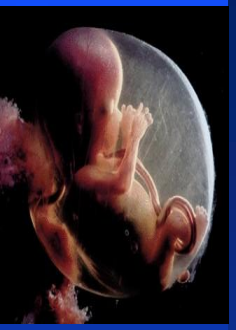


GERONTOLÓGIA

7. Biogerontológia: öregedési elméletek

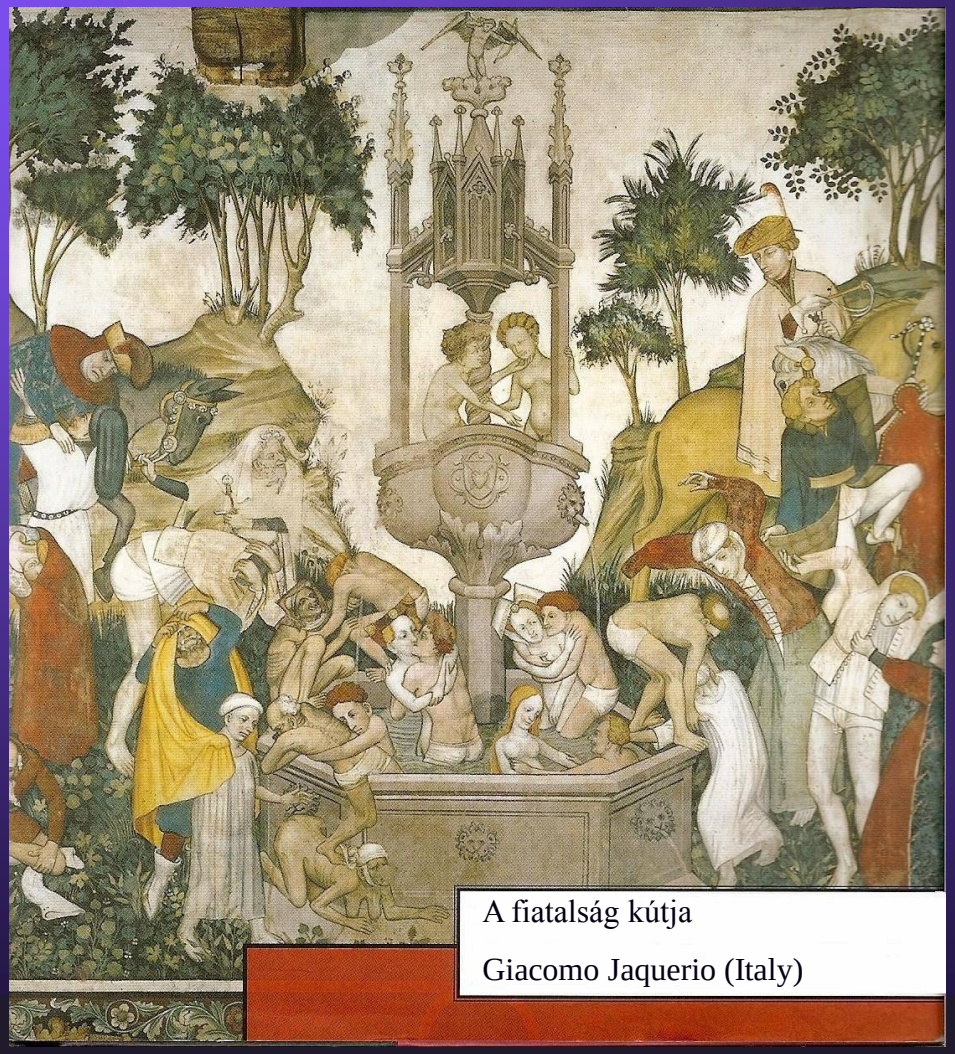
Dr. SEMSEI IMRE

Debreceni Egyetem

Egészségügyi Kar

DEBRECENI EGYETEM

KEZDETI PRÓBÁLKOZÁSOK

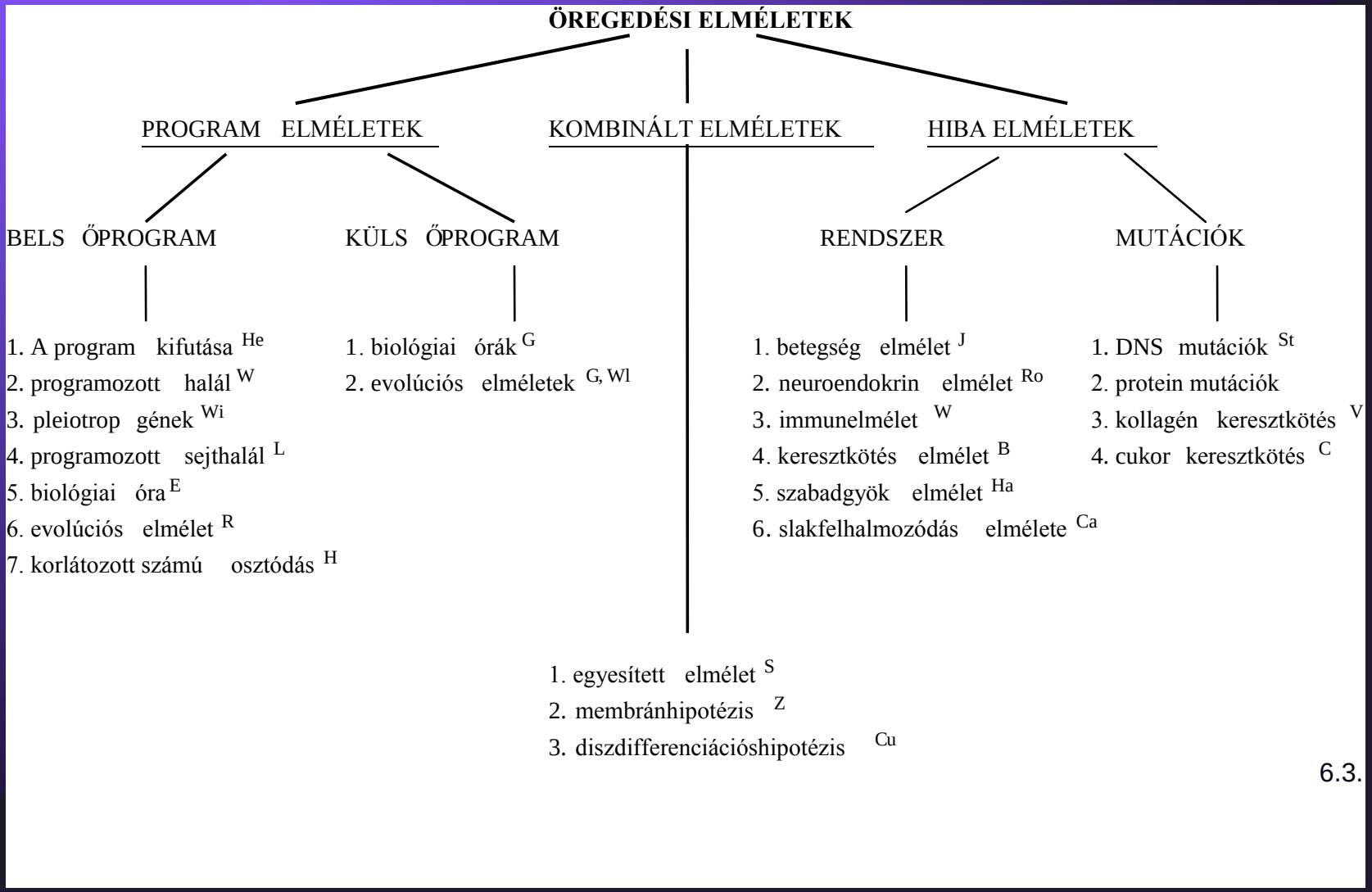


A fiatalság kútja

Giacomo Jaquerio (Italy)

