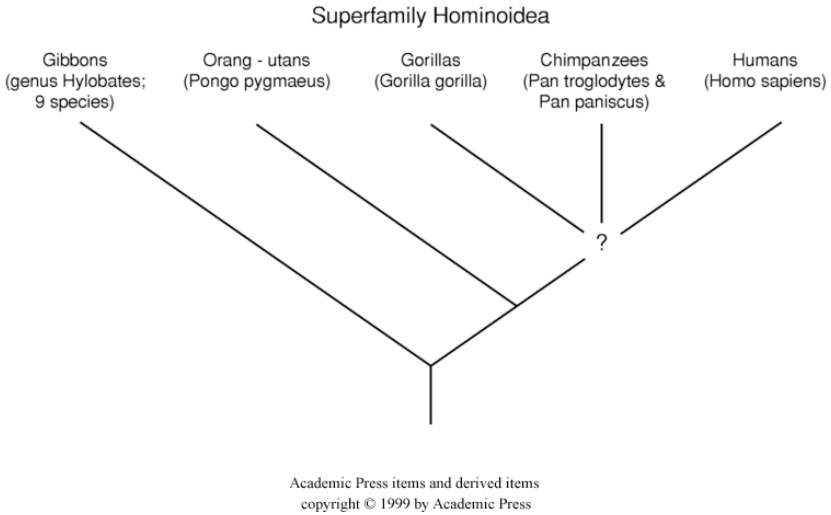
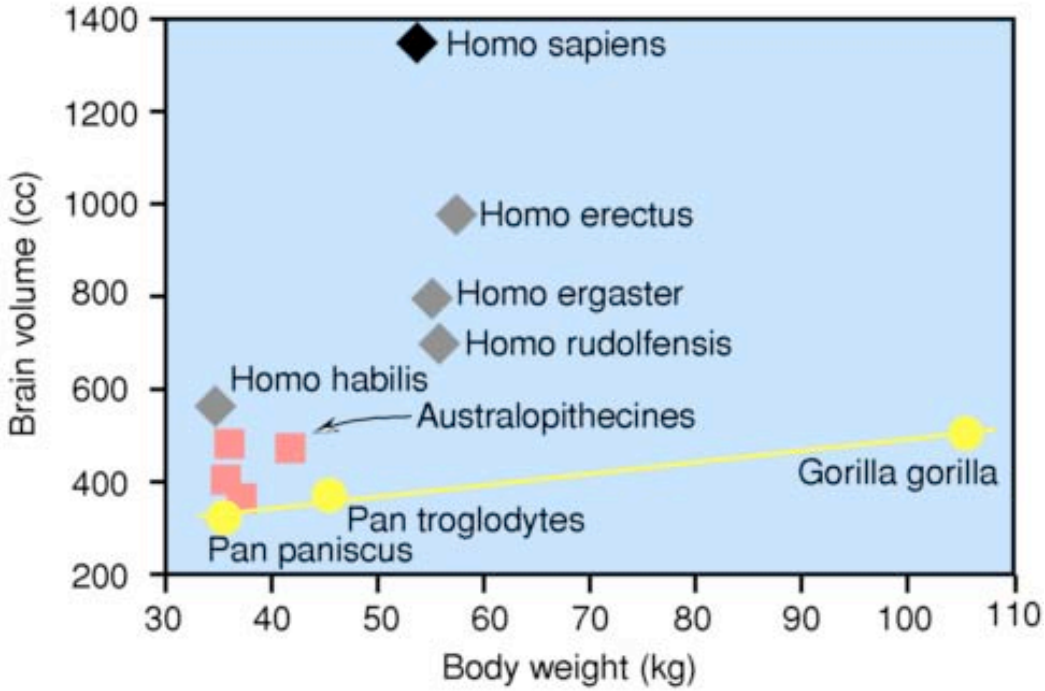
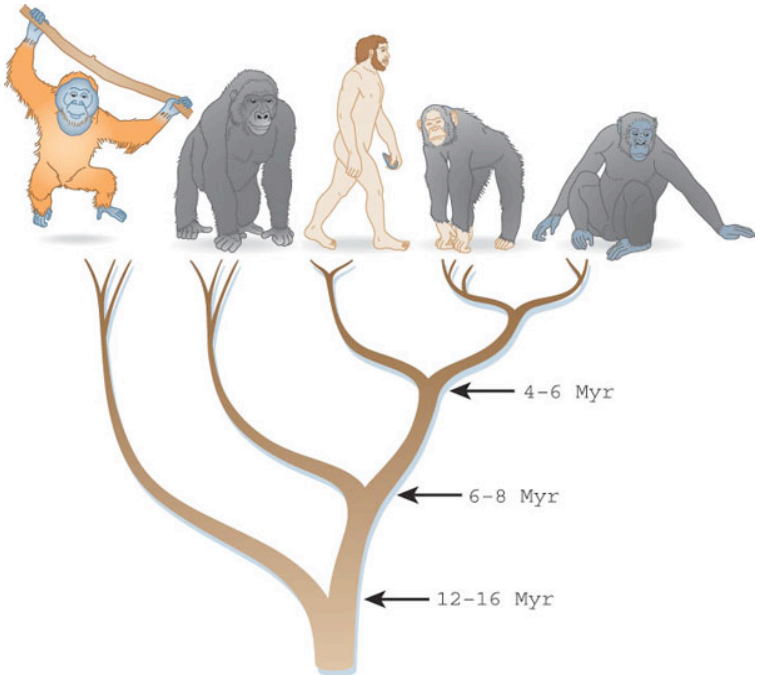
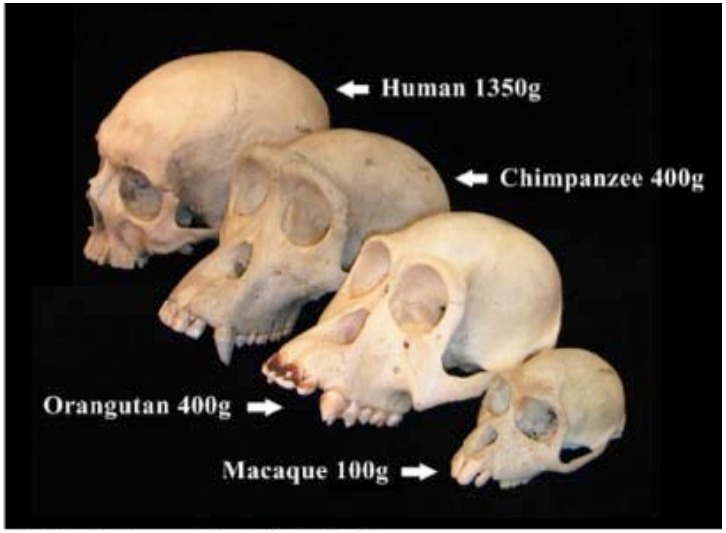
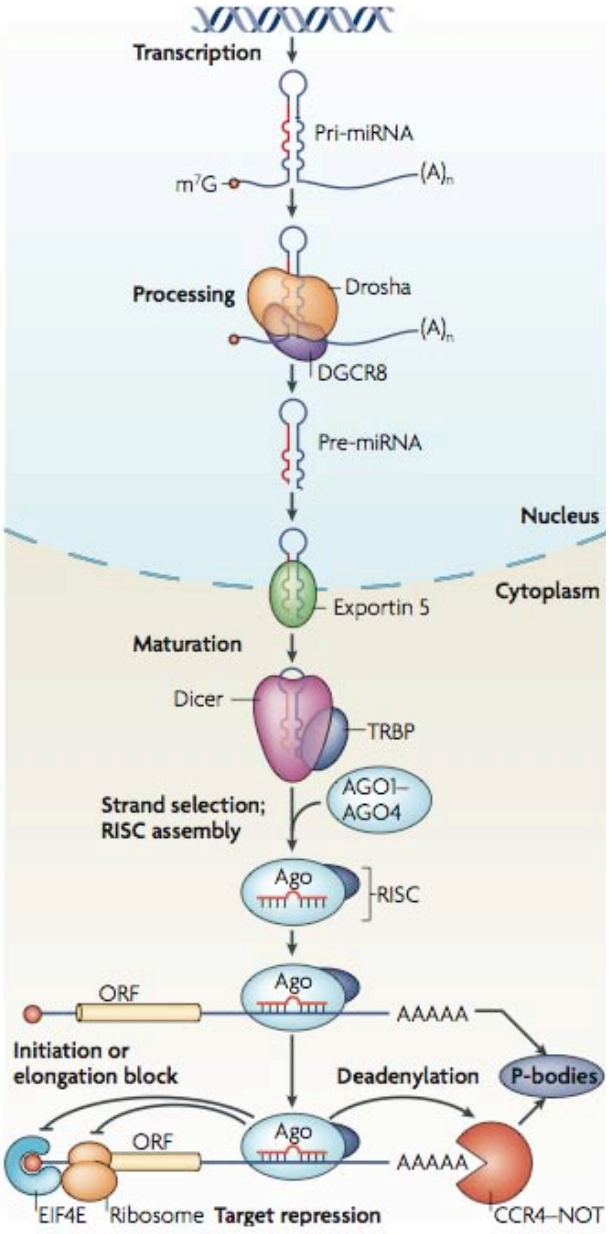
Cours du 7 novembre 2011











