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EPIGENÈTICA

PER A DOCENTS DE BATXILLERAT

David Bueno i Torrens

CVE-DOGC-A-22263095-2022

Biologia

Els avenços de la biologia s'han accelerat notablement en les darreres dècades. En aquest camp s'han produït grans canvis de paradigma (com el descobriment de la cèl·lula, el desenvolupament de la teoria de l'evolució, el naixement de la biologia molecular i el descobriment dels virus i els prions, entre d'altres) que han revolucionat el concepte d'organisme viu i la comprensió del seu funcionament.

CVE-DOGC-A-22263095-2022

Genètica i cicle cel·lular

- Relació entre les característiques químiques, l'estructura i la funció biològica dels diferents tipus d'àcids nucleics.

- Anàlisi del mecanisme de replicació de l'ADN mitjançant el model procariota.

- Anàlisi, utilitzant un model procariota, de les etapes generals de l'expressió gènica i de les característiques del codi genètic i resolució de problemes relacionats amb aquestes.

- Resolució de problemes de monohibridisme i dihibridisme en casos d'herència autosòmica i lligada al sexe.

- Interpretació de l'evolució com un canvi en la freqüència gènica, tot resolent problemes senzills de genètica quantitativa.

- Argumentació sobre la relació entre les mutacions, la replicació de l'ADN, l'evolució i la biodiversitat.

- Valoració de la importància de la regulació de l'expressió gènica en la diferenciació cel·lular.

- Comparació de les característiques generals del genoma i de l'expressió gènica en procariotes i eucariotes.

- Seqüenciació de les fases del cicle cel·lular i anàlisi dels mecanismes de regulació.

- Comparació de la meiosi i la mitosi: fases i funció.

- Estudi del càncer i la relació amb les mutacions i l'alteració del cicle cel·lular.

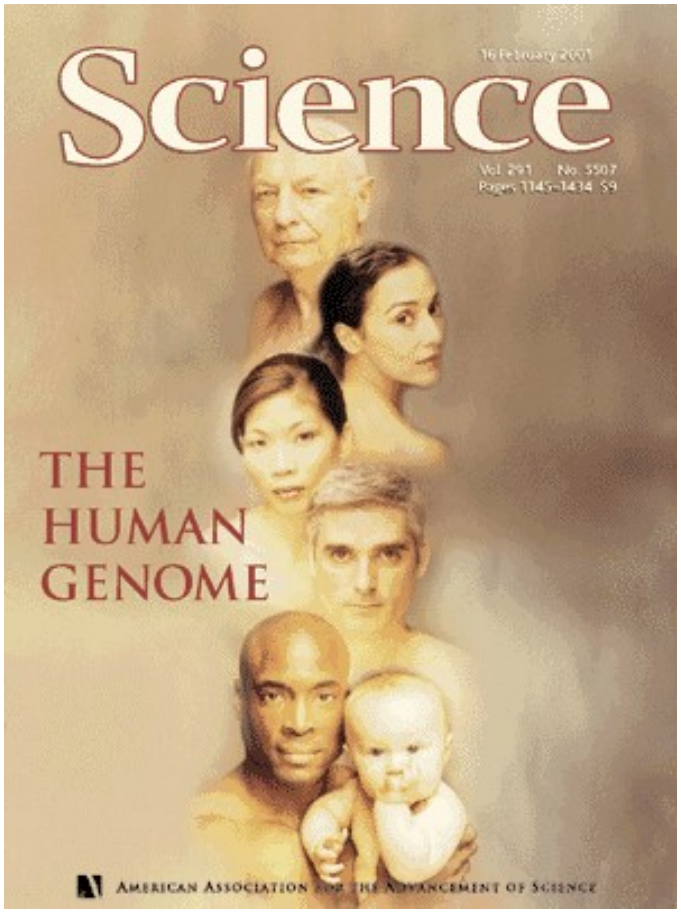
– Anàlisi, utilitzant un model procariota, de les etapes generals de l'expressió gènica i de les característiques del codi genètic i resolució de problemes relacionats amb aquestes.

-Valoració de la importància de la regulació de l'expressió gènica en la diferenciació cel·lular.

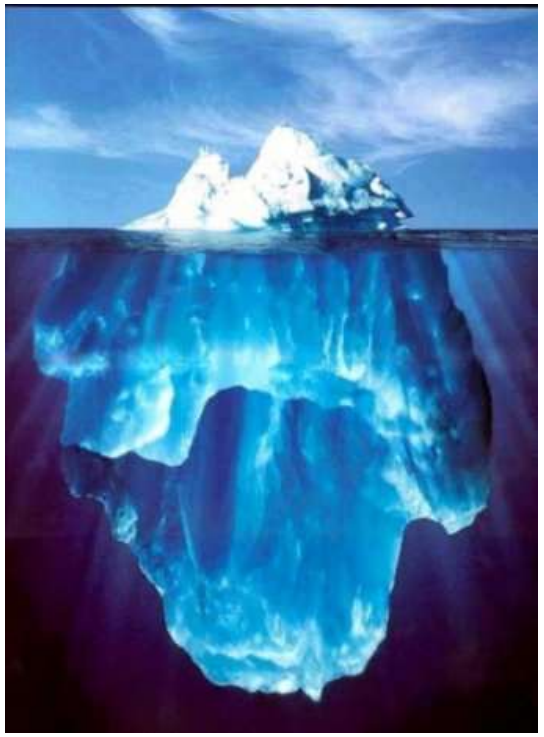
Interpretar i relacionar l'expressió gènica i el codi genètic amb la síntesi de proteïnes. Comparar transcripció i traducció en procariotes i eucariotes: mecanisme i enzims implicats. Regulació de l'expressió gènica en procariotes (exemple dels operons) i eucariotes (promotors, *enhancers* o intensificadors, factors de transcripció, activadors i repressors). Les marques epigenètiques com a sistema de regulació de l'expressió gènica. Mecanismes genètics i epigenètics de la diferenciació cel·lular.