KEZDETI PRÓBÁLKOZÁSOK

AZ ÖREGEDÉSI ELMÉLETEK CSOPORTOSÍTÁSA



ÖREGEDÉSI ELMÉLETEK

I. Program elméletek

Evolúciós elmélet

Nature Vol. 270 24 November 1977

review article

Dr. I. SEMSEI

Evolution of ageing

T. B. L. Kirkwood*

An evolutionary view of ageing suggests that mortality may be due to an energy-saving strat of reduced error regulation in somatic cells. This supports Orgel's 'error catastrophe' hypothesis offers a new basis for the study of normal and abnormal ageing syndromes and of apparently immo transformed cell lines.

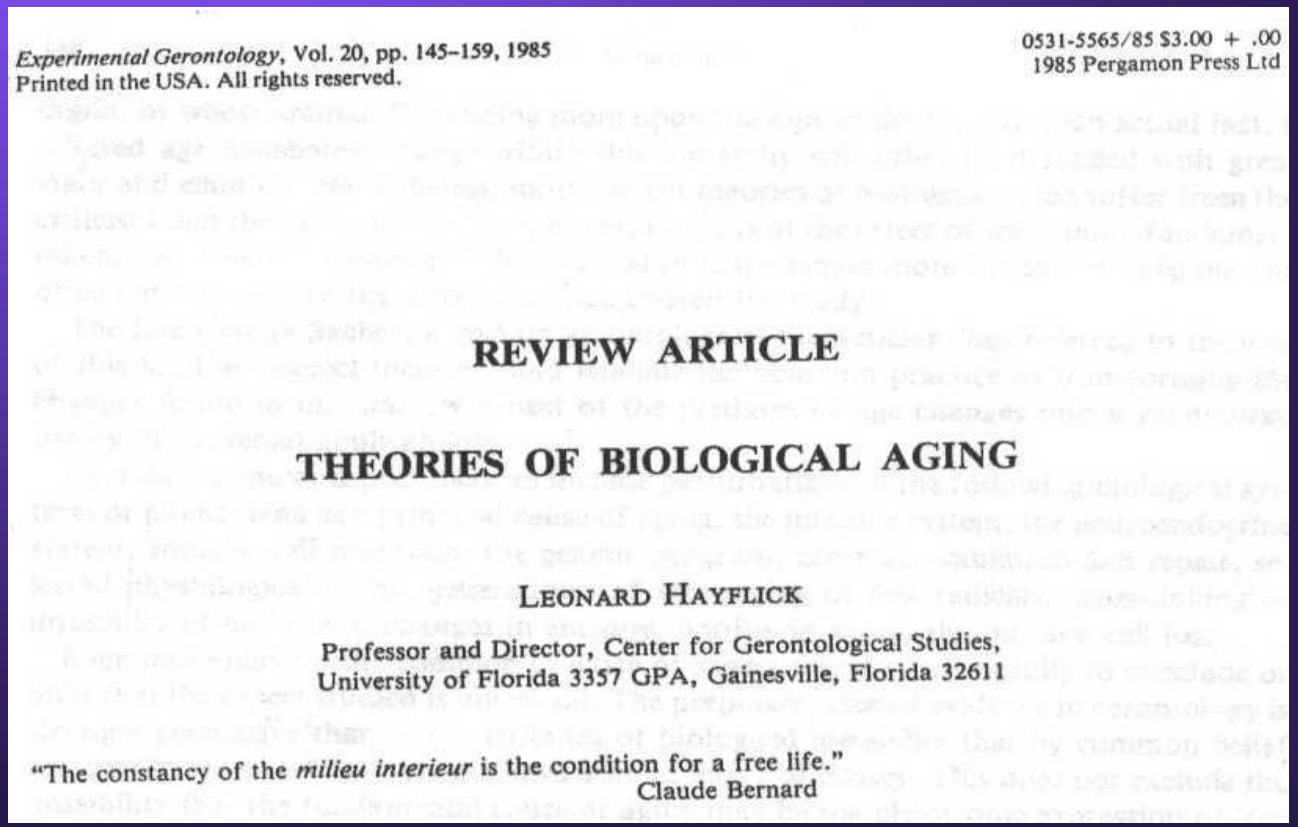
THE question of why we and most other higher animals age is not trivial since many organisms, notably higher plants, live and propagate indefinitely. Perhaps because we are so familiar with ageing it is dangerously easy to fall into circular argument such as that ageing is a mechanism for ridding a population of old and worn-out individuals who would otherwise compete for resources with younger and fitter ones[1]. This makes sense if, for example, we assume

order, and entropy, or disorder. Indeed, given the capa for replication, neo-Darwinist natural selection may be s as the direct result of interaction between the informa stored in living organisms and the entropy of their envi ment. The diversity of present-day species testifies to wide range of evolutionary strategies that are effective survival, and it is my intent in this paper to demonstr why ageing should be one of these.

ÖREGEDÉSI ELMÉLETEK

I. Program elméletek

Limitált osztódás elmélet



Experimental Gerontology, Vol. 20, pp. 145–159, 1985
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0531-5565/85 $3.00 + .00

