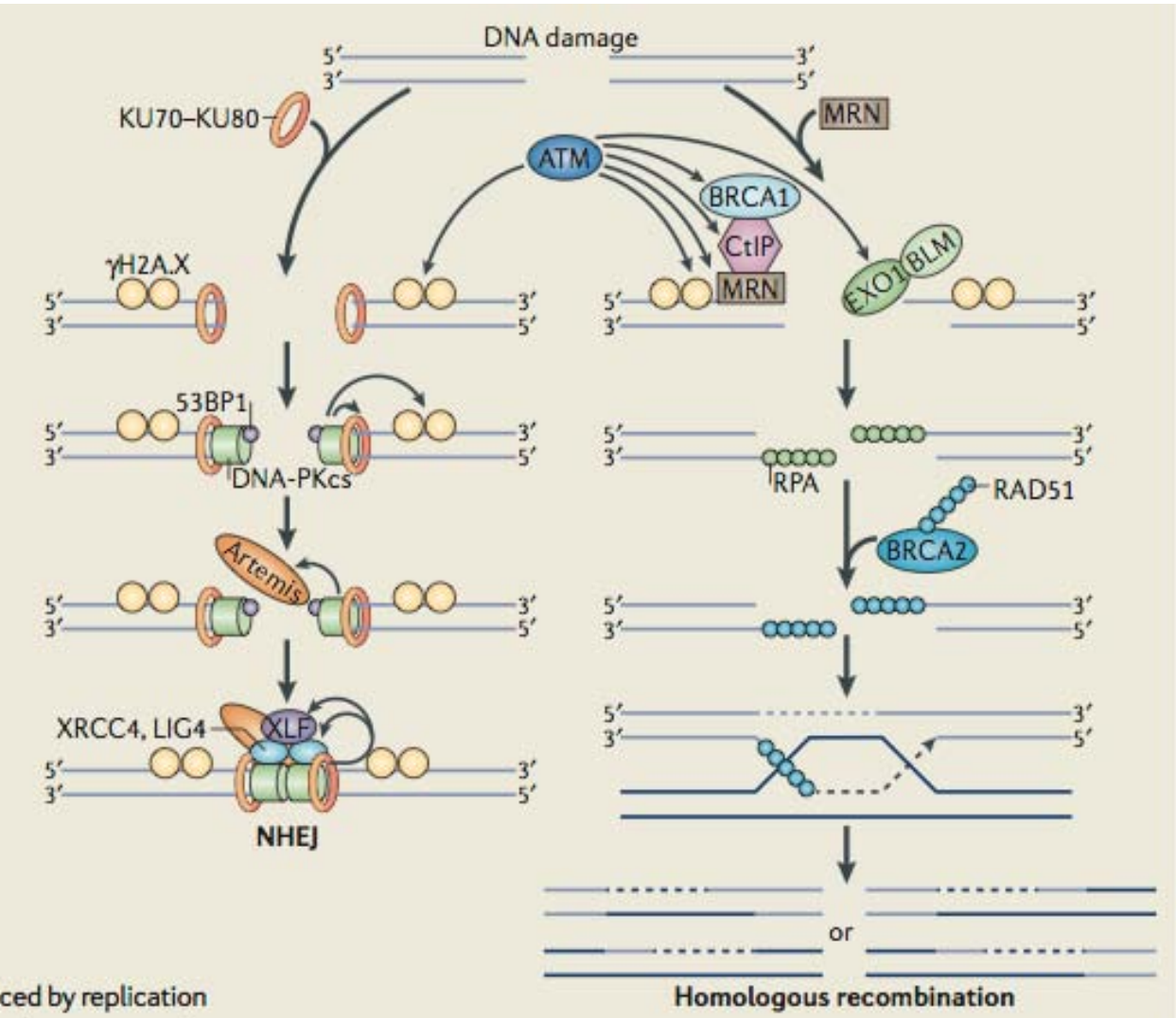
Cours du 21-10-2013

Longévité cérébrale

Charity begins at home: non-coding RNA functions in DNA repair

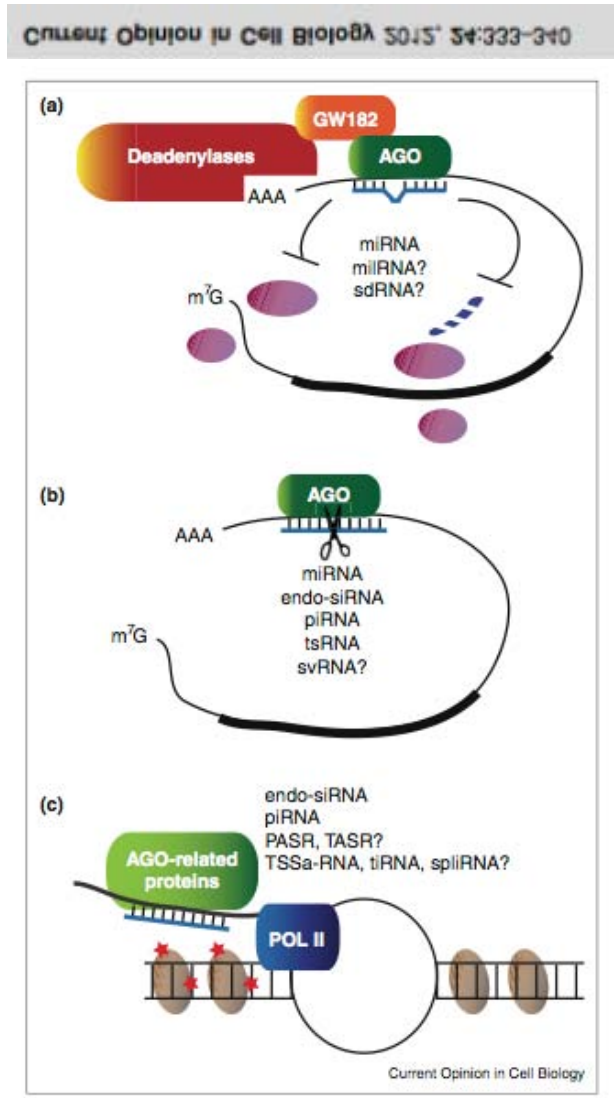
Dipanjan Chowdhury, Young Eun Choi and Marie Eve Brault

Nature Reviews Molecular Cell Biology
2013 vol. 14 (3) pp. 181–9



Small non-coding RNAs mount a silent revolution in gene expression

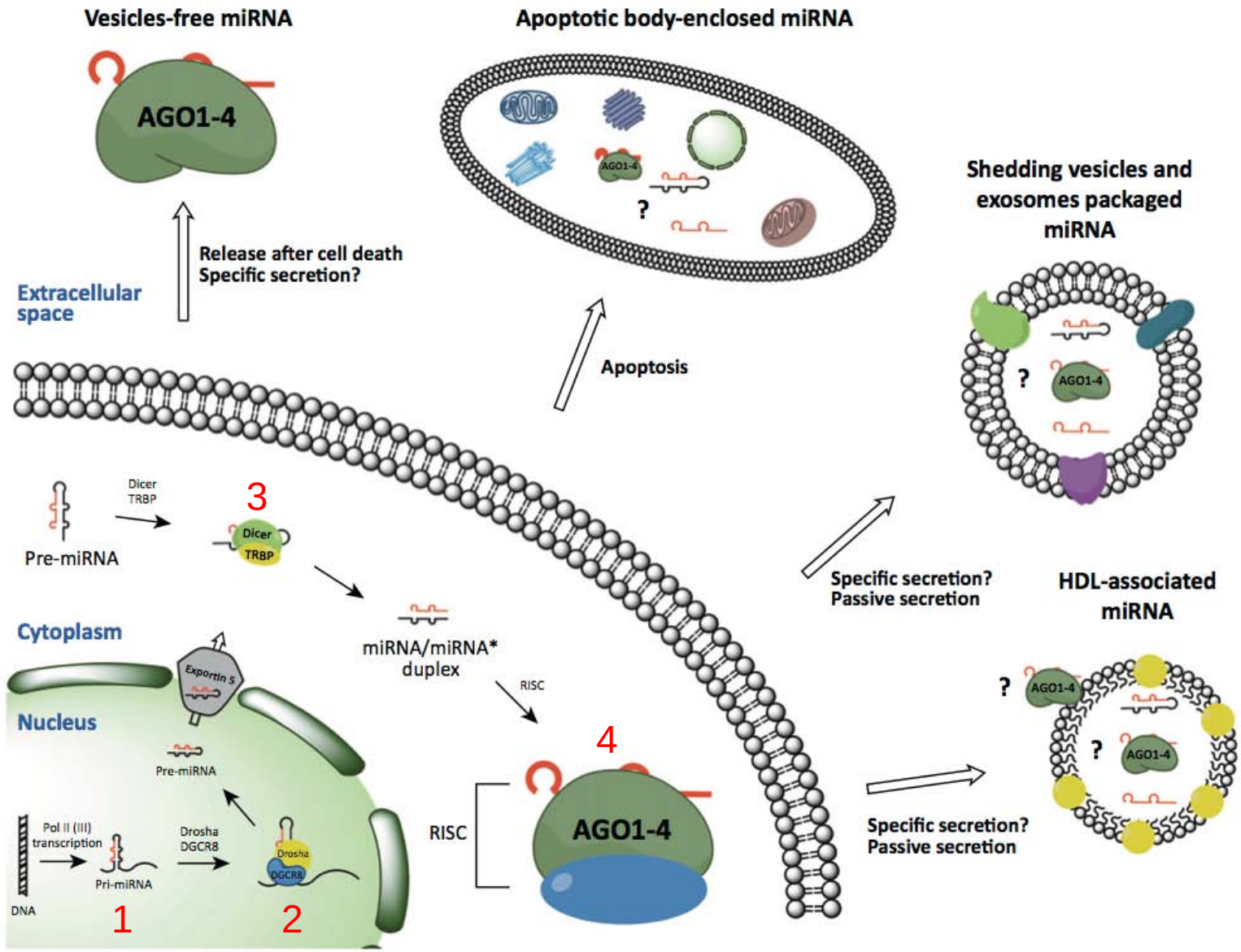
Antti P Aalto[1,a] and Amy E Pasquinelli[2]



