

# VANHUUS ON MAHDOLLISUUS

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Sampo - If - 25.9.2013

COSMOLOGY  
Goldilocks Black Holes


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# SCIENTIFIC AMERICAN

January 2012

ScientificAmerican.com



## THE PATHWAY OF YOUTH?

New insights into the body's aging-control system



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# TORm

MECHANISM

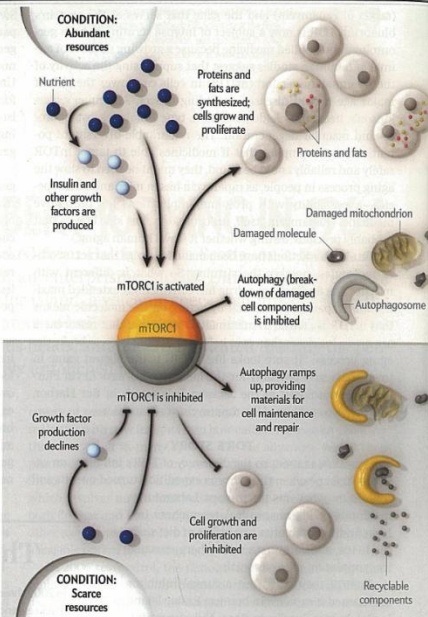
## TOR Story: A Jekyll and Hyde Molecule

Rapamycin extends life in yeast and animals by inhibiting a protein called TOR; calorie restriction, too, slows aging in part by acting on TOR. Research into how the protein functions in cells and into why its inhibition slows aging indicates that TOR is both angelic and diabolical. It is a nutrient sensor critical to organismal growth and development early in life (near right). Yet its continued activity after maturity can impair cell function (far right) and thus damage tissues. Investigators suspect that these late-life effects on TOR contribute to aging and its associated diseases in humans. The figures here, which focus on mammalian TOR (mTOR) are highly simplified; mTOR is affected by and affects a complex network of molecules in cells. (The pointed arrows represent stimulation; the others represent inhibition.)

### The Good Guy: A Key Nutrient Sensor Early in Life

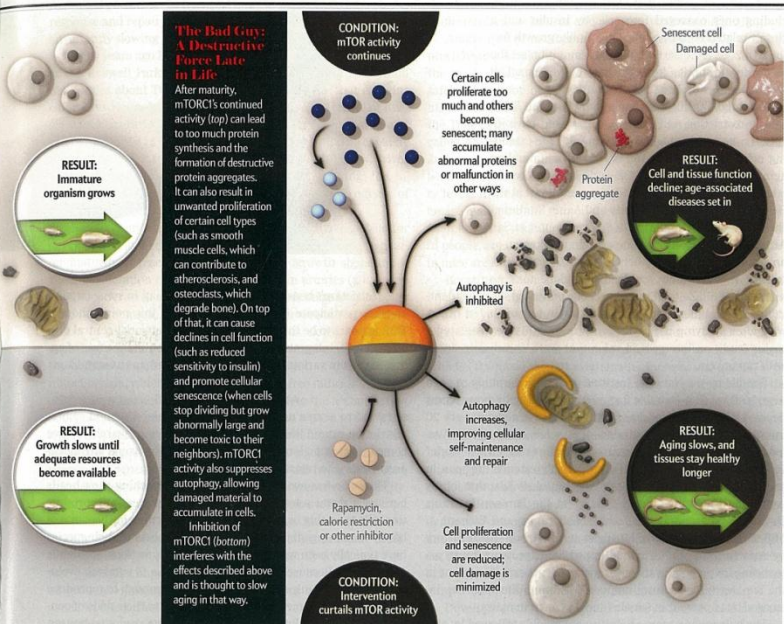
Mammalian TOR exerts many of its effects as part of a complex called mTORC1. When food is plentiful (top), which evokes increased production of insulin and related proteins (known as growth factors), mTORC1 reacts to the nutrients and the growth factors by stimulating the synthesis of cellular components (especially proteins and fat) and prompting cell growth and division. At the same time, the complex instructs cells to pull back on autophagy—a process that degrades damaged mitochondria (the cell's energy factories) and molecules.

When food or other resources are scarce (bottom), mTORC1 quiets down, causing cells to focus on self-preservation over replication. Meanwhile autophagy increases to provide an emergency supply of raw materials for cellular repair and energy generation.



### The Bad Guy: A Destructive Force Late in Life

After maturity, mTORC1's continued activity (top) can lead to too much protein synthesis and the formation of destructive protein aggregates. It can also result in unwanted proliferation of certain cell types (such as smooth muscle cells, which can contribute to atherosclerosis, and osteoclasts, which degrade bone). On top of that, it can cause declines in cell function (such as reduced sensitivity to insulin) and promote cellular senescence (when cells stop dividing but grow abnormally large and become toxic to their neighbors). mTORC1 activity also suppresses autophagy, allowing damaged material to accumulate in cells.



# Albert Einstein

Aika on suhteellista, –  
Niin on ikäkin  
Yksilöitten erot kasvavat iän myötä

- MUUTOKSET

ELINIÄN ODOTUS

VANHUSTEN TERVEYS

IKÄRAKENTEEN MUUTOS

HOITOISUUDEN IKÄRAJAN NOUSU

ISO D

ARVOJEN MUUTOS

VAKA VANHA VÄINÄMÖINEN

TRENDIKÄS POSTMODERNI JUPPI

ONKO MUUTOSTA? (vrt,Einstein & Co)

# BIOLOGIAN ANTAMAT MAHDOLLISUUDET

LÄÄKETIEDE

GENETIIKKA

YMPÄRISTÖ

VANHUKSET

ELÄMISEN MAHDOLLISUUDET

EIVÄT VÄLTTÄMÄTTÄ RIIPU FYSIOLOGIASTA!!!

KOKEMUS

SOSIAALISET SUHTEET

UUDET INTRESSIT

HARRSTUTOIMINTA

YHTEISÖLLISYYS



MIKÄ ON TÄRKEÄÄ?????????

OMA ASENNE

ITSELLE LUOTU KUVA VANHUUDESTA

AIVOT

TUNTEET

FYSIIKKA

KIPUKYNNYKSEN NOSTAMINEN

SIETORAJAN NOSTAMINEN

I L O

K E I N O T

Anna arvo itsellesi, voidaksesi arvostaa muita

Olet loppuun asti itsestäsi vastuullinen viisas kansalainen  
oman elämän paras asiantuntija

LIIKU JA LIKUTTELE PIENIÄ HARMAITA AIVOSOLLUJA  
HARJOITUS TEKEE MESTARIN

SYÖ JA JUO VIISAASTI

KOSKAAN EI OLE LIIAN MYÖHÄISTÄ ALOITTAU UUSI SUHDE  
UUSI HARRASTE, UUSI VAIHE ELÄMÄSSÄ

OLET VASTUUSSA VAIN ITSELLESI

T O I V O A T A I P A L E E L L E !

