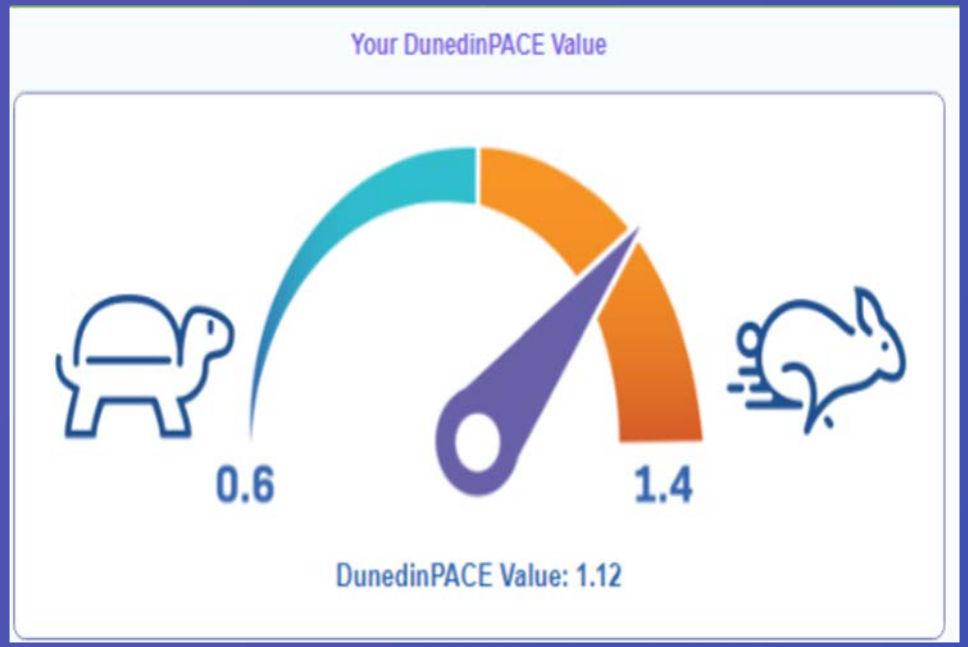
2023 RESULTS

Chronological age	50 (49.67)	
DNA/Methylation age (Intrinsic)	59 (58.68)	9 years older than C age
DNA Extrinsic (Immune Age)	40 (40.13)	10 years less than C age
Pace of Aging	1.12%	Aging 12% faster

2024 RESULTS

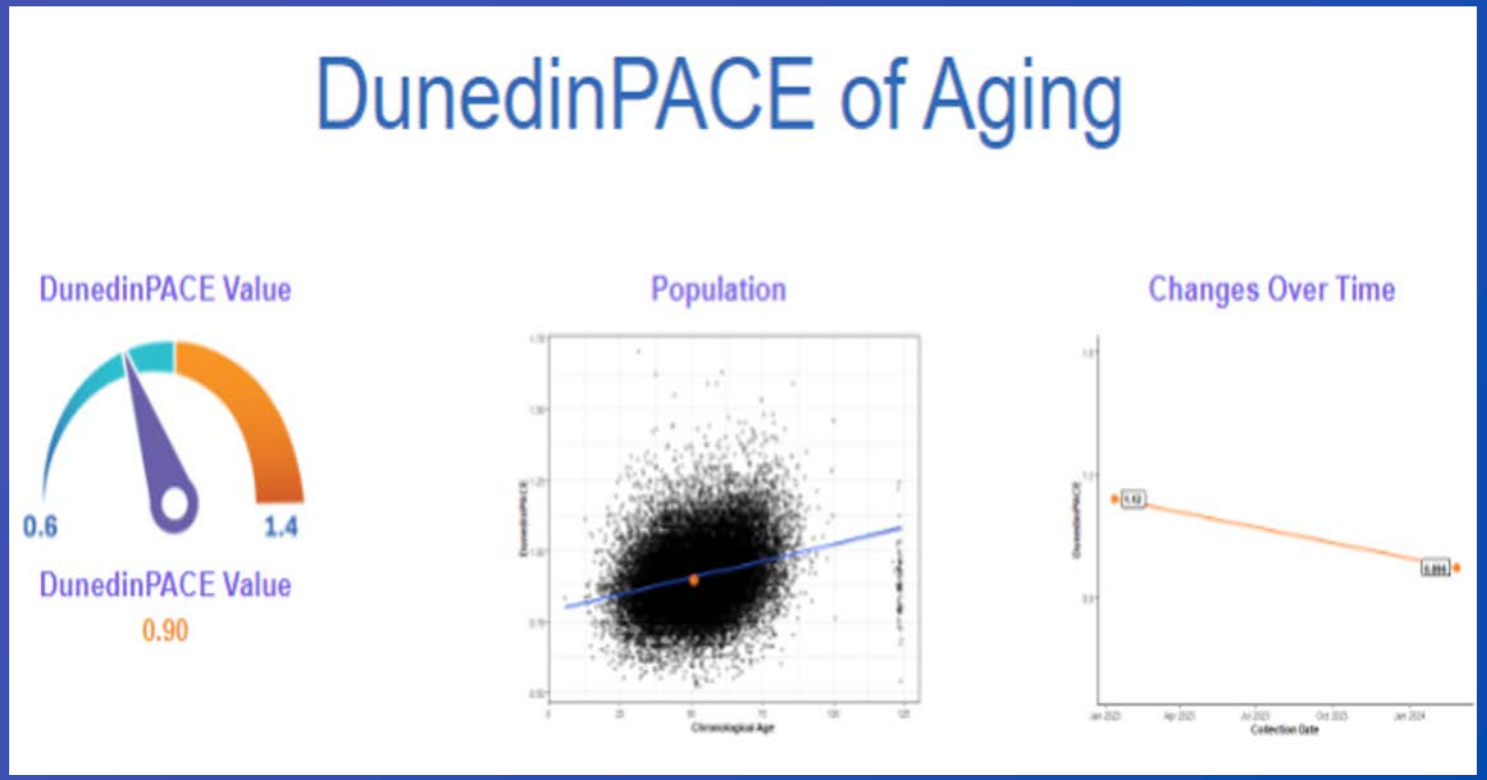
Chronological age	51 (50.80)	
DNA/Methylation age (Intrinsic)	54 (54.43)	3 years older than C age
DNA Extrinsic (Immune Age)	33 (32.95)	18 years less than C age
Pace of Aging	0.90%	Aging 10% slower

Pace of Aging Before



Aging 0.12% FASTER than others his age

Pace of Aging After

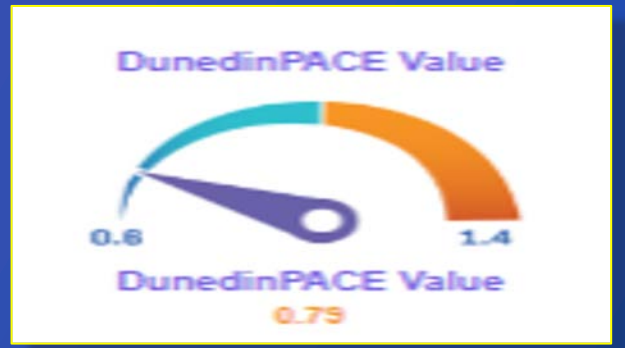


Aging .10% SLOWER than others his age

*Aging above 1.00=54% increased risk of chronic disease and 56% increased risk all-cause mortality over next 7 years

Bill Lawrence

Dunedin PACE of Aging Results



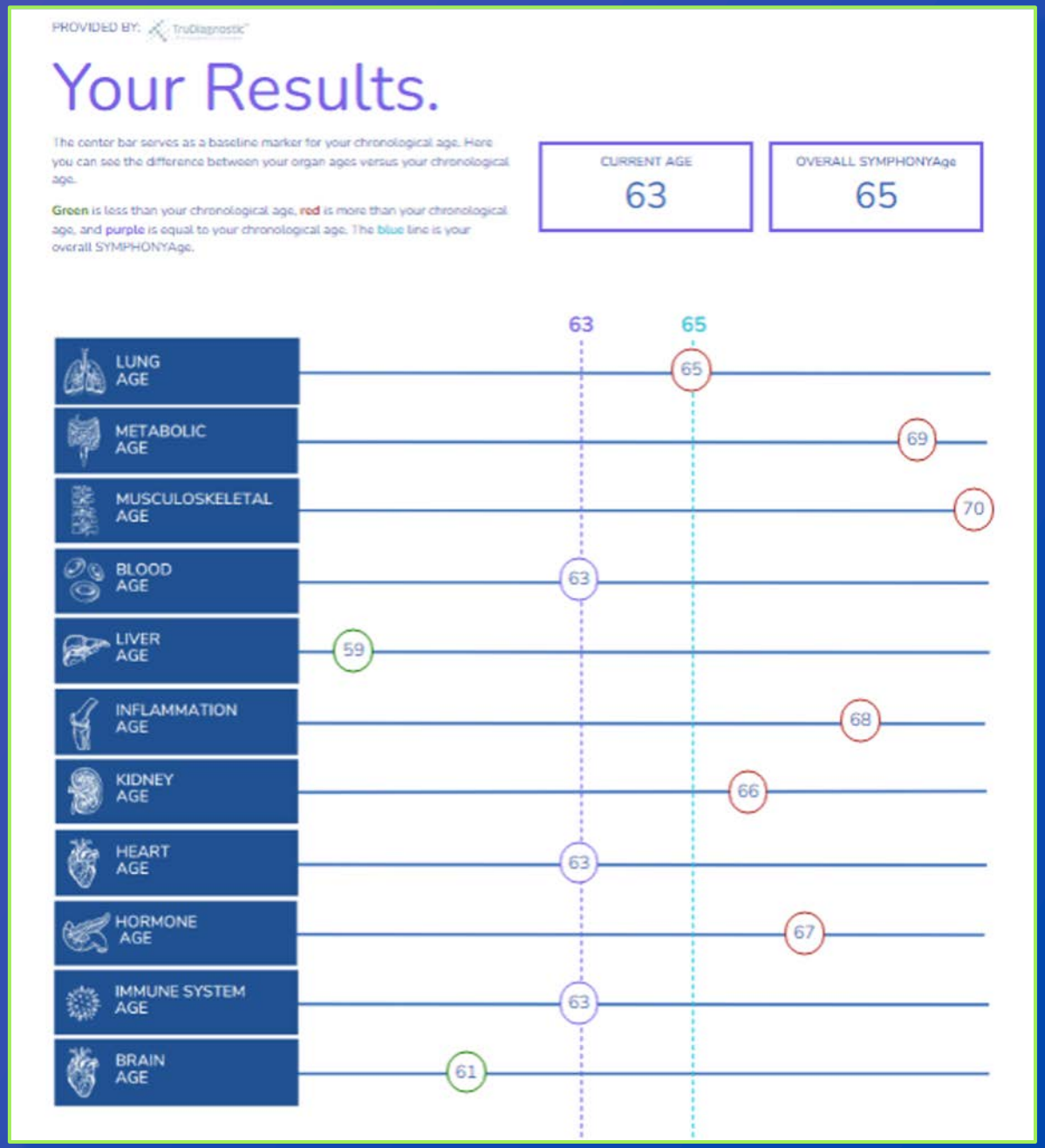
2020	2021	2022	2024
0.92%	0.91%	0.89%	0.79%

TRUAGE BY TRUDIAGNOSTIC

SYMPHONYAge

SYSTEM METHYLATION PROXY OF HETEROGENOUS ORGAN YEARS

In partnership with Yale University,
this report approximates the biological age of eleven organs and systems while also calculating disease-related risks.