https://www.ub.edu/paubiologia/Espec%C3%ADfic2023/Orientacions.pdf

El genoma humà

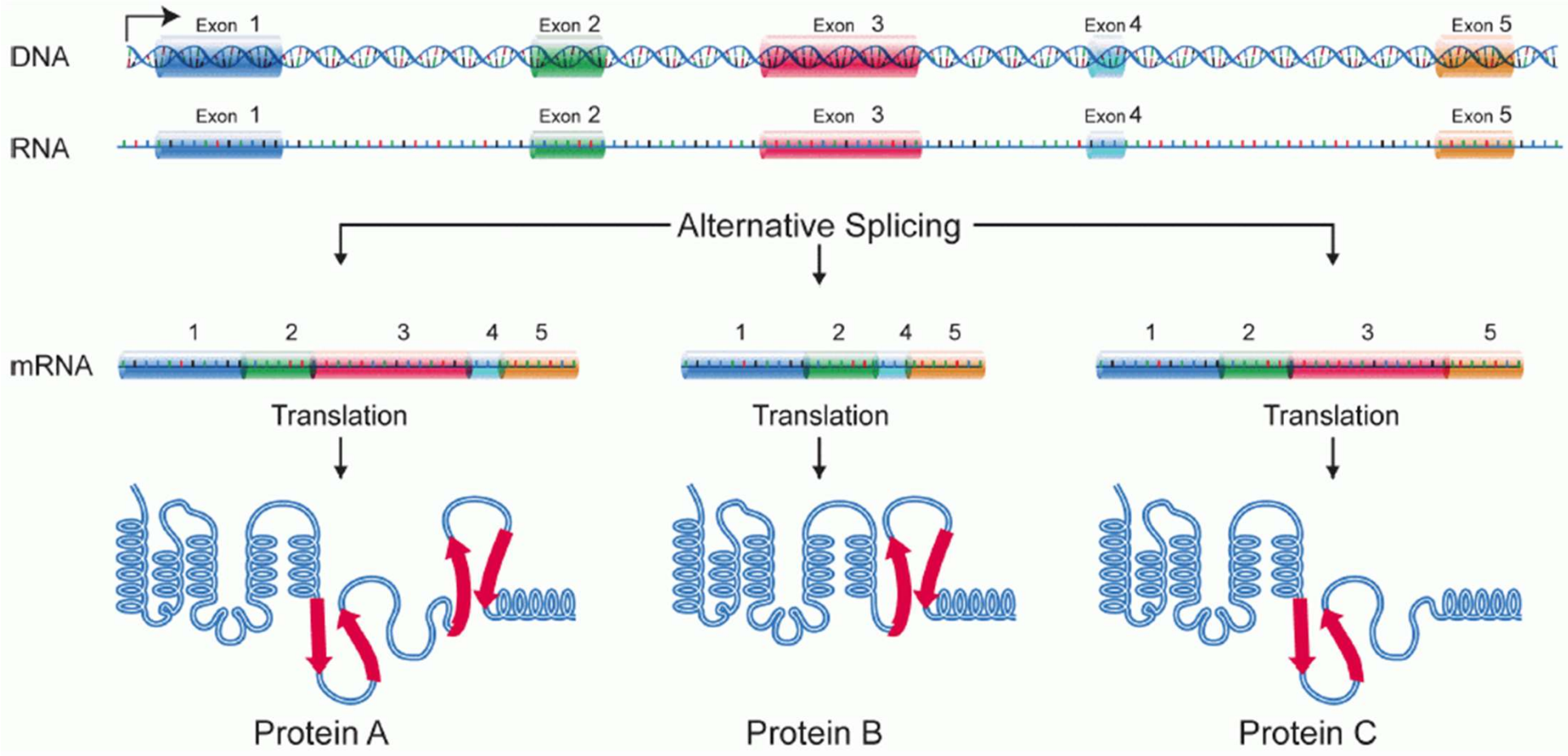


El genoma és
“la punta de l’iceberg”

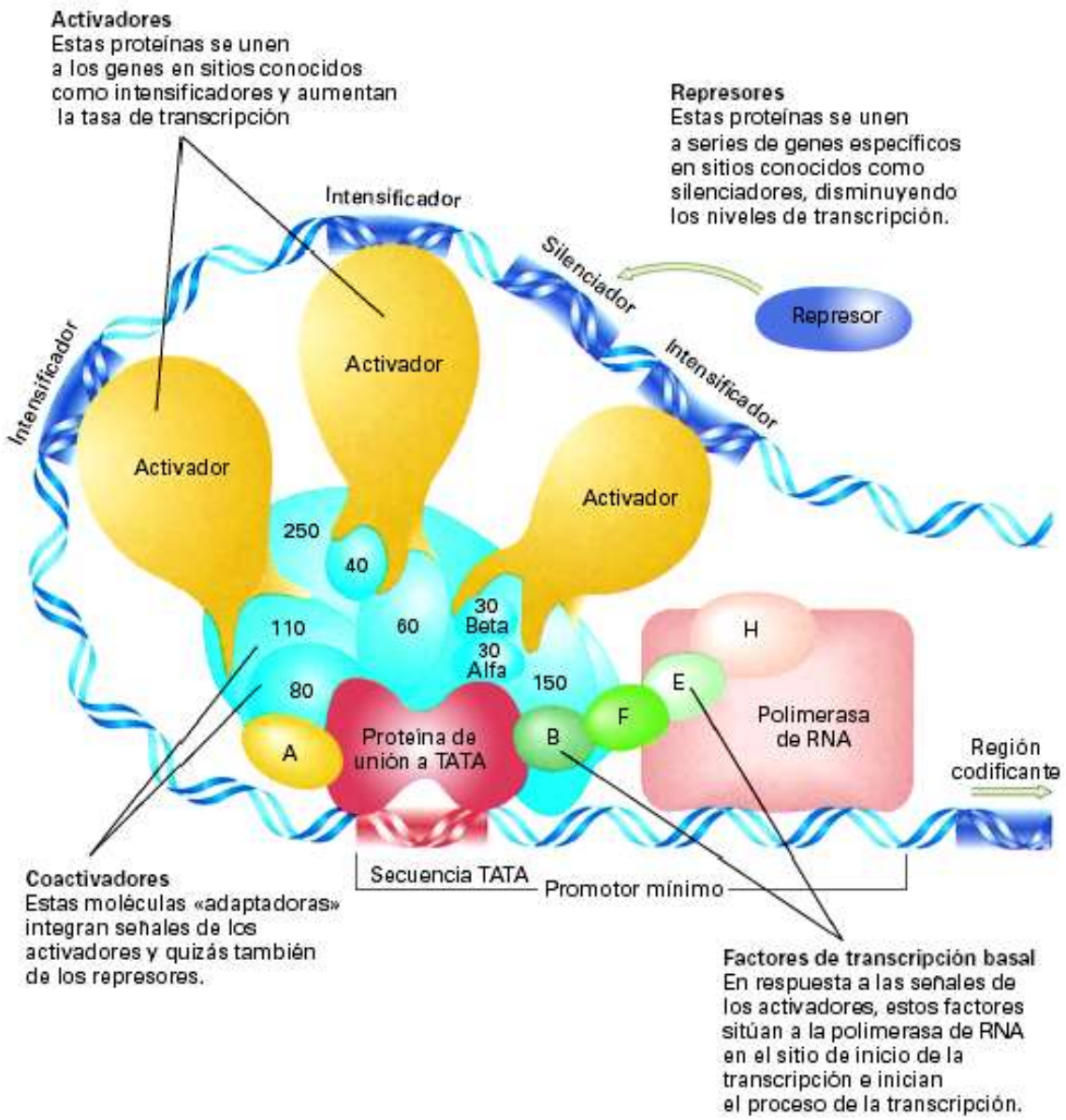


20.300 gens → més de 100.000 proteïnes

Splicing diferencial
(empalmament diferencial, tall i unió diferencial)