REVIEW ARTICLE

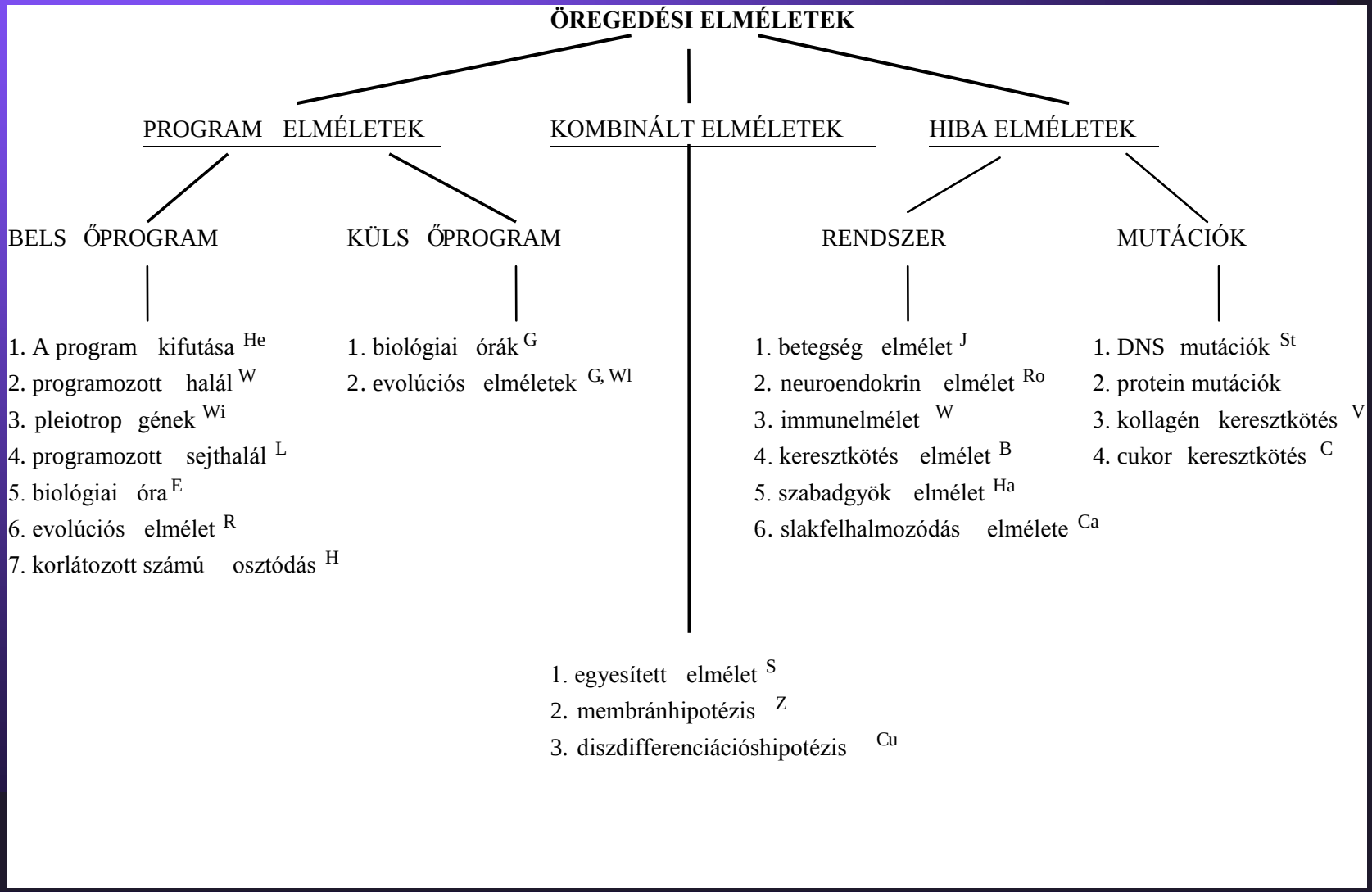
THEORIES OF BIOLOGICAL AGING

LEONARD HAYFLICK

Professor and Director, Center for Gerontological Studies,
University of Florida 3357 GPA, Gainesville, Florida 32611

"The constancy of the *milieu interieur* is the condition for a free life."
Claude Bernard

AZ ÖREGEDÉSI ELMÉLETEK CSOPORTOSÍTÁSA



ÖREGEDÉSI ELMÉLETEK

II. Hiba elméletek

Betegség elmélet

Relations Between Normal Aging and Disease, edited by H. A. Johnson. Raven Press © 1985.

Is Aging Physiological or Pathological?

Horton A. Johnson

Departments of Pathology, St. Luke's–Roosevelt Hospital Center; and College of Physicians and Surgeons, Columbia University, New York, New York 10019

Aging, being a universal phenomenon, is regarded as a "normal" or physiological process distinct from

ÖREGEDÉSI ELMÉLETEK

II. Hiba elméletek

Szabadgyök elmélet

Proc. Natl. Acad. Sci. USA
Vol. 78, No. 11, pp. 7124–7128, November 1981
Medical Sciences

The aging process

(free radicals/evolution/antioxidants/degenerative diseases/longevity)

DENHAM HARMAN

University of Nebraska College of Medicine, Departments of Medicine and Biochemistry, Omaha, Nebraska 68105

Communicated by Melvin Calvin, July 13, 1981

ABSTRACT **Aging is the progressive accumulation of changes with time that are associated with or responsible for the ever-increasing susceptibility to disease and death which accompanies advancing age. These time-related changes are attributed to the aging process. The nature of the aging process has been the subject of considerable speculation. Accumulating evidence now indicates that the sum of the deleterious free radical reactions going on continuously throughout the cells and tissues constitutes the aging process or is a major contributor to it. In mammalian systems the free radical reactions are largely those involving oxygen.**

Dietary manipulations expected to lower the rate of production of free radical reaction damage have been shown (*i*) to increase the life span of mice, rats, fruit flies, nematodes, and rotifers, as well as the "life span" of neurospora; (*ii*) to inhibit development of some forms of cancer; (*iii*) to enhance humoral and cell-mediated immune responses; and (*iv*) to slow development of amyloidosis and the autoimmune disorders of NZB and NZB/NZW mice. In addition, studies strongly suggest that free radical reactions play

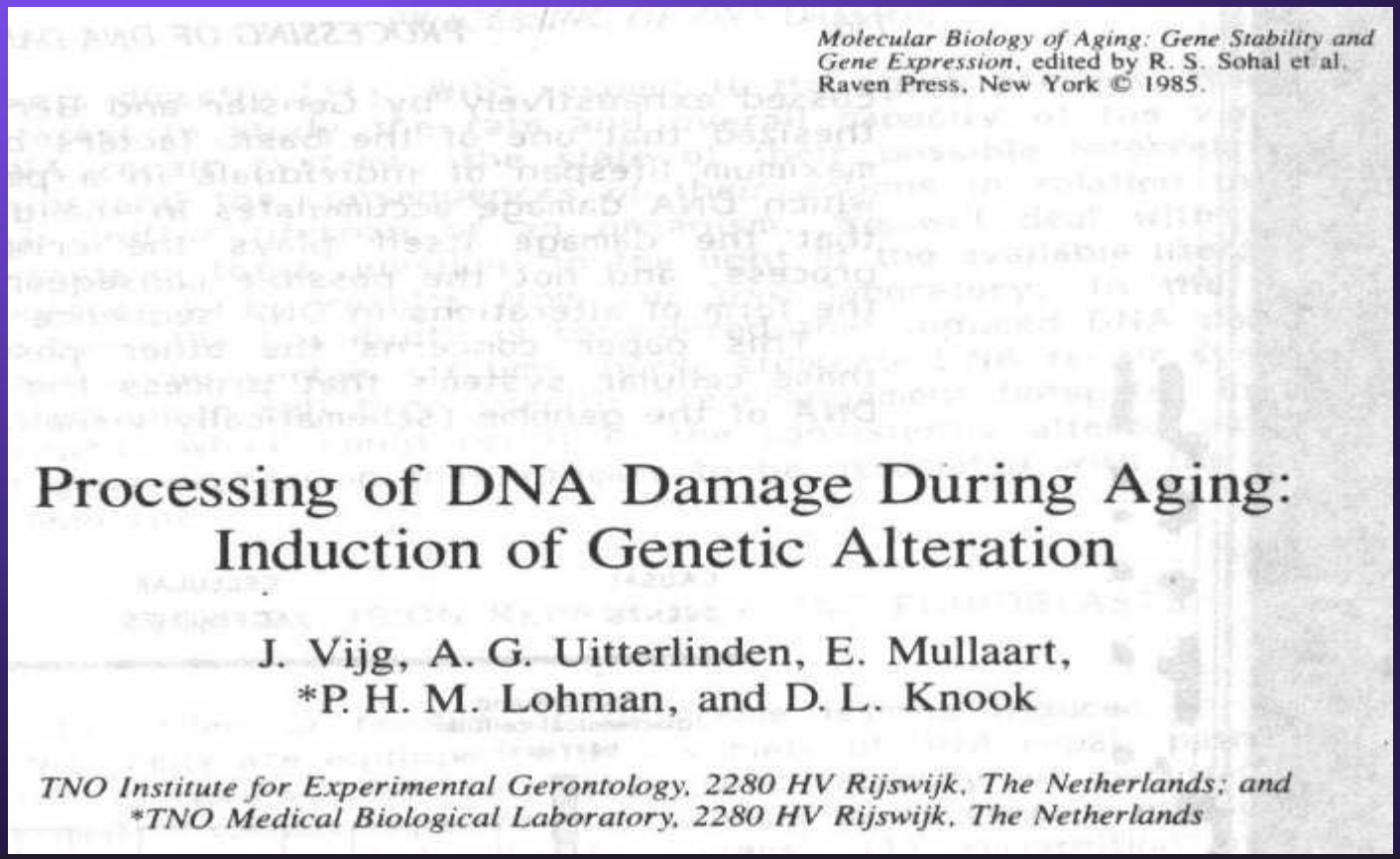
level. Similarly, aging at the multicellular level may be considered the result of the aging processes proceeding in all the cells, with environmental influences now including the effects of the aging cells on each other and the changes with time of the connective tissues. Death of multicellular life occurs because of death or dysfunction, or both, of cells involved in functions vital to the cells as a whole (e.g., functions in mammals such as those of the respiratory center or of the myocardium).