PLP Study Results

(124 participants)

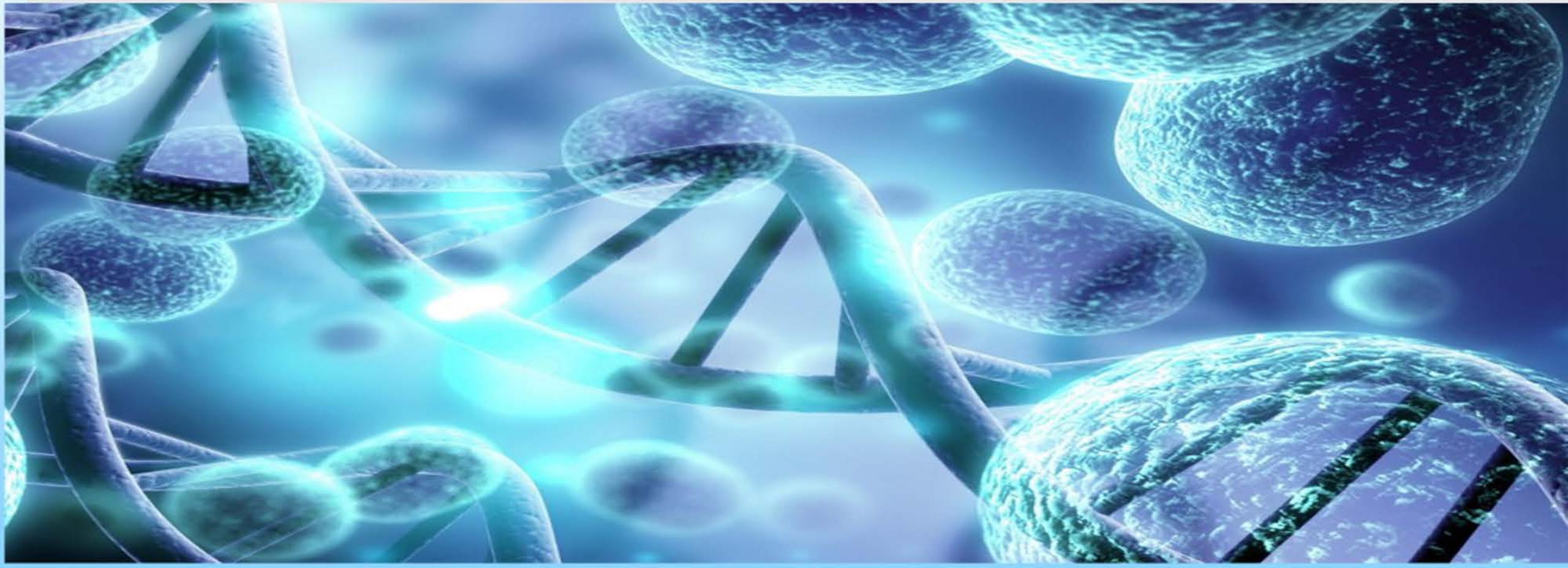
Average **Decrease** in
Epigenetic Age

4.67 IN TWO YEARS

Represents reduction in all-cause
mortality risk

56%

PEPTIDE BIOREGULATORS



MORTALITY REDUCTION

ST. PETERSBURG INSTITUTE OF BIOREGULATION AND GERONTOLOGY

Clinical Trial Neuro Endocrinol Lett. 2003 Jun-Aug;24(3-4):233-40.

PMID: 14523363

Peptides of pineal gland and thymus prolong human life

Vladimir Kh Khavinson [1], Vyacheslav G Morozov

Abstract

Objectives and design: Researchers of the St. Petersburg Institute of Bioregulation and Gerontology of the North-Western Branch of the Russian Academy of Medical Sciences and the Institute of Gerontology of the Ukrainian Academy of Medical Sciences (Kiev) clinically assessed the geroprotective effects of thymic (Thymalin) and pineal (Epithalamin) peptide bioregulators in 266 elderly and older persons during 6-8 years. The bioregulators were applied for the first 2-3 years of observation.

Results: The obtained results convincingly showed the ability of the bioregulators to normalize the basic functions of the human organism, i.e. to improve the indices of cardiovascular, endocrine, immune and nervous systems, homeostasis and metabolism. Homeostasis restoration was accompanied by a 2.0-2.4-fold decrease in acute respiratory disease incidence, reduced incidence of the clinical manifestations of ischemic heart disease, hypertension disease, deforming osteoarthrosis and osteoporosis as compared to the control. Such a significant improvement in the health state of the peptide-treated patients correlated with decreased mortality rate during observation: 2.0-2.1-fold in the Thymalin-treated group; 1.6-1.8-fold in the Epithalamin-treated group; 2.5-fold in the patients treated with Thymalin plus Epithalamin as compared to the control. A separate group of patients was treated with Thymalin in combination with Epithalamin annually for 6 years and their mortality rate decreased 4.1 times as compared to the control.

- 266 elderly/older persons
- Duration: 6-8 yrs
- 2-6 yrs of peptide supplementation

Results:

- ↓ ischemic heart disease
- ↓ hypertension
- ↓ osteoporosis

Decreased Mortality Rates

- 2.0-2.1x in Thylamin group
- 1.6-1.8x in Epithalamin group
- 2.5x for both Thylamin + Epithalamin group
- 4.1x for Thylamin+Epithalamin group treated for 6 yrs

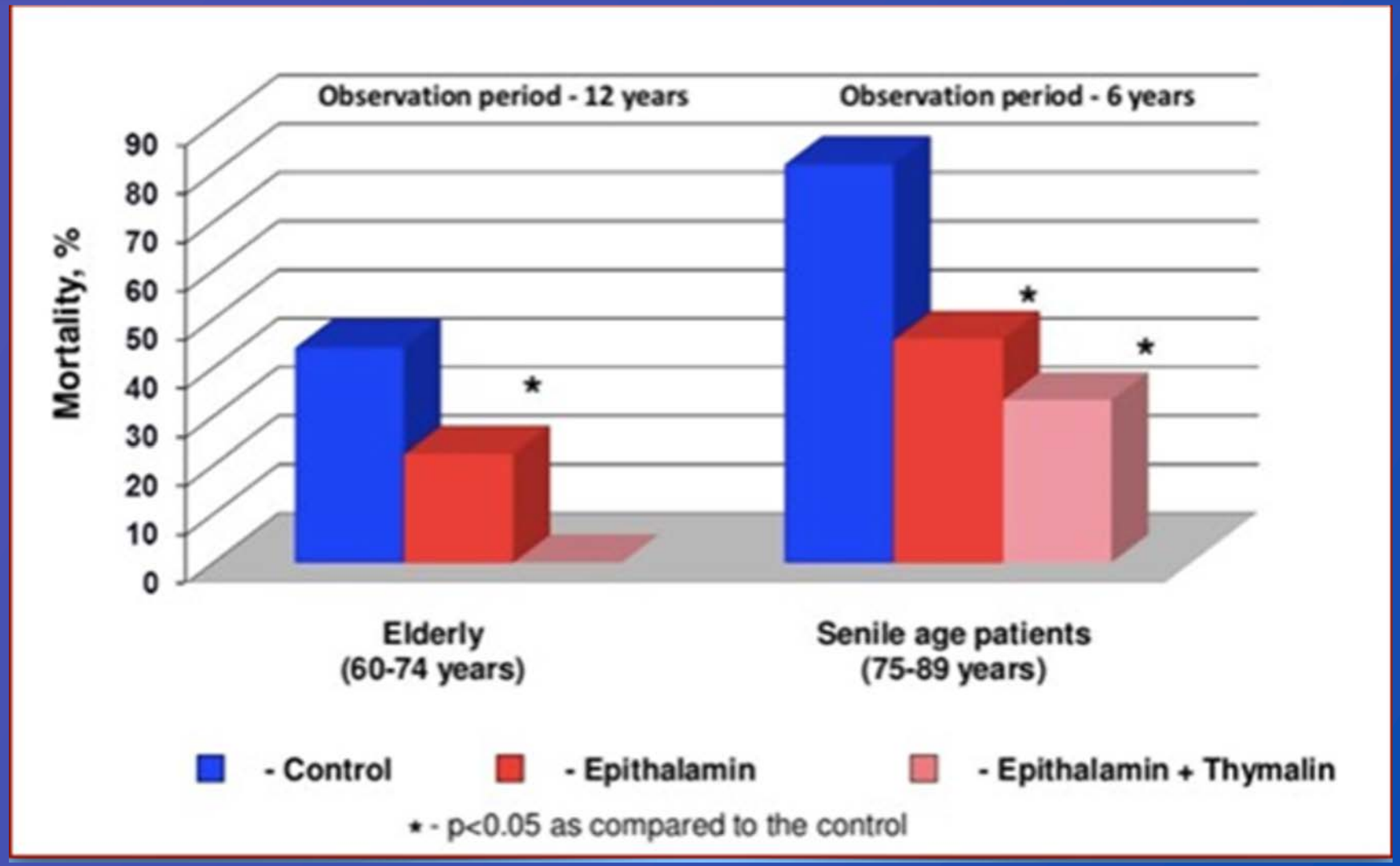
Mortality Reduction in Elderly People (60-74 Years)

Group of patients	Indices	Control (polyvitamins)	Administration **pineal gland peptide**	Administration thymus and pineal complex peptides
Elderly people (60-74 years)	Initial mean age, years	**69.3** ± 2.2	**71.1** ± 1.4	*No studies*
	Mortality rate in the course of 8 years, %	**13.6**	**8.5***	
	Mortality rate in the course of 12 years, %	**44.1**	**22.3***	
Old people (75-89 years)	Initial mean age, years	**80.2** ± 1.6	**81.5** ± 2.1	**82.1** ± 2.3
	Mortality rate in the course of 6 years, %	**81.8**	**45.8***	**33.3***

Mortality Reduction in Old People (75-89 Years)

Group of patients	Indices	Control (polyvitamins)	Administration pineal gland peptide	Administration **thymus and pineal complex** peptides
Elderly people (60-74 years)	Initial mean age, years	**69.3** ± 2.2	**71.1** ± 1.4	*No studies*
	Mortality rate in the course of 8 years, %	**13.6**	**8.5***	
	Mortality rate in the course of 12 years, %	**44.1**	**22.3***	
Old people (75-89 years)	Initial mean age, years	**80.2** ± 1.6	**81.5** ± 2.1	**82.1** ± 2.3
	Mortality rate in the course of 6 years, %	**81.8**	**45.8***	**33.3**

The Influence of Peptide Bioregulators on Mortality in Elderly and Senile Age Patients



Peptide Bioregulator Morbidity Study

Main Group

11,192

Employees

Received a complex of 6 peptide bioregulators to improve the functional state of immune system, brain, blood vessels, bronchi, liver, cartilage tissue (in capsules for oral administration).