Extracellular miRNAs: the mystery of their origin and function

Trends Biochem Sci
2012 vol. 37 (11) pp. 460–5

Turchinovich A, Weiz L, Burwinkel B



Membrane vesicles as conveyors of immune responses

Clotilde Théry, Matias Ostrowski and Elodie Segura

NATURE REVIEWS | IMMUNOLOGY VOLUME 9 | AUGUST 2009 | 581

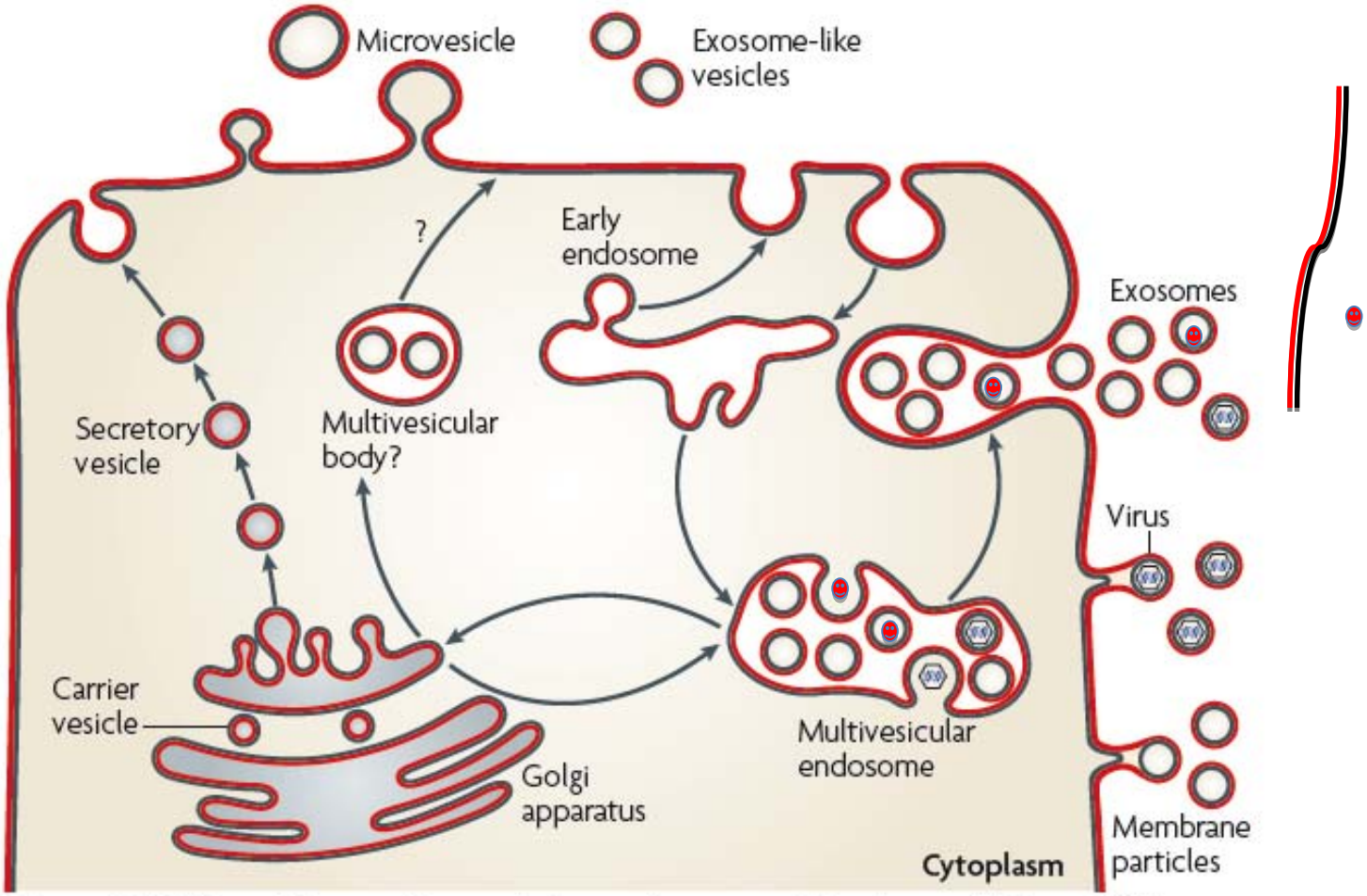


Figure 1 | **Different types of secreted membrane vesicles.** Intracellular trafficking

miRNA response to DNA damage

Trends in Biochemical Sciences, September 2011, Vol. 36, No. 9

Guohui Wan[1], Rohit Mathur[1], Xiaoxiao Hu[1], Xinna Zhang[2] and Xiongbin Lu[1]

Table 1. miRNAs target key genes involved in the DNA damage response

Targets	Function in DNA damage response	miRNAs	Refs.
ATM	Mediator/transducer	miR-421	[27]
H2AX	Mediator, DNA repair	miR-24	[28]
RAD52	DNA repair	miR-210, miR-373	[63]
RAD23B	DNA repair	miR-373	[63]
MSH2	DNA mismatch repair	miR-21	[64]
BRCA1	DNA repair	miR-182	[65]
p53	Cell cycle checkpoint, apoptosis	miR-504, miR-125b	[30,31]
p63	Transcription factor	miR-92, miR-302	[32,33]
E2F	Transcription factor	miR-17-92, miR-20a, miR-34a	[66,67]
p21	Cell cycle	miR-17, miR-20a/b, miR-106a/b, miR-93, miR-215, miR-192	[35,68]
CDK2	Cell cycle	miR-124a, miR-885-5p	[69,70]
CDK6	Cell cycle	miR-124a, miR-29, miR-449a/b	[71–73]
Cdc25A	Cell cycle checkpoint	miR-21, miR-449a/b	[73,74]
Cdc42	Cell cycle checkpoint	miR-29	[75]
Cyclin E	Cell cycle	miR-15a, miR-16	[76,77]
Cyclin D	Cell cycle	miR-15a, miR-16	[78]
Cyclin G1	Cell cycle	miR-122	[79]
Wee1	Cell cycle checkpoint	miR-195	[80]
p27	Cell cycle	miR-221/222, miR-181	[81,82]
p57	Cell cycle	miR-221/222	[81]
Wip1	Cell cycle checkpoint	miR-16	[61]
Bcl-2	Apoptosis	miR-15a, miR-16-1	[83]

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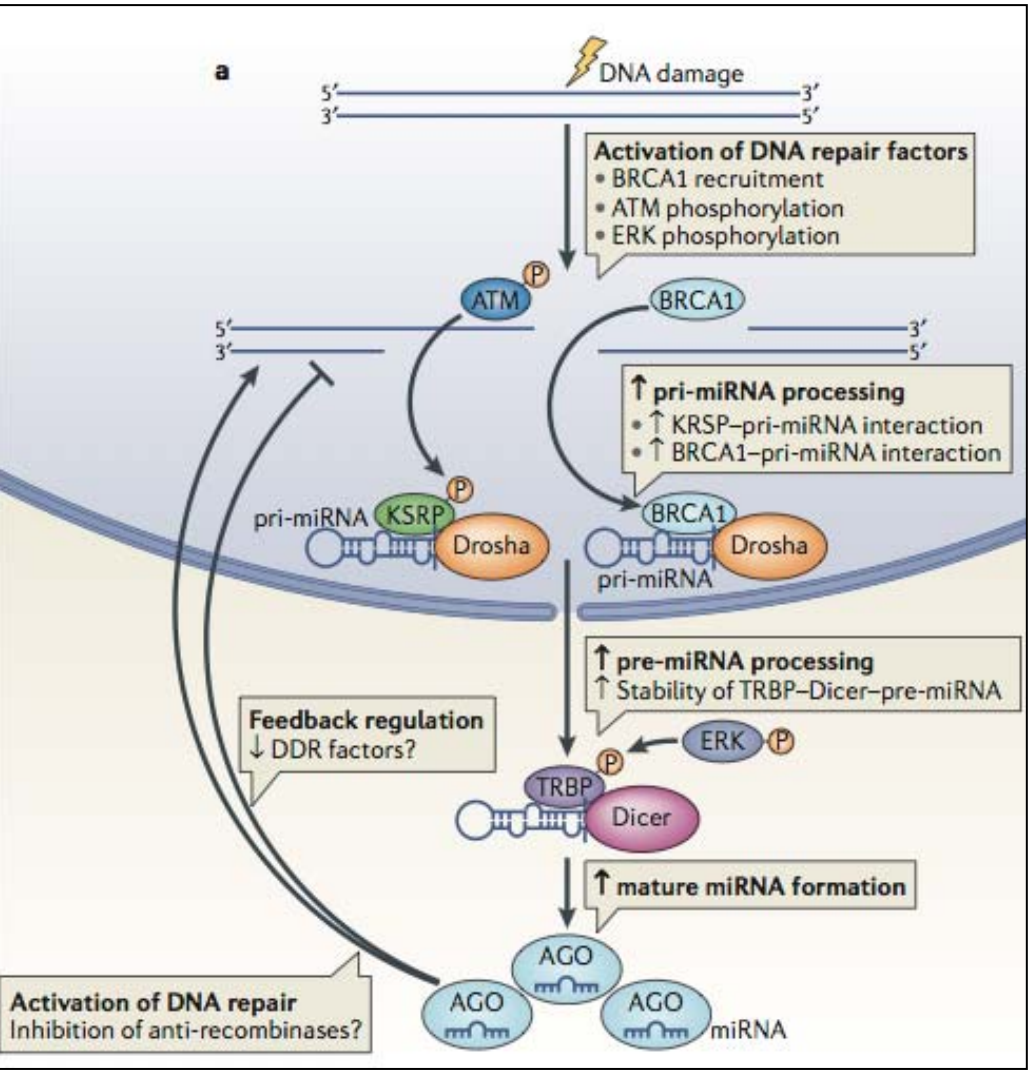

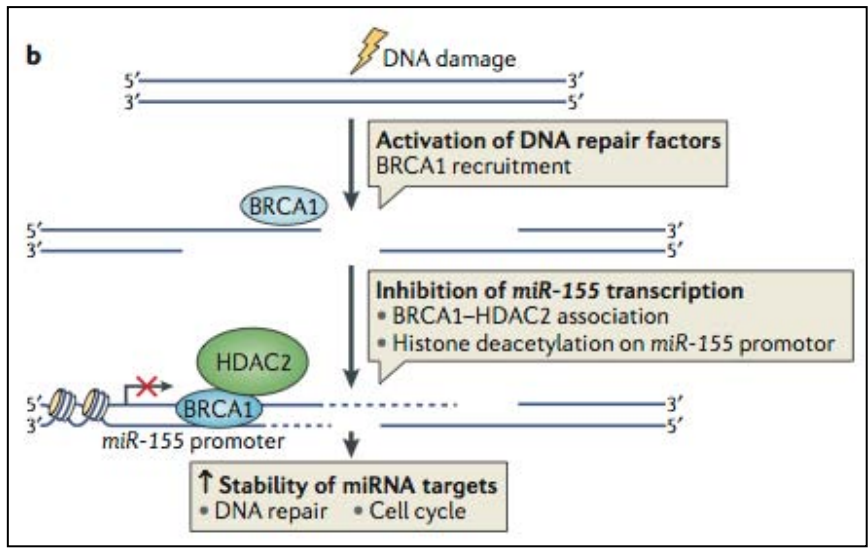

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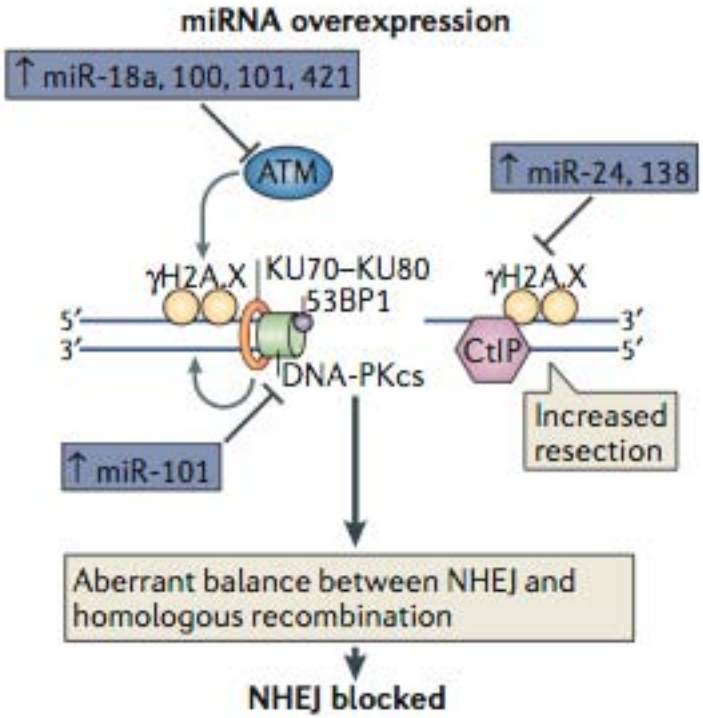