The nature of the aging process has been the subject of considerable speculation (1). Suggested possibilities include (*i*) encodement of aging in DNA (made manifest in a manner similar to development), (*ii*) progressive breakdown in accuracy in protein synthesis, (*iii*) crosslinkage of macromolecules, (*iv*) in higher organisms, "attack" of the immune system on self-antigens, and (*v*) free radical reaction damage. This paper is mainly limited to a discussion of the last-named possibility not only because accumulating evidence indicates that aging is largely

ÖREGEDÉSI ELMÉLETEK

II. Hiba elméletek

DNS hiba elmélet



Molecular Biology of Aging: Gene Stability and Gene Expression, edited by R. S. Sohal et al.
Raven Press, New York © 1985.

Processing of DNA Damage During Aging: Induction of Genetic Alteration

J. Vijg, A. G. Uitterlinden, E. Mullaart,
*P. H. M. Lohman, and D. L. Knook

TNO Institute for Experimental Gerontology, 2280 HV Rijswijk, The Netherlands; and
**TNO Medical Biological Laboratory, 2280 HV Rijswijk, The Netherlands*

ÖREGEDÉSI ELMÉLETEK

II. Hiba elméletek

Kollagén elmélet

Gerontologia
Editor: F. VERZÁR, Basel
S. KARGER – BASEL/NEW YORK (Printed in Switzerland)
SEPARATUM

Gerontologia *15:* 233–239 (1969)

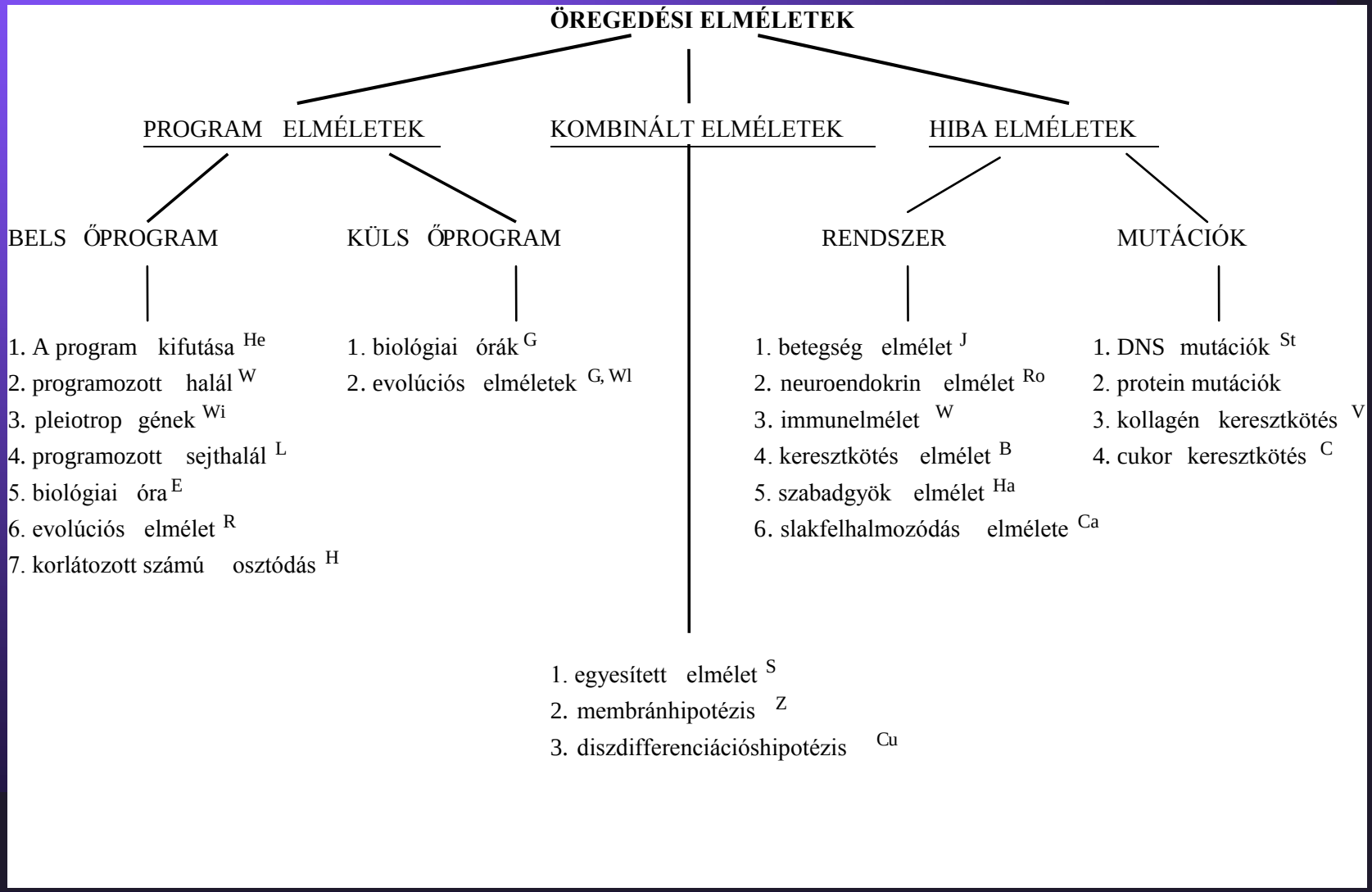
The Stages and Consequences of Ageing of Collagen

F. VERZÁR

Institute of Experimental Gerontology, Basel

Much has been said during recent years about the age changes of collagen, its structure and chemistry. It seems to be appropriate to raise the question whether we are now in a position to use this knowledge for the understanding of the physiology and pathology of ageing in man

AZ ÖREGEDÉSI ELMÉLETEK CSOPORTOSÍTÁSA



ÖREGEDÉSI ELMÉLETEK

III. Kombinált elméletek

Egyesített elmélet

From the Department of Biology, University of Southern California, Los Angeles, 90007, Ca., U. S. A.