Control Group

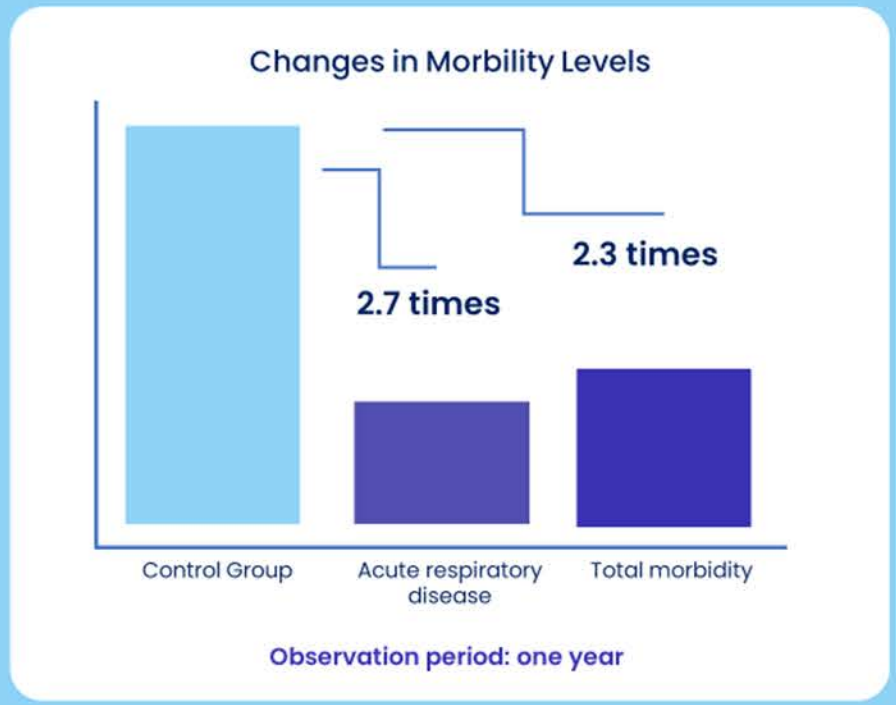
3,000

Employees

Received multivitamins for 30 days (for oral administration).

Gazprom Study

The influence of peptide bioregulators on morbidity of employees of «Gazprom» under the influence of adverse factors



Bull Exp Biol Med. 2004 May;137(5):503-6.

Peptide promotes overcoming of the division limit in human somatic cell.

Khavinson VKh[1], Bondarev IE, Butyugov AA, Smirnova TD.

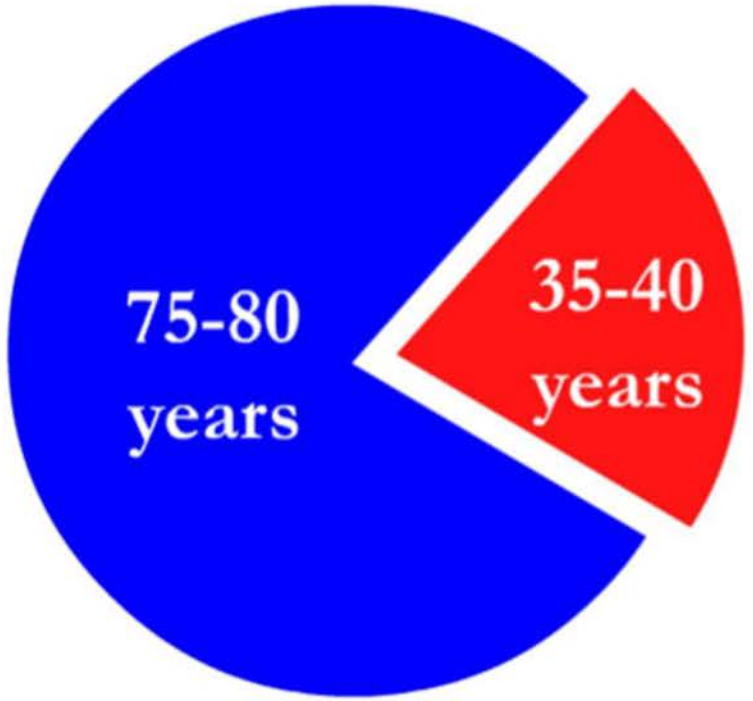
Author information

Abstract

We previously showed that treatment of normal human diploid cells with Epithalon (Ala-Glu-Asp-Gly) induced expression of telomerase catalytic subunit, its enzymatic activity, and elongation of telomeres. Here we studied the effect of this peptide on proliferative potential of human fetal fibroblasts. Primary pulmonary fibroblasts derived from a 24-week fetus lost the proliferative potential at the 34th passage. The mean size of telomeres in these cells was appreciably lower than during early passages (passage 10). Addition of Epithalon to aging cells in culture induced elongation of telomeres to the size comparable to their length during early passages. Peptide-treated cells with elongated telomeres made 10 extra divisions (44 passages) in comparison with the control and continued dividing. Hence, Epithalon prolonged the vital cycle of normal human cells due to overcoming the Heyflick limit.

"Peptide-treated cells with elongated telomeres made 10 extra divisions (44 passages) in comparison with the control and continued dividing. Hence, Epithalon prolonged the vital cycle of normal human cells due to overcoming the Hayflick limit."

Specific limit of human lifespan - 110-120 years



CHANGES IN THE EXPRESSION AND STRUCTURE OF GENES

DISTURBANCES:
- biorhythms
- water
- food

ADVERSE FACTORS:
- stress
- environmental factors
- radiation

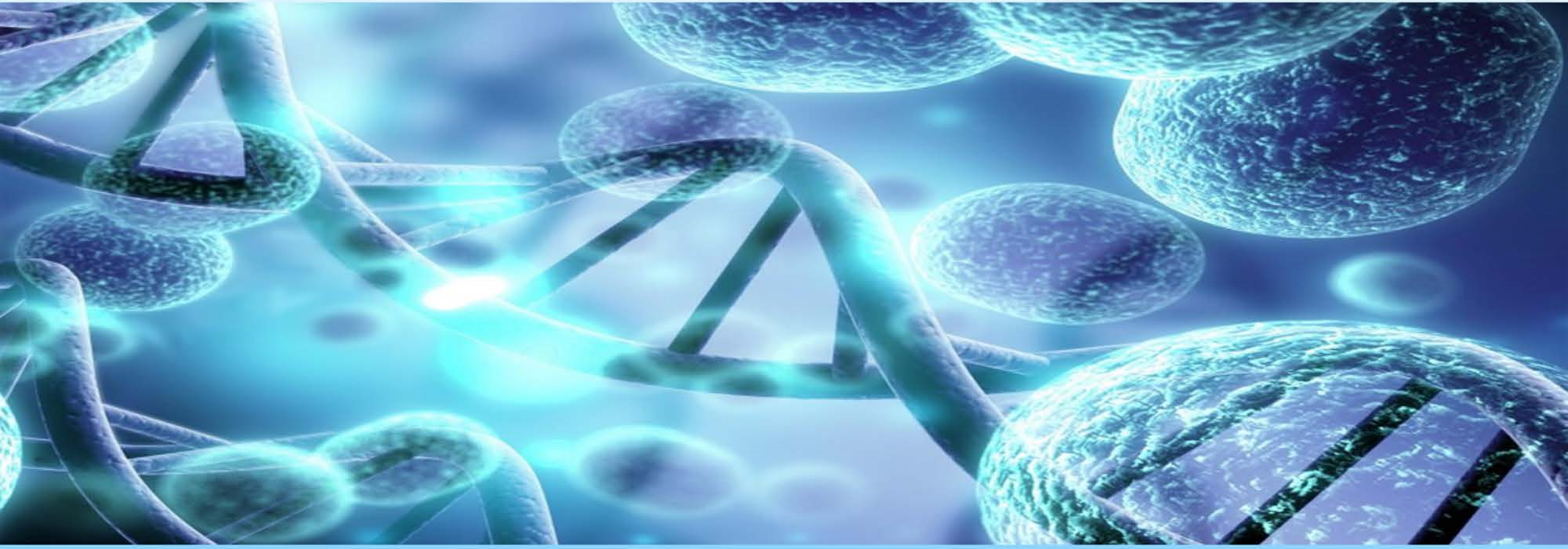
- the average human lifespan nowadays (premature ageing)

- biological reserve of human life

Khavinson V. Peptidergic regulation of ageing (2009)

KHAVINSON PEPTIDES®

PEPTIDE BIOREGULATORS



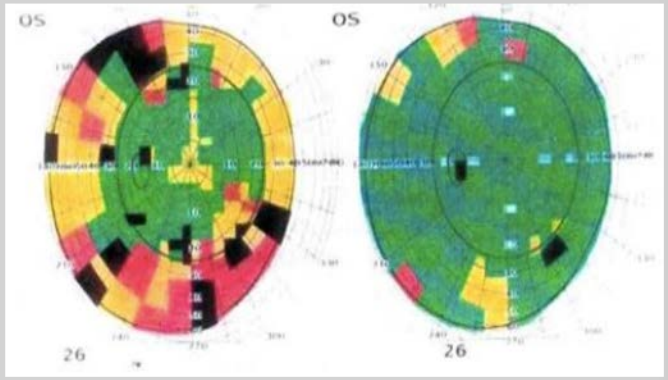
ORGAN REGENERATION

ST. PETERSBURG INSTITUTE OF BIOREGULATION AND GERONTOLOGY

Vladimir Khavinson V.Kh., Neroev V.V., Trofimova S.V., Yu.Yu. Osokina. UNIQUE METHOD FOR RESTORATION OF RETINAL FUNCTIONS IN CASE OF DIFFERENT DISEASES. Russian Academy of Sciences 2009.