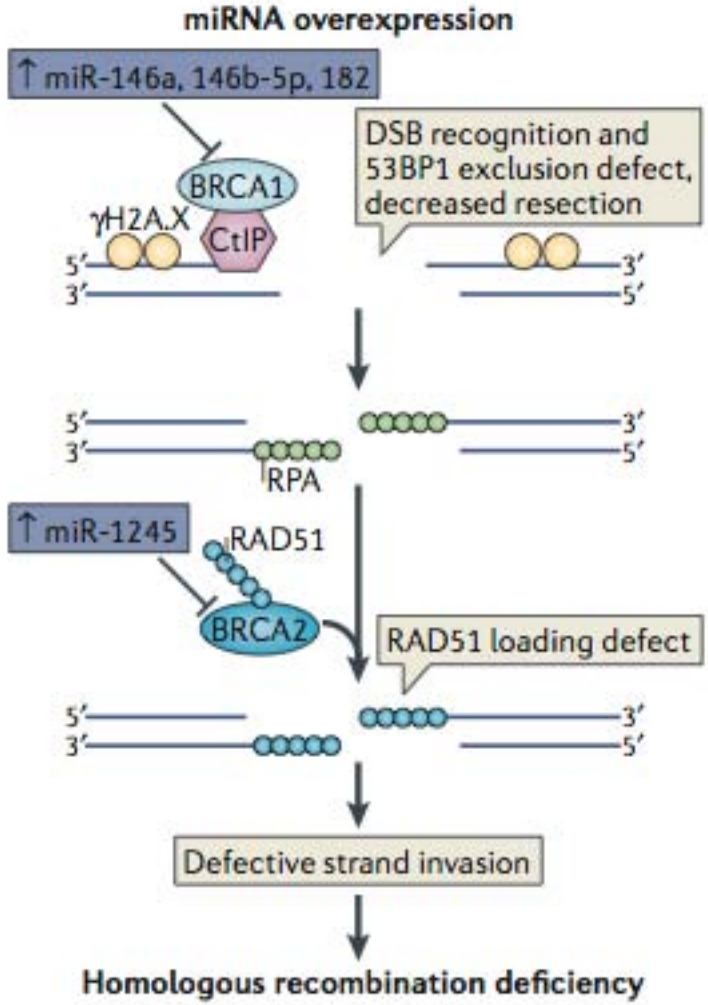

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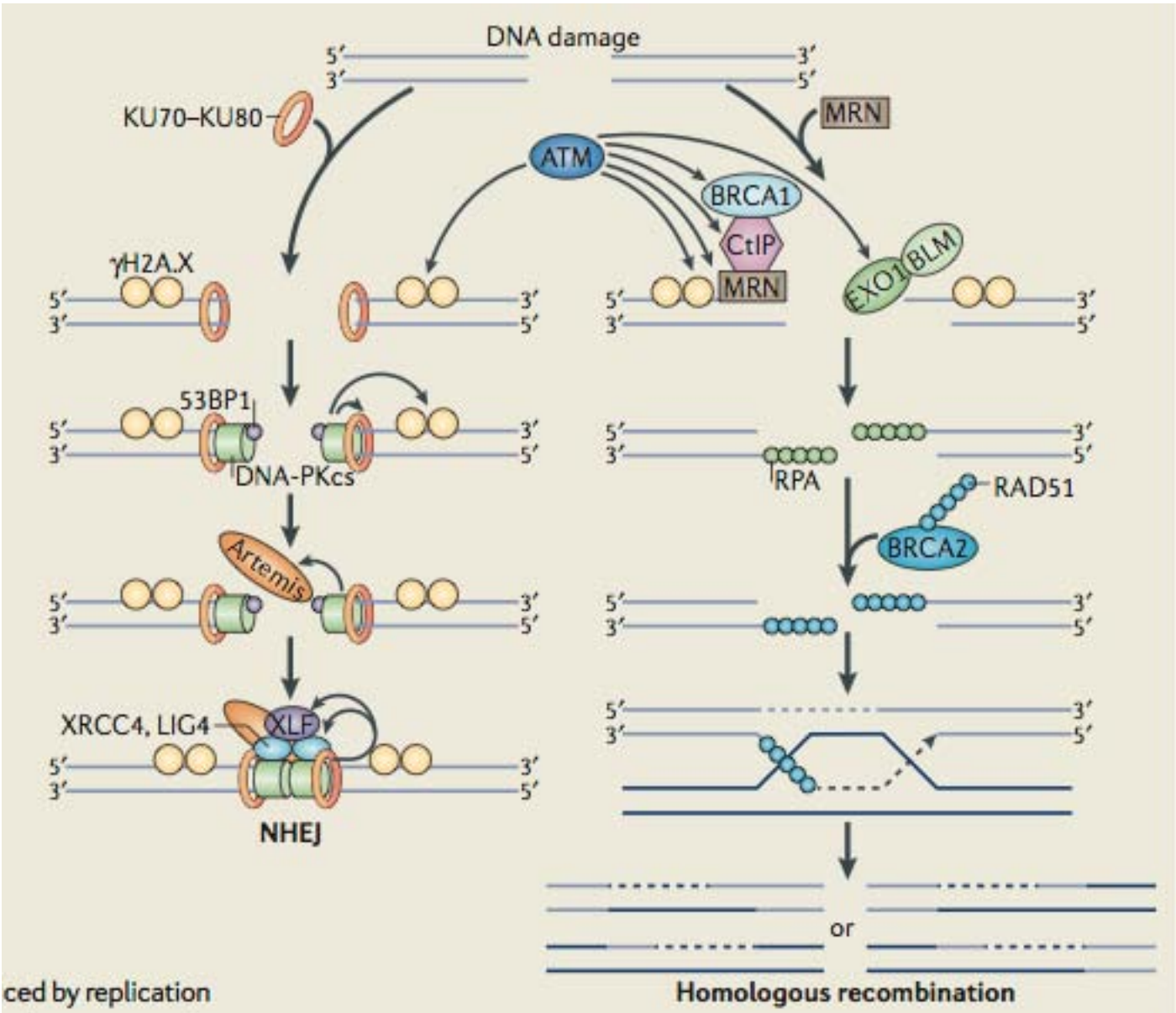
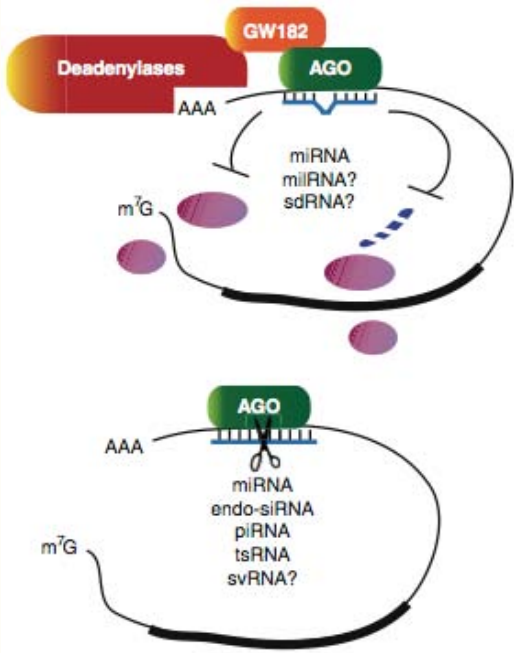
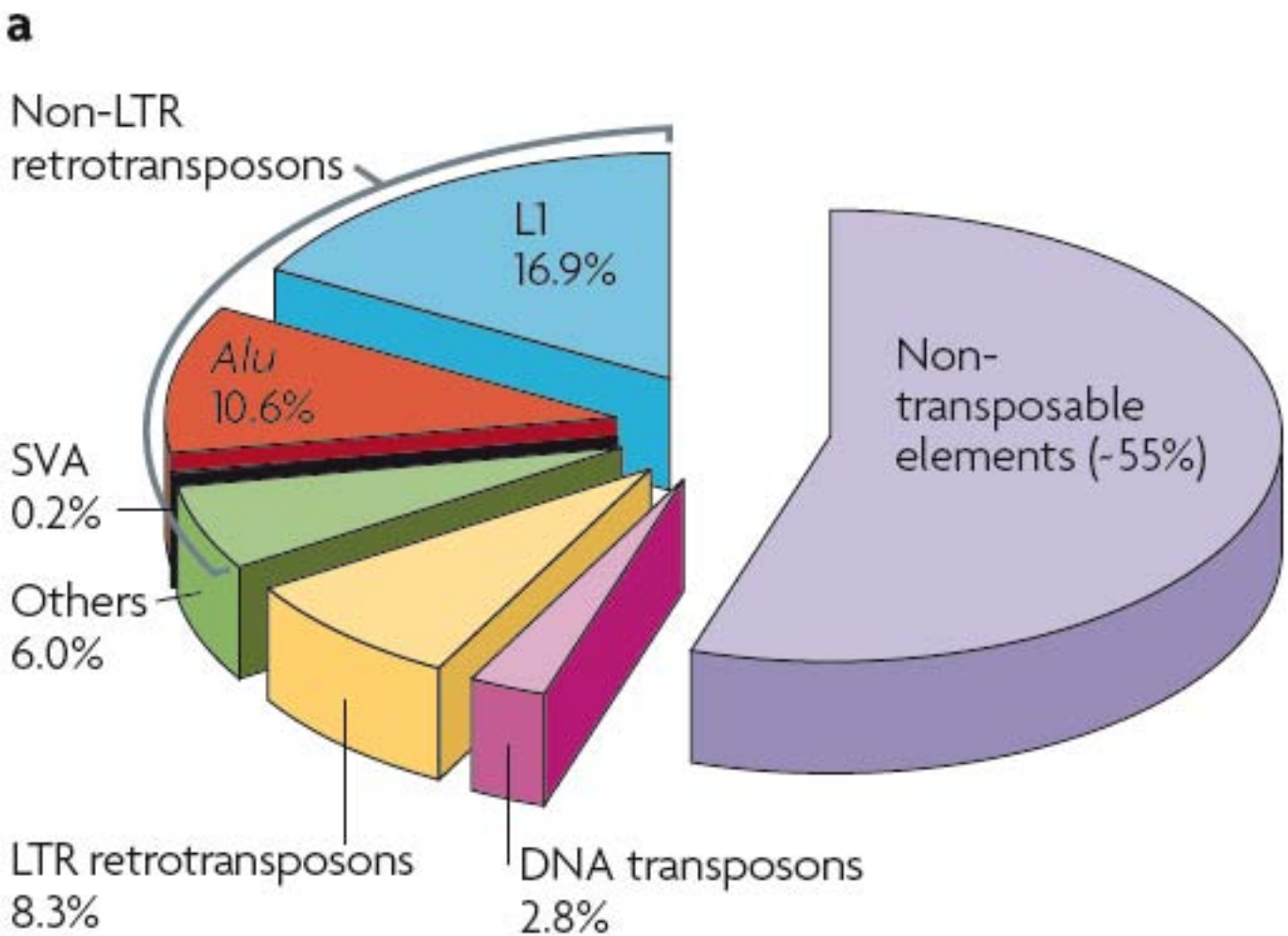




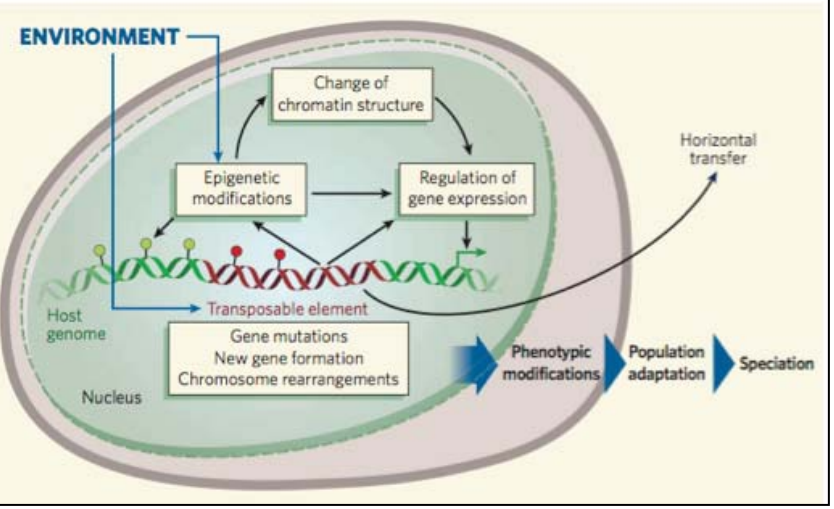
Cordaux @ Batzer, Nature Reviews genetics, 10: 691-703, 2009



Junk DNA as an evolutionary force

Christian Biémont and Cristina Vieira

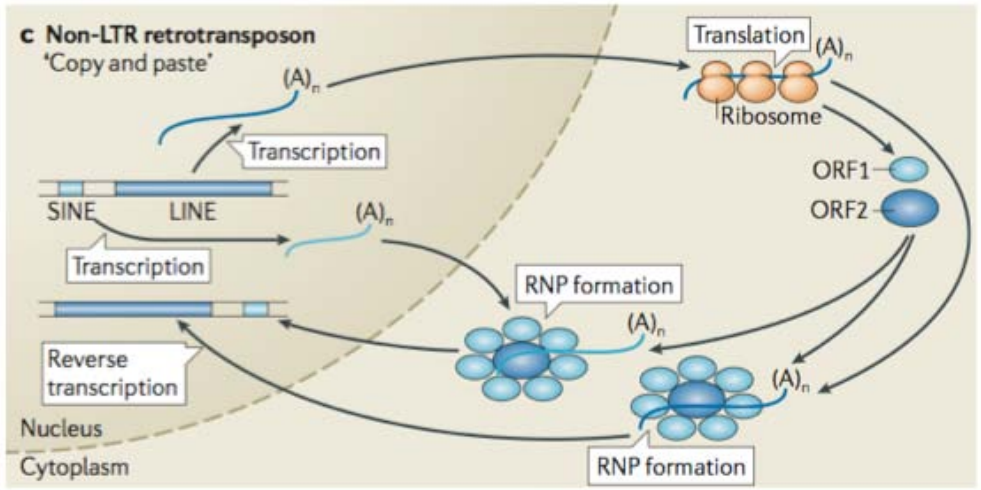
Vol 443|5 October 2006 nature



PIWI-interacting small RNAs: the vanguard of genome defence

Nat Rev Mol Cell Biol
2011 vol. 12 (4) pp. 246-58

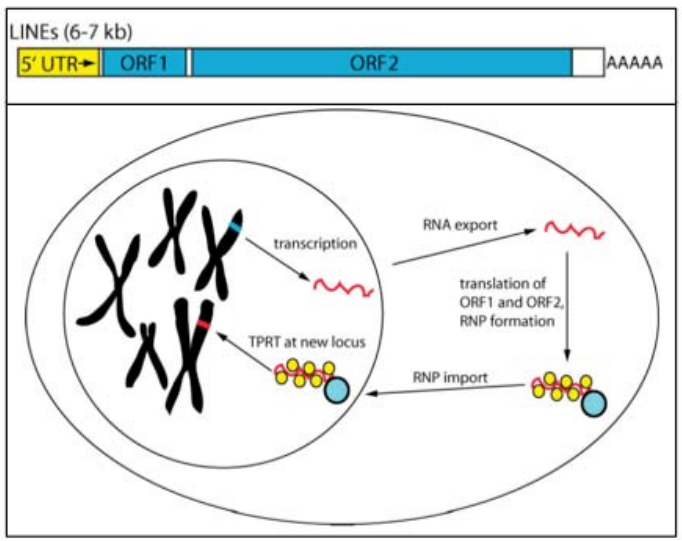
Siomi MC, Sato K, Pezic D, Aravin AA



LINE-1 retrotransposons: modulators of quantity and quality of mammalian gene expression?