Elements of a Unified Theory of Aging: Integration of Alternative Models

B. L. STREHLER

Introduction

During the past decade very substantial progress has been made in the critical testing of alternative models or theories of the origins of the aging process. As a result of discoveries at the cellular, molecular and integrative levels of function, the range of alternative mechanisms listed in an earlier publication (1) has been condensed considerably and the role of various potential sites of failure has been established on a quantitative basis in many instances. While it is still too early to state an overall hypothesis of human aging with certainty, enough diverse threads of evidence now exist to permit one to state a cautious synthesis of the essential outline

ÖREGEDÉSI ELMÉLETEK

III. Kombinált elméletek

Disdifferenciáció hipotézis

Molecular Biology of Aging: Gene Stability a
Gene Expression, edited by R. S. Sohal et al.
Raven Press, New York © 1985.

Dysdifferentiative Hypothesis of Aging: A Review

Richard G. Cutler

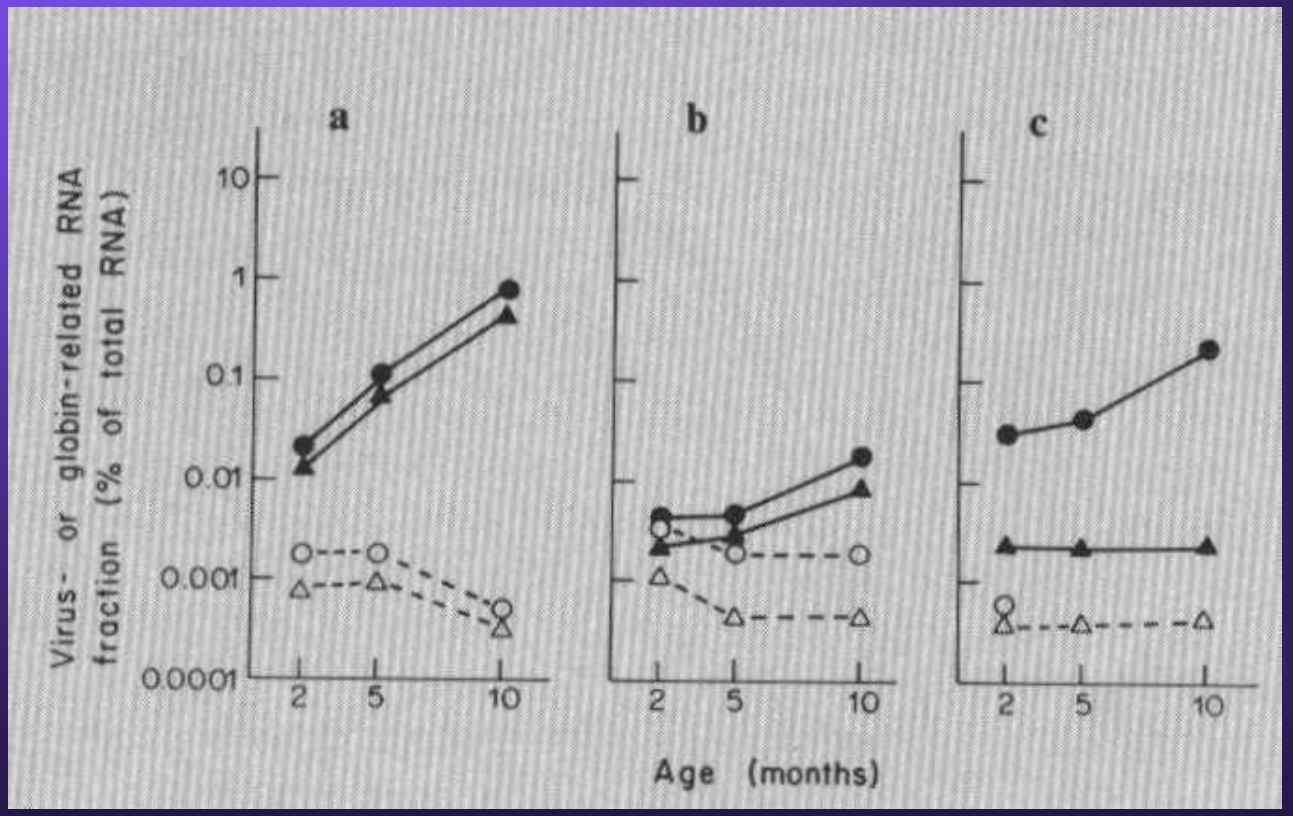
Gerontology Research Center, National Institute on Aging, Francis Scott Key Medical Center, Baltimore, Maryland 21224

One of the objectives I had in helping organize the symposium and this volume on "Molecular Biology of Aging: Gene Stability and Gene Expression" was to review the basic concepts and experimental data concerning the "Dysdifferentive Hypothesis of Aging" (49-52). Briefly, this hypothesis states that a primary