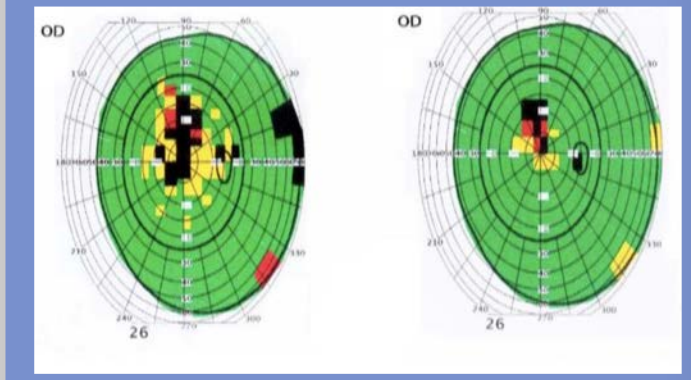
PEPTIDE BIOREGULATOR PROTOCOL

Restoration of Retinal Function



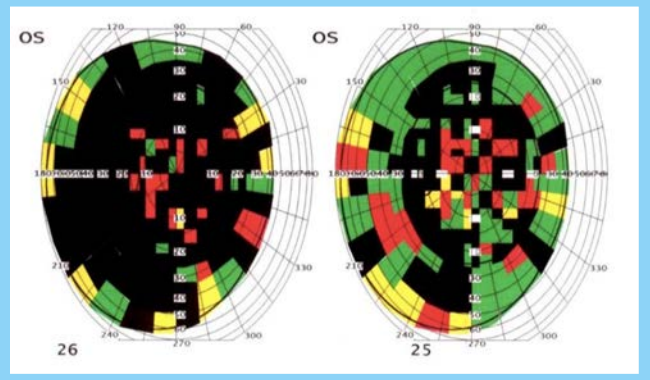
STUDY 1

Diabetic Retinopathy



STUDY 2

Age-Related Macular Degeneration

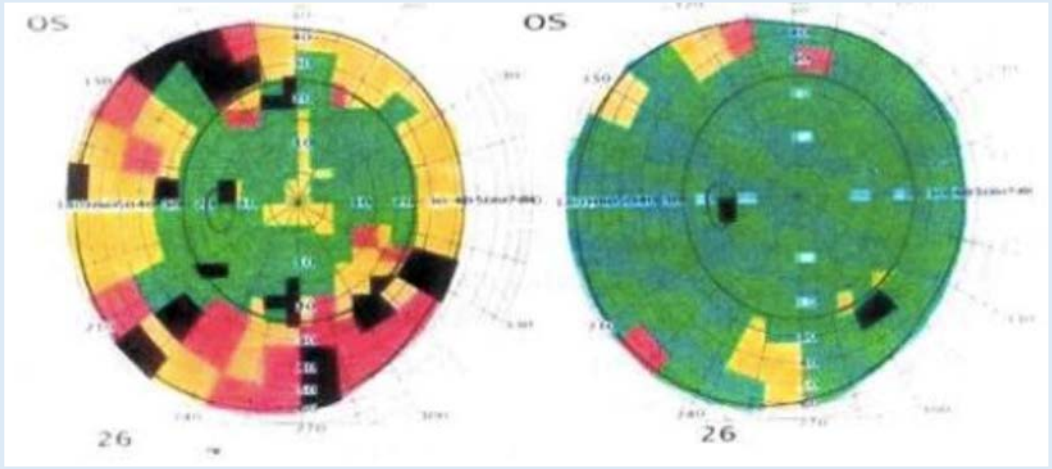


STUDY 3

Retinitis Pigmentosa

PEPTIDE BIOREGULATOR PROTOCOL

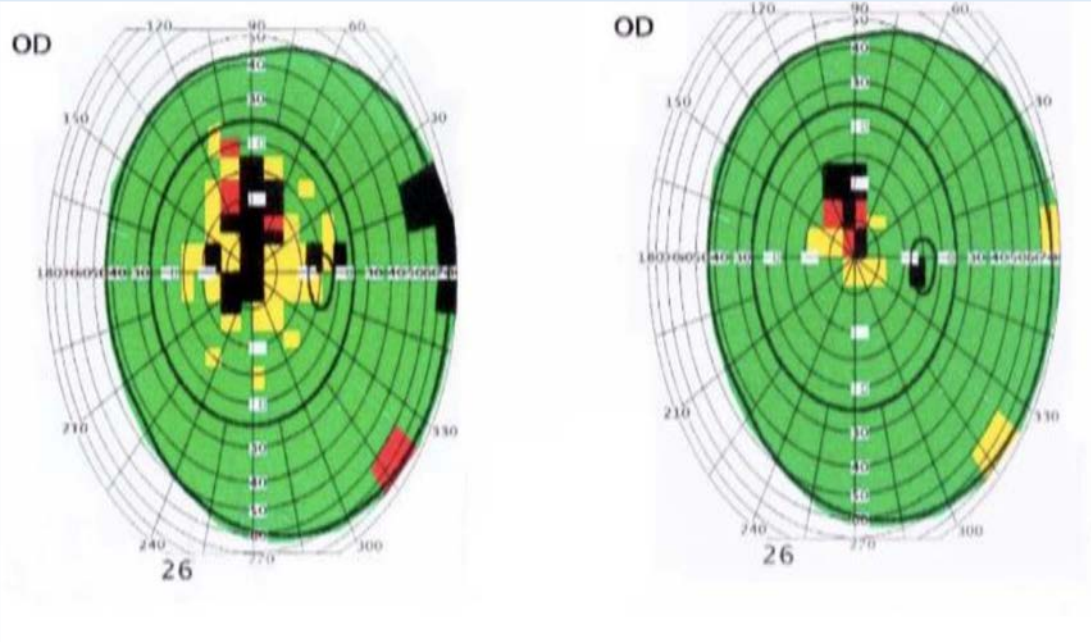
Diabetic Retinopathy



Brightness Degradation	Before Treatment	%	After Treatment	%
Normal	97	51.3	177	93.7
Scotoma 1	52	27.5	7	3.7
Scotoma 2	20	10.6	3	1.6
Abs Scotoma	20	10.6	2	1.1

PEPTIDE BIOREGULATOR PROTOCOL

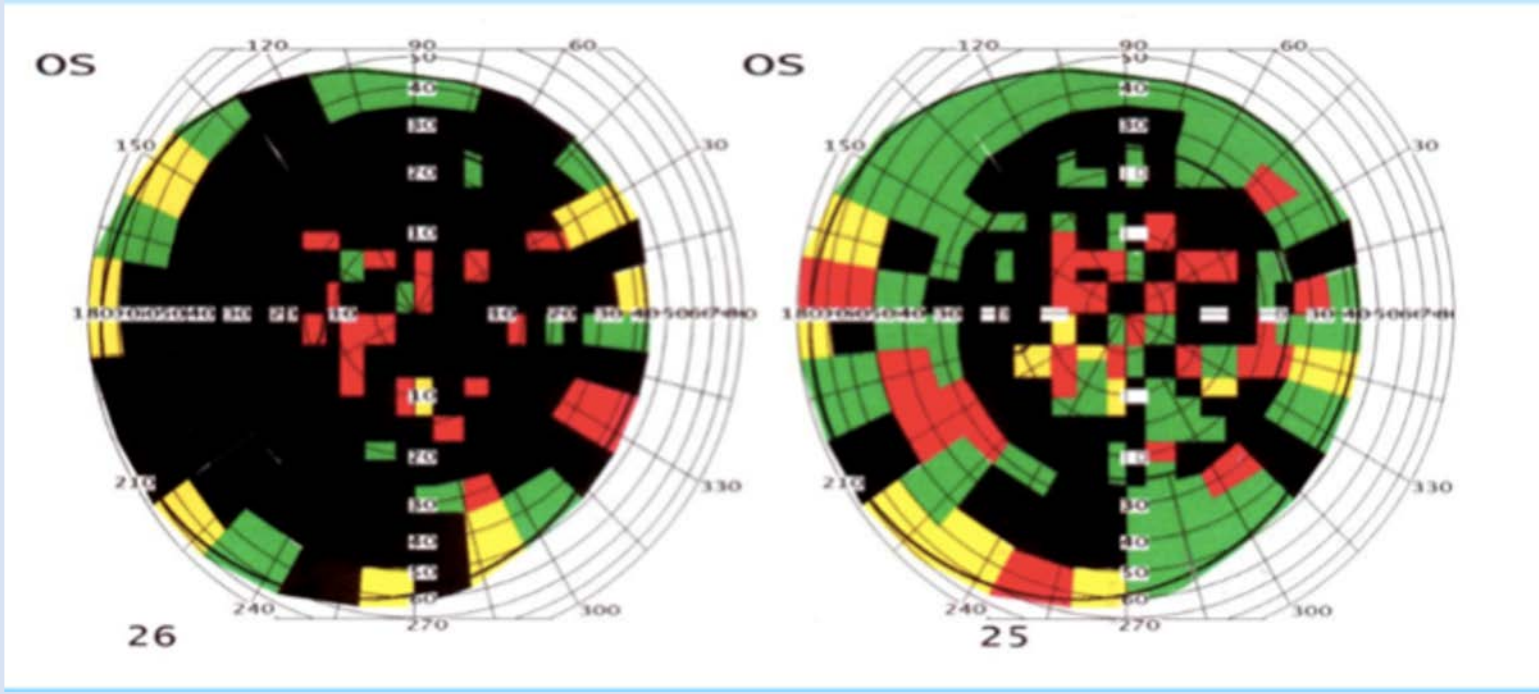
Age-Related Macular Degeneration



Brightness Degradation	Before Treatment	%	After Treatment	%
Norm	134	70.9	171	90.5
Scotoma 1	30	15.9	8	4.2
Scotoma 2	4	2.1	4	2.1
Abs. scotoma	21	11.1	6	3.2

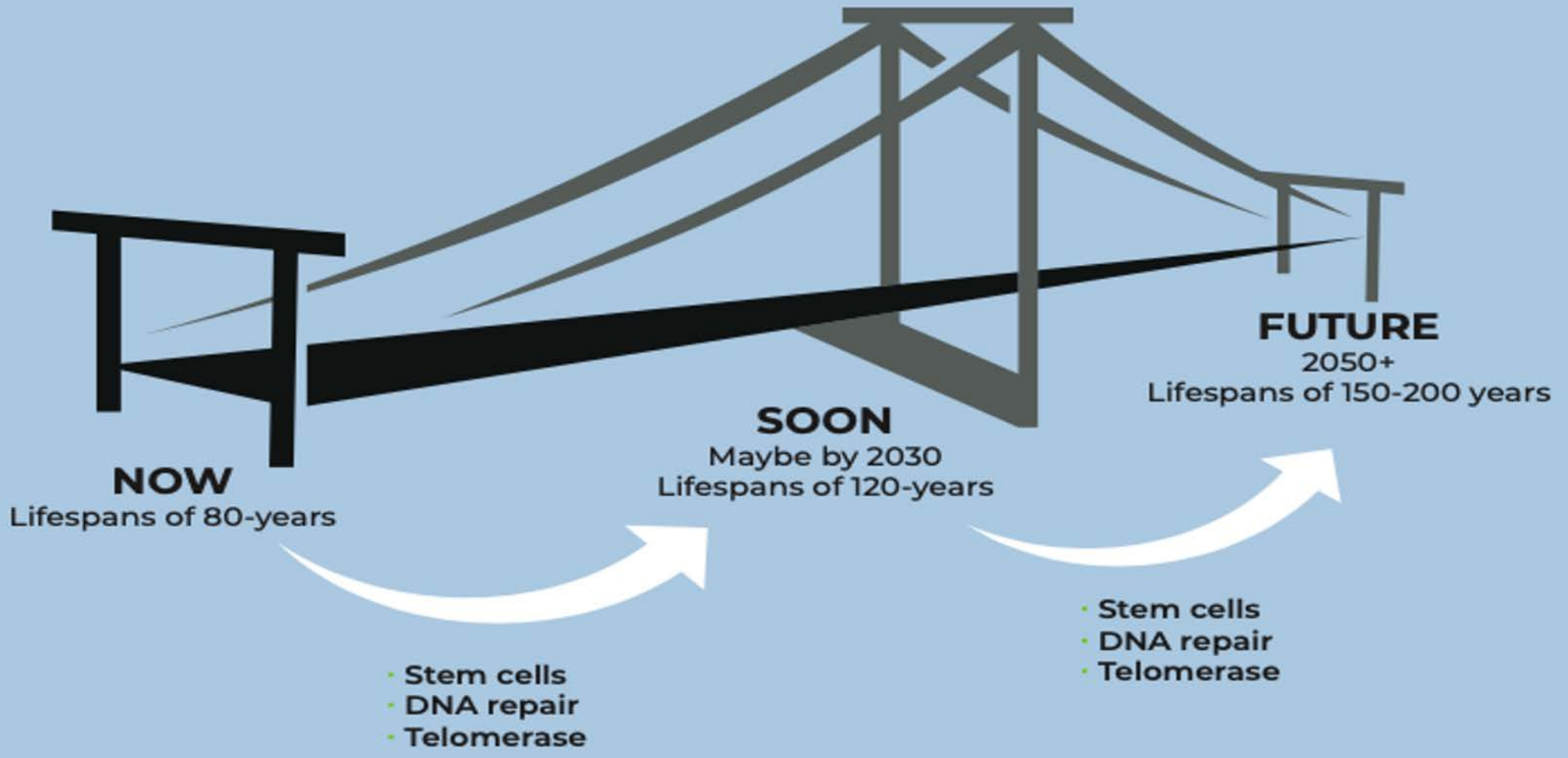
PEPTIDE BIOREGULATORS PROTOCOL

Retinitis Pigmentosa



The Longevity Bridge

Practical immortality: Healthy human lifespans of 150 years or longer may be achieved- if we employ anti-aging therapeutics as the bridge between now and the immediate future.