Regulació de l'expressió gènica



Encyclopedia of DNA Elements

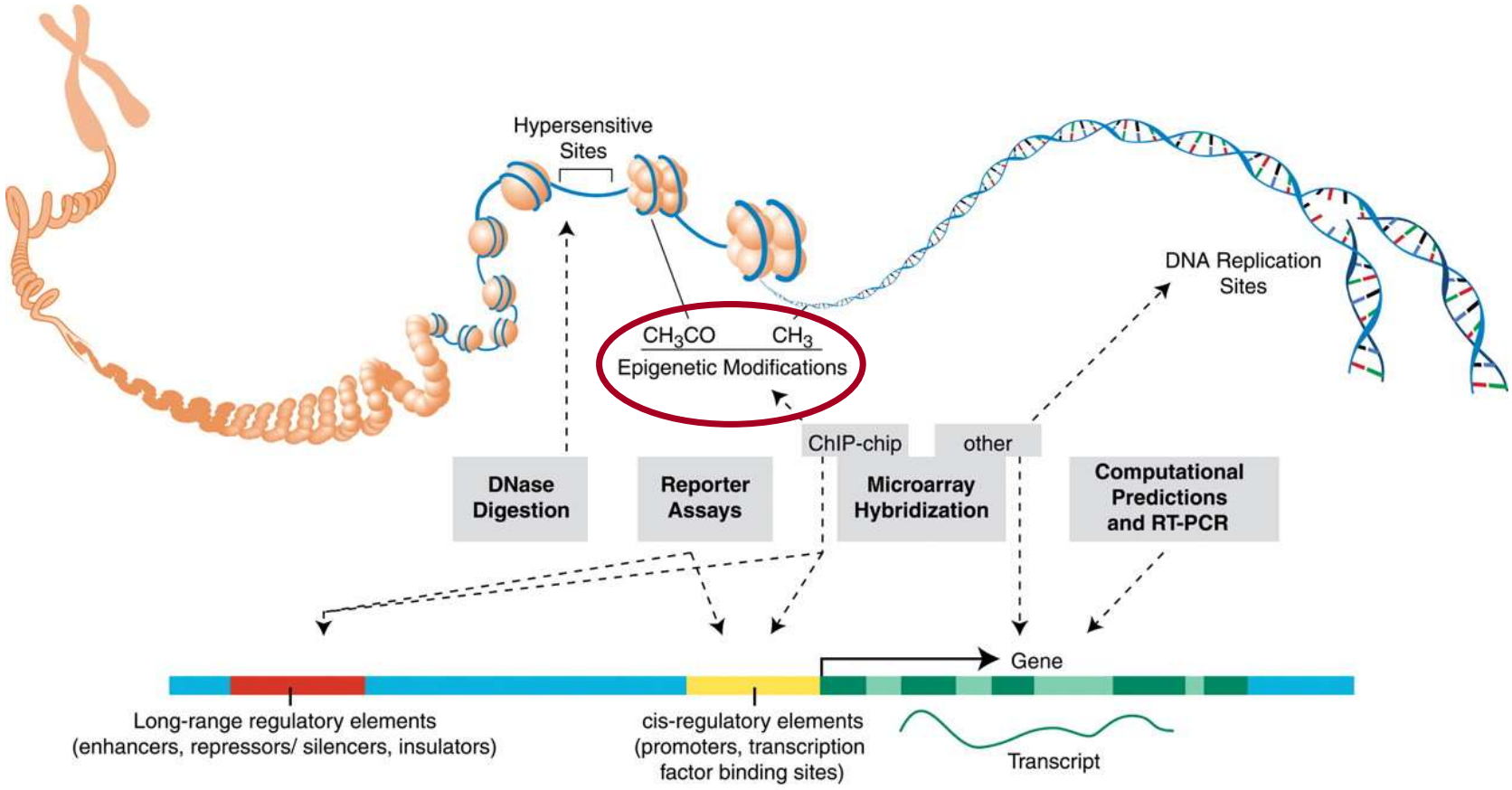
Abans del projecte ENCODE

- 1,1% del genoma eren exons
- 24% del genoma eren introns
- 75% restant no tenia funció coneguda - “DNA escombraria” (*junk DNA*) ????

Resultats del projecte ENCODE

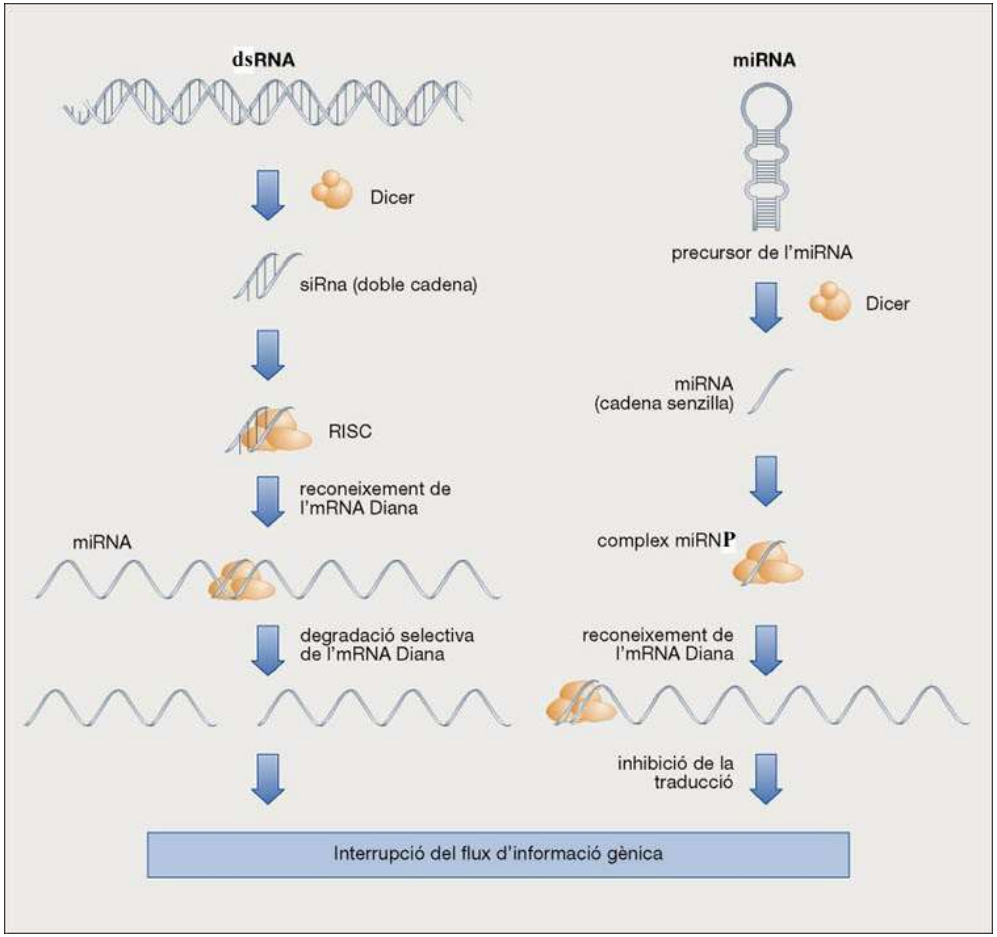
- 80% del genoma té una funció
    - 2,9% del genoma són exons
    - 20% del genoma són introns
    - 20% del genoma són promotors i intensificadors
    - I la resta, regions implicades en la topologia del DNA, miRNAs i zones per modificacions epigenètiques

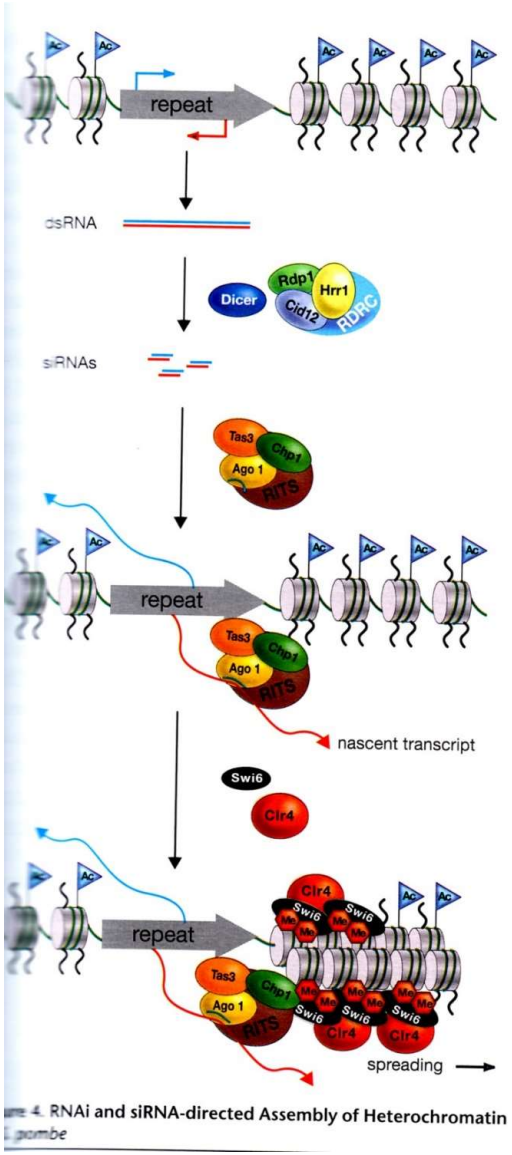
Fig. 1. Functional genomic elements being identified by the ENCODE pilot phase.