ÖREGEDÉSI ELMÉLETEK

III. Kombinált elméletek

Disdifferenciáció hipotézis



a = thymus

b = agy

c = máj

MuLV

- mag
- citoplazma

Globin

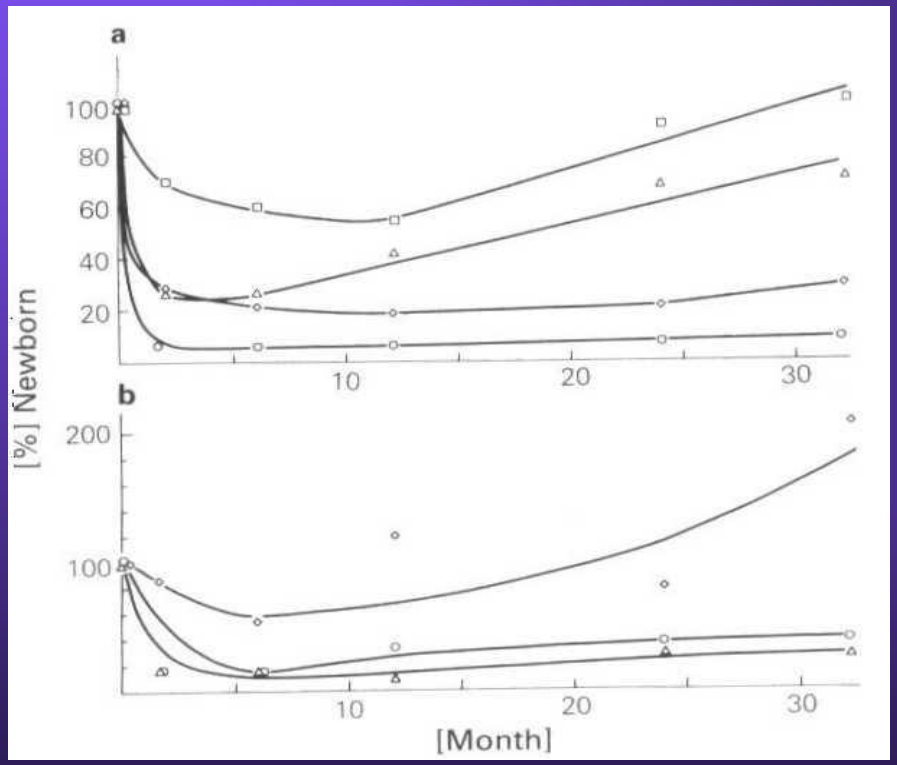
- mag
- citoplazma

ÖREGEDÉSI ELMÉLETEK

III. Kombinált elméletek

Disdifferenciáció hipotézis

c-myc



bőr

máj

vese

agy

vékonybél

lép

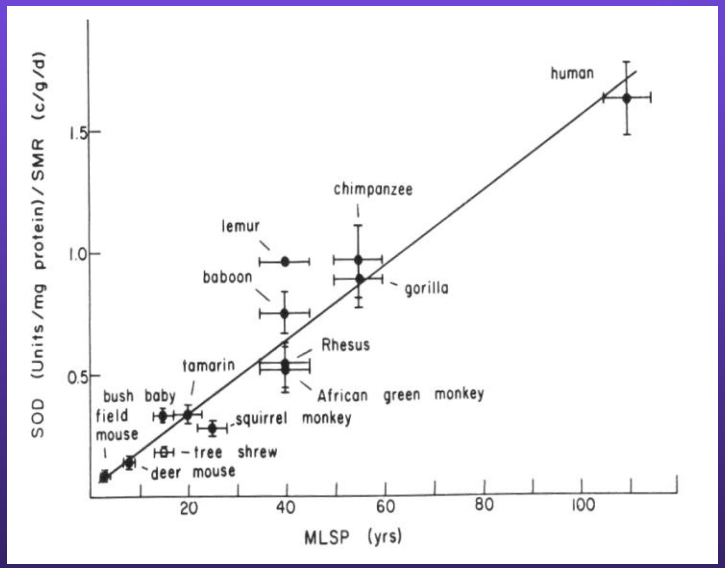
szív

ÖREGEDÉSI ELMÉLETEK

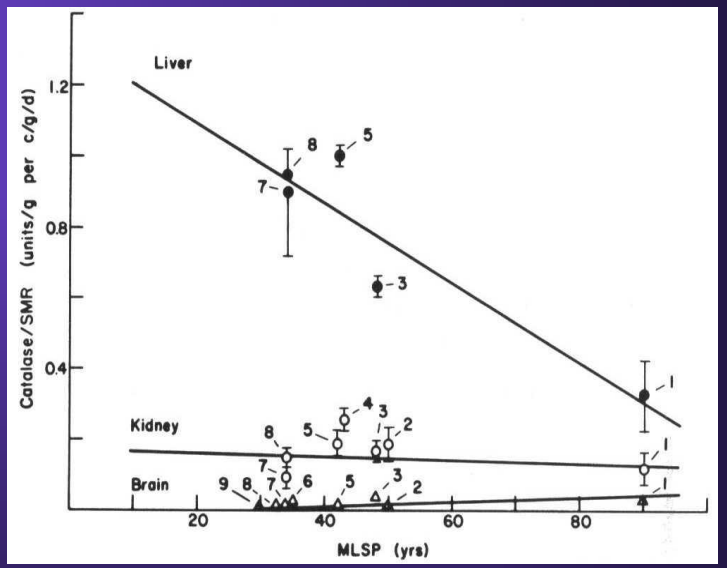
III. Kombinált elméletek

Disdifferenciáció hipotézis

kitüntetett gének - evolúció



szuperoxid dizmutáz



kataláz

ÖREGEDÉSI ELMÉLETEK

III. Kombinált elméletek

Az öregedés membránhipotézise

Liver and Aging – 1990, Kenichi Kitani, editor

A REVIEW ON THE RECENT ADVANCES IN THE MEMBRANE HYPOTHESIS OF AGING

IMRE ZS.-NAGY

F. Verzár International Laboratory for Experimental Gerontology (VILEG), Italian Section, Research Department of I.N.R.C.A., Via Birarelli 8, I-60121 Ancona (Italy)