MicroRNA control of signal transduction

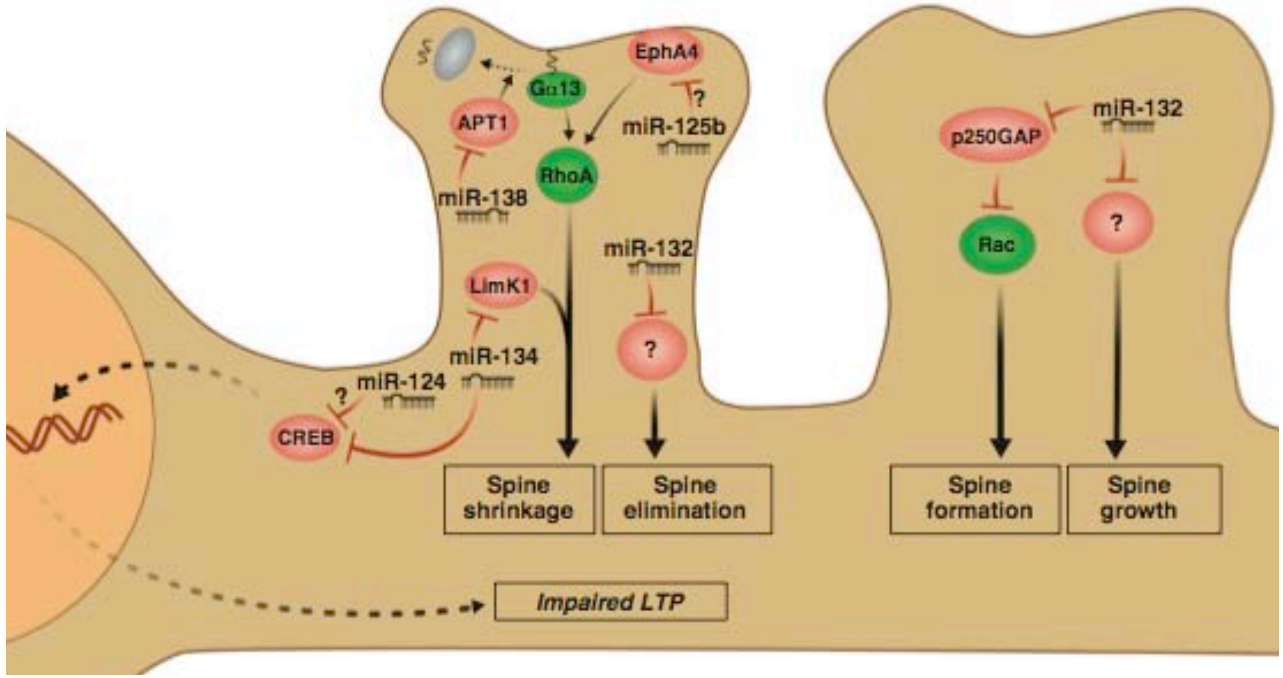
Inui M, Martello G, Piccolo S

Nat Rev Mol Cell Biol
2010 vol. 11 (4) pp. 252–63

microRNAs in neurons: manifold regulatory roles at the synapse

Siegel G, Saba R, Schratt G

Current Opinion in Genetics & Development
2011 vol. 21 (4) pp. 491–7

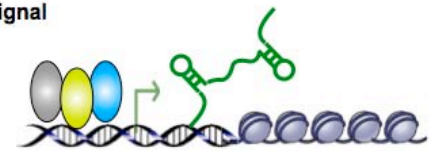


Molecular Mechanisms of Long Noncoding RNAs

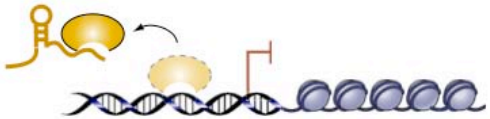
Wang KC, Chang HY

Molecular Cell
2011 vol. 43 (6) pp. 904-14

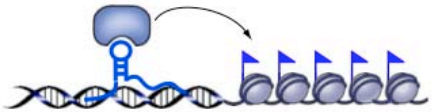
I. **Signal**



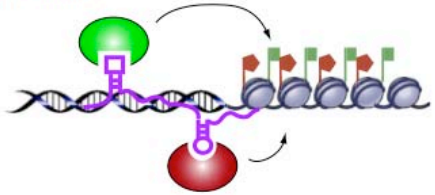
II. **Decoy**



III. **Guide**



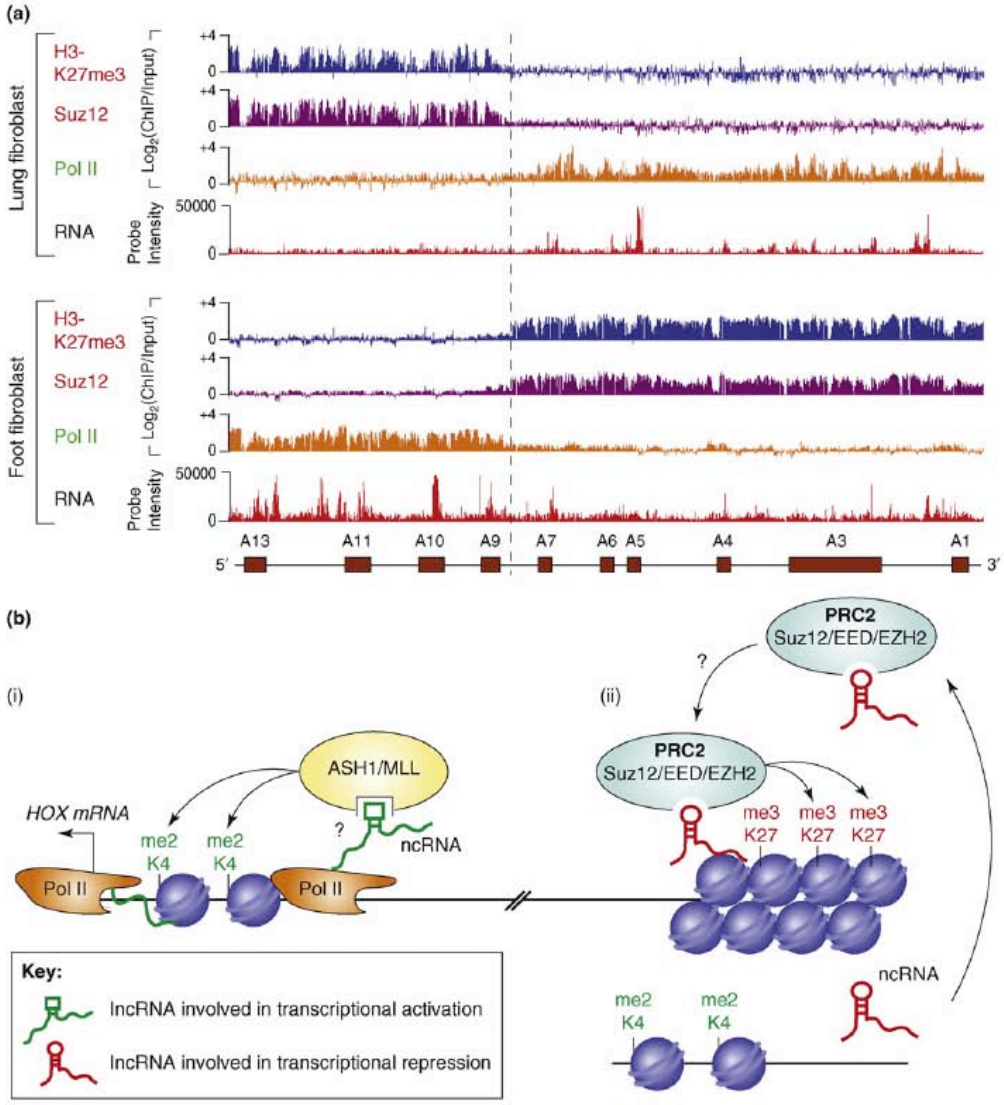
IV. **Scaffold**



Regeneration, repair and remembering identity: the three Rs of *Hox* gene expression

Kevin C. Wang[1,2], Jill A. Helms[3] and Howard Y. Chang[1]