Jeffrey S. Han and Jef D. Boeke*

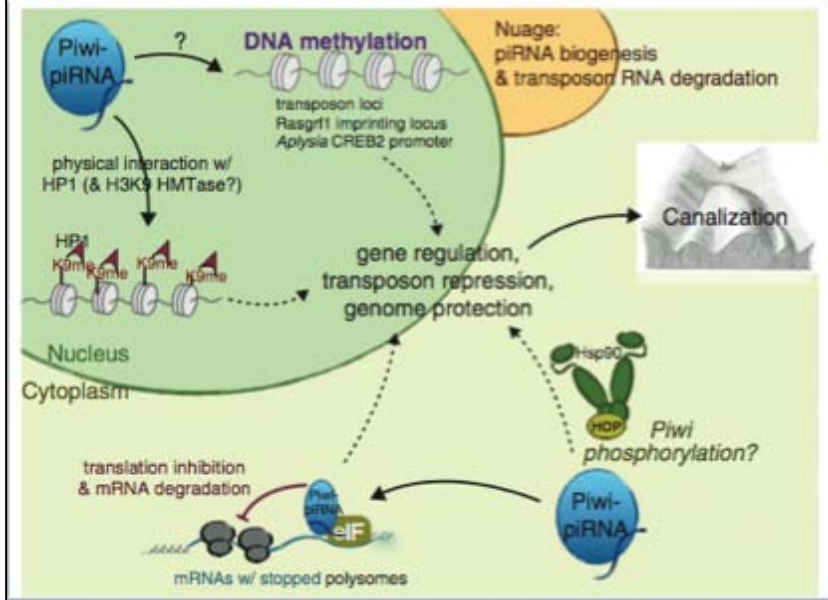
BioEssays 27:775–784, © 2005



Beyond transposons: the epigenetic and somatic functions of the Piwi-piRNA mechanism

Current Opinion in Cell Biology
2013 vol. 25 (2) pp. 190–4

Peng JC, Lin H



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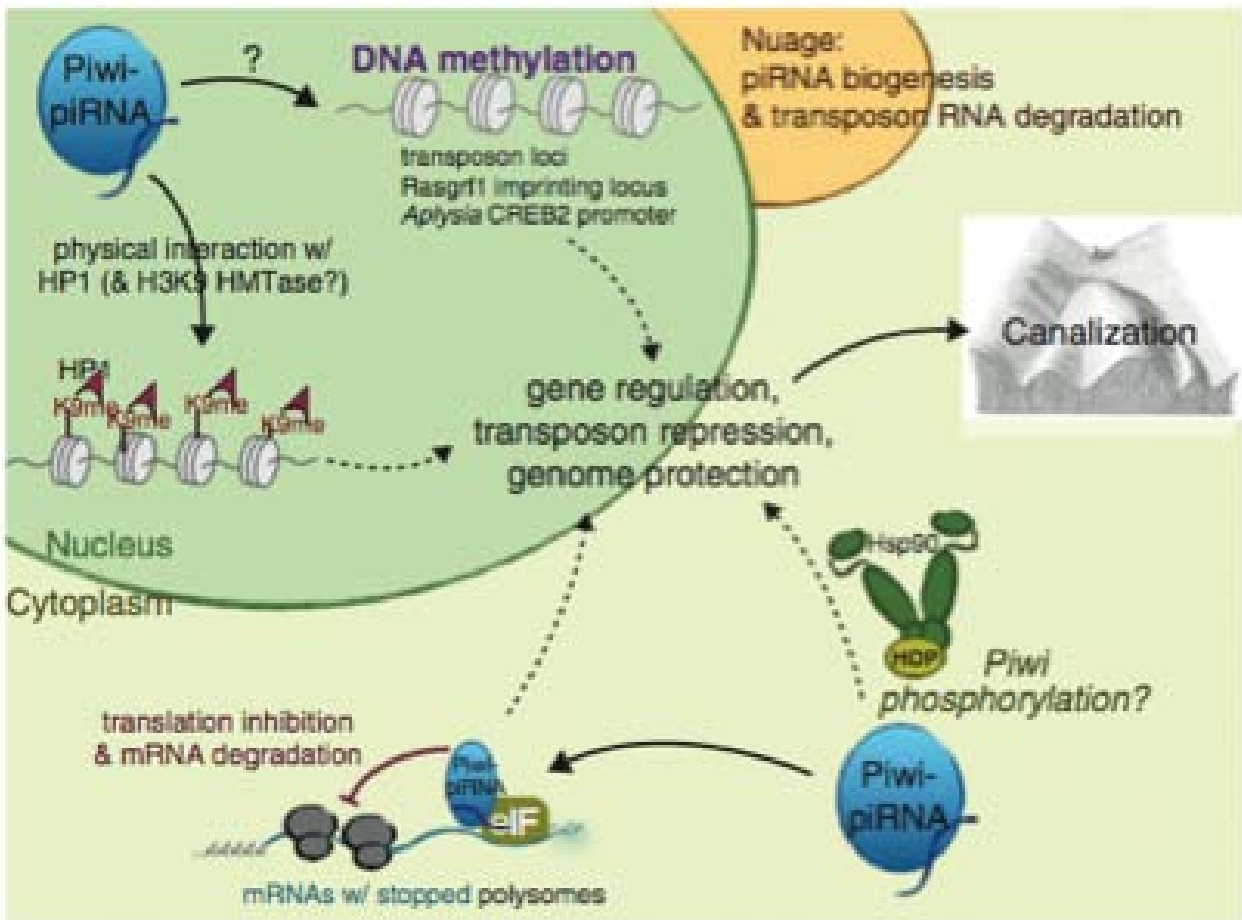

A LINE-1 component to human aging do LINE elements exact a longevity cost for evolutionary advantage?

St Laurent G, Hammell N, Mccaffrey TA

Mech Ageing Dev
2010 vol. 131 (5) pp. 299-305

Activation of transposable elements during aging and neuronal decline in Drosophila

Li W, Prazak L, Chatterjee N, Grüninger S, Krug L, Theodorou D, Dubnau J

Nat Neurosci
2013 vol. 16 (5) pp. 529-31

L1 retrotransposition in nondividing and primary human somatic cells

Kubo S, Seleme MC, Soifer HS, Perez JL, Moran JV, Kazazian HH, Kasahara N

Proc Natl Acad Sci USA
2006 vol. 103 (21) pp. 8036-41

Active human retrotransposons: variation and disease

Hancks DC, Kazazian HH

Current Opinion in Genetics & Development
2012 vol. 22 (3) pp. 191-203

Somatic expression of LINE-1 elements in human tissues

Belancio VP, Roy-Engel AM, Pochampally RR, Deininger P

Nucleic Acids Research
2010 vol. 38 (12) pp. 3909-22

DICER1 deficit induces Alu RNA toxicity in age-related macular degeneration

Kaneko H, Dridi S, Tarallo V, Gelfand BD, Fowler BJ, Cho WG, Kleinman ME, Ponicsan SL, Hauswirth WW, Chiodo VA, Kariko K, Yoo JW, Lee D, Hadziahmetovic M, Song Y, Misra S, Chaudhuri G, Buaas FW, Braun RE, Hinton DR, Zhang Q, Grossniklaus HE, Provis JM, Madigan MC, Milam AH, Justice NL, Albuquerque RJC, Blandford AD, Bogdanovich S, Hirano Y, Witta J, Fuchs E, Littman D, Ambati BK, Rudin CM, Chong MMW, Provost P, Kugel JF, Goodrich JA, Dunaief JL, Baffi JZ, Ambati J

Nature
2011 vol. 471 (7338) pp. 325-30

A Role for Neuronal piRNAs in the Epigenetic Control of Memory-Related Synaptic Plasticity

Rajasethupathy P, Antonov I, Sheridan R, Frey S, Sander C, Tuschl T, Kandel ER

Cell
2012 vol. 149 (3) pp. 693-707

Transcriptional activation of short interspersed elements by DNA-damaging agents

Rudin CM, Thompson CB

Genes Chromosomes Cancer
2001 vol. 30 (1) pp. 64-71

L1 mobile element expression causes multiple types of toxicity

Wallace NA, Belancio VP, Deininger PL

Gene
2008 vol. 419 (1-2) pp. 75-81

Human Alu element retrotransposition induced by genotoxic stress

Hagan C, Sheffield R, Rudin CM

Nat Genet
2003 vol. 35 (3) pp. 219-20

The human LINE-1 retrotransposon creates DNA double-strand breaks

Gasior SL, Wakeman TP, Xu B, Deininger PL

J Mol Biol
2006 vol. 357 (5) pp. 1383-93

LINE-1 retrotransposition in human neuroblastoma cells is affected by oxidative stress

Giorgi G, Marcantonio P, Del Re B

Cell Tissue Res
2011 vol. 346 (3) pp. 383-91

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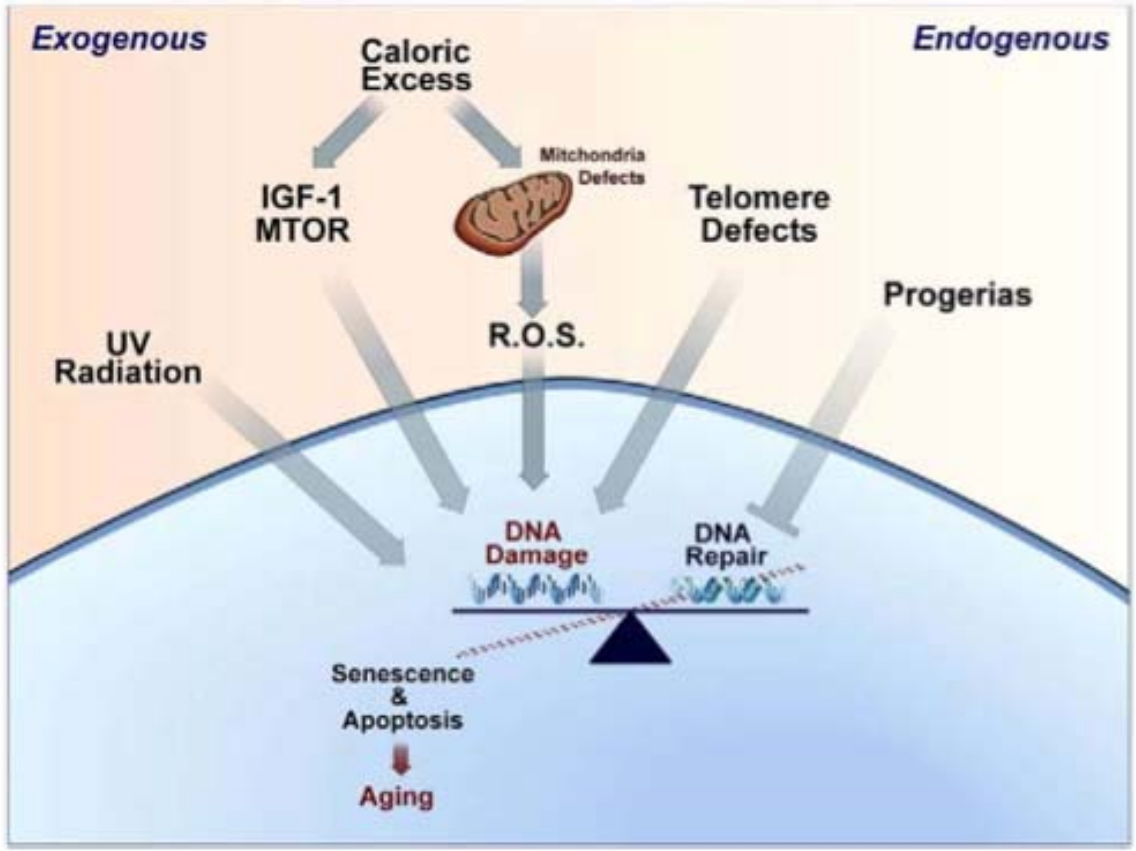

Fig. 1. Aging theories converge on accumulated DNA damage. Exogenous factors, such as UV irradiation, caloric excess, alterations in the IGF/mTOR pathways, and oxidative stress can alter the cellular balance between DNA damage and repair. Similarly, endogenous pathways such as telomere regulation and genetic defects in DNA repair can lead to cellular senescence and premature aging. These effects are of added importance when they alter stem cell regenerative activity.

