GENESI 5

GENESI 5:21 Enoch aveva sessantacinque anni quando generò Matusalemme.

GENESI 5:22 Enoch camminò con Dio; dopo aver generato Matusalemme, visse ancora per trecento anni e generò figli e figlie.

GENESI 5:23 L'intera vita di Enoch fu di trecentosessantacique anni.

GENESI 5:24 Poi Enoch cammino con Dio e non fu più perché Dio l'aveva preso.

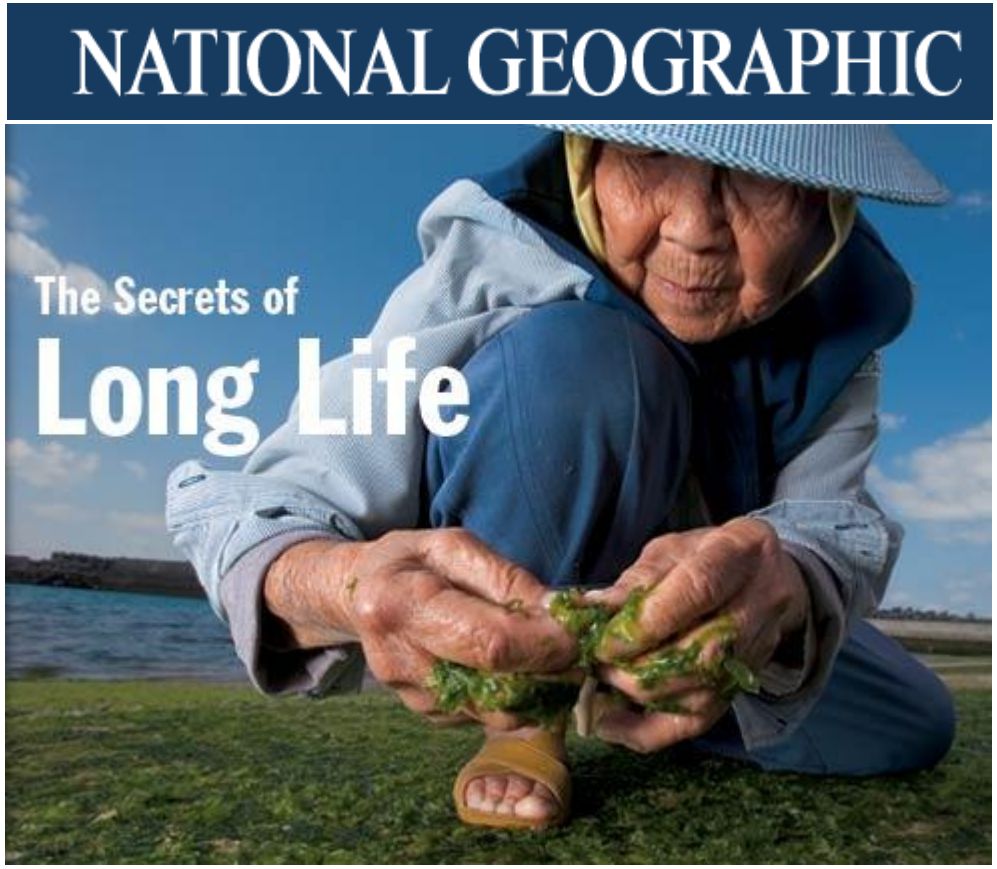
GENESI 5:25 Matusalemme aveva centottantasette anni quando generò Lamech;

GENESI 5:26 Matusalemme, dopo aver generato Lamech, visse ancora settecentottantadue anni e generò figli e figlie.