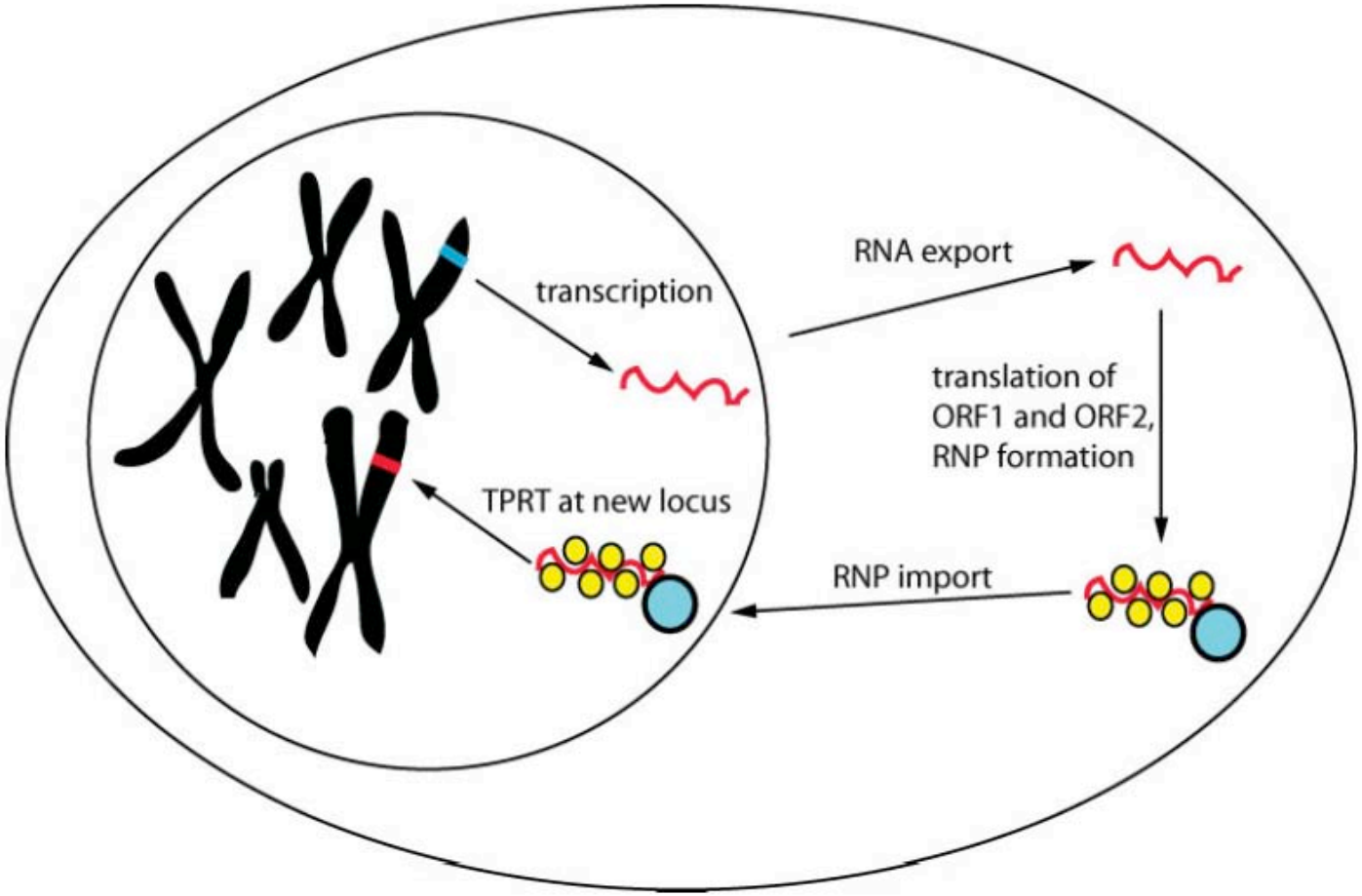
Trends in Cell Biology Vol.19 No.6



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

Jeffrey S. Han and Jef D. Boeke*

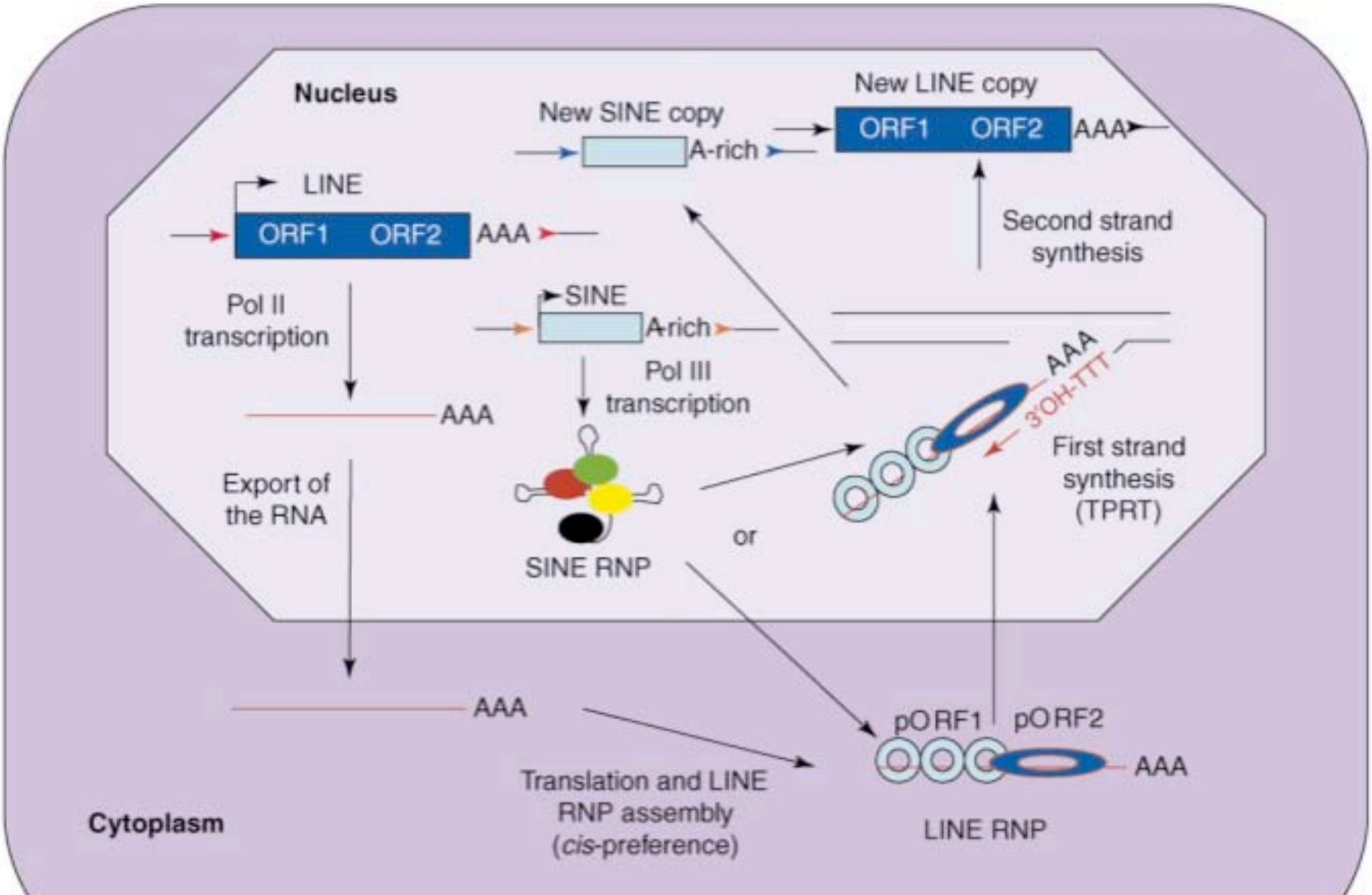
BioEssays 27:775–784, © 2005



Common evolutionary trends for SINE RNA structures

Trends Genet
2007 vol. 23 (1) pp. 26–33

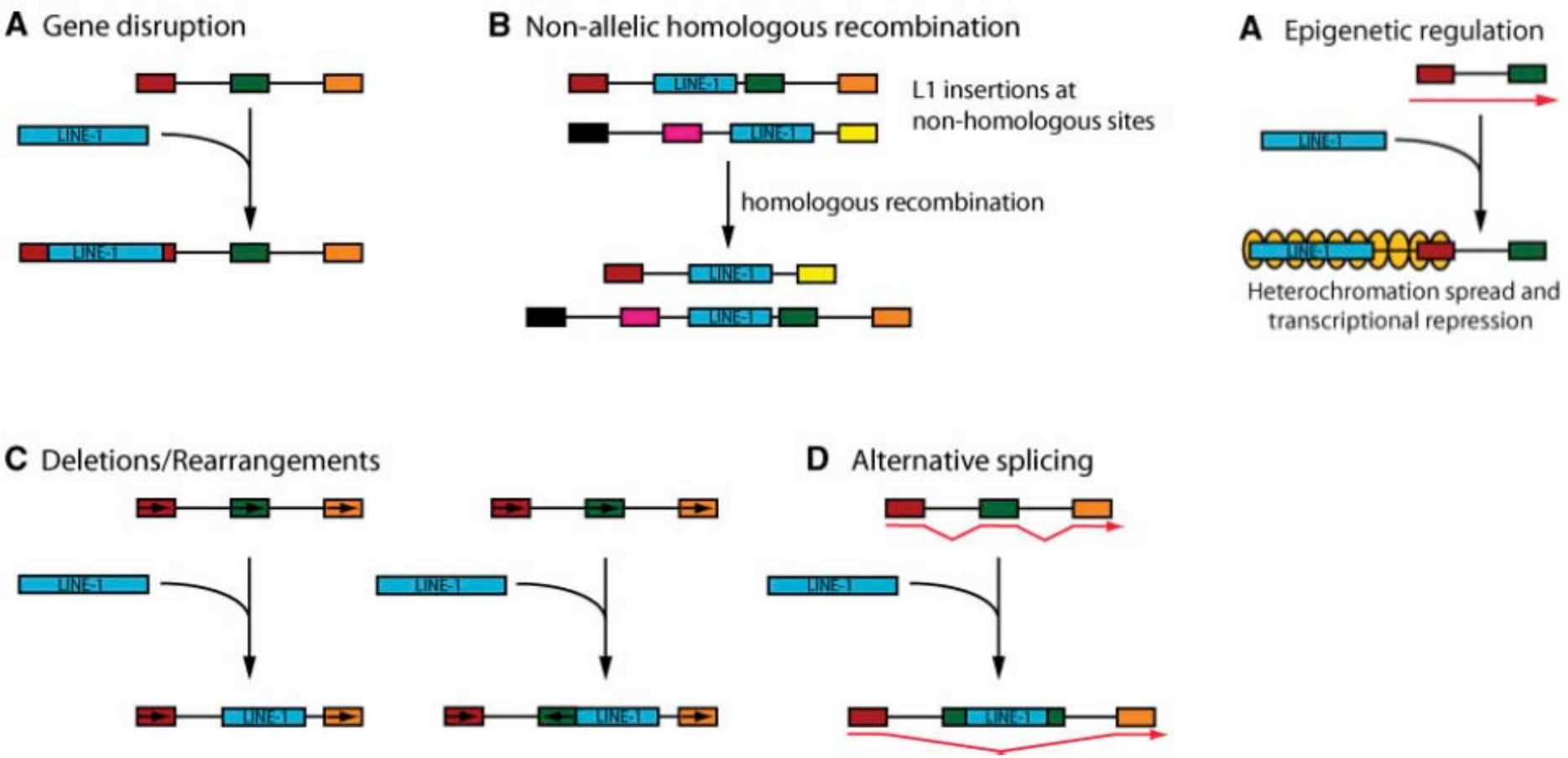
Sun FJ, Fleurdépine S, Bousquet-Antonelli C,
Caetano-Anollés G, Deragon JM



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

BioEssays 27:775–784, © 2005

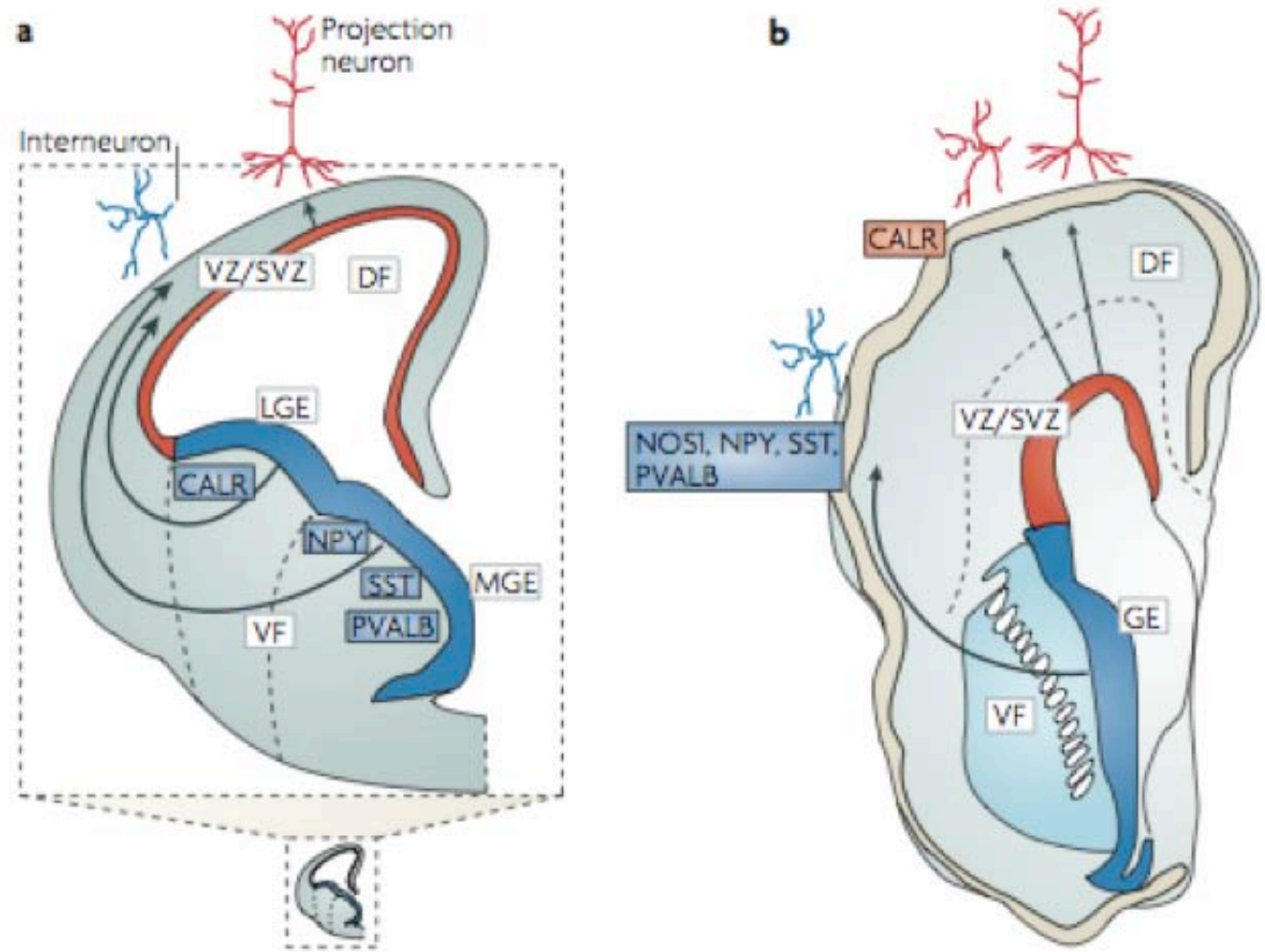
Jeffrey S. Han and Jef D. Boeke*



Evolution of the neocortex: a perspective from developmental biology

Rakic P

Nat Rev Neurosci
2009 vol. 10 (10) pp. 724-35



The Origin of Speech

CONSTANCE HOLDEN

27 FEBRUARY 2004 VOL 303 SCIENCE www.sciencemag.org

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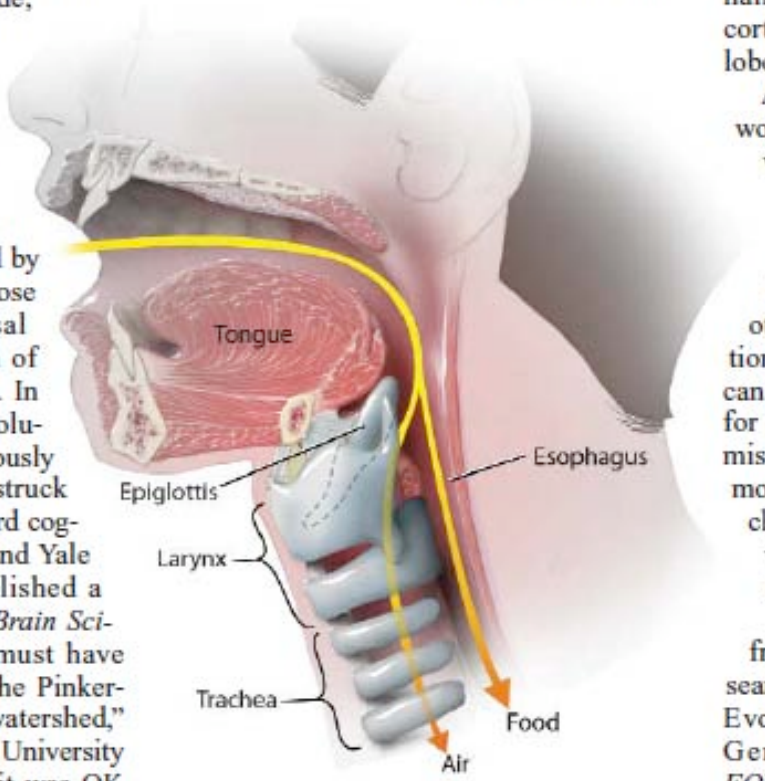