Science 2004;306:636-640

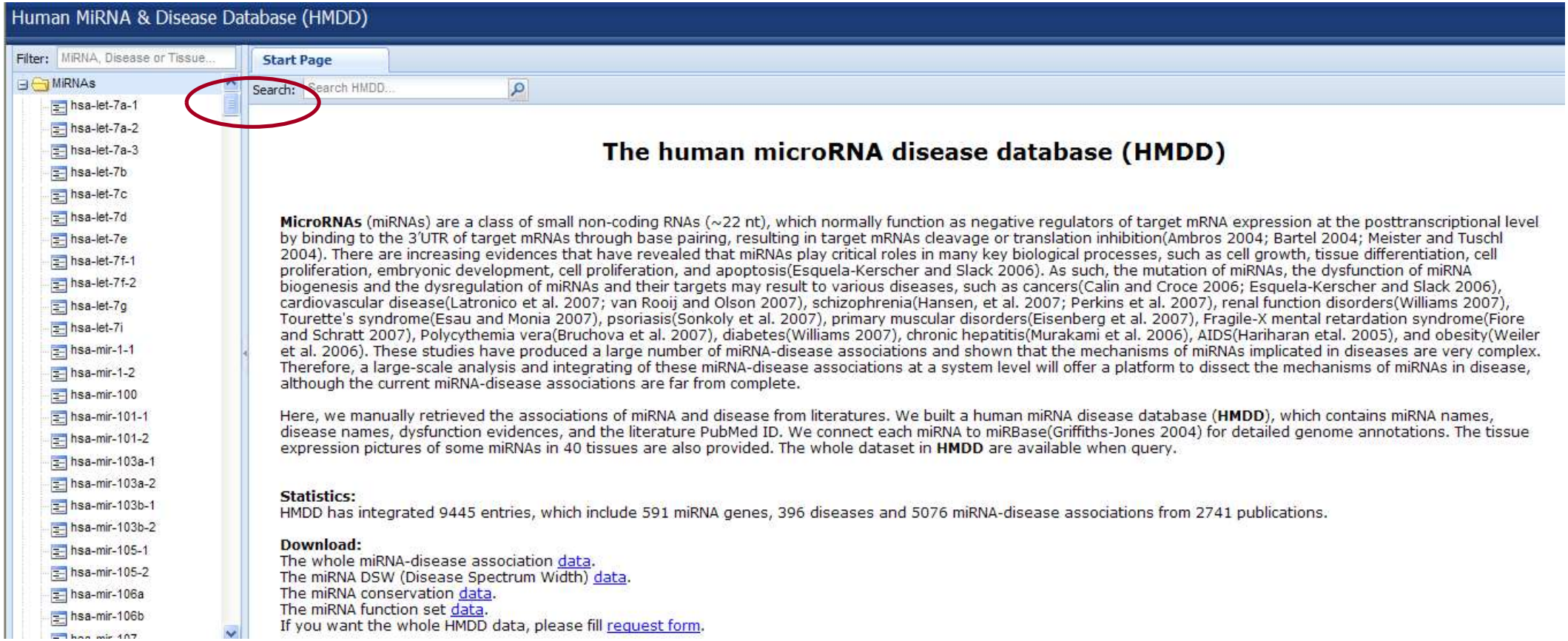
Els miRNA





RNAi and siRNA-directed Assembly of Heterochromatin
pombe

Els miRNA


Human MiRNA & Disease Database (HMDD)
Filter: MiRNA, Disease or Tissue...
MiRNAs
hsa-let-7a-1
hsa-let-7a-2
hsa-let-7a-3
hsa-let-7b
hsa-let-7c
hsa-let-7d
hsa-let-7e
hsa-let-7f-1
hsa-let-7f-2
hsa-let-7g
hsa-let-7i
hsa-mir-1-1
hsa-mir-1-2
hsa-mir-100
hsa-mir-101-1
hsa-mir-101-2
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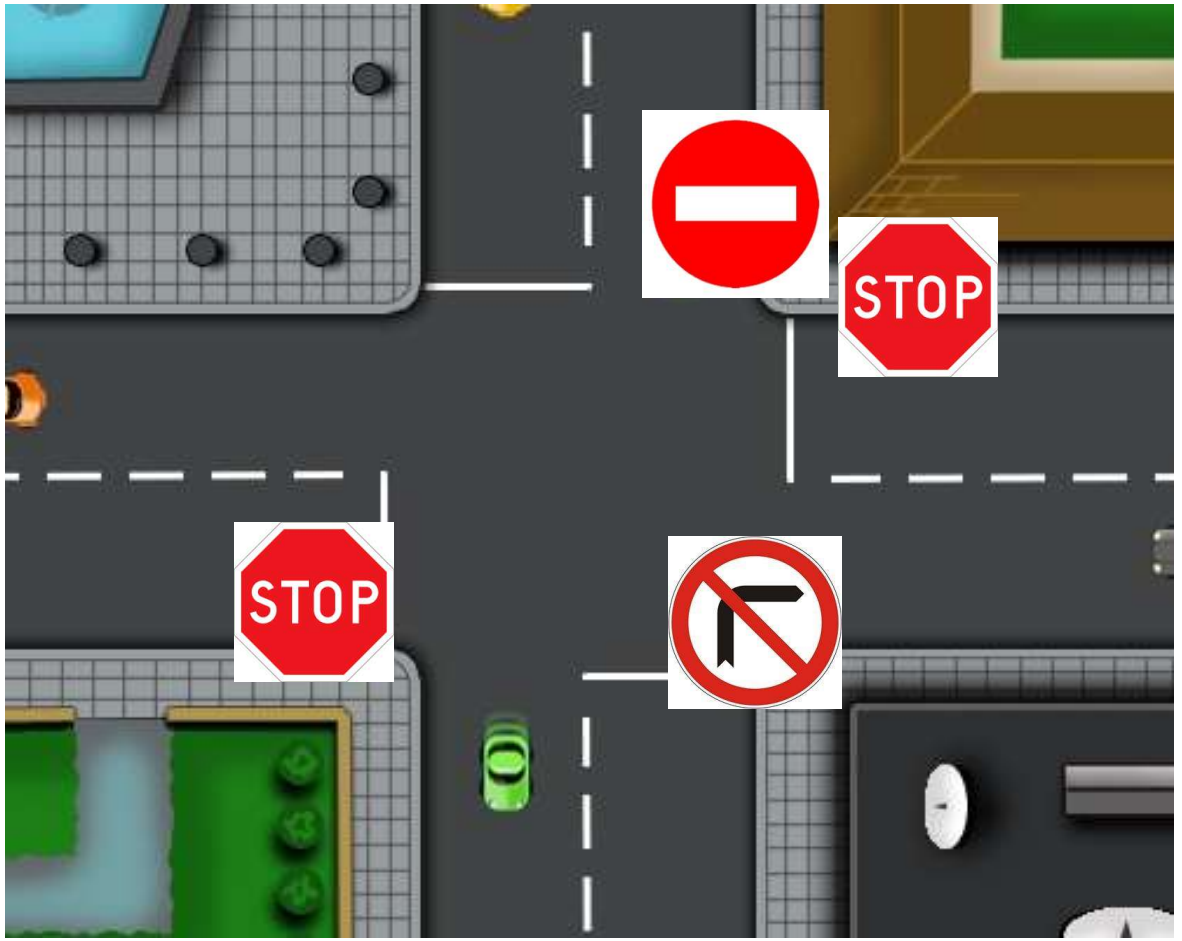
hsa-mir-103b-1
hsa-mir-103b-2
hsa-mir-105-1
hsa-mir-105-2
hsa-mir-106a
hsa-mir-106b
Start Page
Search: Search HMDD...
The human microRNA disease database (HMDD)
MicroRNAs (miRNAs) are a class of small non-coding RNAs (~22 nt), which normally function as negative regulators of target mRNA expression at the posttranscriptional level by binding to the 3'UTR of target mRNAs through base pairing, resulting in target mRNAs cleavage or translation inhibition(Ambros 2004; Bartel 2004; Meister and Tuschl 2004). There are increasing evidences that have revealed that miRNAs play critical roles in many key biological processes, such as cell growth, tissue differentiation, cell proliferation, embryonic development, cell proliferation, and apoptosis(Esquela-Kerscher and Slack 2006). As such, the mutation of miRNAs, the dysfunction of miRNA biogenesis and the dysregulation of miRNAs and their targets may result to various diseases, such as cancers(Calin and Croce 2006; Esquela-Kerscher and Slack 2006), cardiovascular disease(Latronico et al. 2007; van Rooij and Olson 2007), schizophrenia(Hansen, et al. 2007; Perkins et al. 2007), renal function disorders(Williams 2007), Tourette's syndrome(Esau and Monia 2007), psoriasis(Sonkoly et al. 2007), primary muscular disorders(Eisenberg et al. 2007), Fragile-X mental retardation syndrome(Fiore and Schratt 2007), Polycythemia vera(Bruchova et al. 2007), diabetes(Williams 2007), chronic hepatitis(Murakami et al. 2006), AIDS(Hariharan etal. 2005), and obesity(Weiler et al. 2006). These studies have produced a large number of miRNA-disease associations and shown that the mechanisms of miRNAs implicated in diseases are very complex. Therefore, a large-scale analysis and integrating of these miRNA-disease associations at a system level will offer a platform to dissect the mechanisms of miRNAs in disease, although the current miRNA-disease associations are far from complete.
Here, we manually retrieved the associations of miRNA and disease from literatures. We built a human miRNA disease database (HMDD), which contains miRNA names, disease names, dysfunction evidences, and the literature PubMed ID. We connect each miRNA to miRBase(Griffiths-Jones 2004) for detailed genome annotations. The tissue expression pictures of some miRNAs in 40 tissues are also provided. The whole dataset in HMDD are available when query.
Statistics:
HMDD has integrated 9445 entries, which include 591 miRNA genes, 396 diseases and 5076 miRNA-disease associations from 2741 publications.
Download:
The whole miRNA-disease association data.
The miRNA DSW (Disease Spectrum Width) data.
The miRNA conservation data.
The miRNA function set data.
If you want the whole HMDD data, please fill request form.