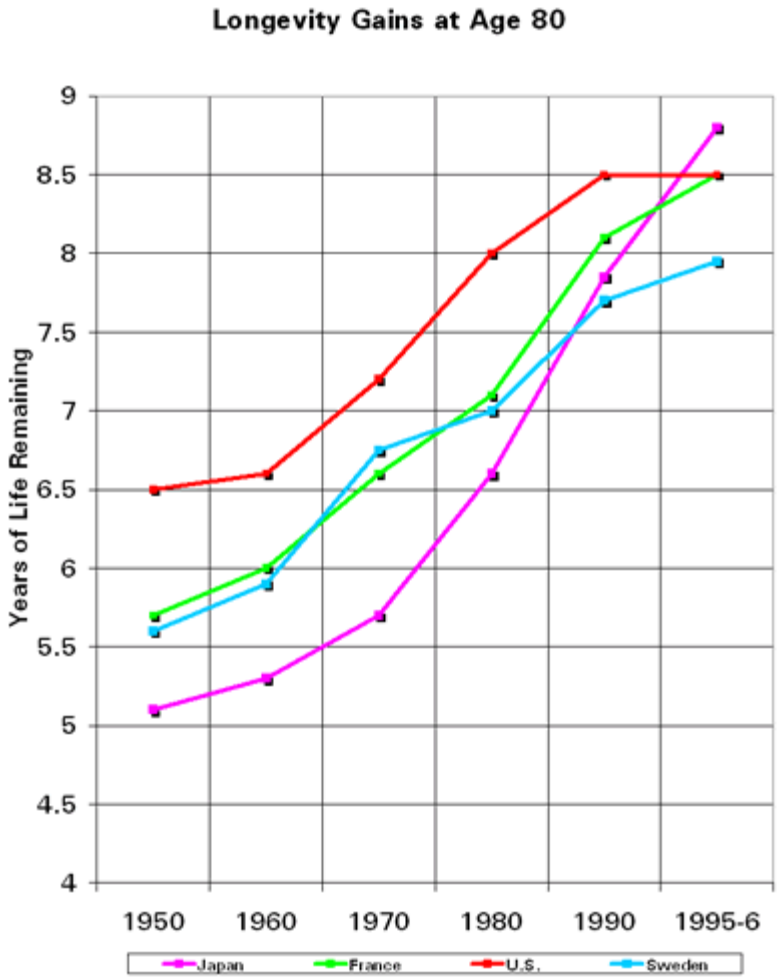
GENESI 5:27 L'intera vita di Matusalemme fu di novecentosessantanove anni; poi morì.

Mikhail Verba, un ricercatore russo di San Pietroburgo appassionato dello studio dei testi biblici, sostiene che Noè aveva circa 60 anni, non 500, quando salpò con l'arca per sottrarsi al diluvio universale, Matusalemme raggiunse la rispettabile età di 120 anni, ma non i 969 che la tradizione gli attribuisce,

nella Bibbia non c'è alcuna esagerazione o simbolismo mistico a livello numerico, basta rileggerla tenendo conto della matematica sumera e tutto appare perfettamente normale



Residents of Okinawa, Sardinia, and Loma Linda, California, live longer, healthier lives than just about anyone else on Earth. What do they know that the rest of us don't?


Longevity Gains at Age 80
Years of Life Remaining
9
8.5
8
7.5
7
6.5
6
5.5
5
4.5
4
1950
1960
1970
1980
1990
1995-6
Japan
France
U.S.
Sweden

Puzzles, promises and a cure for ageing

Jan Vijg[1]† & Judith Campisi[1,2]

Recent discoveries in the science of ageing indicate that lifespan in model organisms such as yeast, nematodes, flies and mice is plastic and can be manipulated by genetic, nutritional or pharmacological intervention. A better understanding of the targets of such interventions, as well as the proximate causes of ageing-related degeneration and disease, is essential before we can evaluate if abrogation of human senescence is a realistic prospect.