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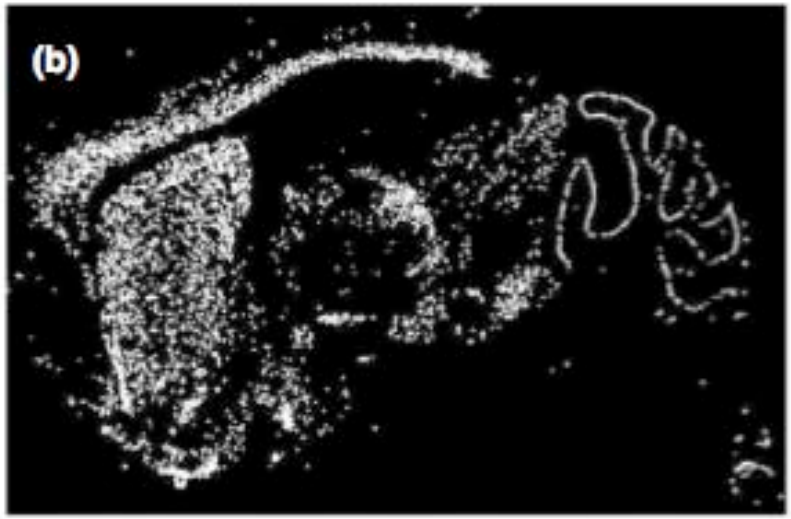
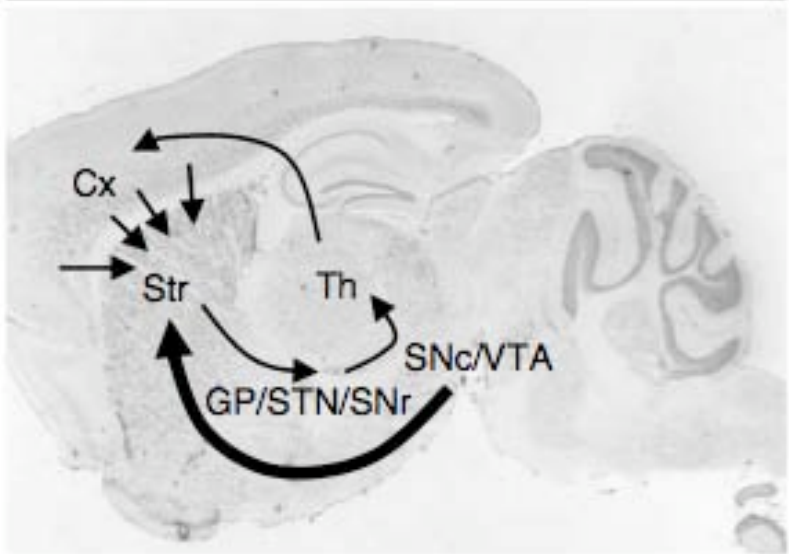
FOXP2 and the role of cortico-basal ganglia circuits in speech and language evolution

Enard W

Current Opinion in Neurobiology
2011 pp.

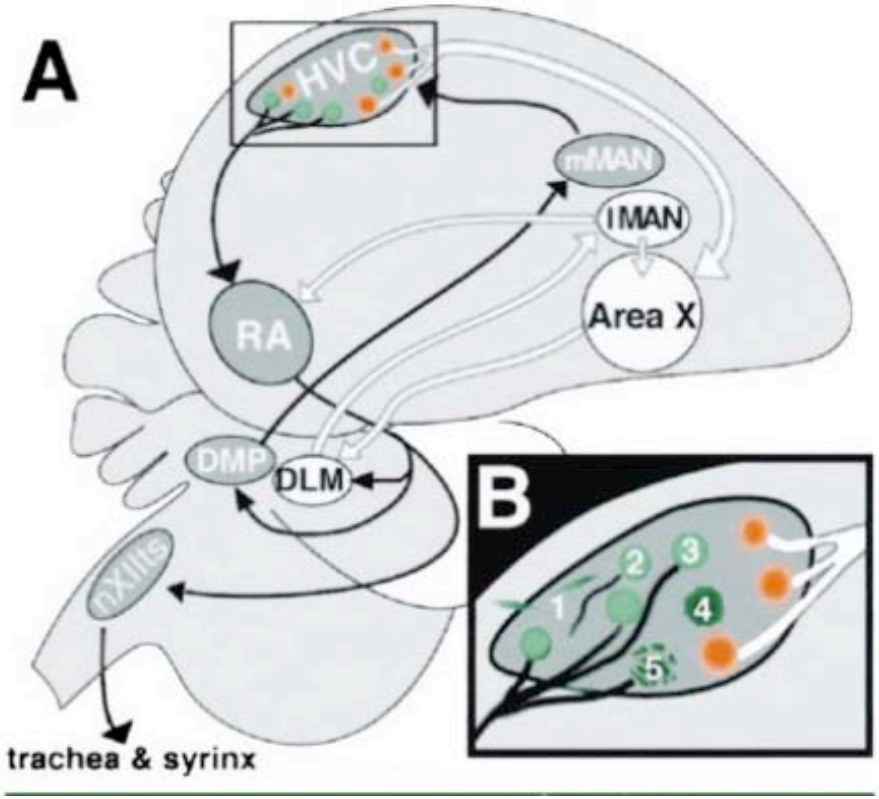


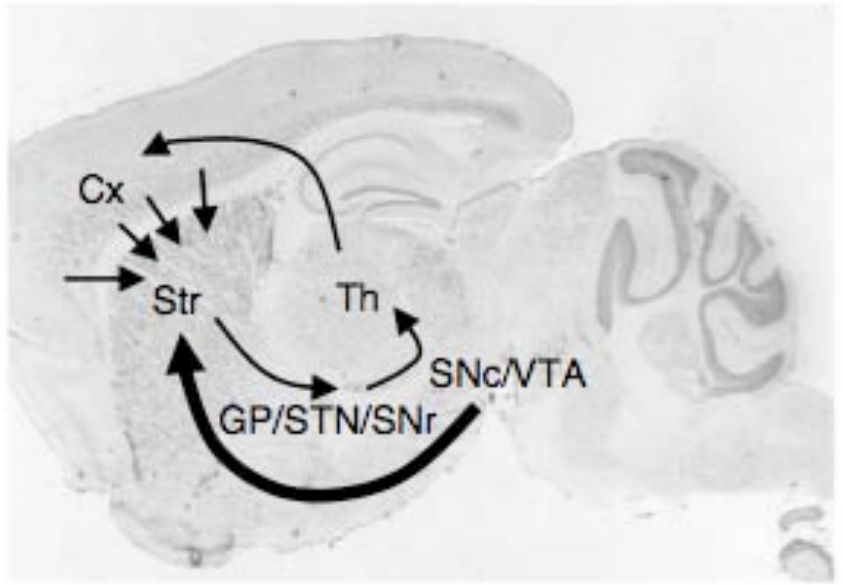


Neuron, Vol. 25, 481–492, February, 2000, Copyright ©2000 by Cell Press

Targeted Neuronal Death Affects Neuronal Replacement and Vocal Behavior in Adult Songbirds

Constance Scharff,*§ John R. Kirn,† Matthew Grossman,* Jeffrey D. Macklis,‡‖ and Fernando Nottebohm*‖

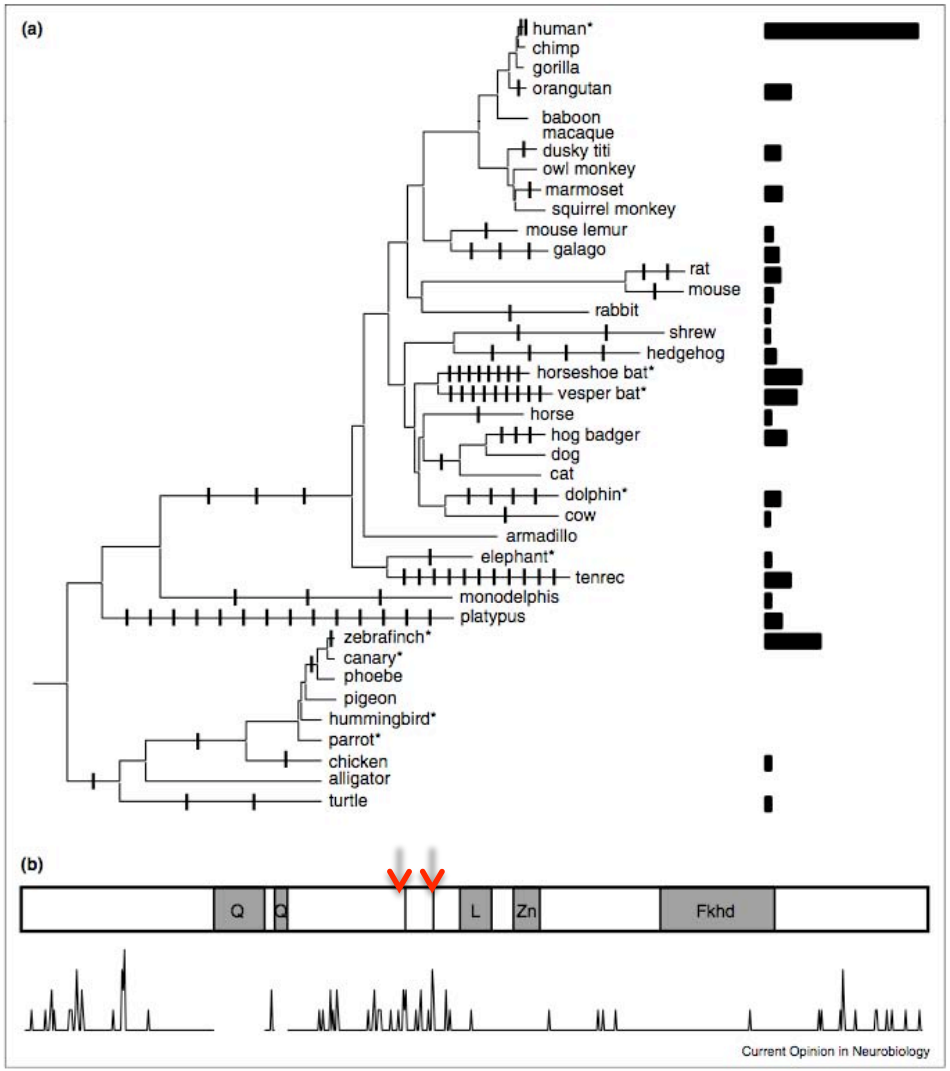




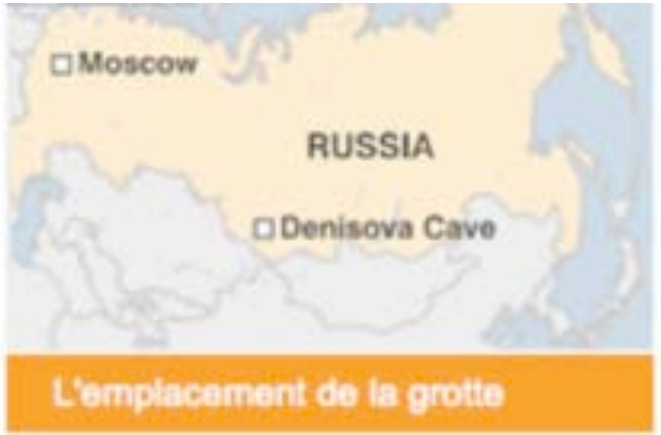
FOXP2 and the role of cortico-basal ganglia circuits in speech and language evolution