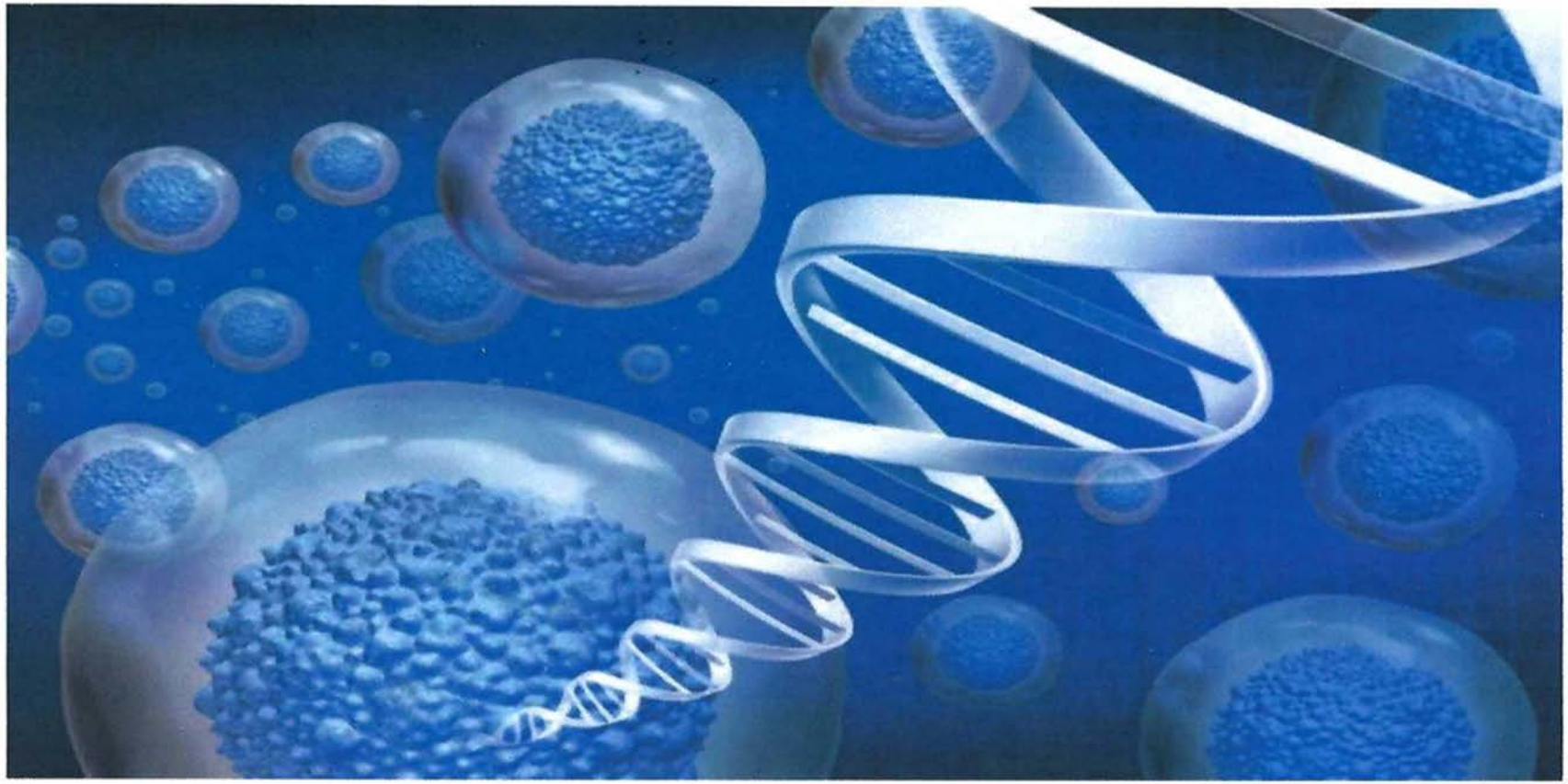
nature biotechnology

HEALTH CARE

Billionaires Bankroll Cell Rejuvenation Tech as the Latest Gambit to Slow Aging

Start-ups bet that carefully controlled cell reprogramming may lead to age reversal, but hurdles remain

By Michael Eisenstein, Nature Biotechnology on January 21, 2022



CROSSING THE LONGEVITY BRIDGE

- ❑ QUANTUM COMPUTERS
- ❑ ARTIFICIAL INTELLIGENCE
- ❑ CRISPR (GENOME SEQUENCING)
- ❑ GENE SEQUENCING/THERAPY
- ❑ STEM CELLS
- ❑ REPLACEMENT ORGANS
- ❑ ROBOTICS
- ❑ NANOTECHNOLOGY
- ❑ 3D PRINTING
- ❑ MICROBOTS

NewLimit

AGEX THERAPEUTICS

INVESTMENT TO DATE- $10 BILLION

AgeX Therapeutics
Altos Labs
Calico
Gameto
Iduna Therapeutics
Retro Bio
Turn Biotechnologies
NewLimit
Shift Bioscience
Life Biosciences
YouthBio Therapeutics

gameto

ALTOS™

YouthBio THERAPEUTICS

Current Aging Expectations

Telomerase Activation & DNA Methylation Longevity Interventions

Peptide Paradigm of Aging

Cellular Repair and Epigenetic Rejuvenation

Professor Vladimir Khavinson
Dr. Bill Lawrence

"Russian and American clinical studies confirm 10-20 years of additional healthy lifespan."

DR. BILL LAWRENCE
RUSSIAN PEPTIDE BIOREGULATORS
PEPTIDE LONGEVITY PROGRAMS

YOUTUBE POWERPOINT PRESENTATIONS

Telomerase Activation Study (TAP)

Epigenetic Methylation Study (EMS)

Interviews of Dr. Lawrence presenting interim clinical study data are now posted on YouTube and can be found there with the following search terms: “Bill Lawrence Peptides”

Bioregulator Peptides & Russian Longevity Research with Dr. Bill Lawrence PART 1

Dr. Bill Lawrence: Reverse Aging Bioregulator Peptides Effect on DNA Methylation PART 2

Cellular Reprogramming and Epigenetic Rejuvenation

Peptide Bioregulator Protocol

Organ Regeneration, Mortality Reduction, Biological Age Reversal

Bill Lawrence
JD, MS, Ph.D

St. Petersburg Institute of Bioregulation and Gerontology
Russia

Viruses

2021 Oct 13, 2021

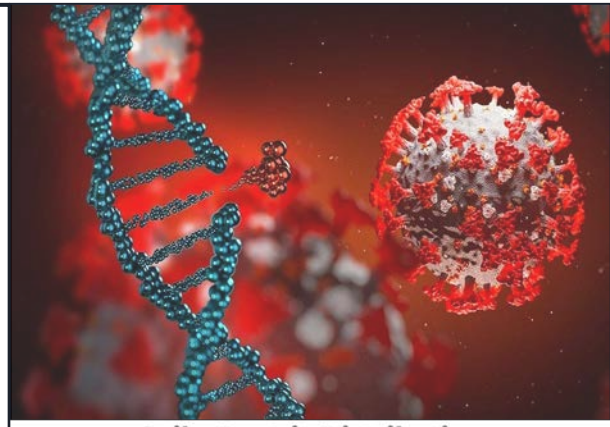
PMID: 34696485

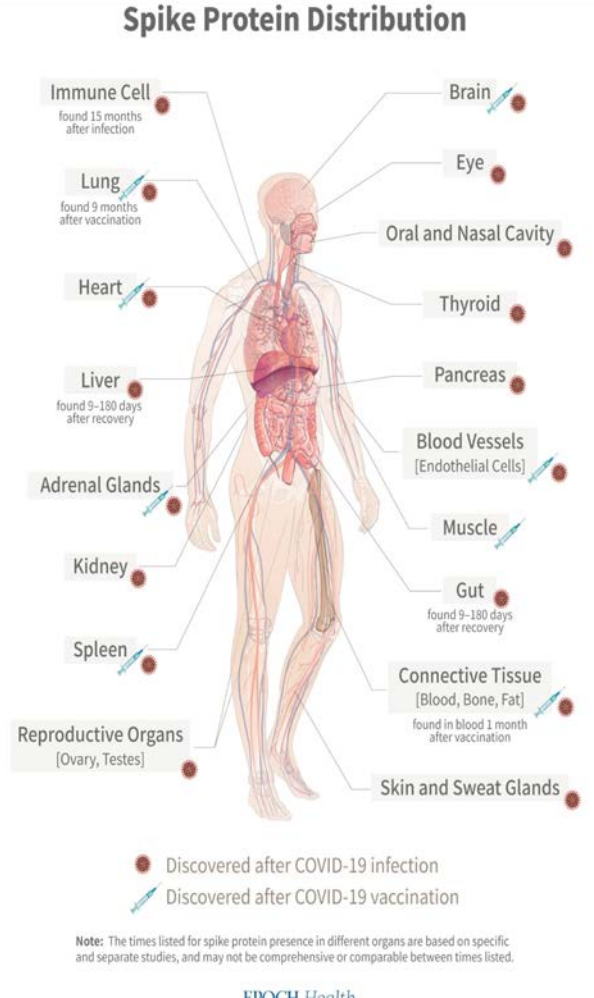
SARS-CoV-2 Spike Impairs DNA Damage Repair and Inhibits V(D)J Recombination In Vitro

Hui Jiang [1,2], Ya-Fang Mei [2]