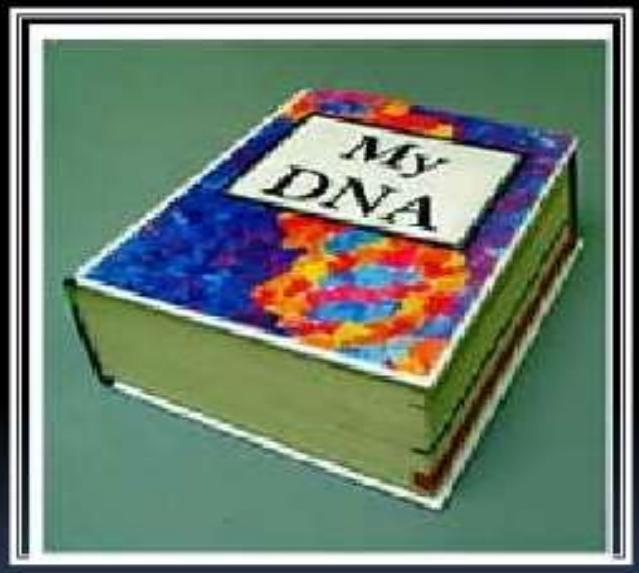
Marques (modificacions) epigenètiques

Marques (modificacions) epigenètiques



Marques (modificacions) epigenètiques

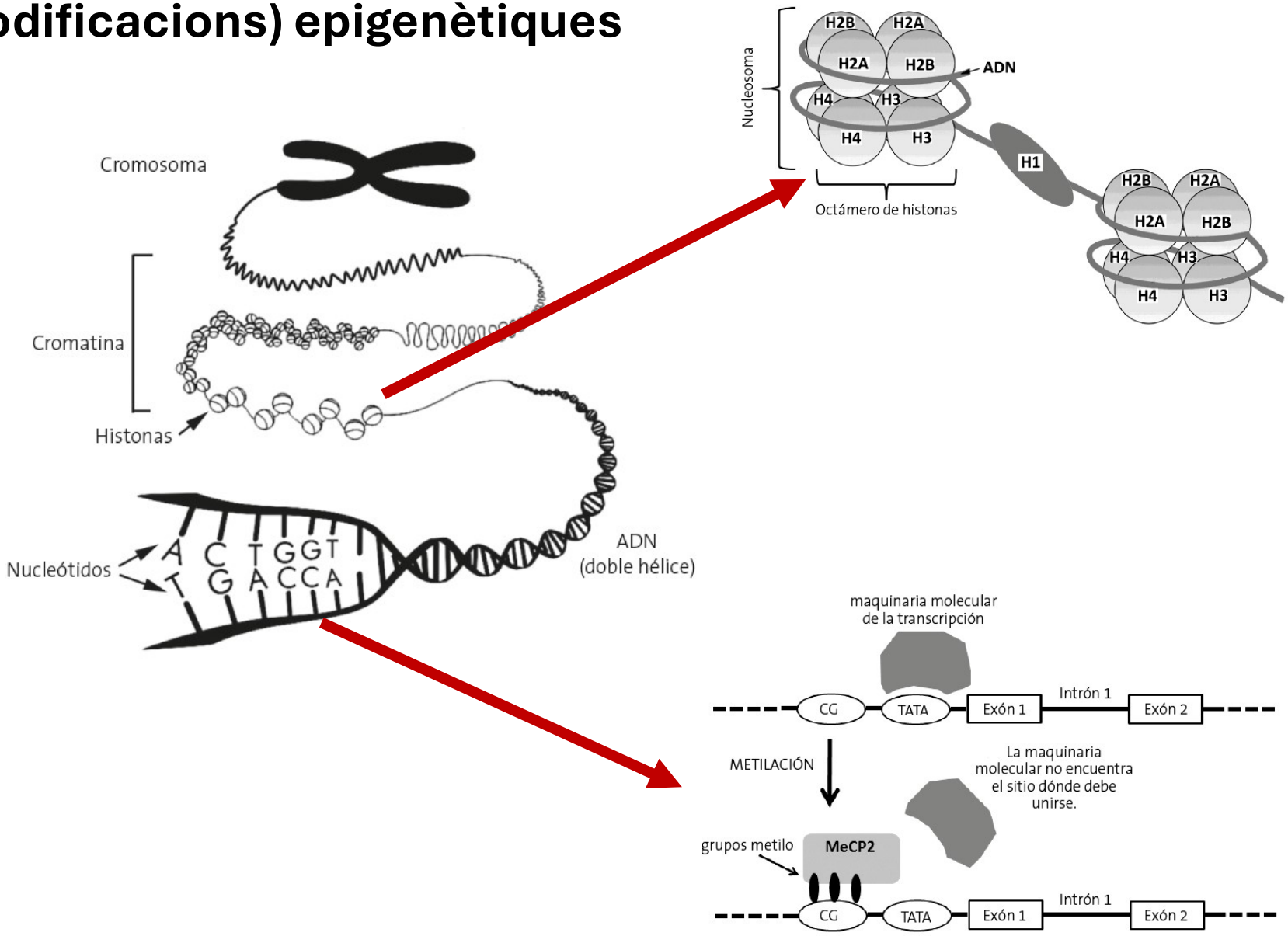
GENETICS VERSUS EPIGENETICS



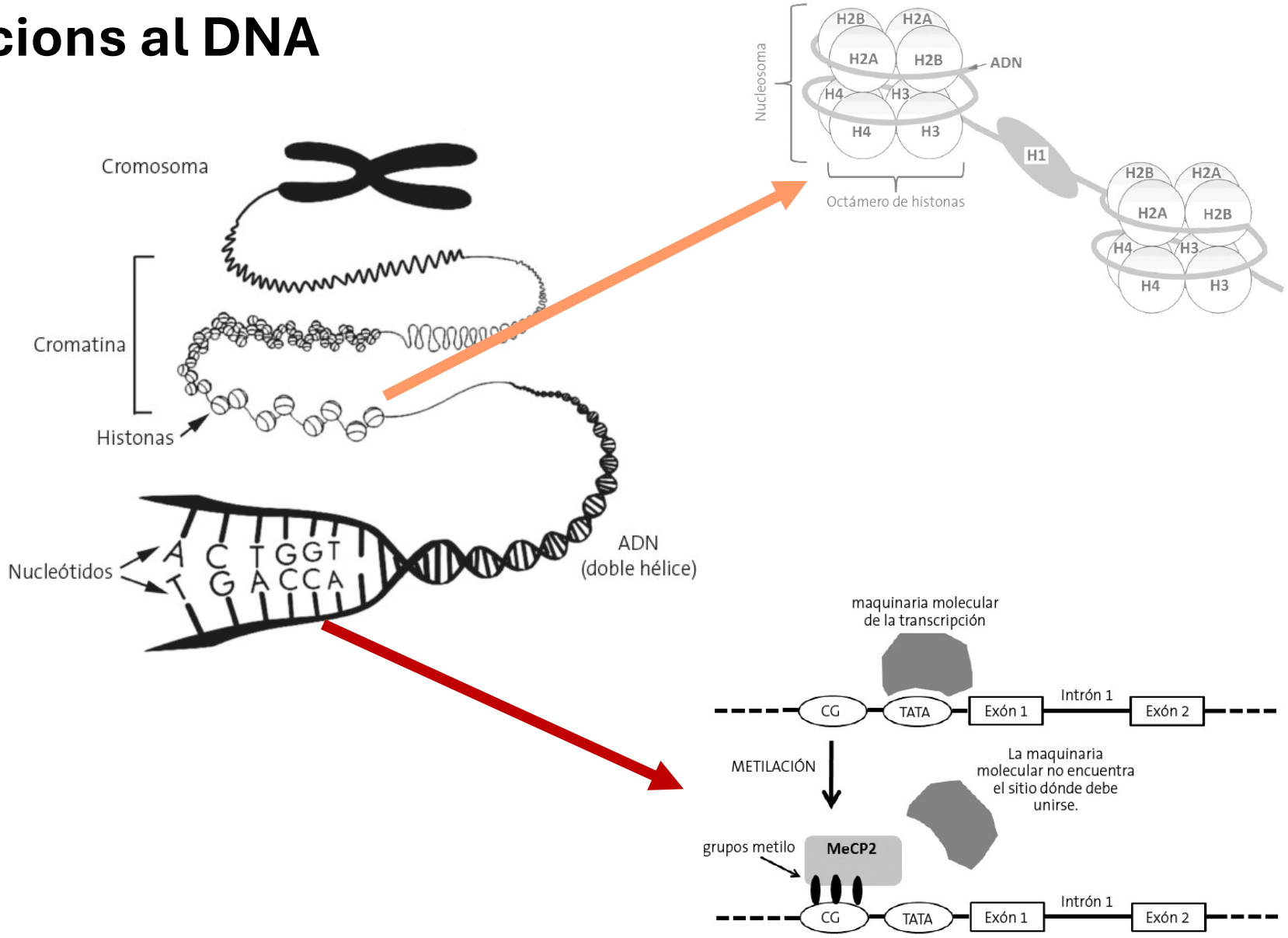
GENETICS

EPIGENETICS

Marques (modificacions) epigenètiques



Metilacions al DNA



Metilacions al DNA

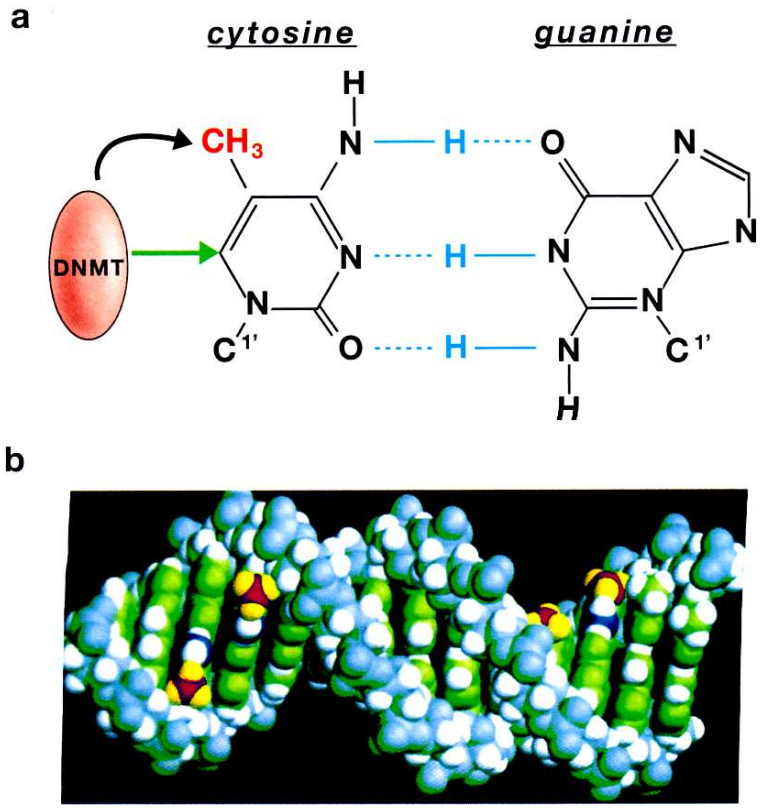


Figure 1. Cytosine Methylation in DNA

a) Addition of a methyl group (*red*) at the 5 position of the cytosine pyrimidine ring (*black arrow*) does not sterically interfere with GC base-pairing (*blue lines*). DNA methyltransferases associate covalently with the carbon-6 position (*green arrow*) during methyl group transfer. (*b*) A model of B-form DNA methylated at cytosines in two