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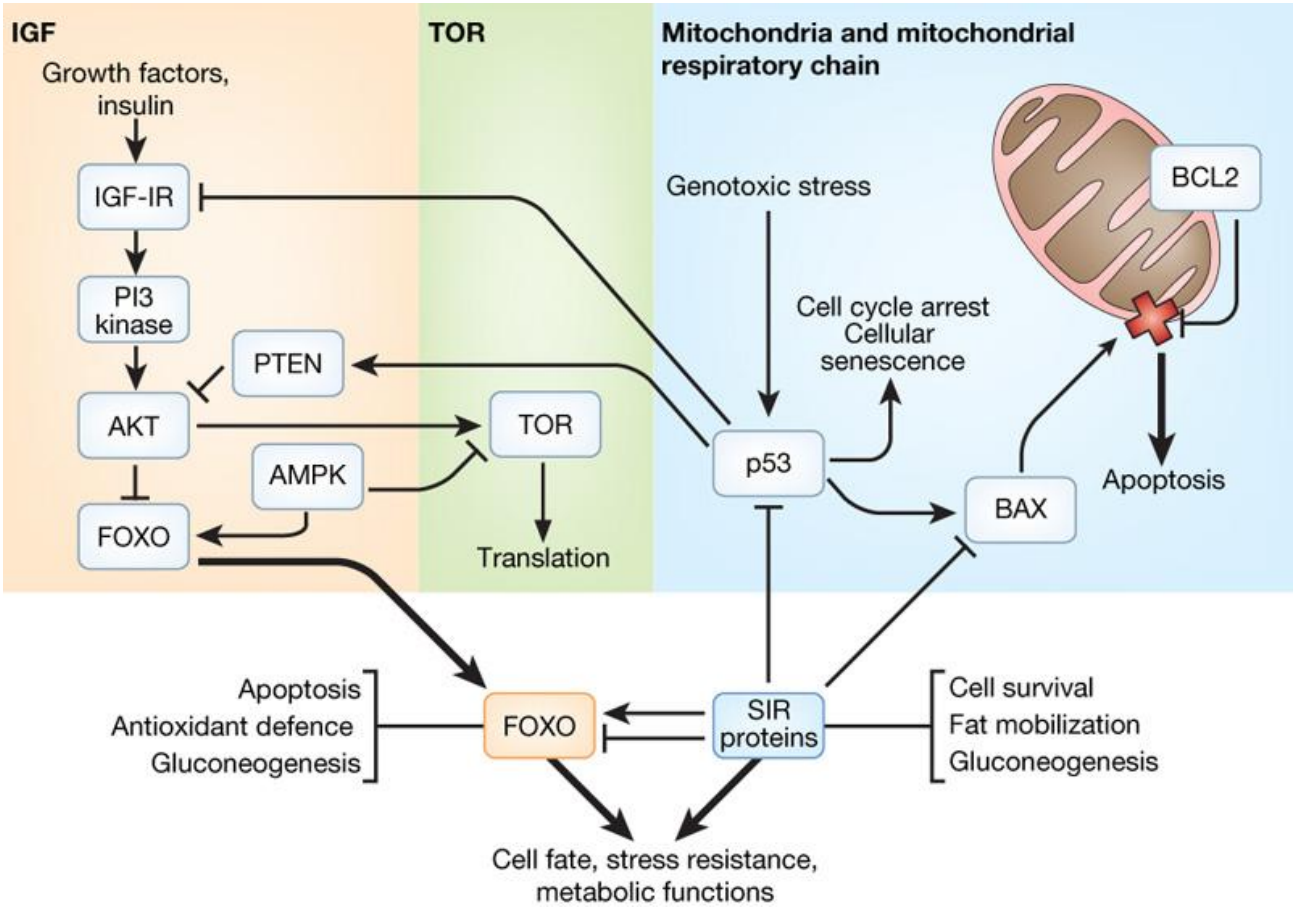
- Future prospects

- Strategies to counter intrinsic aging
- Future Research

Potentially conserved pro-ageing pathways, their interconnections and possible targets for intervention.