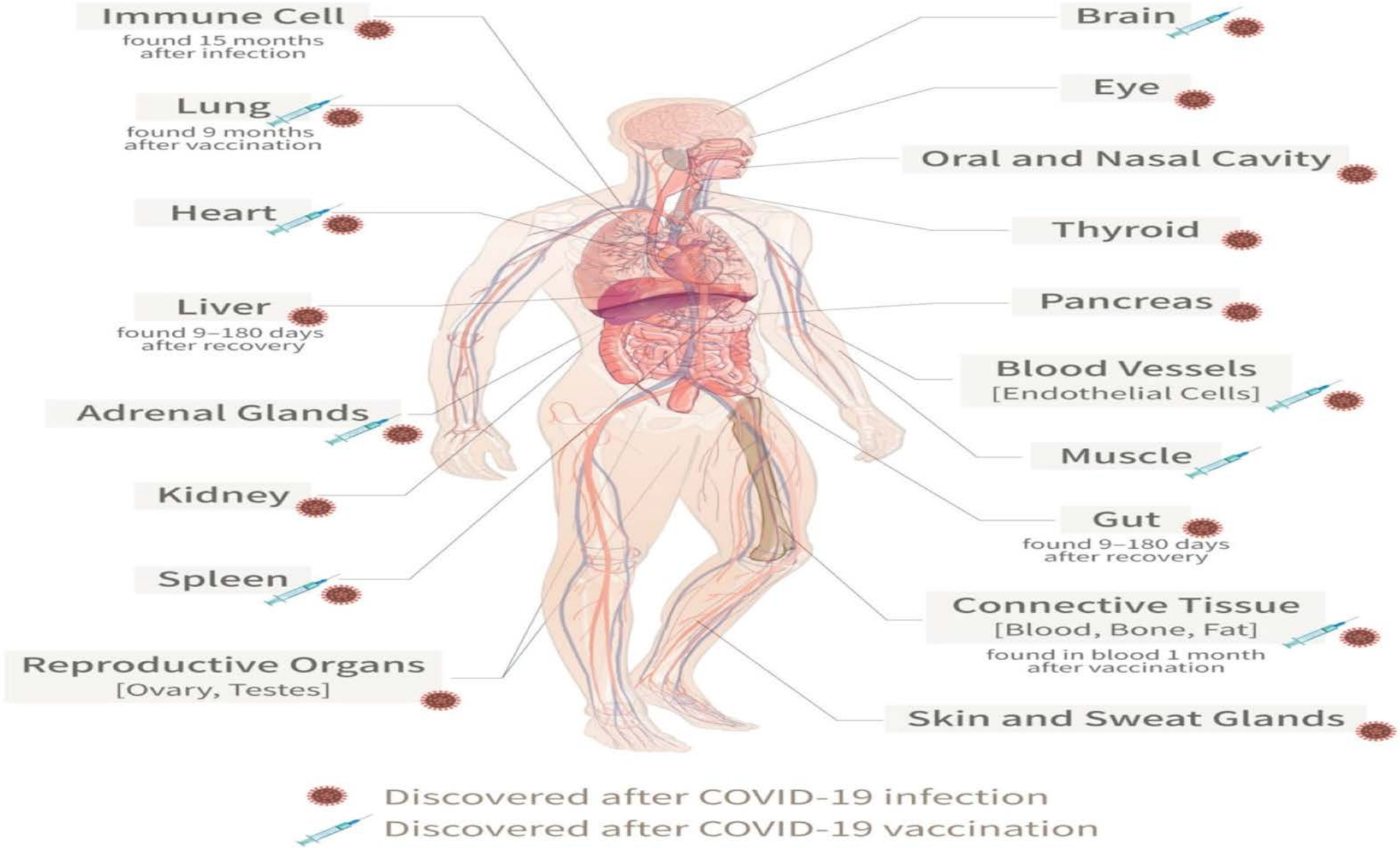
Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has led to the coronavirus disease 2019 (COVID-19) pandemic, severely affecting public health and the global economy. Adaptive immunity plays a crucial role in fighting against SARS-CoV-2 infection and directly influences the clinical outcomes of patients. Clinical studies have indicated that patients with severe COVID-19 exhibit delayed and weak adaptive immune responses; however, the mechanism by which SARS-CoV-2 impedes adaptive immunity remains unclear. Here, by using an in vitro cell line, we report that the SARS-CoV-2 spike protein significantly inhibits DNA damage repair, which is required for effective V(D)J recombination in adaptive immunity. Mechanistically, we found that the spike protein localizes in the nucleus and inhibits DNA damage repair by impeding key DNA repair protein BRCA1 and 53BP1 recruitment to the damage site. Our findings reveal a potential molecular mechanism by which the spike protein might impede adaptive immunity and underscore the potential side effects of full-length spike-based vaccines.





Spike Protein Distribution



EU Open Research Repository

Published December 3, 2024 | Version 1

SARS-CoV2 spike protein pathogenicity research collection

Description

Originally part of the outer coat of the SARS-CoV2 virus, where it functions as a “key” to “unlock” (infect) cells, spike proteins are also produced in large amounts by the mRNA “vaccines,” triggering a short-lived immune response in the form of antibodies. However, a growing body of evidence has shown that the spike protein is harmful by itself, independent of the rest of the virus.

The following (I. Alphabetical List) collects over **250 peer-reviewed scientific studies confirming that the spike protein is highly pathogenic on its own;** most *in vitro* studies cited here used recombinant spike proteins or spike proteins in pseudo viral vectors and produced pathological effects not reliant on the SARS-CoV 2 viral machinery.

The second section (II. Categories) organizes the research into broad categories, including affected tissues and organ systems, mechanisms, and evidence from clinical pathology. Because these areas overlap, many articles appear more than once in the second section.

Science, Public Health Policy and the Law

December 3, 2024

By Nicolas Hulscher, MPH

Study Urges Immediate Halt to COVID-19 mRNA Injections Over Alarming Levels of DNA Contamination

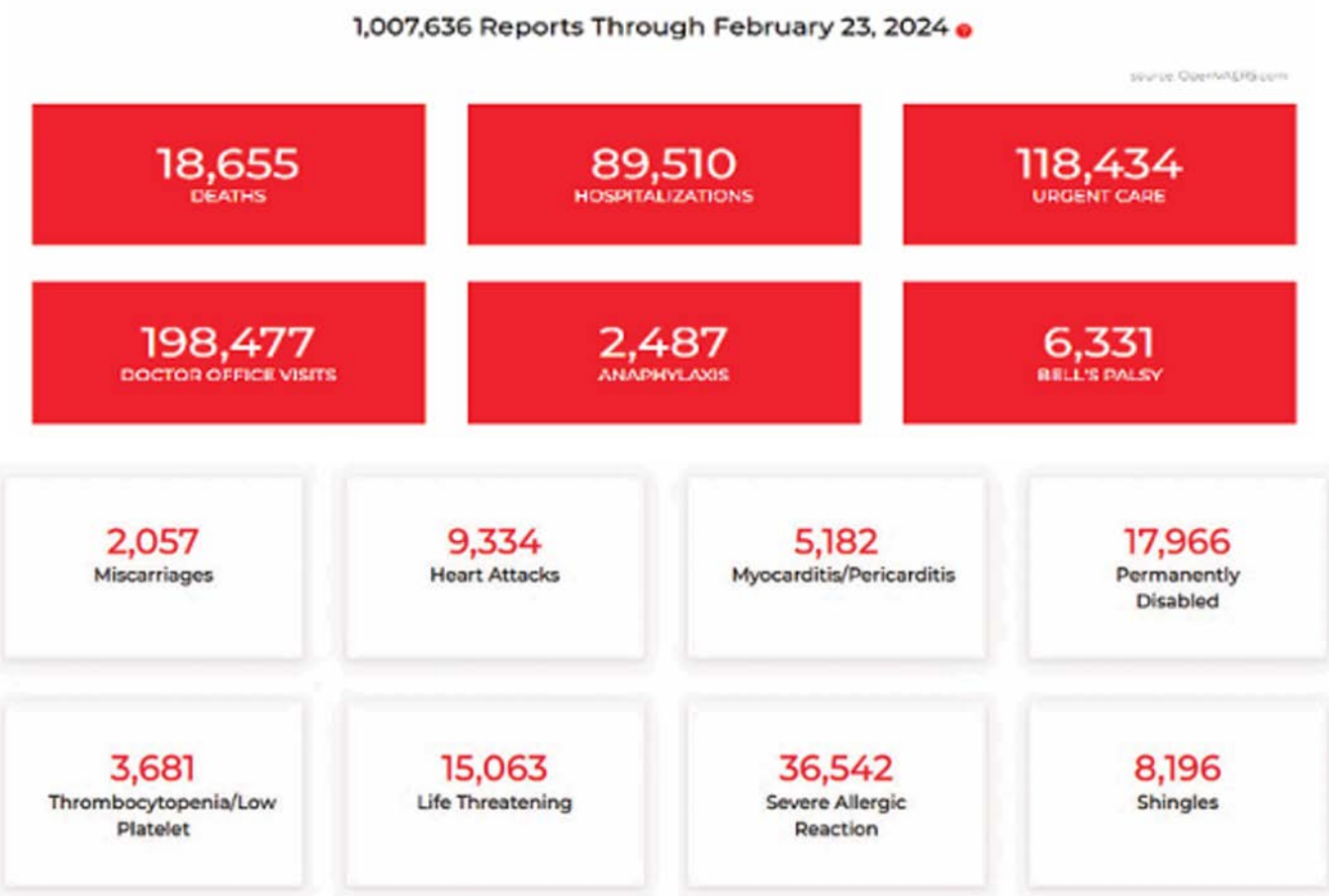
Researchers find DNA contamination in COVID-19 mRNA injections exceeding regulatory limits by over 300%, confirming findings from earlier studies.

Calls for an immediate global moratorium on COVID-19 mRNA injections continue to intensify. Today, Kammerer et al published the study titled, BioNTech RNA-Based COVID-19 Injections Contain Large Amounts Of Residual DNA Including An SV40 Promoter/Enhancer Sequence, in the journal

Pfizer/BioNTech's COVID-19 modRNA Vaccines: Dangerous Genetic Mechanism of Action Released before Sufficient Preclinical Testing

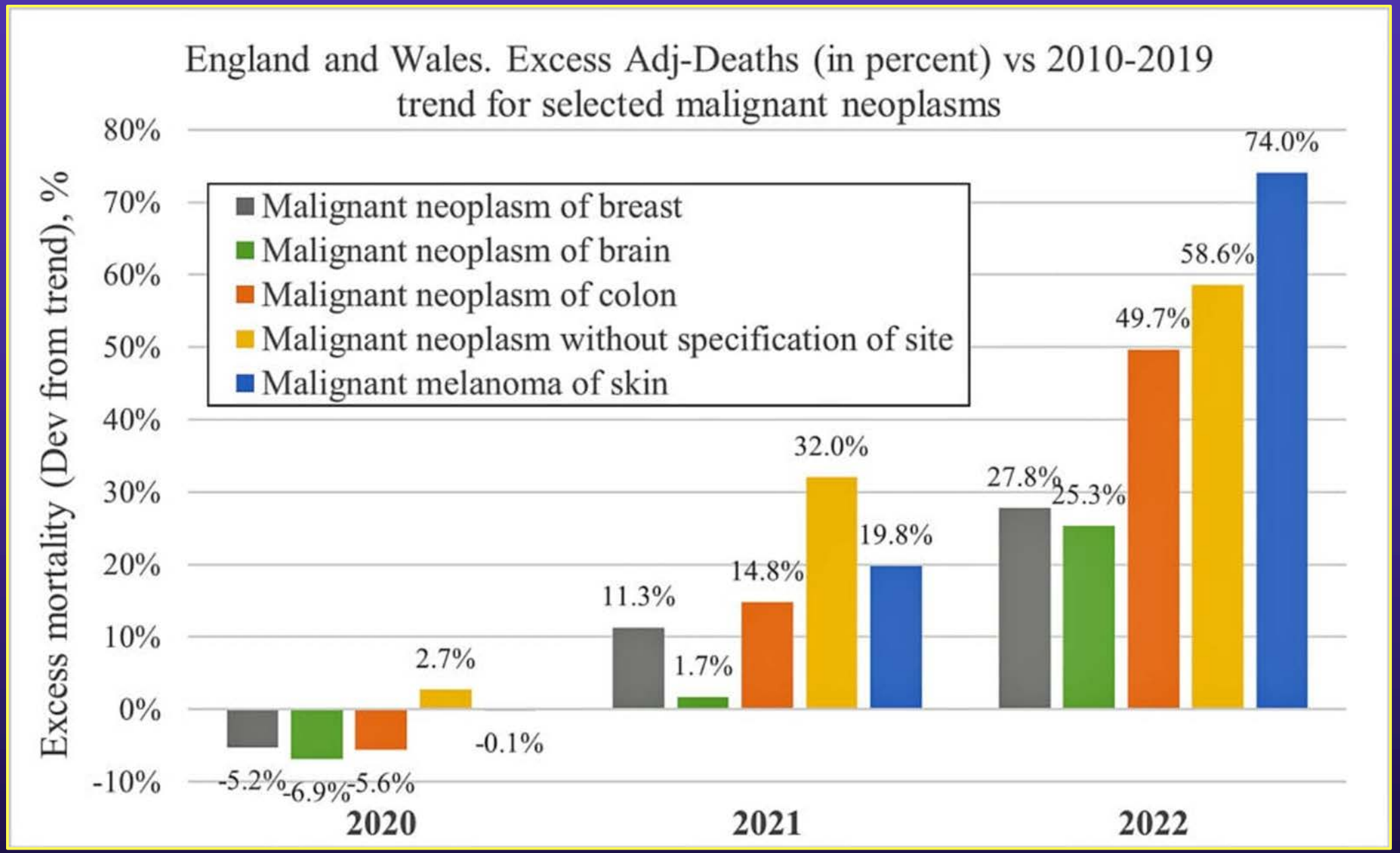
Journal of American Physicians and Surgeons
AAPS The voice for private physicians since 1943

Philip R. Oldfield, D.Phil.; L. Maria Gutschi, B.Sc.Phm, Pharm.D.; Peter A. McCullough, M.D., M.P.H.; David J. Speicher, Ph.D.