self-complementary CpG sequences. The paired methyl moieties (*magenta* and *yellow*) lie in the major groove of the double helix.

Metilacions al DNA

CpG
GpC

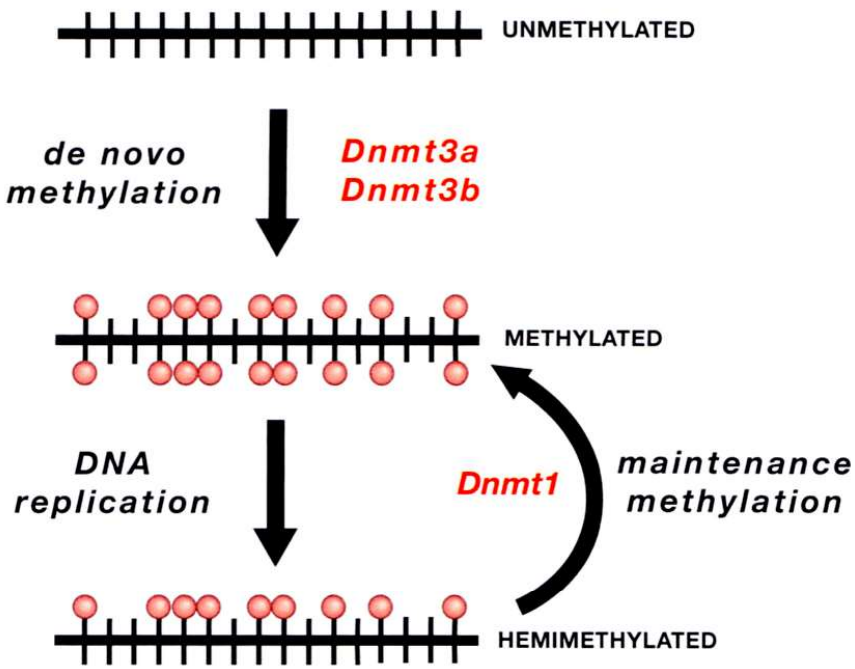


Figure 2. De novo Methylation and Maintenance Methylation of DNA

A stretch of genomic DNA is shown as a line with self-complementary CpG pairs marked as vertical strokes. Unmethylated DNA (*top*) becomes methylated "de novo" by Dnmt3a and Dnmt3b to give symmetrical methylation at certain CpG pairs. Upon semiconservative DNA replication, a progeny DNA strand is base-paired with one of the methylated parental strands (the other replication product is not shown). Symmetry is restored by the maintenance DNA methyltransferase Dnmt1, which completes half-methylated sites, but does not methylate unmodified CpGs.