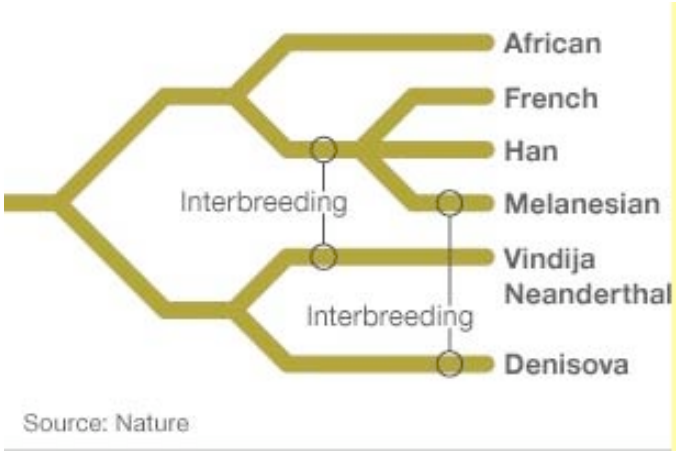
Current Opinion in Neurobiology
2011 pp.

Enard W



FoxP2 evolution in vertebrates. **(A)** Amino acid changes outside the polyglutamin tracts (Q) were mapped on the phylogeny of available Foxp2 sequences from vertebrates [63,64*,65]. Branch lengths are from [63**], if available and are compatible with the rate of synonymous substitutions in *FoxP2* (data not shown). Frog and zebrafish FoxP2 was used as outgroups. The bars on the right site depict the ratio of amino acid changes to the length of the terminal branches. Asterics indicate species with evidence for vocal learning. **(B)** Amino acid changes in the tree are plotted for each position of the human FOXP2 protein sequence. Domains are shaded (polyglutamin (Q), Leucine zipper (L), Zinkfinger (Zn) and forkhead DNA binding domain (Fkhd) and the two human amino acid changes are shown as lines. Data and trees were analyzed using MEGA5 [66].





FOXP2 and the role of cortico-basal ganglia circuits in speech and language evolution

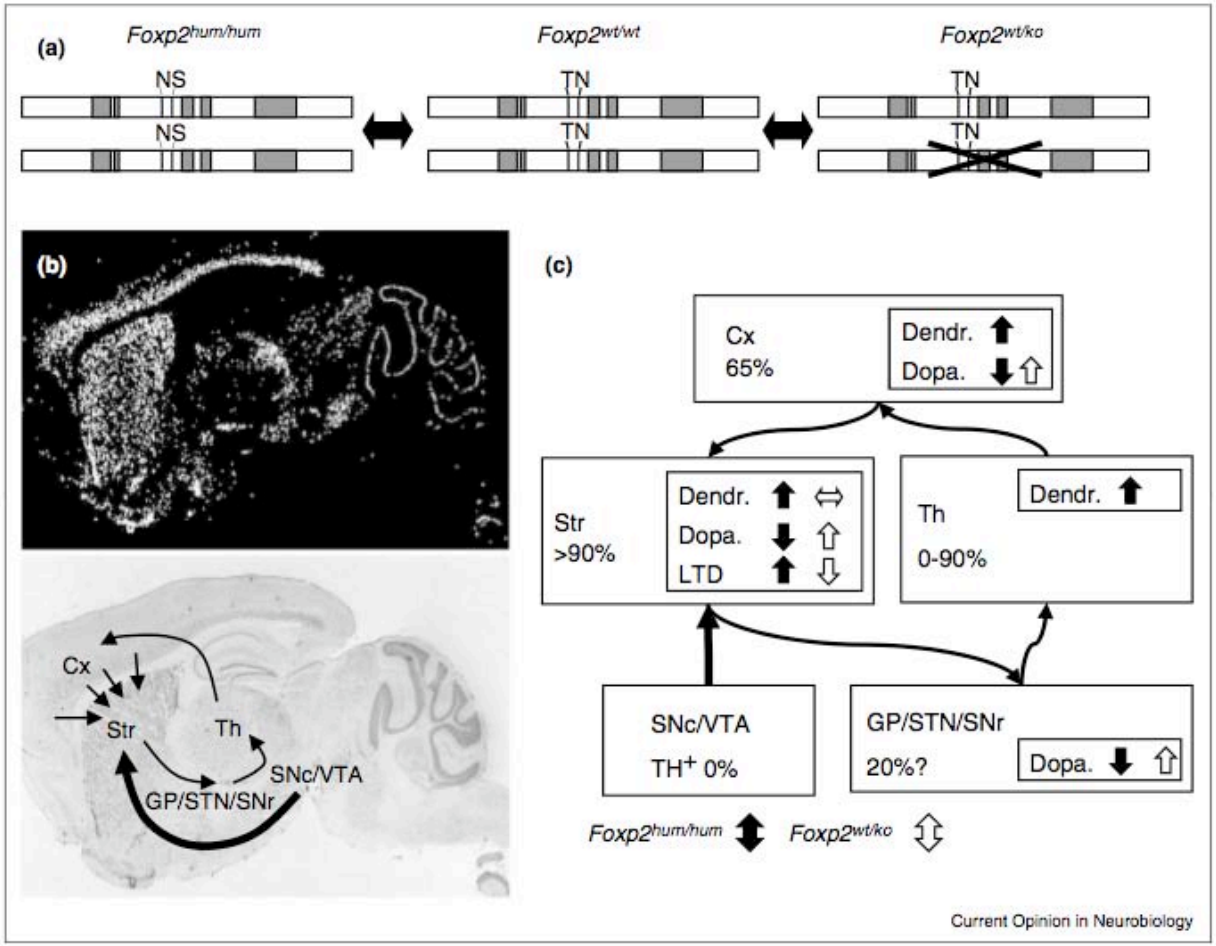
Enard W

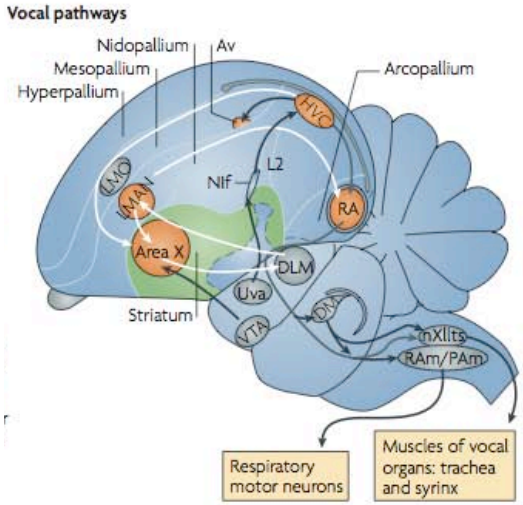
Current Opinion in Neurobiology
2011 pp.

Twitter evolution: converging mechanisms in birdsong and human speech

Bolhuis J, Okanoya K, Scharff C

Nat Rev Neurosci
2010 vol. 11 (11) pp. 747-59





Human specific properties of FOXP2 and CBG circuits. **(A)** Mice humanized for the endogenous Foxp2 gene (*Foxp2*$^{hum/hum}$) are compared to wildtype littermates to infer properties of FOXP2 that arose during human evolution. Mice heterozygous for functional Foxp2 (*Foxp2* $^{wt/ko}$) are compared to wildtype littermates to infer etiological mechanisms of speech and language impairments. **(B)** *Foxp2* gene expression in the adult mouse brain [84]. The *in situ* hybridization image includes a schematic representation of CBG circuits, showing the input from the cortex (Cx) to the striatum (Str) over the output nuclei in the globus pallidus (GP), subthalamic nucleus (STN) and substantia nigra pars reticulata (SNr), back to the thalamus (Th) and cortex. Dopaminergic projections from TH$^+$ cells in the substantia nigra pars compacta (SNc) and ventral tegmental area (VTA) to the striatum are shown by a thick arrow. **(C)** Schematic representation of CBG circuits with the percent of Foxp2-positive cells and the effects seen in *Foxp2*$^{hum/hum}$ (black arrows) and *Foxp2* $^{wt/ko}$ (white arrows) mice. Values for cortex (layer VI projection neurons), striatum (medium spiny neurons) and thalamus (parafascicular nucleus 90%, other nuclei 0%) are taken from [32••]. Dopaminergic (TH$^+$) cells do not express Foxp2 in adult mice [38] and expression of Foxp2 in the output nuclei has been described as scattered [29•]. For the effects on dendrite length (Dendr.), tissue dopamine levels (Dopa.) and synaptic plasticity (LTD) see main text and [32,50••].

CONCLUSIONS: These results suggested that the FOXP2 gene may confer vulnerability to schizophrenic patients with auditory hallucinations.

Psychiatr Genet
2006 vol. 16 (2) pp. 67-72

☆☆☆☆☆

CONCLUSIONS: FOXP2 might be involved in the language disorder in patients with schizophrenia. Epigenetic factors might be also implicated in the developing of this disorder.

BMC Med Genet
2010 vol. 11 pp. 114

☆☆☆☆☆

CONCLUSION: This study did not identify specific disease risk variants of trinucleotide repeats in OTX1, EN1, DLX2, HOXA1, and FOXP2 candidate genes in neurodevelopmental psychiatric disorders.

Psychiatr Genet
2008 vol. 18 (6) pp. 295-301

☆☆☆☆☆

CONCLUSIONS: The FOXP2-CNTNAP2 pathway provides a mechanistic link between clinically distinct syndromes involving disrupted language.

N Engl J Med
2008 vol. 359 (22) pp. 2337-45

☆☆☆☆☆

Genetic variation in FOXP2 alters grey matter concentrations in schizophrenia patients

Španiel F, Horáček J, Tintěra J, Ibrahim I, Novák T, Čermák J, Klírová M, Höschl C

Prague Psychiatric Centre, Prague, Czech Republic. spaniel@pcp.lf3.cuni.cz

Neurosci Lett
2011 vol. 493 (3) pp. 131-5

☆☆☆☆☆

The role of the urokinase receptor in epilepsy, in disorders of language, cognition, communication and behavior, and in the central nervous system

Bruneau N, Szepetowski P

INSERM Unité 901, Marseille, France.