Mammalian non-LTR retrotransposons: for better or worse, in sickness and in health

Belancio VP, Hedges D, Deininger P

Genome Research
2008 vol. 18 (3) pp. 343-58

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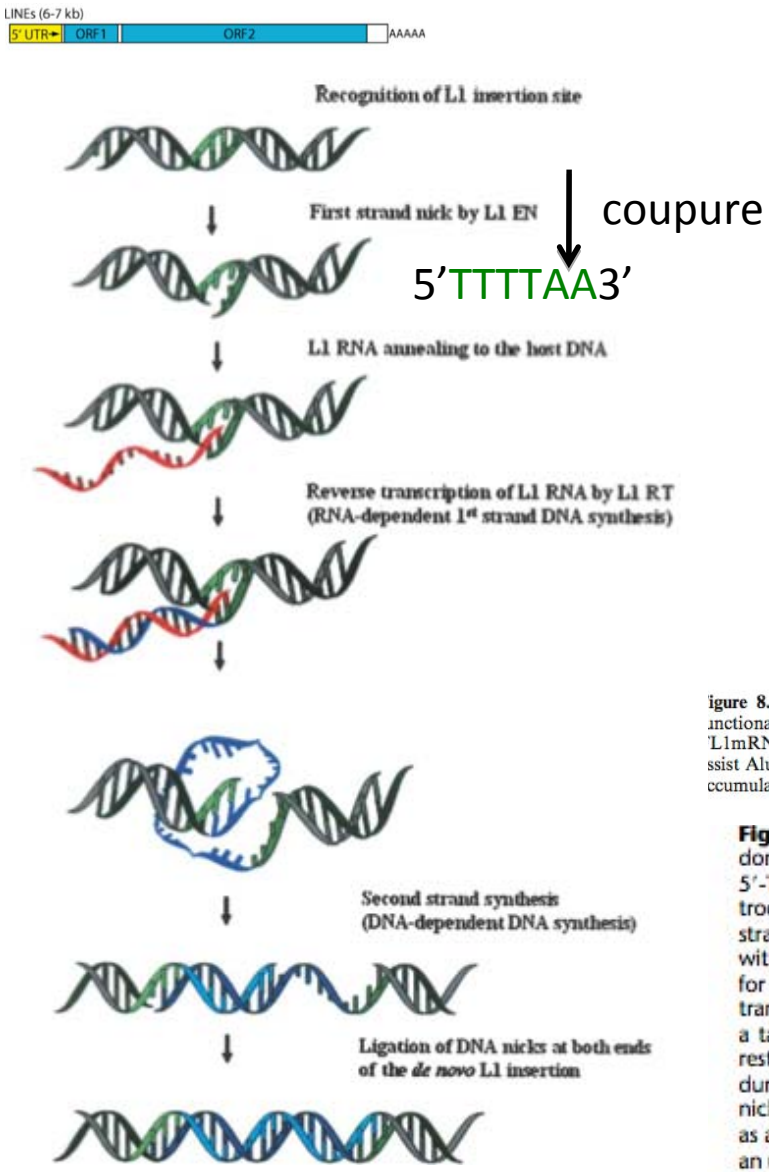

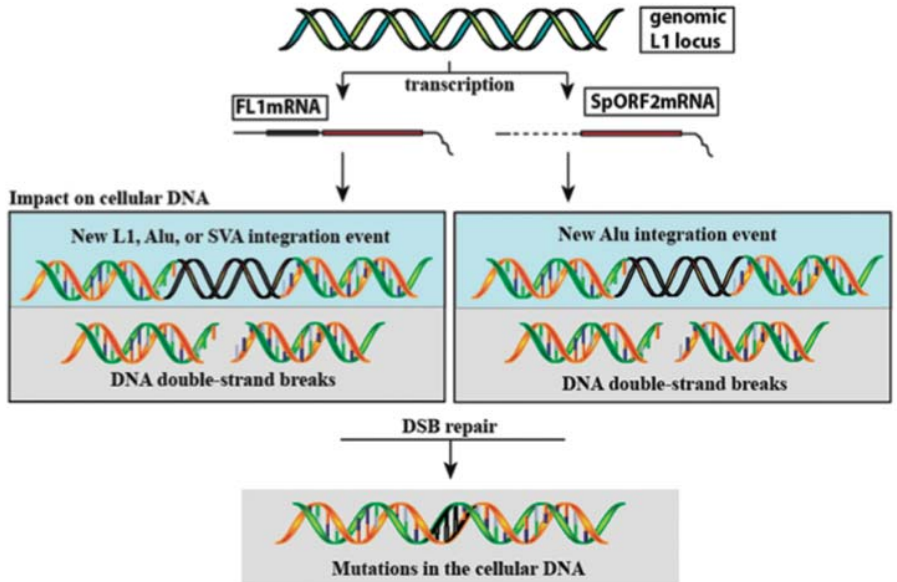

igure 8. A summary of the biologically relevant L1-related mRNA products and their respective impact on the host genome. Transcription of the unctional L1 locus results in the production of either the full-length mRNA (FL1mRNA), the splice ORF2 mRNA (SpORF2mRNA) or both. 'L1mRNA protein products can mobilize L1, Alu, and SVA elements, while SpORF2mRNA only produces ORF2 protein and as a result can only ssist Alu retrotransposition. Expression of either L1 mRNA can generate ORF2, which leads to introduction of DNA DSBs potentially resulting in ccumulation of mutations in the cellular genome.

Figure 3. Steps of the LINE-1 integration process. The L1 endonuclease domain encoded by the ORF2 protein loosely recognizes a consensus 5′-TTTTAA-3′ sequence (shown in green) in the genomic DNA and introduces a first-strand nick between the T and A nucleotides of the minus strand. The resulting free 3′ end of the host DNA is proposed to base-pair with the poly(A) tail of the L1 mRNA (shown in red) and serves as a primer for the first-strand cDNA synthesis (shown in blue) by the L1 reverse transcriptase that uses L1 mRNA as the template. This process is known as a target-primed reverse transcription (TPRT). Mechanistic details of the rest of the L1 integration process are not well defined yet. At some point during L1 integration, either L1 ORF2 or a cellular activity introduces a nick into the plus strand and the structure is resolved to utilize the 3′ end as a primer for the second-strand DNA synthesis (shown in light blue) by an unknown polymerase activity. Finally, the two nicks in the cellular DNA are repaired to complete the L1 integration event.

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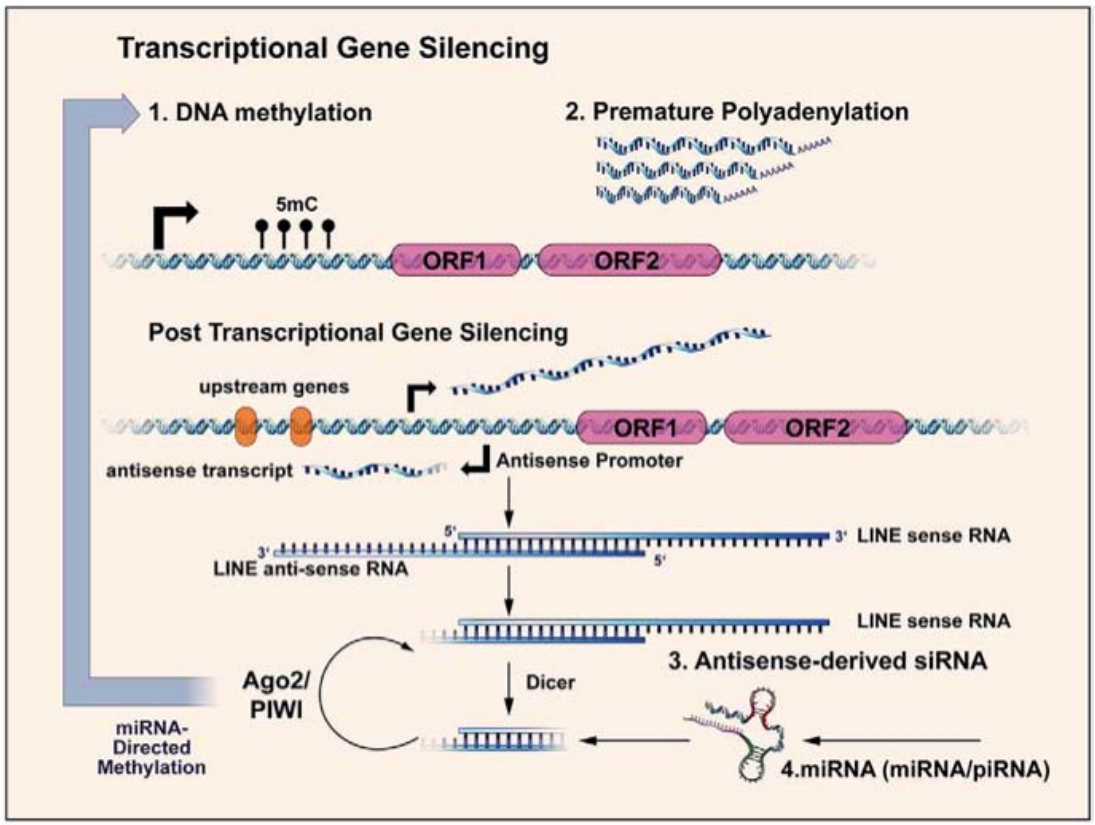


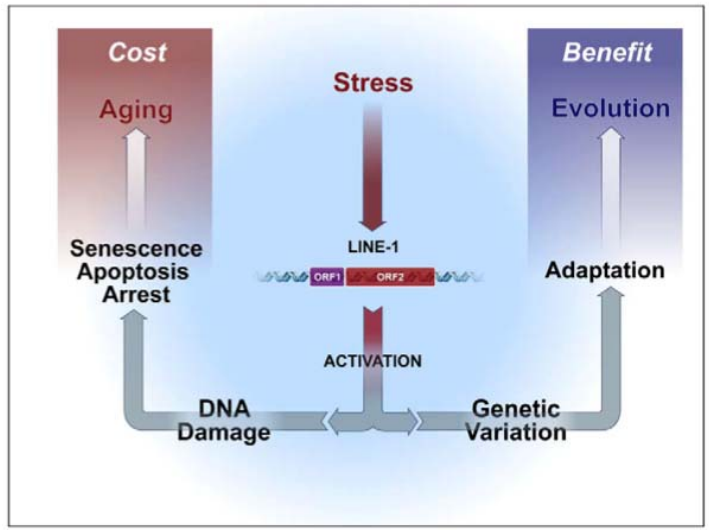
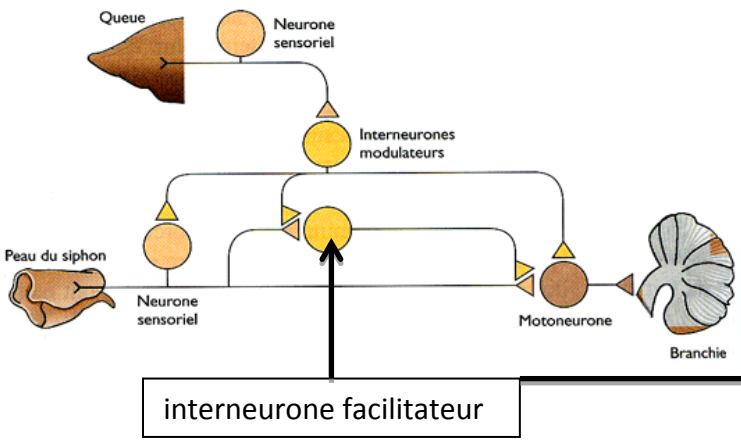


Fig. 4. LINE*age* theory. Acute stress can activate L1 elements leading to transient DNA damage, and rarely to insertional events, which accumulate over a lifetime. L1 ORF1p and ORF2p are equipped with capabilities to alter the genomic landscape that has accelerated evolution, but at the expense of accumulated DNA damage and altered cell fate. This dichotomous nature of L1 may represent another example of antagonistic pleiotropy.