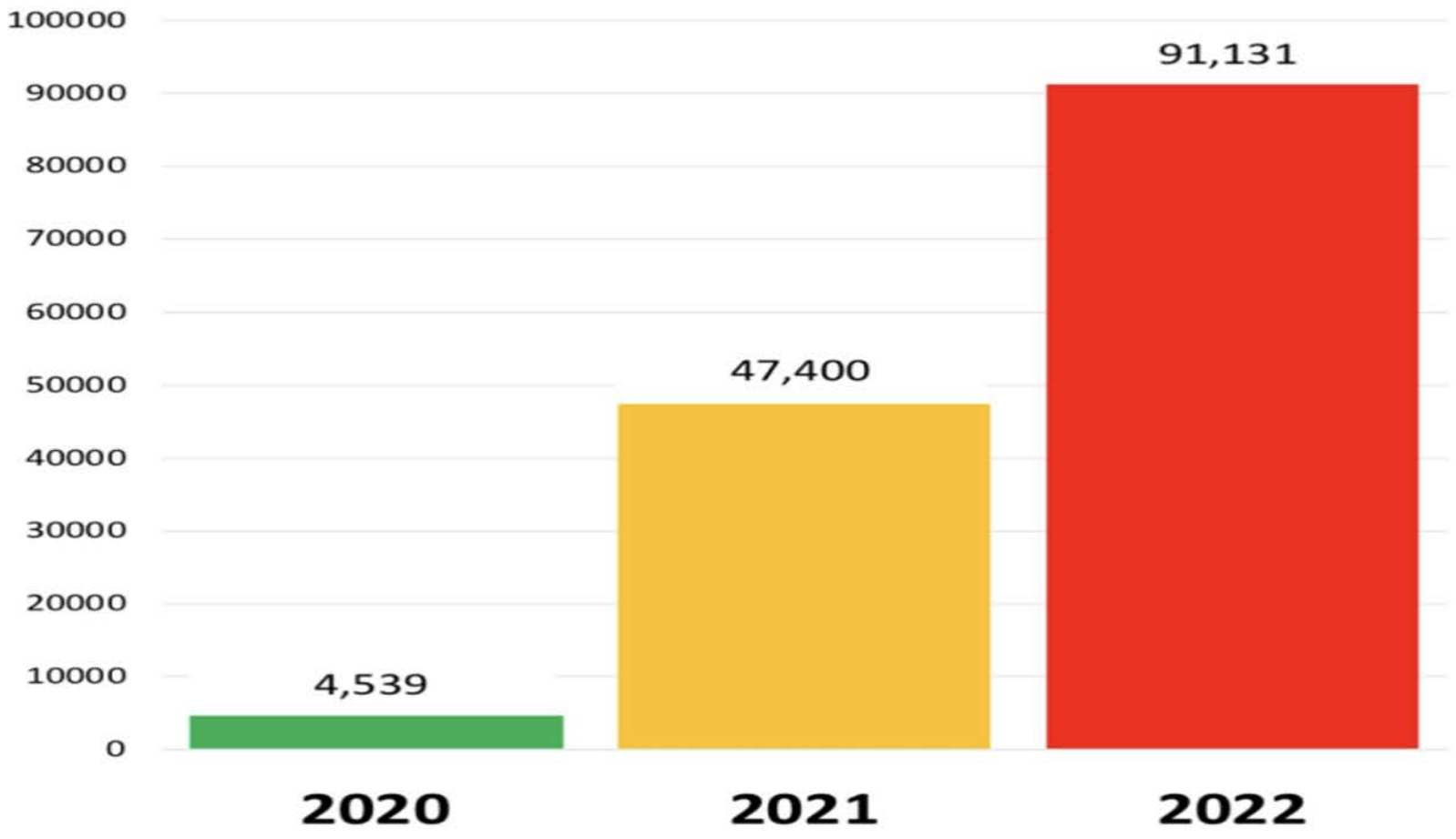
Conclusion

For any other medicinal product, the regulatory submission would have been considered incomplete and most probably rejected. Therefore, a moratorium on the use of Pfizer/BioNTech COVID-19 vaccines and boosters should be enacted at minimum, but ideally, they should be removed from the market and their use in humans should be stopped. It should be the responsibility of the pharmaceutical industry, not independent scientists, to determine whether a medical intervention is safe. Based upon Pfizer/BioNTech's data, safety of their COVID-19 modRNA vaccine has not been proven.


England and Wales. Excess Adj-Deaths (in percent) vs 2010-2019 trend for selected malignant neoplasms
Excess mortality (Dev from trend), %
80%
70%
60%
50%
40%
30%
20%
10%
0%
-10%
Malignant neoplasm of breast
Malignant neoplasm of brain
Malignant neoplasm of colon
Malignant neoplasm without specification of site
Malignant melanoma of skin
-5.2%
-6.9%
-5.6%
2.7%
-0.1%
11.3%
1.7%
14.8%
32.0%
19.8%
27.8%
25.3%
49.7%
58.6%
74.0%
2020
2021
2022

JAPAN: Age Adjusted Excess Deaths
January 1 to October 30

Source: https://exdeaths-japan.org/en/graph/numberof/



DNA REPAIR BY ACTIVATING p53
and
SARS-CoV-2 Spike Protein Protocol

EACH PROTOCOL IS SEPARATE AND EACH IS ESSENTIAL

Bill Lawrence JD, MS, Ph. D

March 2024

DNA REPAIR SYSTEM PROTOCOL

Gene p53 is a system's central component that eliminates pathologically damaged cells from an organism. Multiple signal pathways monitor the state of a cell. When damage or a fault is found that could cause heritable changes, the p53 protein is activated to either coordinate the repair process or induce cell suicide. Loss of the p53 gene results in uncontrolled accumulation of genetic damage causing failure of control by the organism, malignant cell growth, and death of the organism. It is a primary cancer tumor suppressor. p53 plays a prominent role as a facilitator of DNA repair by halting the cell cycle to allow time for the repair machinery to restore genome stability. ***Therefore, it is critical to activate p53 to aid DNA repair.***

1. Selenium to activate p53

Four 200 mcg a day [total of 800 mcg]. The NOAEL (i.e., No Adverse Effect Level) of selenium per day is 819+/-126 micrograms daily. You can also substitute Brazil Nuts for some of the selenium capsules. An average Brazil Nut has about 100 mcg. I recommend no more than four Brazil Nuts daily, as long-term use can produce toxic effects.

2. Flaxseed – increases p53

Technically flaxseed contains *pre*-lignans. Once eaten, lignans are produced in the intestine by the stomach's bacteria acting on the pre-lignans. These lignans (a) inhibit the activation of the AKT enzyme and (b) increase the expression of the p53 protein by blocking its degradation in the cell. The result is that flaxseed inhibits AKT and increases p53**.** Start with half Tbsp and increase up to two Tbsp daily.

SPIKE PROTEIN ABATEMENT PROTOCOL

1. Augmented NAC

Augmented NAC increases the antioxidant properties of N-Acetylcysteine and its natural ability to denature the Spike protein (99%) by breaking its disulfide bonds, thus disturbing the interaction with the ACE-2 receptor. Take 2 capsules (400mg) daily.

2. Nemorex (NEW Khavinson Peptide for Covid VACCINE SPIKE PROTEIN)

Suppresses replication of spike protein and effective immunomodulatory peptide bioregulator.

3. Nattokinase, (5,000-10,000 fu daily) Serrapeptase (120,000 spu) , and Lumbrokinase-Boluoke (300,000 lku)

See the clinical study: Degradative Effect of Nattokinase on Spike Protein of SARS-CoV-2 (Aug 2022)

4. FibroProtek

This product may not directly activate p53 but can inhibit the Covid and Pfizer/Moderna spike proteins.
Several studies identified the flavanol quercetin and the structurally related flavone luteolin as potential potent blockers of the spike proteins in the Covid virus and the Pfizer and Moderna vaccines: 2 caps daily

5. Ivermectin

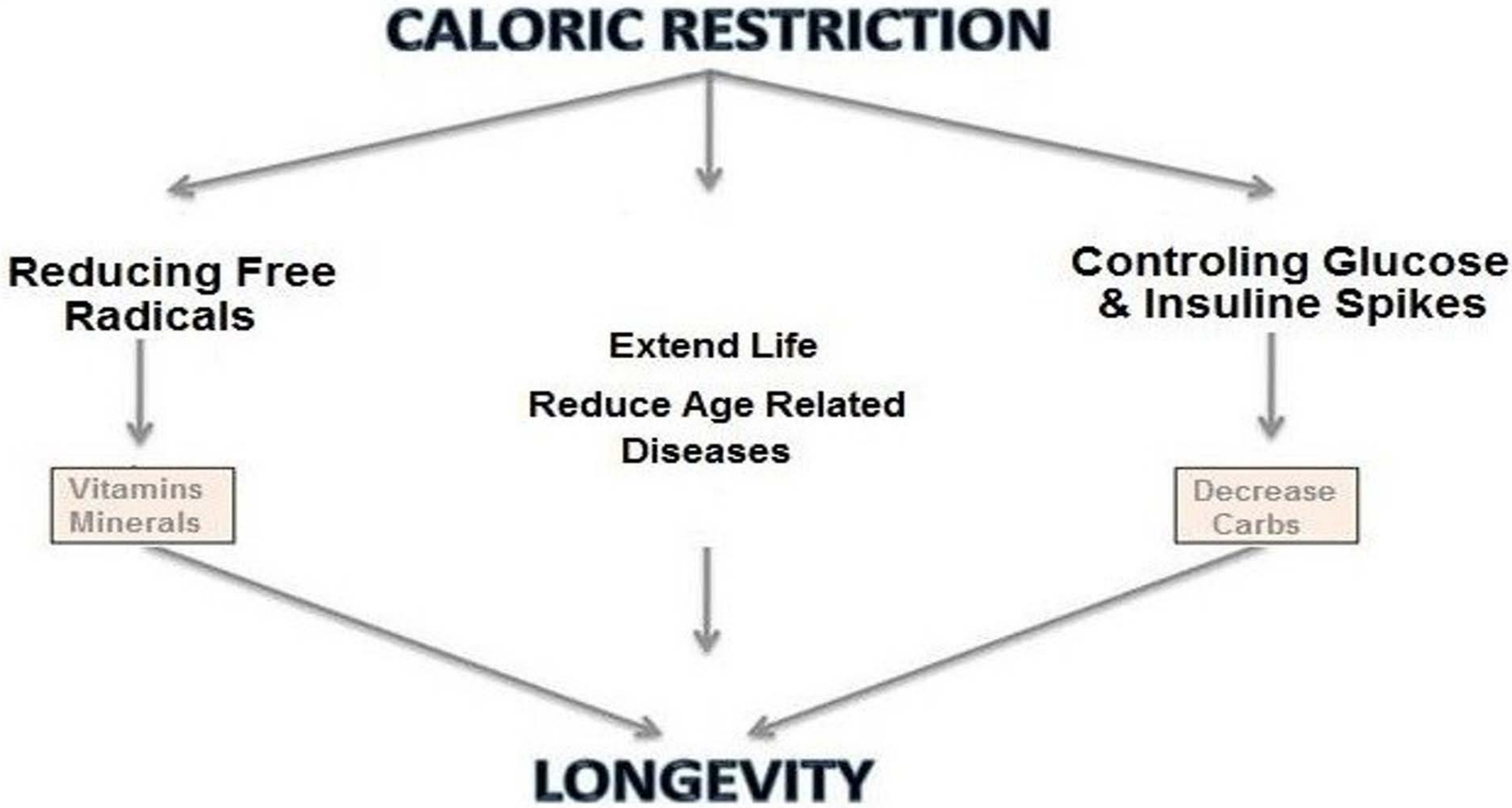
This agent has powerful antiparasitic and antiviral properties. In addition, evidence indicates that ivermectin binds the ACE2 receptor site that the spike protein needs to bind to proceed with entry into the cell and the replication of viral protein. One or two 3 mg capsules prophylactic per week for spike protein without symptoms. With symptoms (organ or system degeneration), increase dosage for one month to 18 mg every other day.