Curr Pharm Des
2011 vol. 17 (19) pp. 1914-23

☆☆☆☆☆

Disruption of CNTNAP2 and additional structural genome changes in a boy with speech delay and autism spectrum disorder

Poot M, Beyer V, Schwaab I, Damatova N, Van't Slot R, Prothero J, Holder SE, Haaf T

Neurogenetics
2010 vol. 11 (1) pp. 81-9

☆☆☆☆☆

De novo mutations in FOXP1 in cases with intellectual disability, autism, and language impairment

Hamdan FF, Daoud H, Rochefort D, Piton A, Gauthier J, Langlois M, Foomani G, Dobrzeniecka S, Krebs MO, Joober R, Lafrenière RG, Lacaille JC, Mottron L, Drapeau P, Beauchamp MH, Phillips MS, Fombonne E, Rouleau GA, Michaud JL

Am J Hum Genet
2010 vol. 87 (5) pp. 671-8

☆☆☆☆☆

homozygous haplotype sharing identifies candidate genes in autism spectrum disorder

and 1,218 novel ASD candidate genes in the discovery analysis including CADM2, ABHD14A, CHRFAM7A, GRIK2, GRM3, EPHA3, FGF10, KCND2, PDZK1, IMMP2L and FOXP2. Furthermore, 10 of the previously reported ASD genes and 300 of the novel candidates identified in the discovery analysis were replicated in an independent sample of 1,182 trios. Our results demonstrate that regions of HH are significantly enriched for previously reported ASD candidate genes and the observed association is independent of gene size (odds ratio 2.10). Our findings highlight the applicability of HH mapping in complex disorders such as ASD and offer an alternative approach to the analysis of genome-wide association data.

Human genetics
2011 pp.

☆☆☆☆☆

Genetic advances in the study of speech and language disorders

Newbury D, Monaco A

Neuron
2010 vol. 68 (2) pp. 309-20

Developmental speech and language disorders cover a wide range of childhood conditions with overlapping but heterogeneous phenotypes and underlying etiologies. This characteristic heterogeneity hinders accurate diagnosis, can complicate treatment strategies, and causes difficulties in the identification of causal factors. Nonetheless, over the last decade, genetic variants have been identified that may predispose certain individuals to different aspects of speech and language difficulties. In this review, we summarize advances in the genetic investigation of stuttering, speech-sound disorder (SSD), specific language impairment (SLI), and developmental verbal dyspraxia (DVD). We discuss how the identification and study of specific genes and pathways, including *FOXP2*, *CNTNAP2*, *ATP2C2*, *CMIP*, and lysosomal enzymes, may advance our understanding of the etiology of speech and language disorders and enable us to better understand the relationships between the different forms of impairment across the spectrum.

Table 1. Summary of Loci Implicated in Speech and Language Disorders

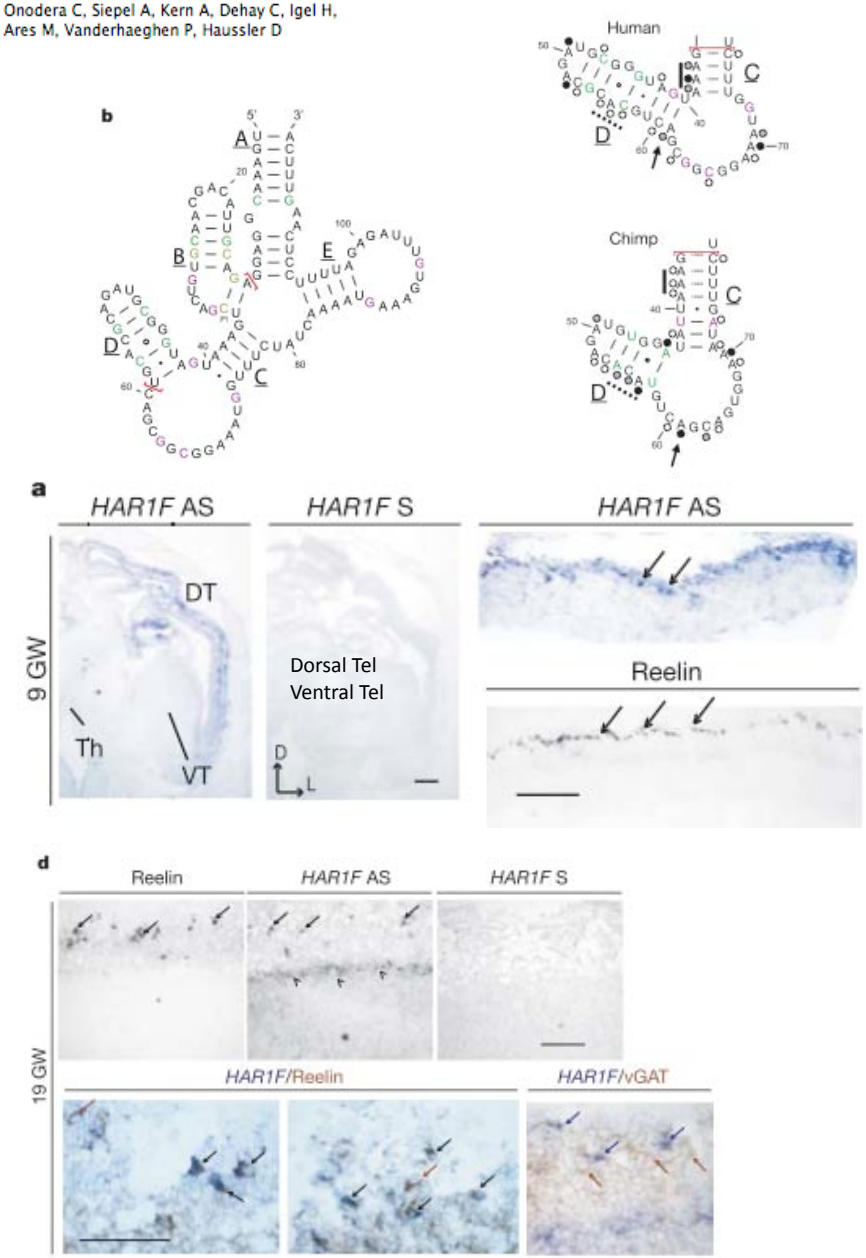
Chromosome Region	Gene	Disorder	Gene Identification Method	Identification References	Replication Notes
1p	NA	Speech-sound disorder	Targeted linkage	Miscimarra et al. (2007)	Region also linked to dyslexia
1q	NA	Stuttering	GWLA	Riaz et al. (2005)	
2q	NA	Stuttering	GWLA	Suresh et al. (2006)	Replicated by Wittke-Thompson et al. (2007)
3	NA	Speech-sound disorder	Targeted linkage	Stein et al. (2004)	Region also linked to dyslexia
3p14	*FOXP1*	Developmental delay with speech and language impairment	Translocation mapping and chromosome abnormality screen	Pariani et al. (2009); Carr et al. (2010); Horn et al. (2010); Vernes et al. (2009)	Replicated across several individuals with variable phenotypes
3q	NA	Stuttering	GWLA	Wittke-Thompson et al. (2007)	Linkage to 3q also found by Raza et al. (2010)
5q	NA	Stuttering	GWLA	Riaz et al. (2005)	Replicated by Wittke-Thompson et al. (2007)
6p	NA	Speech-sound disorder	Targeted linkage	Smith et al. (2005)	Region also linked to dyslexia
7q31	*FOXP2*	Verbal dyspraxia	GWLA with subsequent translocation mapping in an unrelated affected individual	Fisher et al. (1998); Lai et al. (2001)	Disrupted in a small no. of individuals with verbal dyspraxia. Not associated with SLI or autism.
7q	NA	Stuttering	GWLA	Riaz et al. (2005)	Replicated by Suresh et al. (2006) in male individuals only
7q36	*CNTNAP2*	SLI	Targeted association of candidate gene	Vernes et al. (2008)	Also associated with a range of other neurodevelopmental disorders. Association with SLI yet to be replicated.
9p	NA	Stuttering	GWLA	Suresh et al. (2006)	
12q23	*GNPTAB*	Stuttering	GWLA and subsequent targeted candidate gene sequencing	Kang et al. (2010); Riaz et al. (2005)	Linkage replicated by Suresh et al. (2006) when conditioning on chr 7q stuttering locus
13	NA	SLI	GWLA	Bartlett et al. (2002)	Replicated by Bartlett et al. (2004)
13q	NA	Stuttering	GWLA	Wittke-Thompson et al. (2007)	Overlaps with Bartlett linkage to SLI
15p	NA	Stuttering	GWLA	Suresh et al. (2006)	
15q	NA	Stuttering	GWLA	Wittke-Thompson et al. (2007)	
15q	NA	Speech-sound disorder	Targeted linkage	Smith et al. (2005)	Region also linked to dyslexia. Replicated by Stein et al. (2006)
16p13	*GNPTG*	Stuttering	Targeted candidate gene sequencing	Kang et al. (2010)	
16p13	*NAGPA*	Stuttering	Targeted candidate gene sequencing	Kang et al. (2010)	
16q24	*ATP2C2*	SLI	GWLA and subsequent targeted association	Newbury et al. (2009); SLIC (2002)	Linkage replicated by Falcaro et al. (2008); Monaco (2007); SLIC (2004). Association with SLI yet to be replicated. ATP2C2 associated with ADHD (Lesch et al., 2008)
16q24	*CMIP*	SLI	GWLA and subsequent targeted association	Newbury et al. (2009); SLIC (2002)	Linkage replicated by Falcaro et al. (2008); Monaco (2007); SLIC (2004). Association with SLI yet to be replicated.
18p	NA	Stuttering	GWLA	Shugart et al. (2004)	
19q13	NA	SLI	GWLA	SLIC (2002)	Linkage replicated by Falcaro et al. (2008; Monaco (2007); SLIC (2004).
21p	NA	Stuttering	GWLA	Suresh et al. (2006)	Linkage in female individuals only

GWLA, genome-wide linkage analysis; SLI, specific language impairment.

An RNA gene expressed during cortical development evolved rapidly in humans

Nature
2006 vol. 443 (7108) pp. 167–72

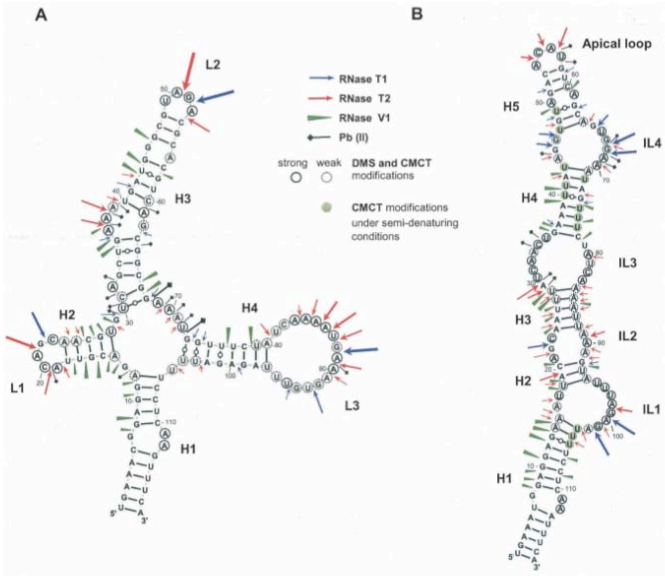
Pollard K, Salama S, Lambert N, Lambot M, Coppens S, Pedersen J, Katzman S, King B, Onodera C, Siepel A, Kern A, Dehay C, Igel H, Ares M, Vanderhaeghen P, Haussler D



Distinctive structures between chimpanzee and human in a brain noncoding RNA

RNA
2008 vol. 14 (7) pp. 1270–5

Beniaminov A, Westhof E, Krol A



Where are the missing pieces of the schizophrenia genetics puzzle?