Fig. 3. Cellular mechanisms limiting selfish retroelements in mammals. L1 transcription is suppressed by DNA methylation and can be interrupted by premature polyadenylation. An antisense promoter residing within the L1 promoter can generate antisense L1 transcripts and lead to transcription of neighboring 5′ sequences. RNA interference can also regulate L1 post-transcriptionally via small RNAs facilitated by Argonaute and PIWI proteins. Small RNAs may in-turn direct DNA methylation.



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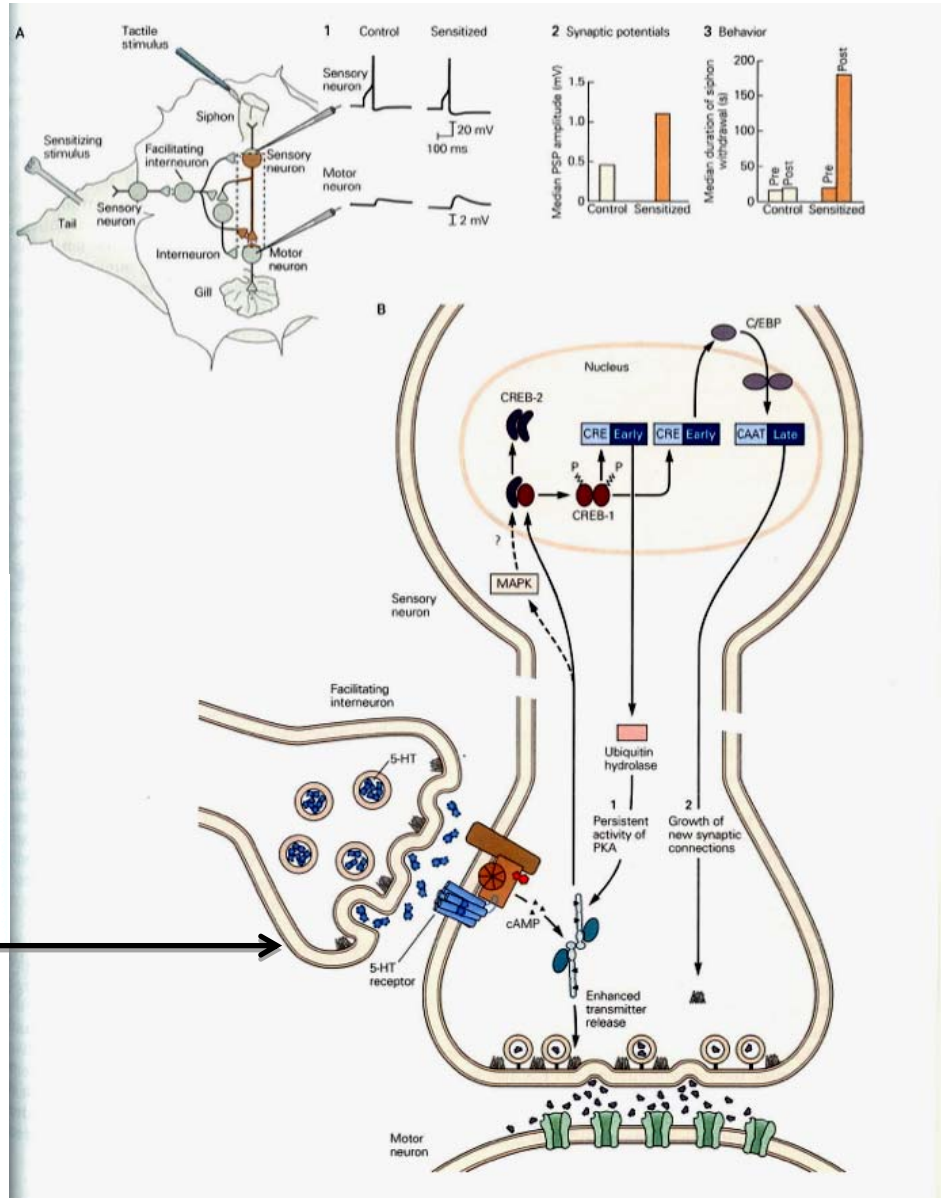

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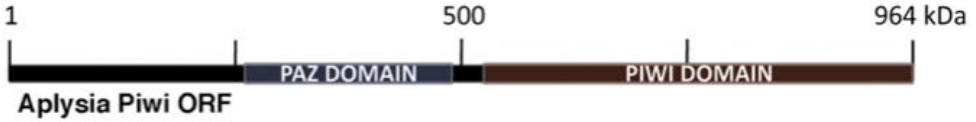
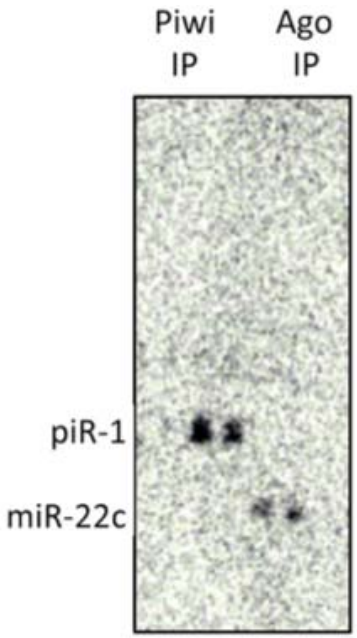
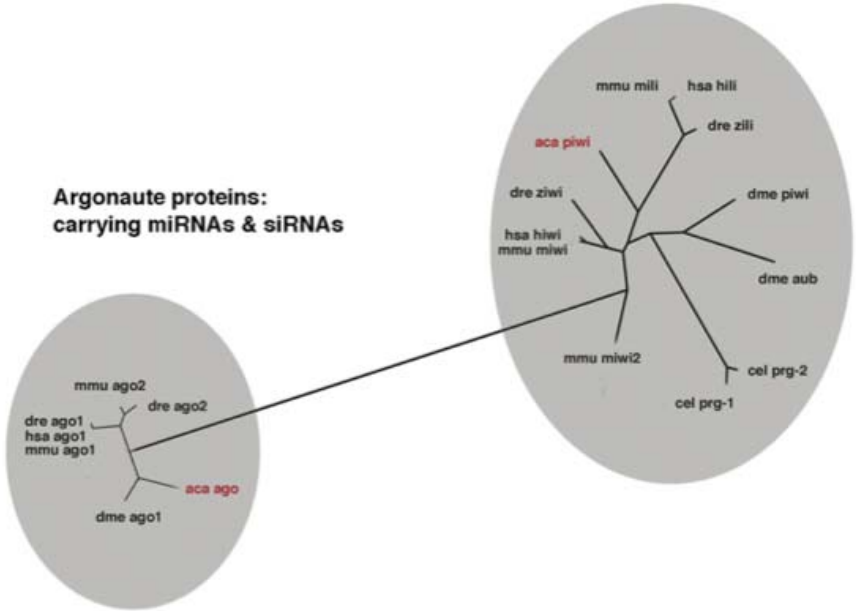


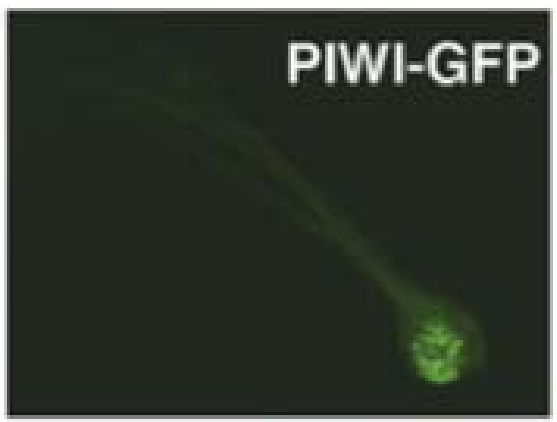
Aplysia Piwi ORF

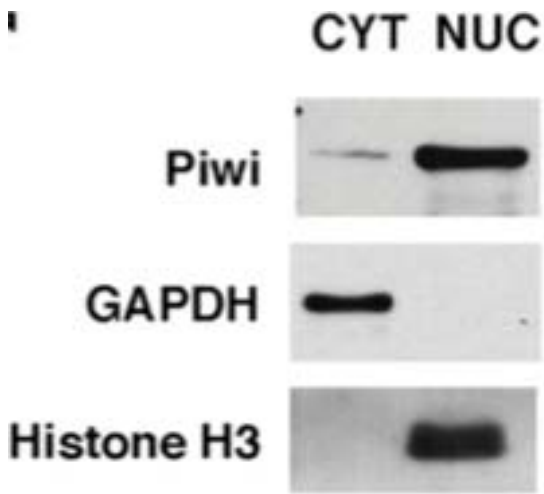


Argonaute proteins: carrying miRNAs & siRNAs



Piwi Proteins: Carrying piRNAs & 21-U RNAs





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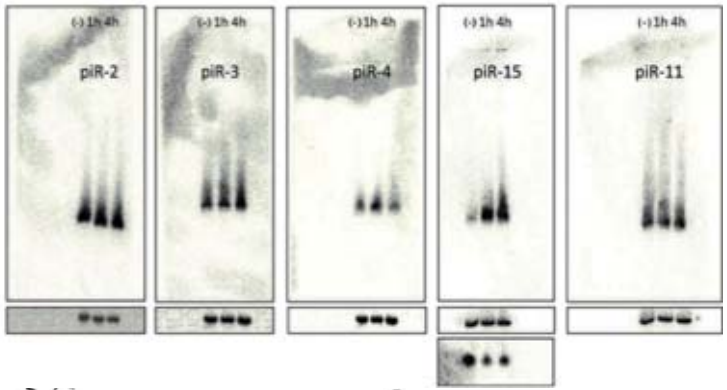

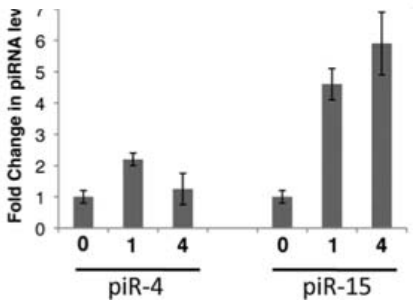

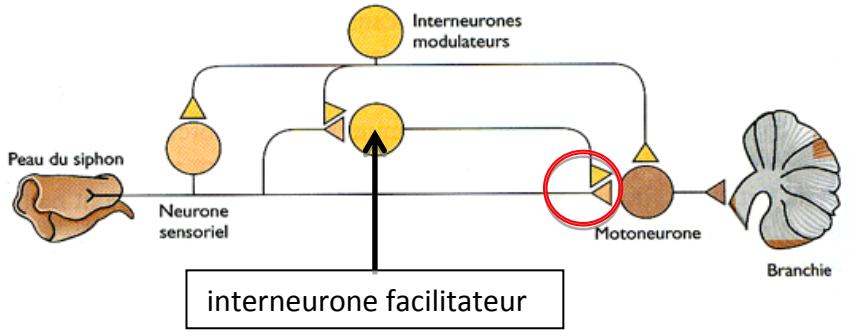

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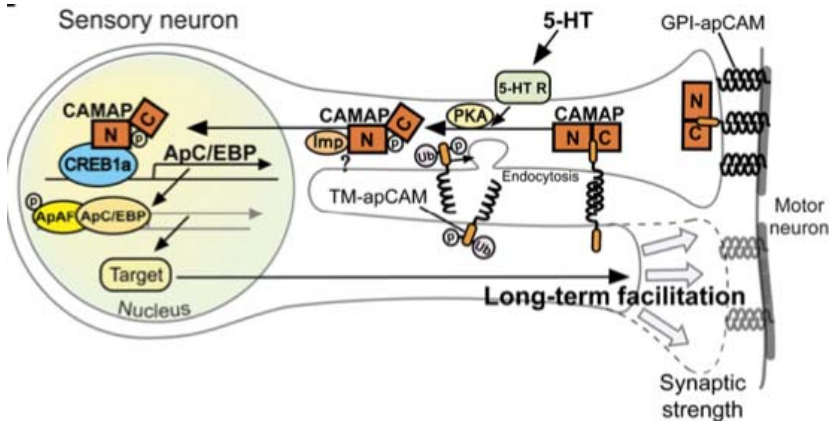