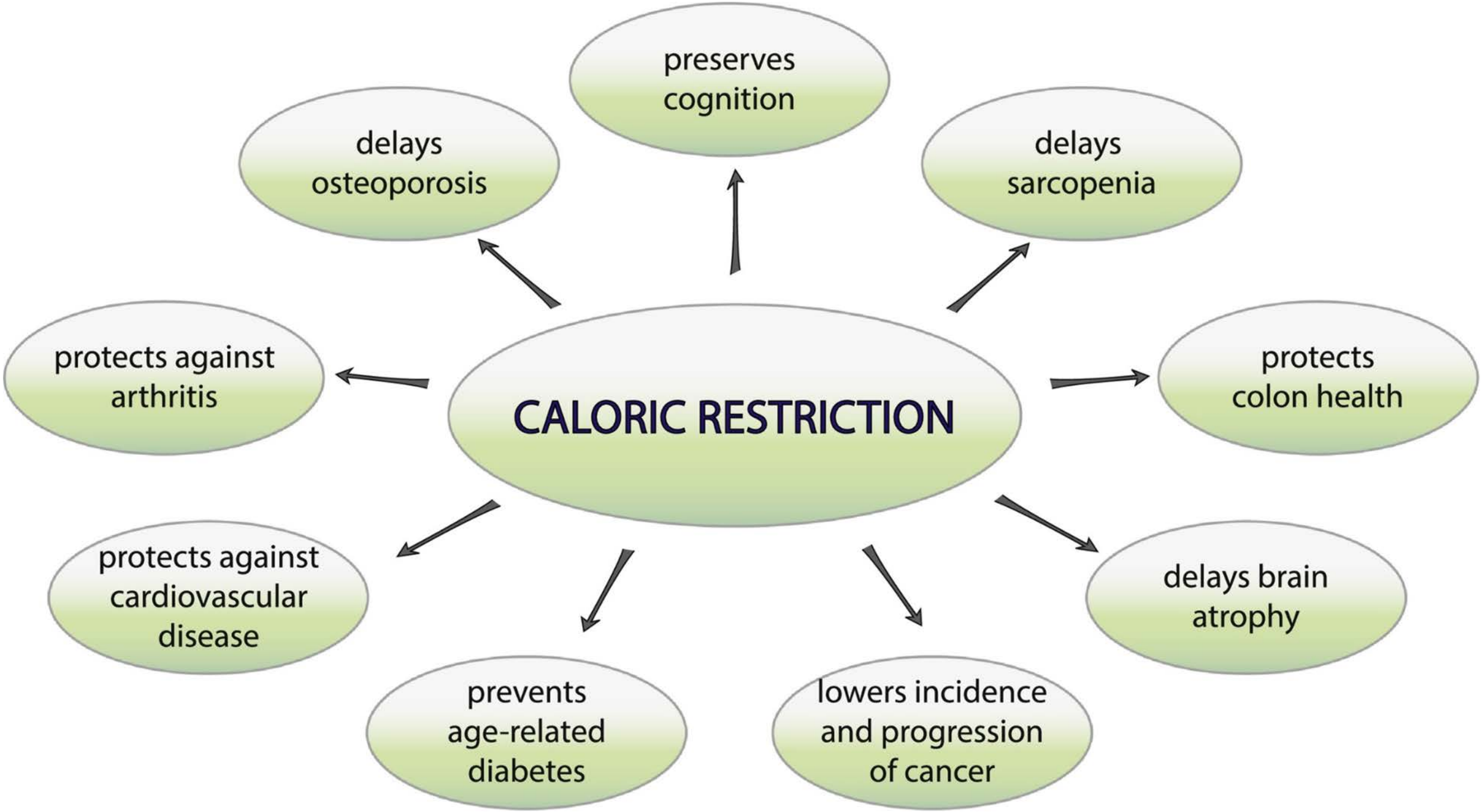
6. Spike Protein Detox

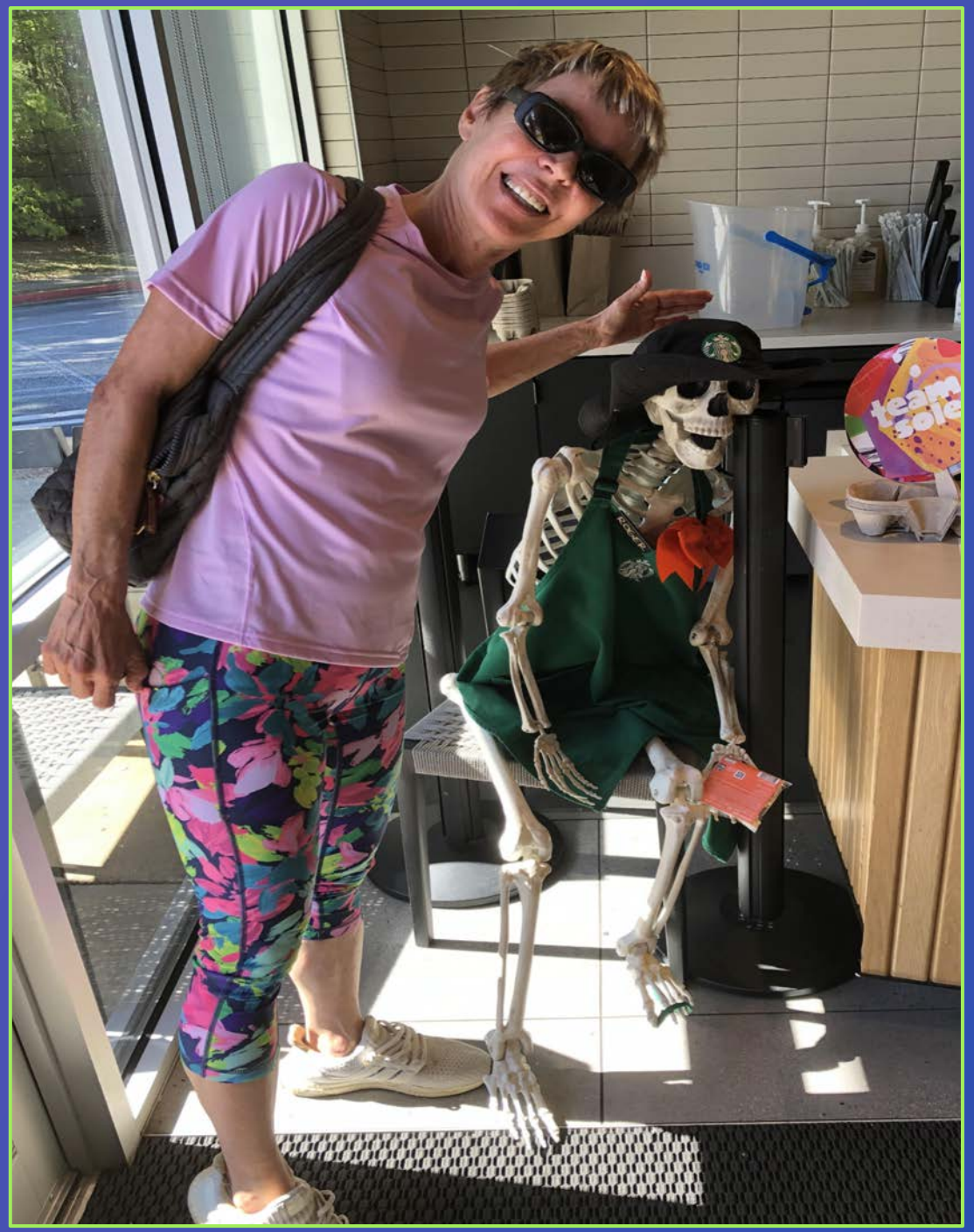
The following compounds are helpful (all with published clinical studies) for spike protein detox. There are other nutraceuticals, herbs, etc., but these are primary

Vitamin D 5000-10,000 IU	Vitamin C
NAC + Bromelain (synergistic) 500 mg 2x	Carpenter's Herb (Prunella vulgaris)
Nigella seed	Quercetin
Giant Knotweed	Suramin
Curcumin and Catechin	Milk thistle extract

Peptide Bioregulators regenerate organs damaged by Covid-19 virus and “vaccines”” spike protein


CALORIC RESTRICTION
Reducing Free Radicals
Vitamins Minerals
Extend Life
Reduce Age Related Diseases
Controling Glucose & Insuline Spikes
Decrease Carbs
LONGEVITY


preserves cognition
delays osteoporosis
delays sarcopenia
protects against arthritis
CALORIC RESTRICTION
protects colon health
protects against cardiovascular disease
delays brain atrophy
prevents age-related diabetes
lowers incidence and progression of cancer



"Bill, Do you think you might have overdone your caloric restriction experiment a bit?"

-Ves

1. Rancourt et al: estimated **17 million** COVID-19 vaccine deaths worldwide by September 2023 .
2. Mostert et al: estimated **3.1 million** excess deaths likely attributed to COVID-19 vaccination/lockdowns among 47 countries of the Western World from 2020 to 2022.
3. Vaccine Adverse Event Reporting System (VAERS): **37,966** reported COVID-19 vaccine deaths - under-reporting factor of 31 yields **1,176,946** COVID-19 vaccine deaths among countries that use VAERS.
4. Skidmore: estimated **278,000** Americans may have died from the COVID-19 vaccine by December 2021.
5. Pantazatos and Seligmann: estimated **146,000 to 187,000** possible vaccine-associated deaths in the United States by August 2021.
6. Hulscher et al (I): estimated **49,240 excess cardiac arrest deaths** possibly due to COVID-19 vaccination in the U.S. from 2021-2023.
7. Hulscher et al (II): found a high likelihood of a **causal link between COVID-19 vaccines and death from analysis of 325 autopsies**.
8. Aarstad and Kvitastein: found a higher COVID-19 vaccine uptake was associated with **increased all-cause mortality**.
9. Alessandria et al: found all-cause death risks to be **higher for those vaccinated with one and two COVID-19 vaccine doses compared to unvaccinated individuals**. The subjects vaccinated with **2 doses lost 37% of life expectancy compared to the unvaccinated population during follow-up.**
10. Lataster: found a positive correlation between **COVID-19 vaccination rates and excess mortality** for every month examined.

COVID-19 VACCINE DEATHS

2021-2023