Current Opinion in Genetics & Development
2011 vol. 21 (3) pp. 310–6

Girard SL, Xiong L, Dion PA, Rouleau GA

further are made on high density SNP arrays. The second pooled DNA GWAS, performed on 660 cases and 1100 controls, identified an intronic SNP of the reelin (*RELN*) gene with a suggestive association (p-value = 2.9×10^{-4}, OR = 1.58) with schizophrenia [8]. This association was female-specific and latter replicated in three independent studies [9–11], thus suggesting that *RELN* is a strong candidate for schizophrenia. Furthermore, *RELN* mutations are also known to cause lissencephaly, a rare brain developmental disorder [12]. The third pooled

The Human Accelerated Region 1 noncoding RNA is repressed by REST in Huntington's disease

Johnson R, Richter N, Jauch R, Gaughwin PM, Zuccato C, Cattaneo E, Stanton LW

Physiol Genomics
2010 pp.

Huntingtin phosphorylation acts as a molecular switch for anterograde/retrograde transport in neurons

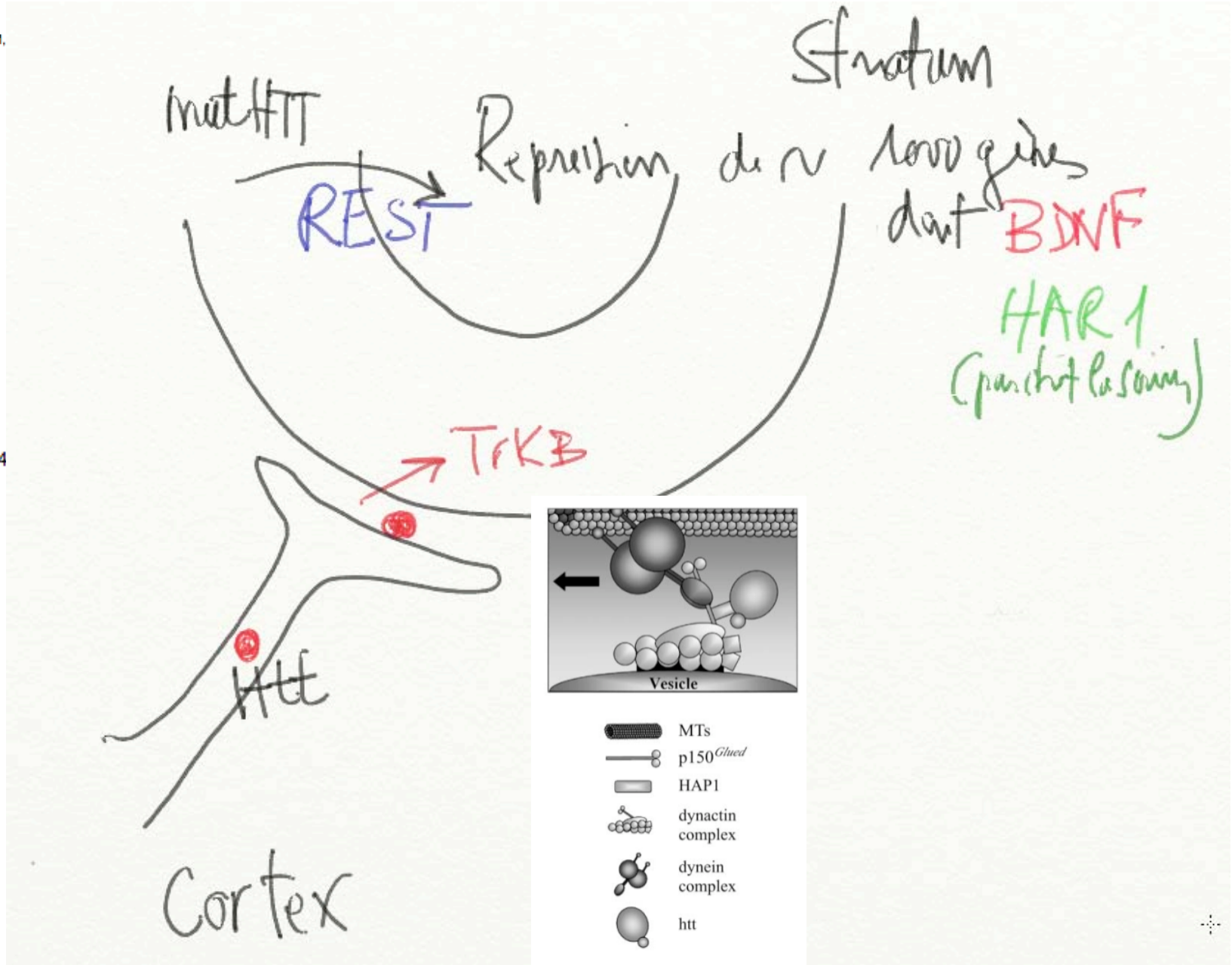
Colin E, Zala D, Liot G, Rangone H, Borrell-Pagès M, Li X, Saudou F, Humbert S

The EMBO Journal
2008 vol. 27 (15) pp. 2124-34

Huntingtin controls neurotrophic support and survival of neurons by enhancing BDNF vesicular transport along microtubules

Gauthier LR, Charrin BC, Borrell-Pagès M, Dompierre JP, Rangone H, Cordelières FP, De Mey J, MacDonald ME, Lessmann V, Humbert S, Saudou F

Cell
2004 vol. 118 (1) pp. 127-38



Jumping-gene roulette

NATURE|Vol 460|27 August 2009

Sandra L. Martin

Jumping genes, which make DNA copies of themselves through an RNA middleman, provide a stochastic process for generating brain diversity among humans. The effect of their random insertion, however, is a bit of a gamble.

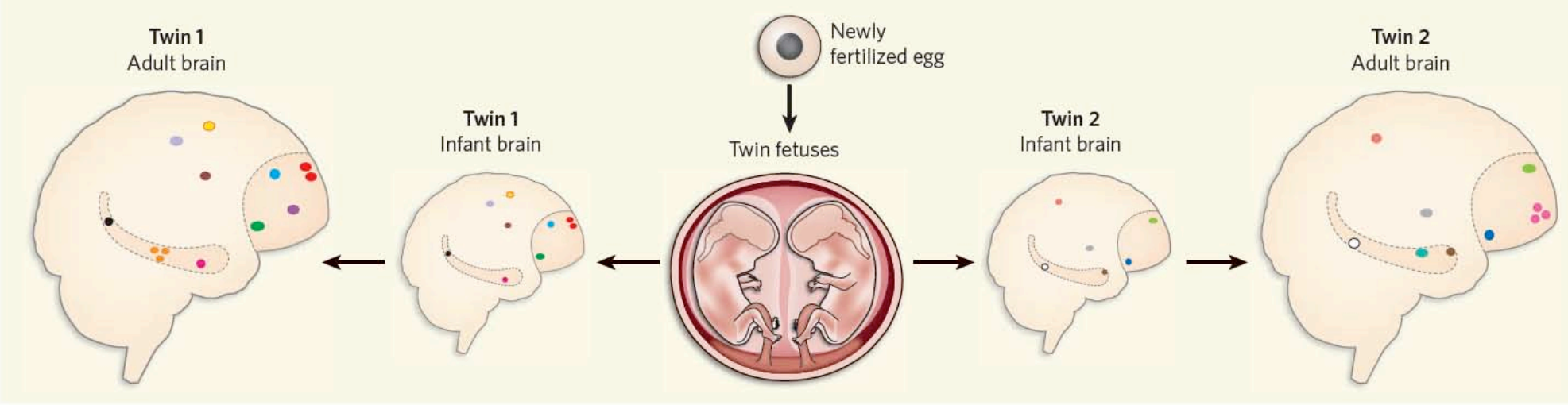


Figure 1 | Human brain variation by retrotransposition. These twins are genetically identical at conception, but at birth their brains differ because of new L1 insertions that take place during the development of the nervous system in the fetus. Ongoing retrotransposition in neural progenitor cells as shown to occur by Coufal *et al.*[1] will further diversify the genetic make-up of their brains in adulthood. Depending on the target genes and the neurons affected by L1 insertions, the twins may differ in brain function or dysfunction. Each unique insertion is represented by a different colour. Darker-shaded areas highlight regions of the brain where L1 retrotransposition may be more likely to occur after birth.

Long non-coding RNAs: insights into functions

Nature Reviews Genetics
2009 vol. 10 (3) pp. 155-9

Mercer TR, Dinger ME, Mattick JS

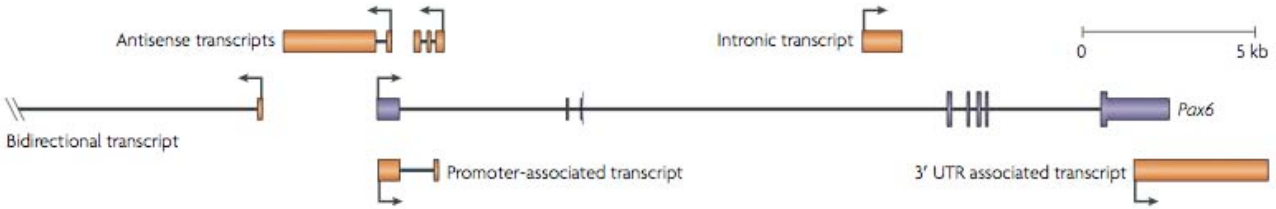


Figure 1 | **Genomic organization of coding and non-coding transcripts.** Schematic diagram illustrating the complexity of the interleaved networks of long non-coding transcripts (orange) that are associated with paired box gene 6 (*Pax6*; purple).

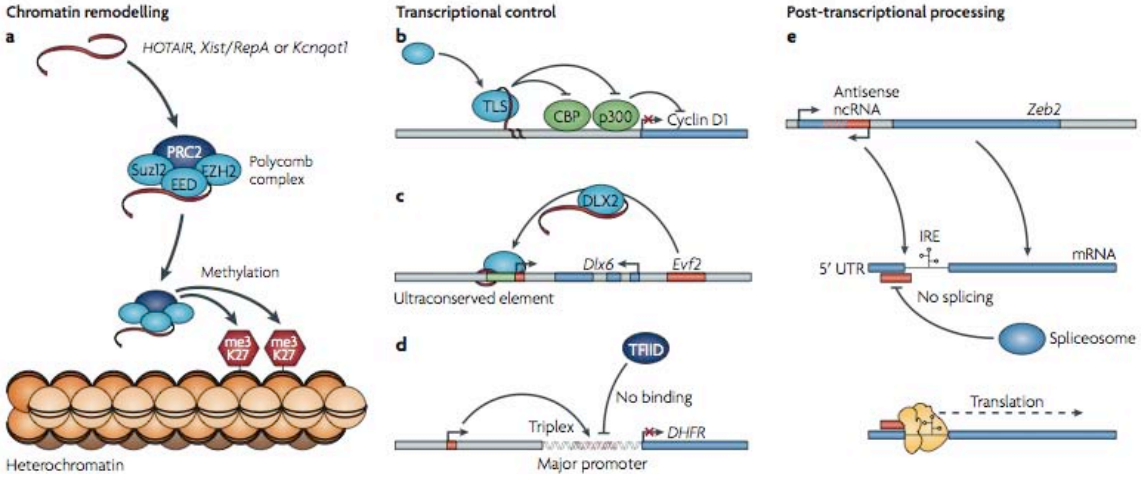


Figure 2 | **Functions of long non-coding RNAs (ncRNAs).** Illustrative mechanisms by which long ncRNAs regulate local protein-coding gene expression at the level of chromatin remodelling, transcriptional control and post-transcriptional processing. **a** | ncRNAs can recruit chromatin modifying complexes to specific genomic loci to impart their catalytic activity. In this case, the ncRNAs *HOTAIR*[21], *Xist* and *RepA* (the small internal non-coding transcript from the *Xist* locus)[25], or *Kcnqot1* (REF. 24) recruit the Polycomb complex to the *HoxD* locus, the X chromosome, or the *Kcnq1* domain, respectively, where they trimethylate lysine 27 residues (me3K27) of histone H3 to induce heterochromatin formation and repress gene expression. **b** | ncRNAs can regulate the transcriptional process through a range of mechanisms. ncRNAs tethered to the cyclin D1 gene recruit the RNA binding protein TLS to modulate the histone acetyltransferase activity of CREB binding protein (CBP) and p300 to repress gene transcription[29]. **c** | An ultraconserved enhancer is transcribed as a long ncRNA, *Evf2*, which subsequently acts as a co-activator to the transcription factor DLX2, to regulate the *Dlx6* gene transcription[30]. **d** | A ncRNA transcribed from the *DHFR* minor promoter in humans can form a triplex at the major promoter to occlude the binding of the general transcription factor TFIID, and thereby silence *DHFR* gene expression[31]. **e** | An antisense ncRNA can mask the 5′ splice site of the zinc finger homeobox mRNA *Zeb2* from the spliceosome, resulting in intron retention. The translation machinery can then recognize and bind an internal ribosome entry site (IRE) in the retained intron, resulting in efficient *Zeb2* translation and expression[35].