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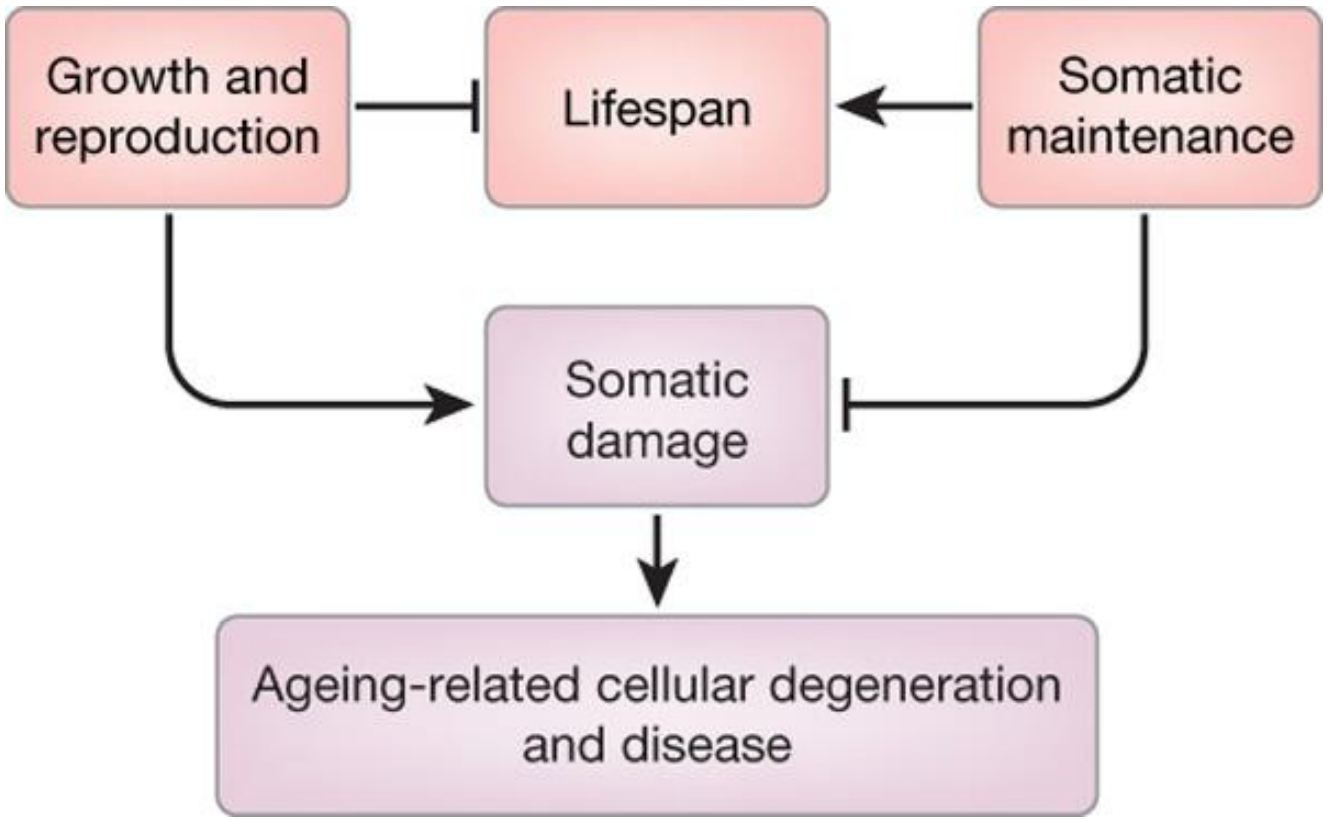
Balancing somatic maintenance with growth and reproduction may determine lifespan.



Conserved ageing phenotypes

Phenotype	*H. sapiens*	*M. musculus*	*D. melanogaster*	*C. elegans*
Decreased cardiac function	Yes	Yes	Yes	NA
Apoptosis, senescence (somatic cells)	Yes	Yes	Yes	?
Cancer, hyperplasia	Yes	Yes	No	No
Genome instability	Yes	Yes	Yes	Yes
Macromolecular aggregates	Yes	Yes	Yes	Yes
Reduced memory and learning	Yes	Yes	Yes	NA
Decline in GH, DHEA, testosterone, IGF	Yes	Yes	?	?
Increase in gonadotropins, insulin	Yes	Yes	?	?
Decreased thyroid function	Yes	Yes	NA	NA
Decrease in innate immunity	Yes	Yes	Yes	Yes
Increase in inflammation	Yes	Yes	No	No
Skin/cuticle morphology changes	Yes	Yes	?	Yes
Decreased mitochondrial function	Yes	Yes	Yes	Yes
Sarcopenia	Yes	Yes	Yes	Yes
Osteoporosis	Yes	Yes	NA	NA
Abnormal sleep/rest patterns	Yes	Yes	Yes	?
Decrease in vision	Yes	Yes	?	NA
Demyelination	Yes	Yes	?	No
Decreased fitness	Yes	Yes	Yes	Yes
Arteriosclerosis	Yes	No	NA	NA
Changes in fat*	Yes	Yes	?	?