p344

Caixes (elements o illes) CpG i metilacions al DNA

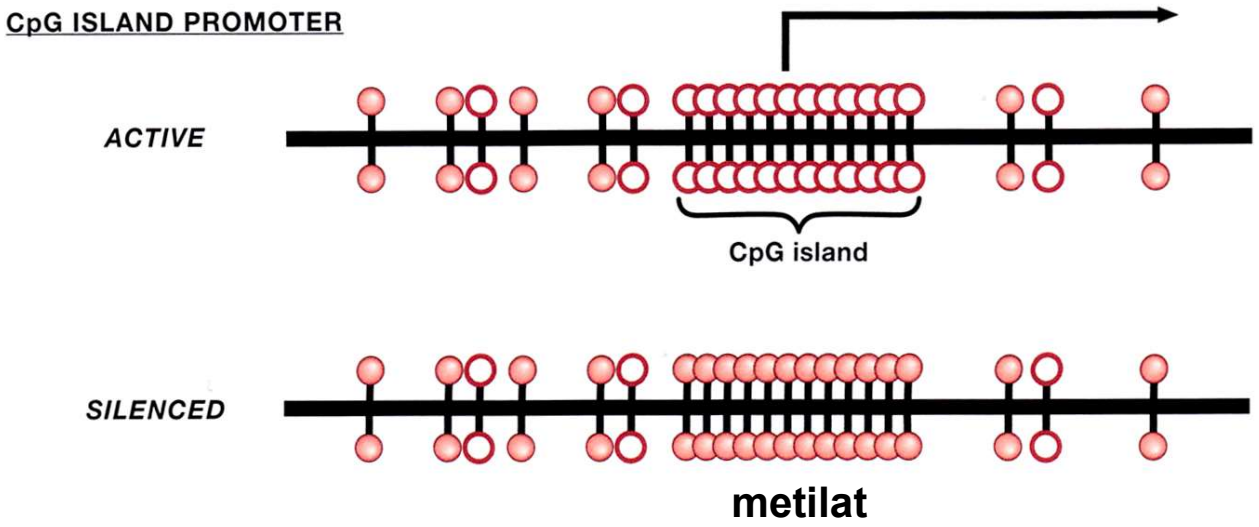


Figure 4. CpG Islands

CpG islands are regions of high CpG density that lack CpG methylation found at promoters of most human genes. Long-term silencing of the gene can be ensured by methylation of the CpG island region. For example, genes on the inactive X chromosome and certain imprinted genes are silenced in this way. Additionally, in cancer cells, certain genes are aberrantly silenced by CpG island methylation.

REVIEW
nature biotechnology

Epigenetic modifications and human disease

Anna Portela[1] & Manel Esteller[1,2]

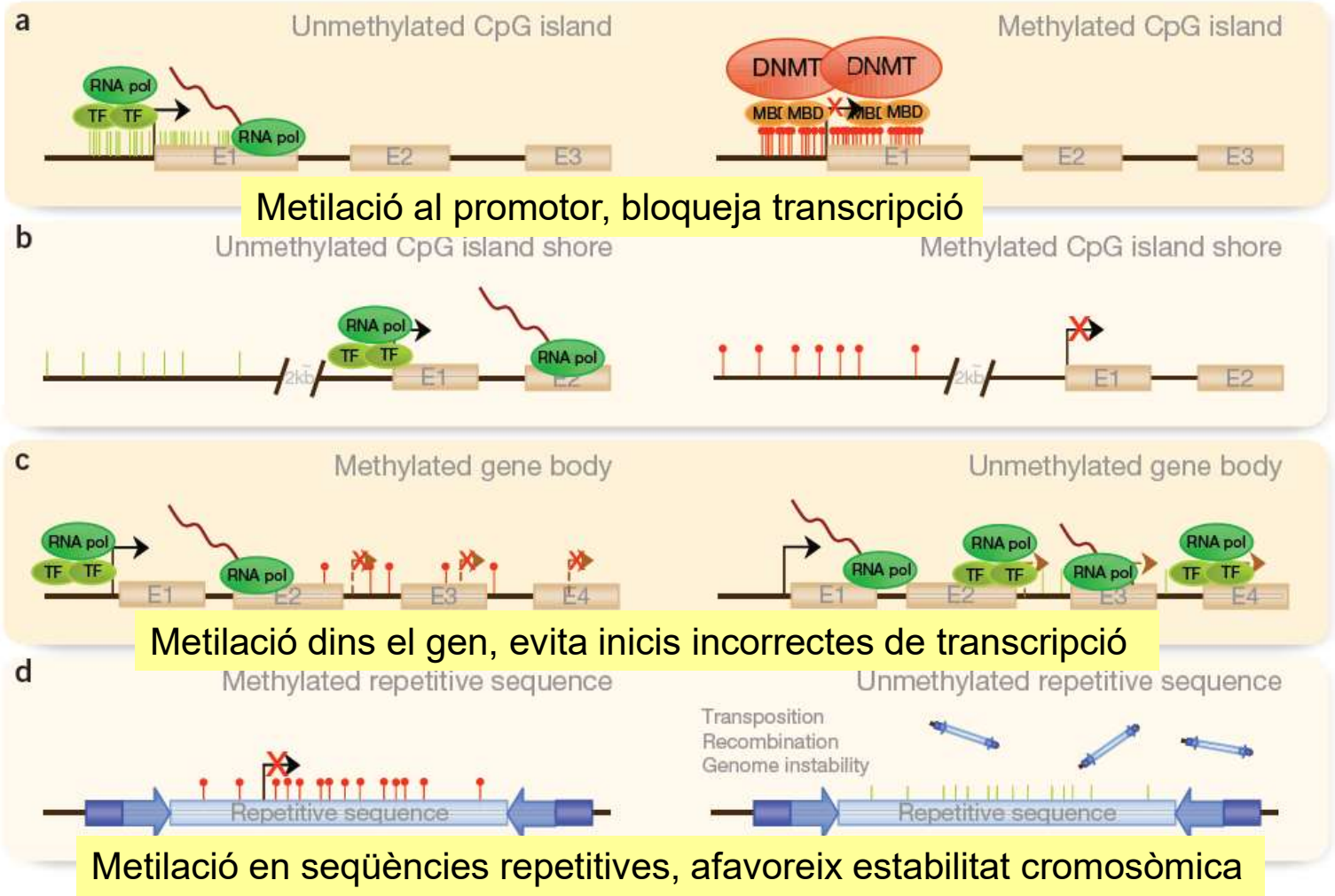
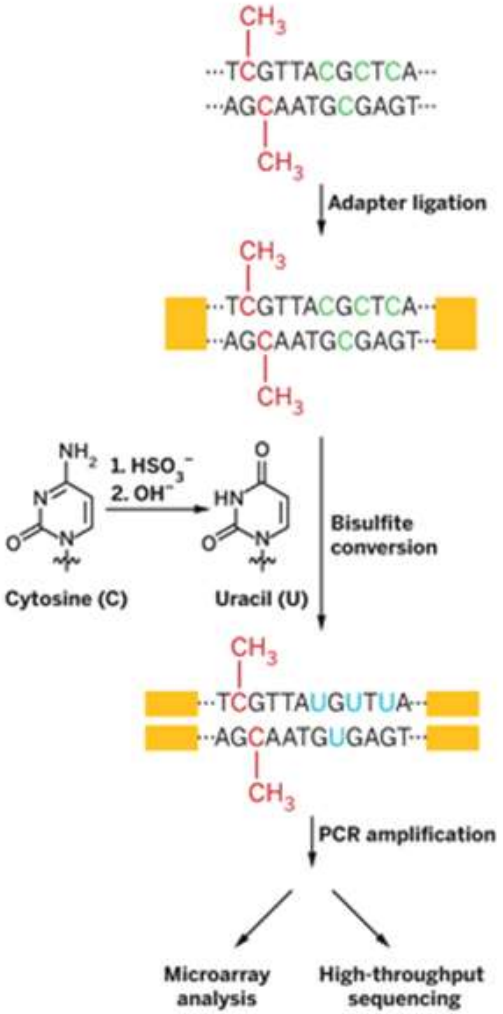
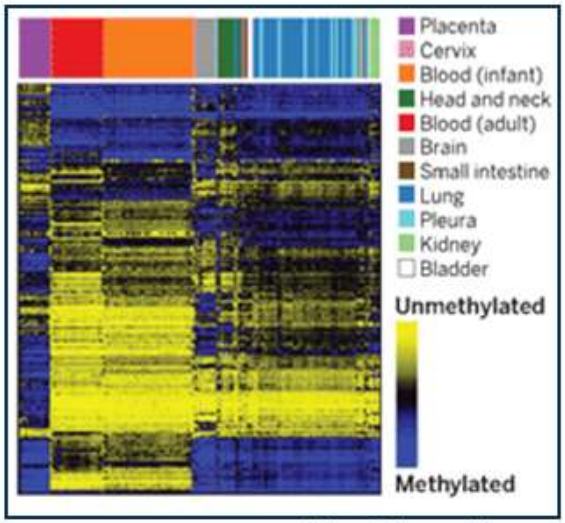


Figure 1 DNA methylation patterns. DNA methylation can occur in different regions of the genome. The alteration of these patterns leads to disease in the cells. The normal scenario is depicted in the left column and alterations of this pattern are shown on the right. (**a**) CpG islands at promoters of genes are normally unmethylated, allowing transcription. Aberrant hypermethylation leads to transcriptional inactivation. (**b**) The same pattern is observed when studying island shores, which are located up to 2 kb upstream of the CpG island. (**c**) However, when methylation occurs at the gene body, it facilitates transcription, preventing spurious transcription initiations. In disease, the gene body tends to demethylate, allowing transcription to be initiated at several incorrect sites. (**d**) Finally, repetitive sequences appear to be hypermethylated, preventing chromosomal instability, translocations and gene disruption through the reactivation of endoparasitic sequences. This pattern is also altered in disease.