RNA regulation of epigenetic processes

Bioessays
2009 vol. 31 (1) pp. 51-9

Mattick JS, Amaral PP, Dinger ME, Mercer TR, Mehler MF

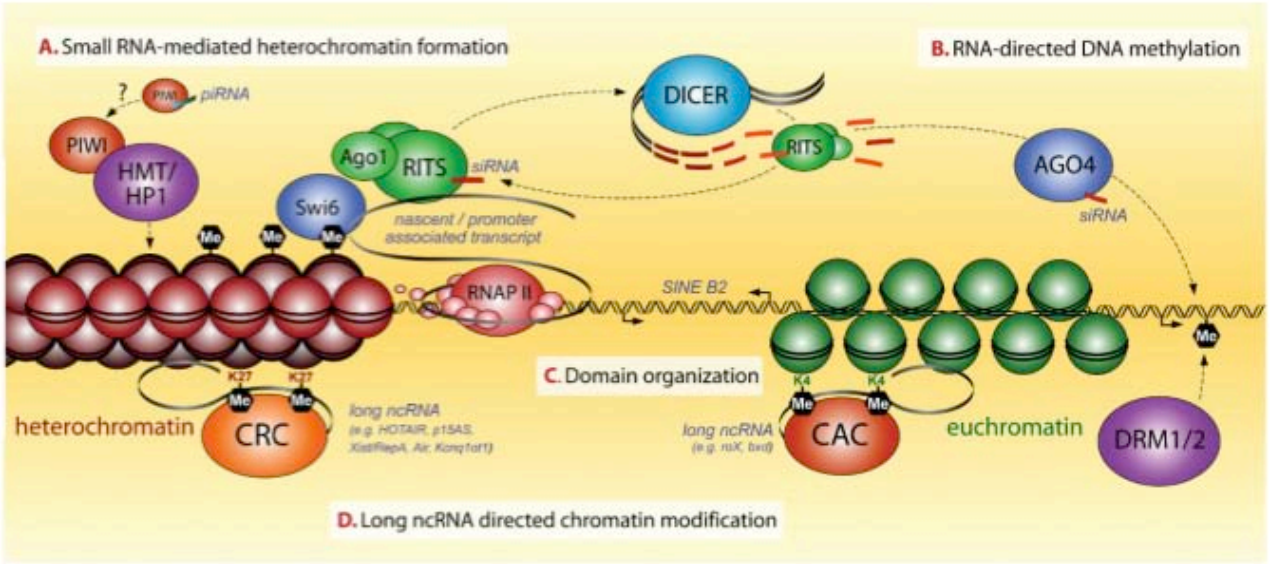
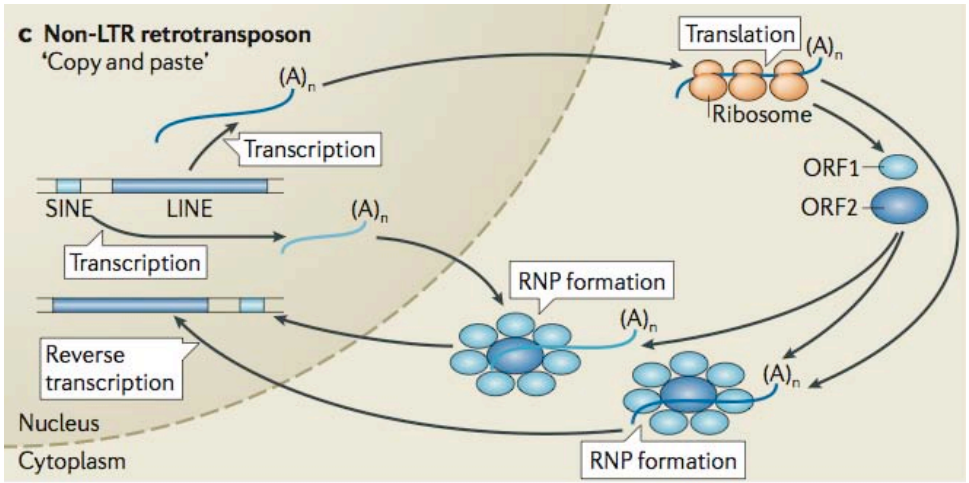


Figure 1. Simplified representation of RNA-mediated processes that direct chromatin modifications in various eukaryotic organisms. (A) Various small RNAs may direct chromatin modifications. RNA duplexes formed by heterochromatic transcription may be processed in a Dicer-dependent manner into siRNAs (short interfering RNAs) that subsequently direct chromatin modifications, possibly by targeting nascent transcripts or DNA directly. SiRNAs may direct histone methylation (Me) *via* RITS (RNA-induced transcriptional silencing complex) in centromere heterochromatin in fission yeast, which results in recruitment of heterochromatin-associated factors such as Swi6 (HP1 homolog) in yeast.[32–34] PIWI proteins and possibly piRNAs (PIWI-associated RNAs) interact with HP1a (heterochromatin protein 1a) and HMT (histone methyltransferases) complexes to induce heterochromatin formation in *Drosophila* and direct DNA methylation in mammalian germ cells.[43,45,46] (B) Alternatively siRNAs originating from RNA polymerase IV transcripts can direct DNA methylation by a DRM2 (domains rearranged methyltransferase 2) dependent mechanism in plants.[47,48] (C) The transcription of SINE B2 elements can establish boundaries between euchromatin and heterochromatin domains in mouse.[80] (D) Long ncRNAs can also recruit chromatin activating complexes (CACs)[56–59] or chromatin repressor complexes (CRCs)[60–66] to target loci in *cis* or *trans*, thereby regulating the chromatin context of local genes.

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



RNA editing, DNA recoding and the evolution of human cognition

Trends in Neurosciences
2008 vol. 31 (5) pp. 227–33

Mattick JS, Mehler MF

Table 1. Human A-to-I edited DNA repair enzymes: functional roles

Gene name	Comment	Functional categories
BRCA1		DSBR (NHEJ, HR); MMR; TCR
Claspin		DSBR (HR)
DDB2[a]		NER; GGR; MMR
DMC1	Rad51 family	Meiotic HR
FANCC[a]		DSBR (HR); TLS
FANCD2		DSBR (HR); TLS
MSH2	Mismatch repair enzymes	MMR; DSBR (HR)
MSH5	Mismatch repair enzymes	MMR; DSBR (HR)
NCoA6[a]		DSBR (NHEJ)
NEIL1		BER; TCR
POLM[a]	X family DNA polymerases	DSBR (NHEJ); TLS
Rad1		BER; TLS
Rad51		DSBR (HR); TLS
RecQL5		DSBR (HR); NER; TCR
Rev3L	Pol-ζ	TLS
TOP3A[a]		DSBR (HR); NER; MMR
UBE2B	Rad6 homolog; ubiquitin [E2]-conjugating enzyme	TLS
USP1[a]		DSBR (HR); TLS
XPA[a]		NER; GGR; TCR
XPB[a]	ERCC3	NER; GGR; TCR
XPV	Pol-η; Y family DNA polymerases	NER; GGR; TLS
XRCC6	Ku70	DSBR (NHEJ)

Abbreviations: DSBR, double-strand break repair; NHEJ, non-homologous end joining; HR, homologous recombination; NER, nucleotide excision repair; BER, base excision repair; MMR, mismatch repair; GGR, global general repair; TCR, transcription-coupled repair; TLS, trans-lesional synthesis.
[a]Gene loci specifically verified to have edited transcripts in neural tissues. Supporting information can be found in Refs [38–41].

Box 1. Categories/roles of edited genes involved in nervous system development and function

(a) System-wide adaptations
i. Neural induction (*SMAD1*; *IFNR1*)
ii. Anterior (forebrain) neural tube patterning (*FGFR1*; *Formin2*; *HHAT*)

(b) Adaptations of regional neural stem cell functions
i. Neural stem cell (NSC) self-renewal (*NuMA1*; *CD44*; *SNX1*)
ii. NSC asymmetric (neurogenic) cell divisions (*Nde1*)
iii. Modulation of NSC proliferation (*CDC2L5*; *RBBP7*; *PKCD1*; *SYK*)

(c) Adaptations of neuronal precursor (neuroblast) development
i. Neuronal precursor (neuroblast; NB) migration (*CXCL1*; *Foxp1*)
ii. NB cell-cycle kinetics (*Par6*; *CDK10*; *CDKL1*; *MCM3*; *DNM2*; *Cullin1*)
iii. Modulation of NB cell-cycle exit (*Sox13*)

(d) Adaptations of the process of neuronal maturation
i. Progressive neuronal differentiation (*TLE2*)
ii. Neuronal morphogenesis (*PAK4*; *SPARC*)
iii. Neuronal cell polarity/neurite process outgrowth (*Neuron navigator1*)
iv. Neuronal axon guidance (*Centaurin-γ2*)
v. Neuronal dendritogenesis (*δ2-Catenin*)
vi. Neuronal synaptogenesis (*Protocadherin* β)
vii. Neuronal subtype specification (*Lhx3*)
viii. Neuronal network connectivity (*Protocadherin α1, 2, 4–6, C1, 2*)

(e) Adaptations of mature neuronal functions
i. Neuronal viability (Beclin1; Casp9, 10; TRAP1; STAG-1; Fas inhibitory molecule 1)
ii. Neuronal excitability (Annexin A4; AMPAR1/GluR1; VDCCβ4; VDKC)
iii. Neuronal cell–cell and cell–environment interactions (Integrin β4)
iv. Cooperative clustering of synaptic neurotransmitter receptors (VDCCβ2)
v. Assembly of multimeric intracellular and cell–cell signaling scaffolds (Syncoilin)
vi. Organization of neuronal somadendritic microdomains (mGluR1)
vii. Neuronal signal transduction (Src homology domain containing E, SHE)
viii. Neuronal plasticity (CaM Kinase II; Synaptotagmin 2; α1-Adaptin; Complexin 1)
ix. Neuronal energy metabolism (CPT1A, C; Dynamin1-like)
x. Neuronal axodendritic transport (Kinesin 1B, 2, 3B, 6; Dynein 10)