*Although changes in fat content and distribution have been reported for long-lived invertebrate mutants, at present there are no data on fat-related changes during normal ageing in these organisms. GH, growth hormone; DHEA, dehydroandrosterone; NA, not applicable.

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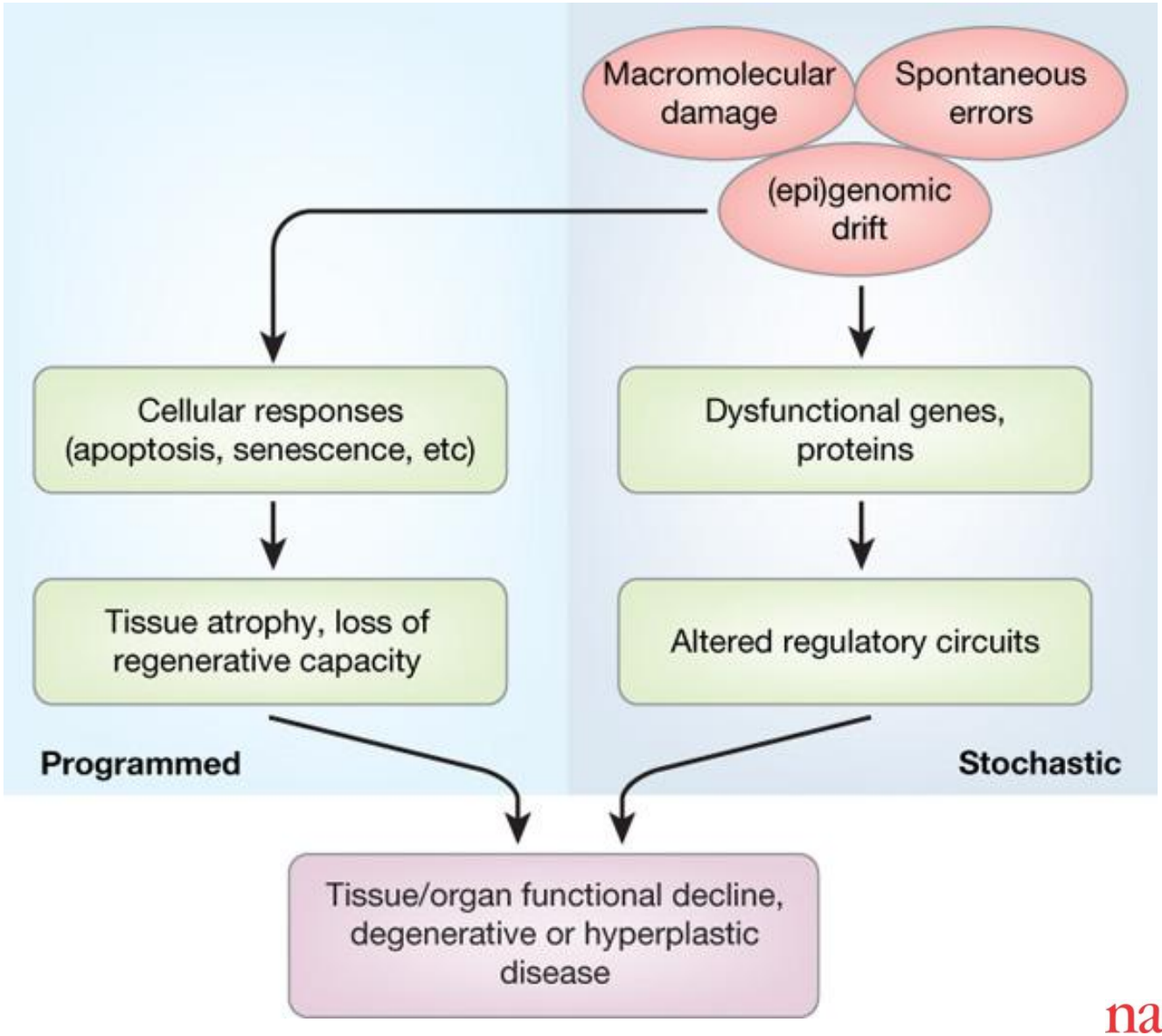
The causes of intrinsic ageing.



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Future research

- Comparative phenotyping
- The aging-disease relationship
- A rational basis for interventions
- Unravel the causes of ageing