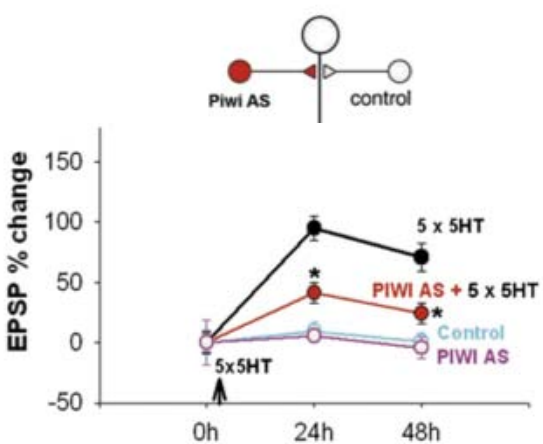

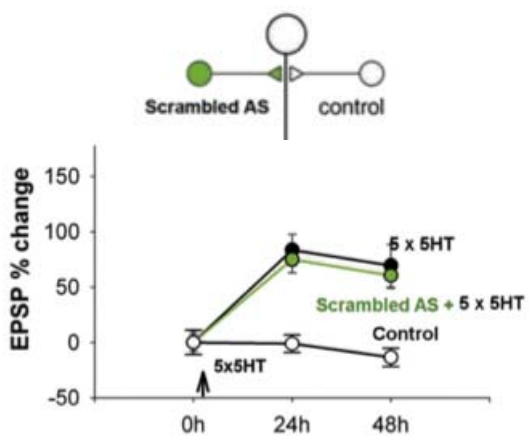

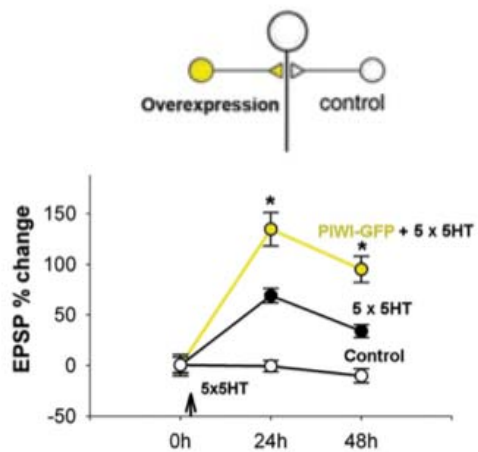

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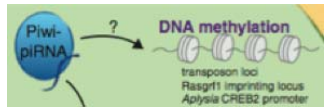

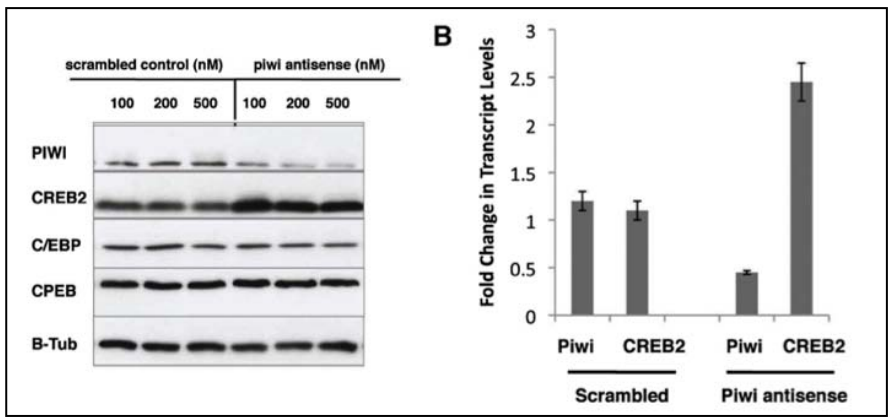

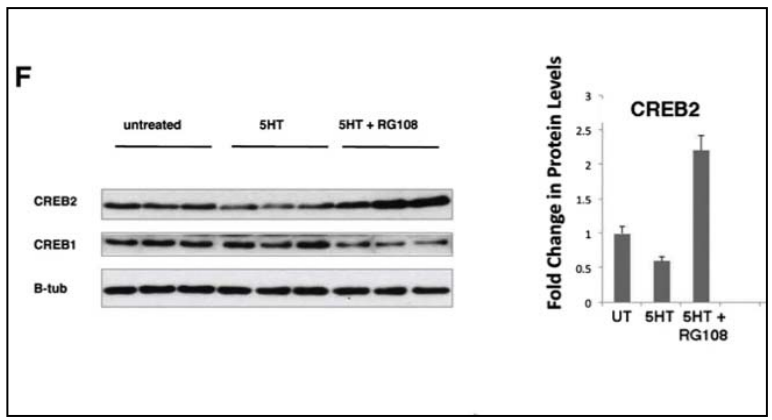

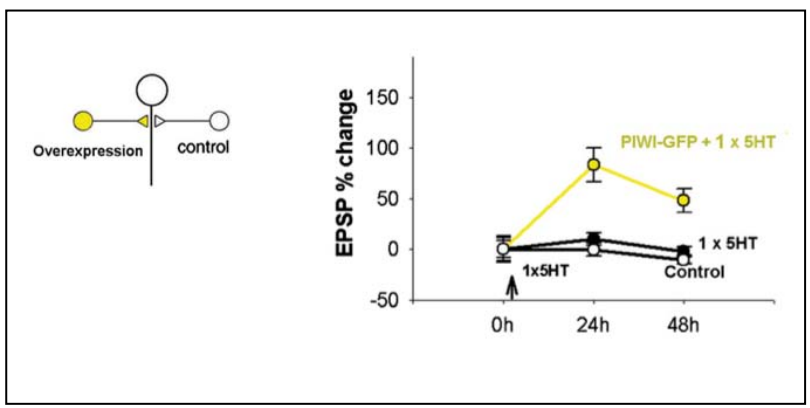

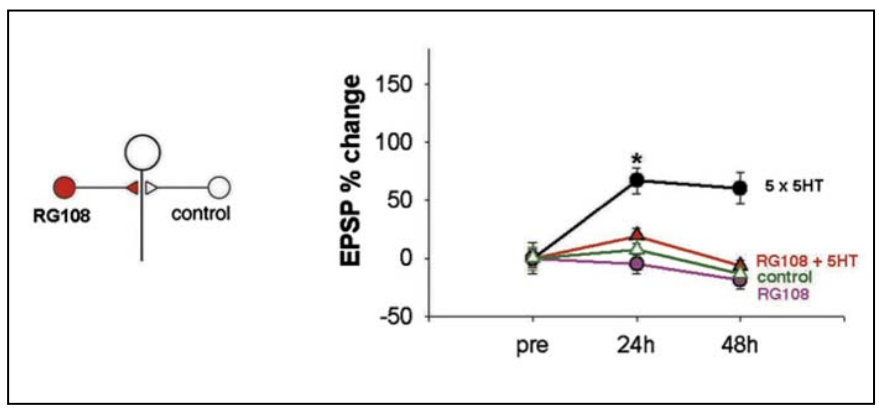

A Role for Neuronal piRNAs in the Epigenetic Control of Memory-Related Synaptic Plasticity

Rajasethupathy P, Antonov I, Sheridan R, Frey S, Sander C, Tuschl T, Kandel ER

Cell
2012 vol. 149 (3) pp. 693-707

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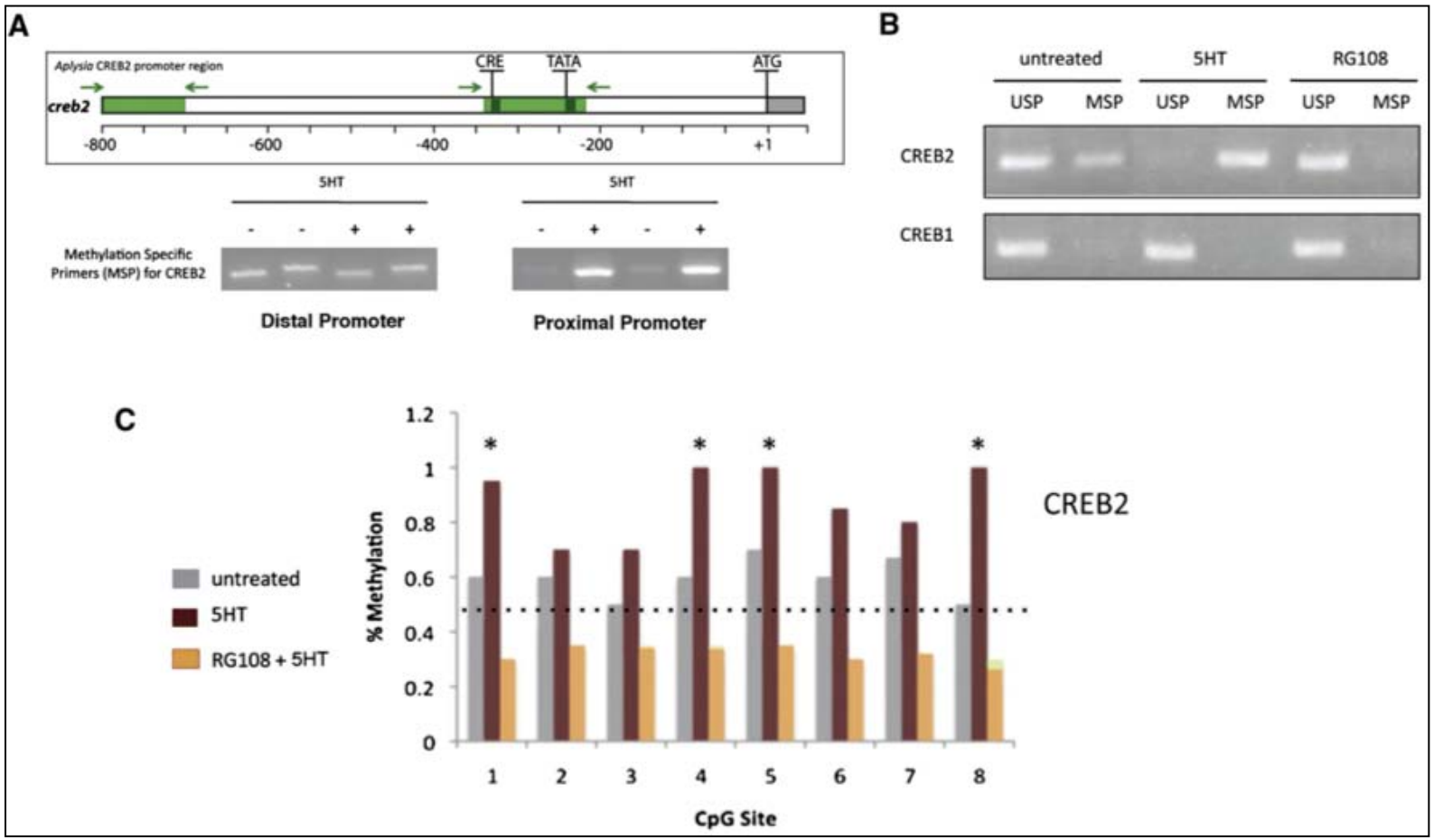

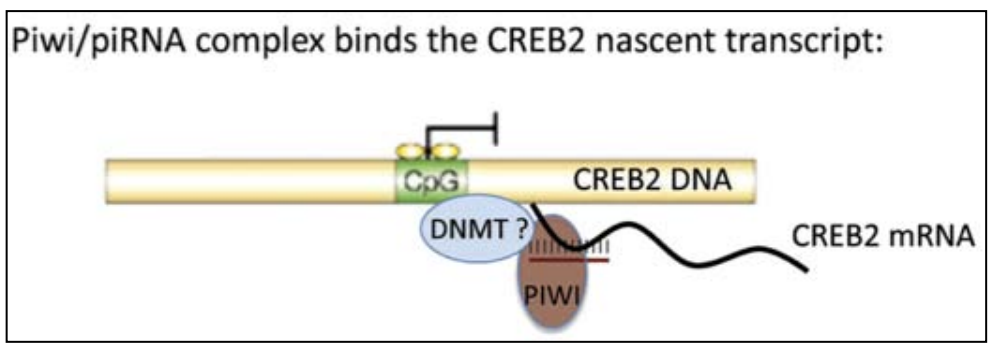

Activation of transposable elements during aging and neuronal decline in Drosophila