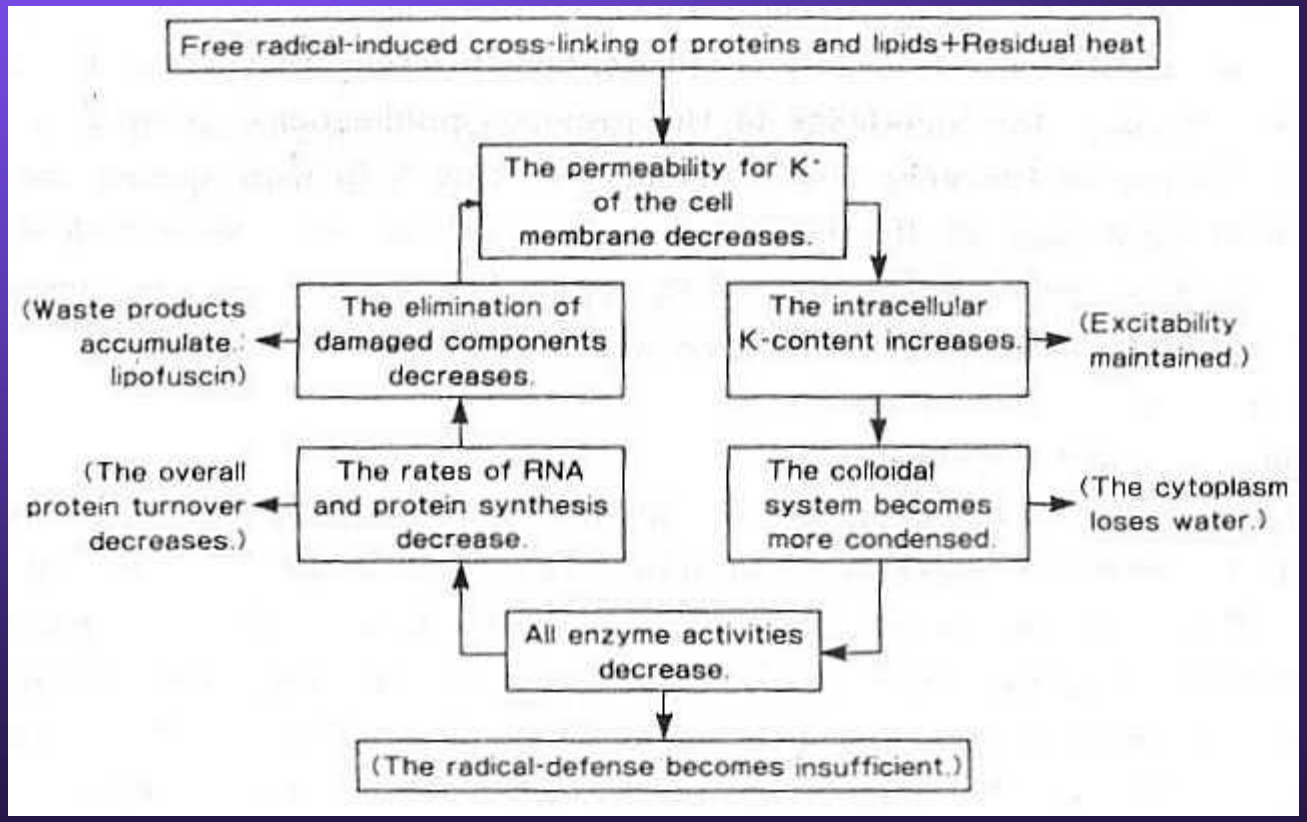
INTRODUCTION

The membrane hypothesis of aging (MHA) explains cell maturation and aging on the basis of the intrinsic biochemical and physicochemical interactions between the cell components and basic oxidative processes of the living systems. This working hypothesis was born in the late seventies (1); it was based on the main achievements of the previous experimental gerontological research, such as the cross-linking theory (2-4), the free radical theory of aging (5, 6), and on the actually known reality of cell physiology. Since its first publication, the MHA has continuously been developed by checking various points of it in

ÖREGEDÉSI ELMÉLETEK

I. Kombinált elméletek

Az öregedés membránhipotézise



ÖREGEDÉSI ELMÉLETEK

I. Kombinált elméletek

Reprinted from Neuroimmunomodulation: Interventions in Aging and Cancer
First Stromboli Conference on Aging and Cancer
Volume 521 of the *Annals of the New York Academy of Sciences*
March 29, 1988

PART VI. PROSPECTS FOR INTERVENTION IN AGING

Dysdifferentiation Hypothesis of Aging and Cancer: A Comparison with the Membrane Hypothesis of Aging

IMRE ZS.-NAGY, RICHARD G. CUTLER,[a]

AZ ÖREGEDÉS EGYESÍTETT ELMÉLETE

SEMSEI IMRE

Debreceni Egyetem

Semsei: *MAD* 117, 93-108 (2000)

AGING THEORIES

PROGRAM THEORIES

INTERNAL PROGRAM

1. run out of program[He]
2. programmed death[W]
3. pleiotrop genes[Wi]
4. programmed cell death[L]
5. biological clocks[E]
6. evolution theories[R]
7. limited number of proliferation[H]

EXTERNAL PROGRAM

1. biological clocks[G]
2. evolution theories[G, Wl]

COMBINED THEORIES

1. unified theories[S]
2. membrane hypothesis[Z]
3. dysdifferentiation hypothesis[Cu]

ERROR THEORIES

SYSTEM

1. disease theories[J]
2. neuroendocrine theories[Ro]
3. immune theories[W]
4. crosslinking theory[B]
5. free radical theory[Ha]
6. waste accumulation[Ca]

MUTATIONS

1. DNA mutations[St]
2. protein utations
3. collagen sslinks[V]
4. sugar crosslinks[C]

Semsei: *MAD* 117, 93-108 (2000)

Felmerül a kérdés:

MI AZ ÖREGEDÉS OKA ?

DNS MUTÁCIÓK

MEMBRÁN KERESZTKÖTÉS

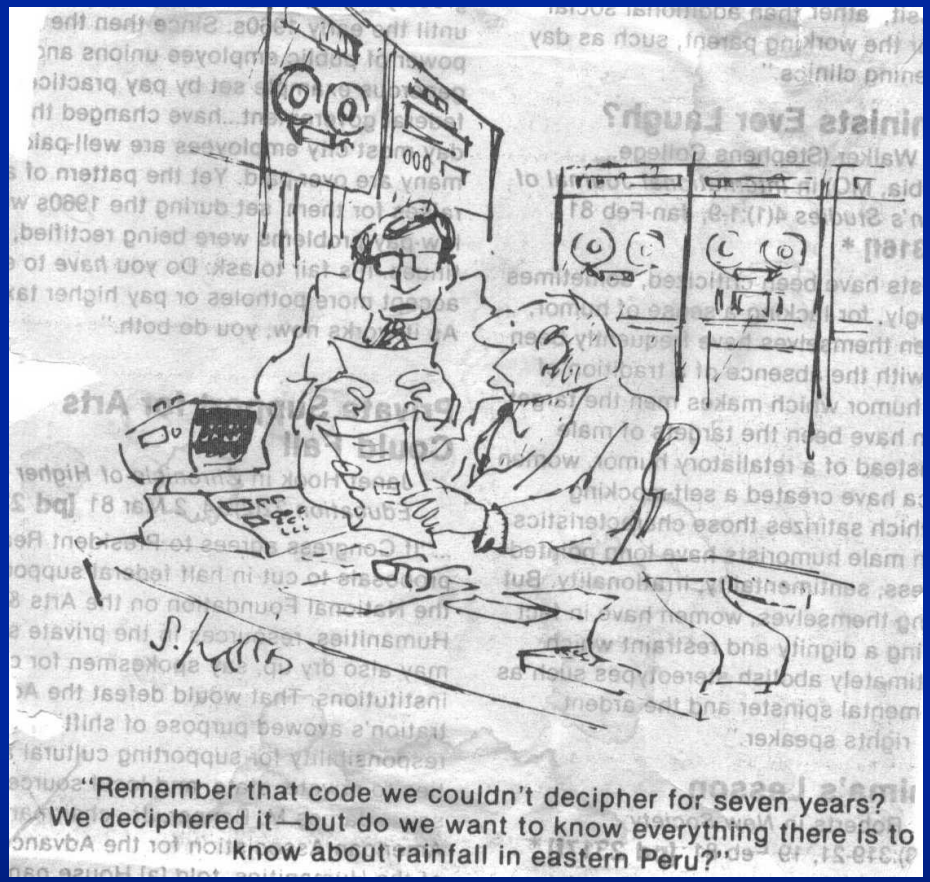
SZABADGYÖKÖK

PROGRAMOZOTT ÖREGEDÉS

STB, STB

Semsei: *MAD* 117, 93-108 (2000)

A ROSSZ KÉRDÉS



6.5.

Semsei: *MAD* 117, 93-108 (2000)

MI AZ ÖREGEDÉS OKA ?

MIK AZ ÖREGEDÉS OKAI?

Ez a helyes kérdés?

