

Mechanism of aging and prevention

Jay Sik Kim

Department of Clinical Pathology, Kyungpook National University, School of Medicine, Taegu, Korea

Aging is a senescence and defined as a normal physiologic and structural alterations in almost all organ systems with age. As Leonard Hayflick, one of the first gerontologists to propose a theory of biologic aging, indicated that a theory of aging or longevity satisfies the changes of above conditions to be universal, progressive, intrinsic and deleterious. Although a number of theories have been proposed, it is now clear that cell aging (cell senescence) is multifactorial. No single mechanism can account for the many varied manifestations of biological aging. Many theories have been proposed in attempt to understand and explain the process of aging. Aging is effected in individual by genetic factors, diet, social conditions, and the occurrence of age-related diseases as diabetes, hypertension, and arthritis. It involves an endogenous molecular program of cellular senescence as well as continuous exposure throughout life to adverse exogenous influences, leading to progressive infringement on the cell's survivability so called wear and tear. So we could say the basic mechanism of aging depends on the irreversible and universal processes at cellular and molecular level. The immediate cause of these changes is probably an interference in the function of cell's macromolecules-DNA, RNA, and cell proteins-and in the flow of information between these macromolecules. The crucial questions, unanswered at present, concerns what causes these changes in truth. Common theories of aging are able to classify as followings for the easy comprehension. 1. Biological, 1) molecular theories - a. error theory, b. programmed aging theory, c. somatic mutation theory, d. transcription theory, e. run-out-of program theory, 2) cellular theories - a. wear and tear theory, b. cross-link theory, c. clinker theory, d. free radical theory, e. waste product theory, 3) system level theory-a. immunologic/autoimmune theory, 4) others - a. telomere theory, b. rate of living theory, c. stress theory, etc. Prevention of aging is theoretically depending on the cause or theory of aging. However no single theory is available and no definite method of delaying the aging process is possible by this moment. The most popular action is anti-oxidant therapy using vitamin E and C, melatonin and DHEA, etc. Another proposal for the reverse of life-span is TCP-17 and IL-16 administration from the mouse bone marrow B cell line study for the immunoglobulin VDJ rearrangement with RAG-1 and RAG-2. Recently conclusional suggestion for the extending of maximum life-span thought to be the calory restriction.

Key Words: aging mechanism, senescence, aging theory, programmed theory, free radical theory, telomere theory, anti-oxidant therapy, calory restriction, TCP-17, IL-16, RAG-1, RAG-2

(老化, aging)	.	Dorland	DNA	가 , catastrophe가
가	가	.	2.	(somatic mutation theory)(8)
(障碍, deterioration)가	(1, 2).	가,	3.	(transcription theory)(6)
	가 .	가 (senescen-	4.	(programmed senescence theory)(7)
ce)	Leonard Hayflick	가 , 1) universal, 2) progressive, 3) intrinsic, 4) deleterious	DNA T	Hayflick(9)
(cell senescence)	가 (3).	가 가	가 가	(biological clock)
	, ,		가 (6).	
		(4).	5.	(run - out of program theory)(10)
		(macromolecule)	가	
DNA, RNA		(5).	6.	(waste product theory)(11)
(aging mechanism)			lipofuscin	
	가	, 90	10%	
	가			
(6).			7.	(wear and tear theory)(12)
1.	(error theory)(7)		DNA DNA	가 .

8. (cross-linkage theory)(6, 7) 가 , ,
 .
 , , 가
 (18). (premature aging
 syndrome) DNA 가
 elastin collagen 가
 Xeroderma pigmentosum Cockayne syndrome
 가
 DNA DNA 가
 RecQ DNA helicase 가 Werner
 syndrome(WB), Bloom syndrome Rothmund-Thomson
 syndrome (RTS) (18).
 가
9. (, free radical theory)(6, 13)
 가
 , , (element) homeostasis가
 (free electron) homeostenosis , (rate of
 living theory)
 O₂ 가
 HO·, HO^{2·}
 , (stress theory)
 . 가 가
 가
 가 (14). 가 (stem cell)
10. (immunologic theory)(14, 15)
 T B
 (foreign cell)
 가 가 2035 135 가
 T NK T
 가 .
 가 .
 innate immunity (16). . Weksler-
 (26)
11. (17-26) 가 .
 가
 (mitochondria theory) 가
 (telomere theory) 가
 .
 가 Radox (total anti-oxidant capacity)
 (life-span gene) 가
 가
 Wallace(17)가 . 가
 (degenerative 가
 disease) , DNA

- 가
가 가 가
가
가
(19-26).
가
가
VDJ rearrangement
innate immunity
가
가
1. 가
가
가
가
가
TCP-17 IL-16
가
가
가
가
2. 가
(anti-oxidant therapy)
E () C가
, DHEA, superoxide
dismutase (SOD)
가
Caenorhabditis elegans
daf-2 가 가
가
가
가
Weksler T TCP-17 IL-16
VDJ
가
가 가
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