Li W, Prazak L, Chatterjee N, Grüninger S, Krug L, Theodorou D, Dubnau J

Nat Neurosci
2013 vol. 16 (5) pp. 529-31

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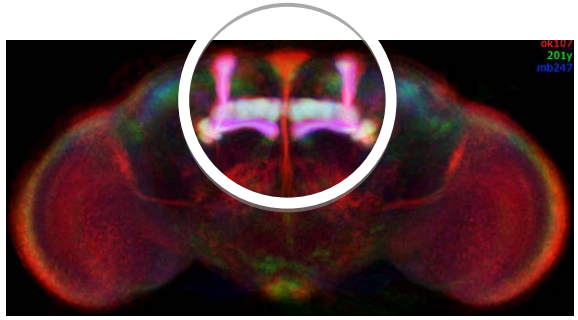
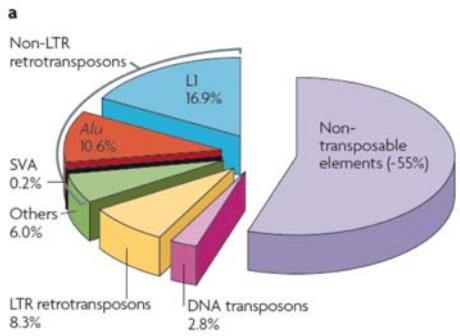
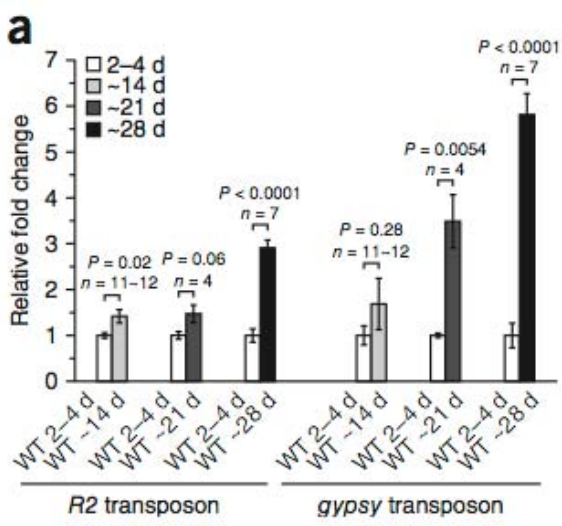
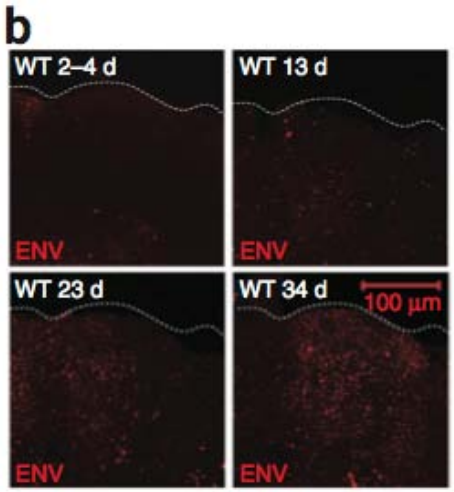
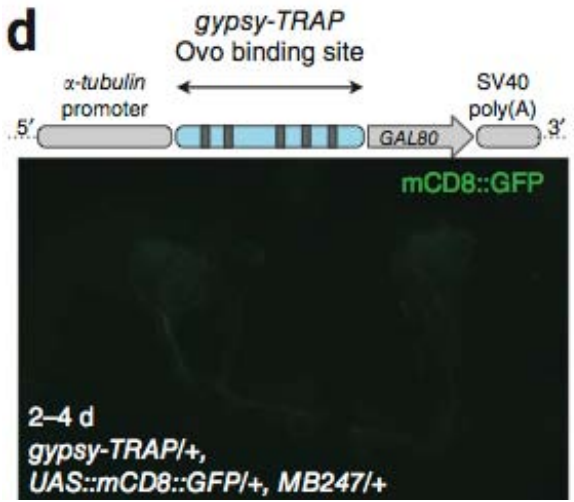


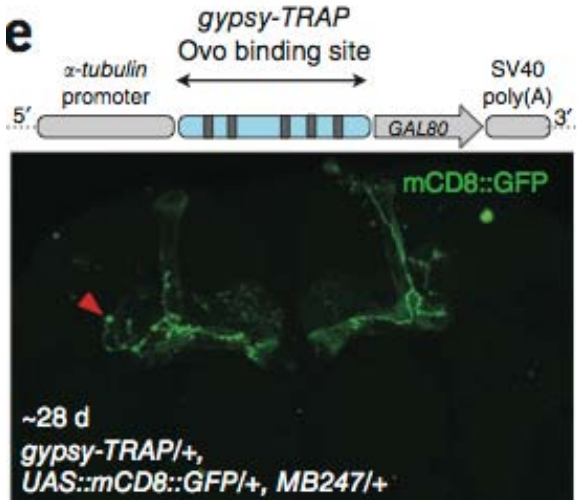
Cordaux @ Batzer, Nature Reviews genetics, 10: 691-703, 2009

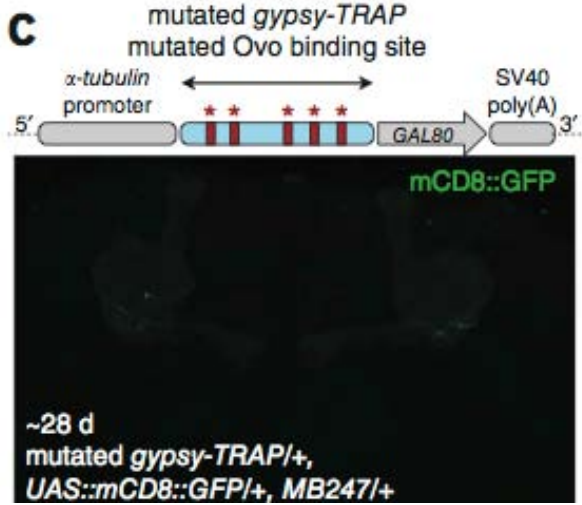












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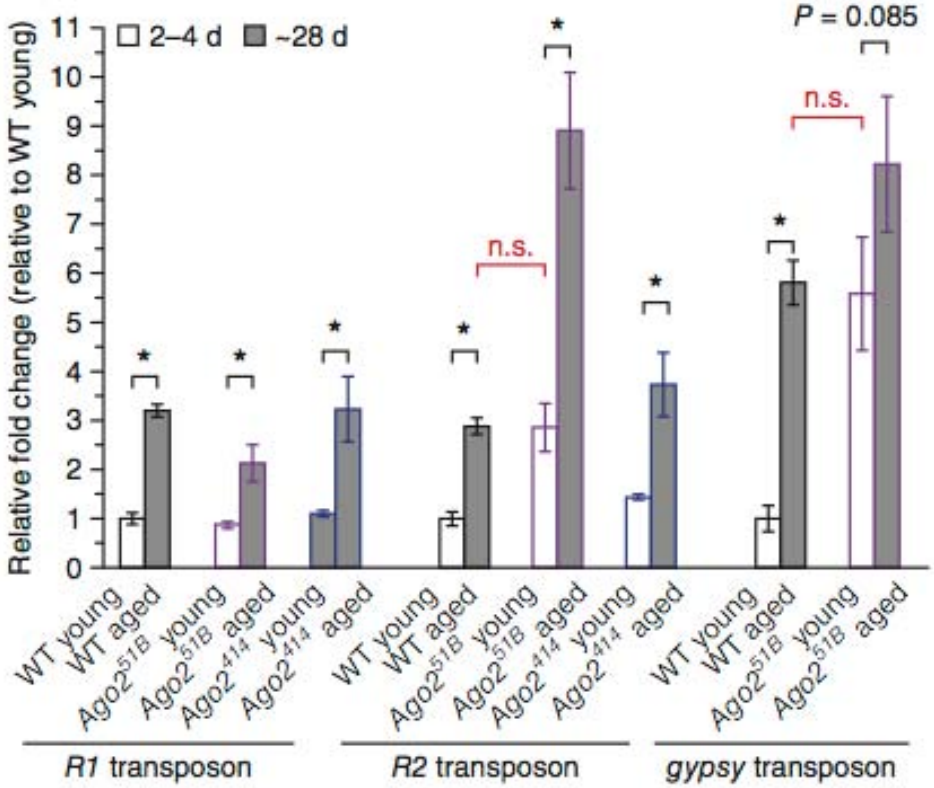

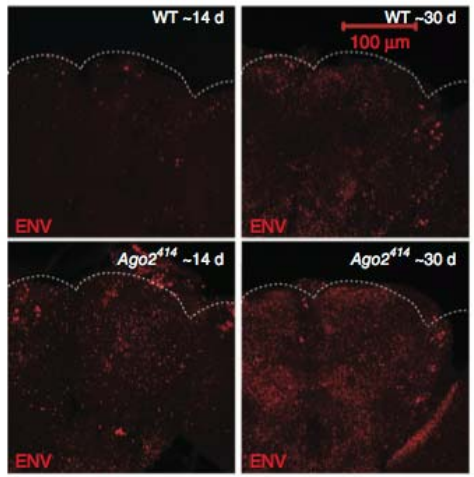

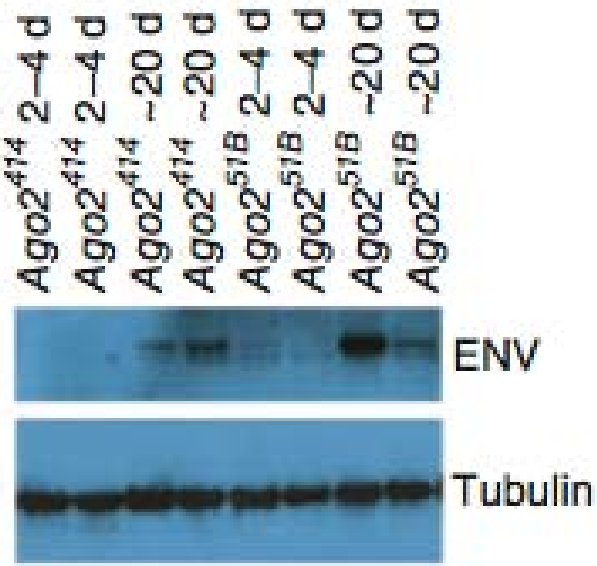

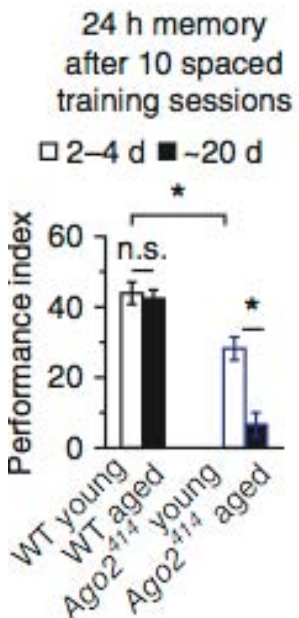

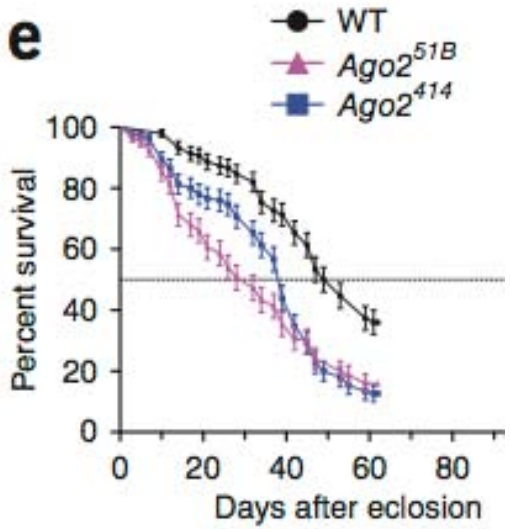

Transposable Elements in TDP-43-Mediated Neurodegenerative Disorders

Wanhe Li[1,2☯], Ying Jin[2☯], Lisa Prazak[2☯], Molly Hammell[2*], Josh Dubnau[2*]

PLoS ONE
2012 vol. 7 (9) pp. e44099