Semsei: *MAD* 117, 93-108 (2000)

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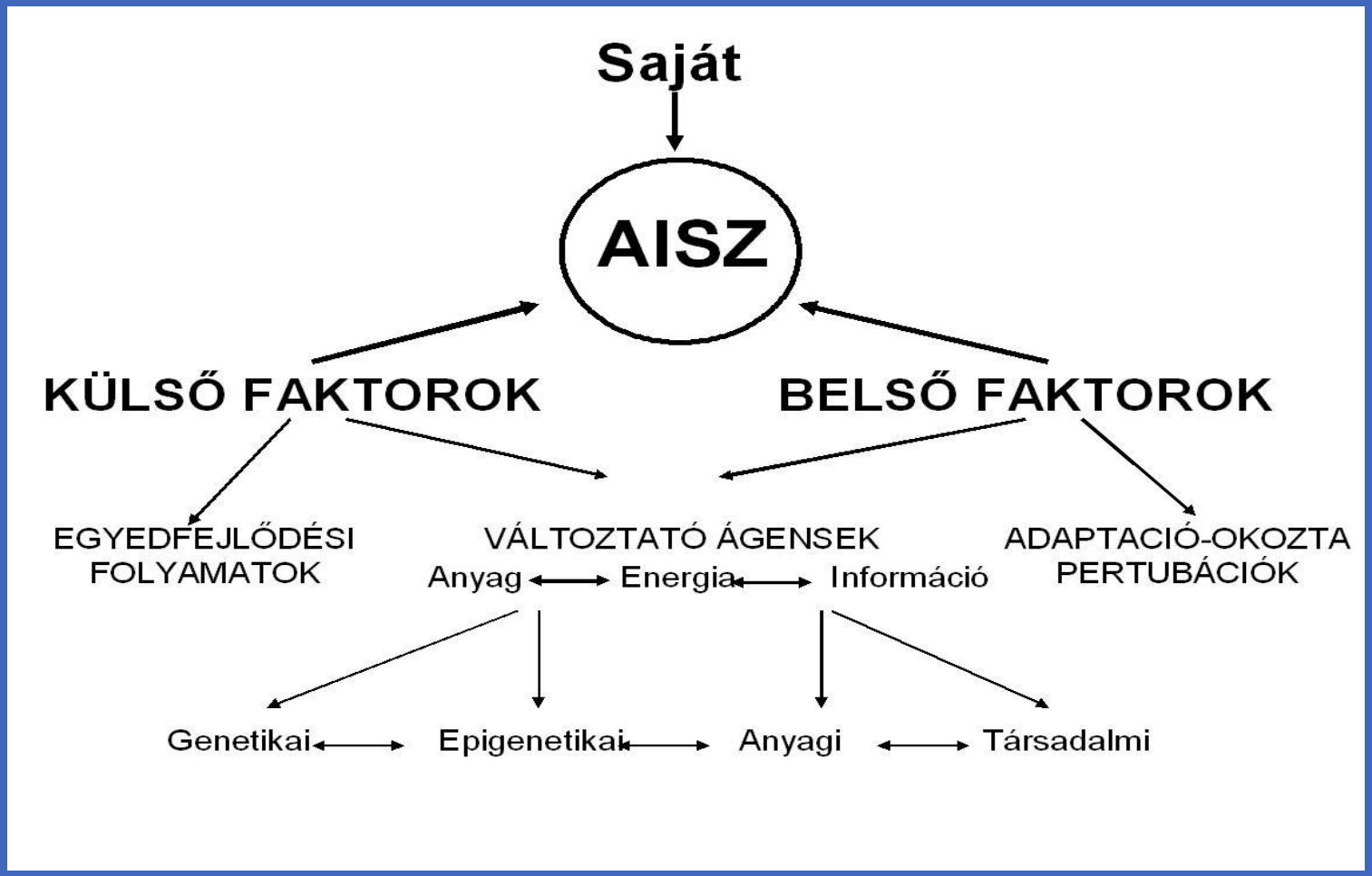
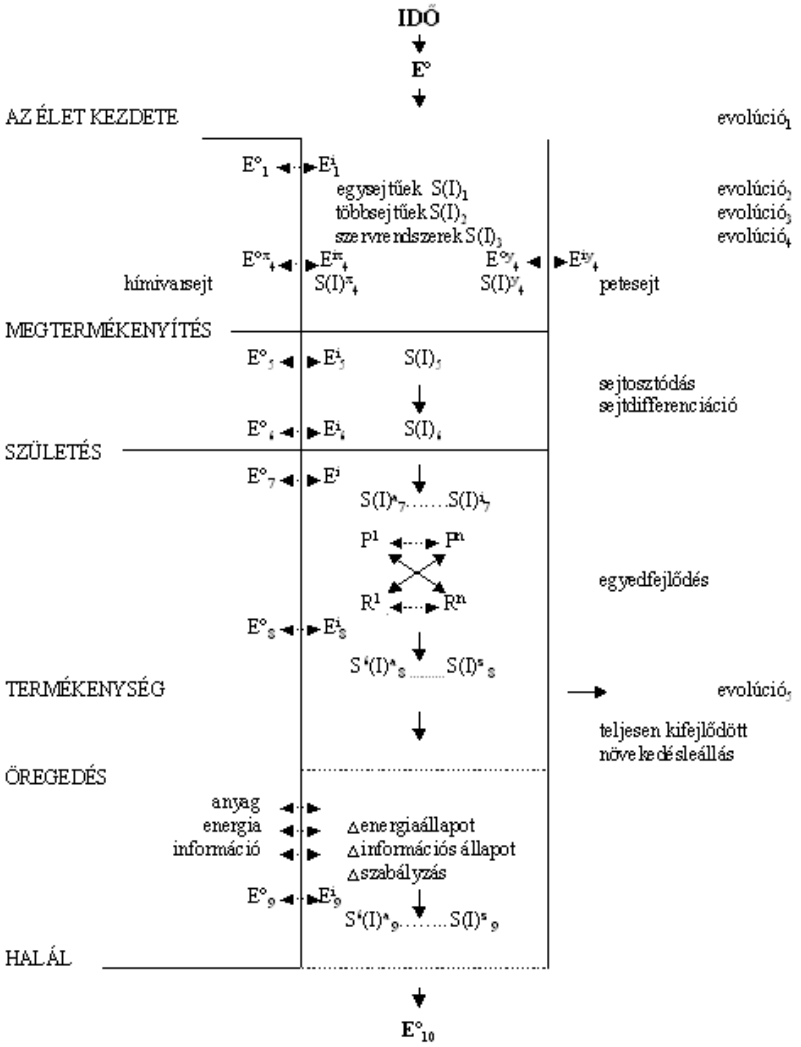


AZ ÖREGEDÉSI FOLYAMAT ALAPVETŐ ZÉNYEZŐI



A SEJT VAGY SZERVEZŐDÉS INFORMÁCIÓS SZINTJEINEK VÁLTOZÁSA AZ ÖREGEDÉS MIATT

BELSŐ ÉS KÜLSŐ
FAKTOROK OKOZTA
KÁROSODÁSOK

PROGRAMOZOTT FOLYAMATOK

AZ ADAPTÁCIÓS FOLYAMATOK OKOZTA
PERTURBÁCIÓK

A BELSŐ INSTABILITÁS OKOZTA VÁLTOZÁSOK

Semsei: *MAD* 117, 93-108 (2000)

A MAXIMÁLIS ÉLETKOR

ALAPVETŐEN MEGHATÁROZZA:	A SZERVEZET INFORMÁCIÓS SZINTJE
BEFOLYÁSOLJA:	KÜLSŐ ÉS BELSŐ FACTOROK
LIMITÁLJA:	A LEGGYENGÉBB LÁNCSZEM

6.7.

Semsei: *MAD* 117, 93-108 (2000)