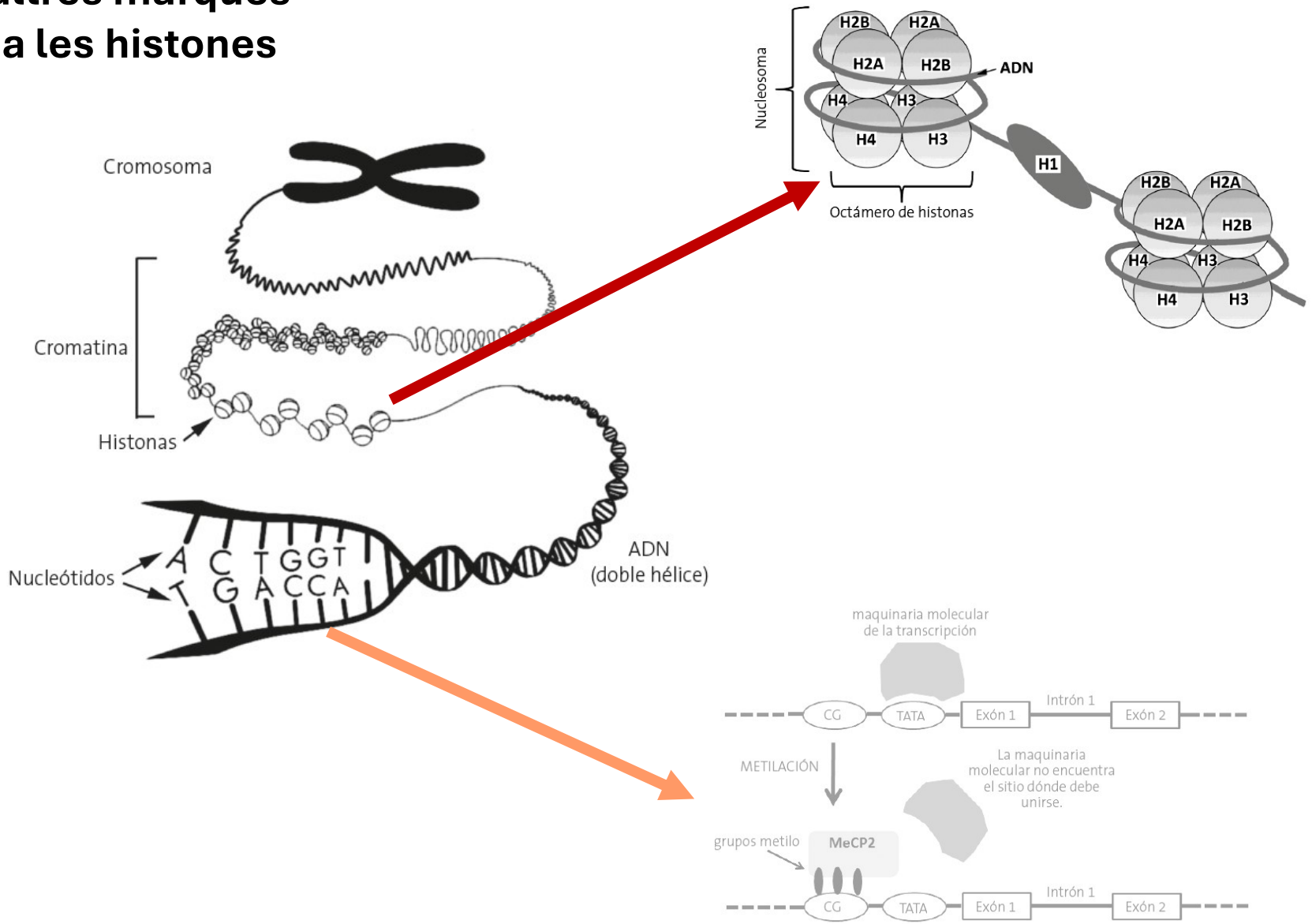
Com se “seqüencien” les metilacions al DNA?



METHYL MAPPING Sodium bisulfite treatment converts unmethylated cytosine residues (green) in genomic DNA to uracil (blue). Methylated cytosines (red) resist conversion and are identified by microarrays or sequencing. Gold blocks indicate common adapter DNA sequences ligated to the ends of each DNA fragment to enable amplification by polymerase chain reaction (PCR).



Acetilacions (i altres marques epigenètiques) a les histones



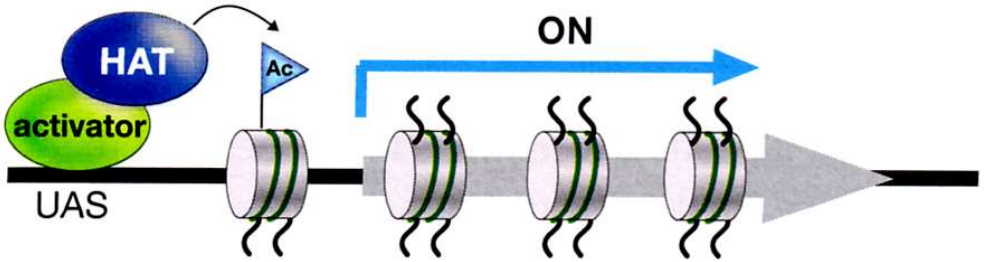
Acetilacions (i altres marques epigenètiques) a les histones

Figure 6. Sites of Histone Tail Modifications

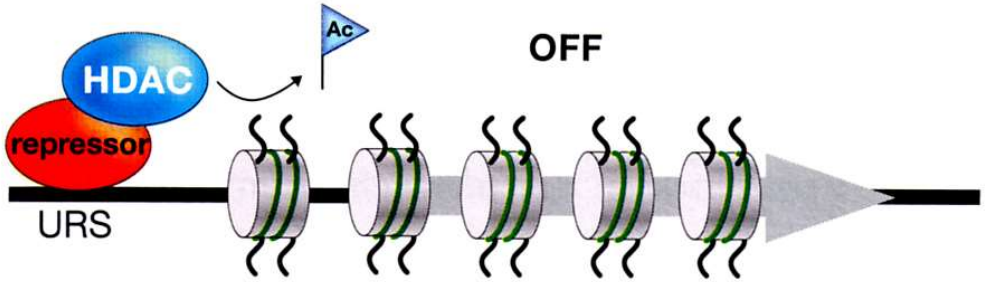
The amino-terminal tails of histones account for a quarter of the nucleosome mass. They host the vast majority of known covalent modification sites as illustrated. Modifications do also occur in the globular domain (*boxed*), some of which are indicated. In general, active marks include acetylation (*turquoise Ac flag*), arginine methylation (*yellow Me hexagon*), and some lysine methylation such as H3K4 and H3K36 (*green Me hexagon*). H3K79 in the globular domain has anti-silencing function. Repressive marks include H3K9, H3K27, and H4K20 (*red Me hexagon*), Green = active mark, red = repressive mark.

Activació i repressió de l'expressió gènica mitjançant marques epigenètiques a les histones

Gene activator recruits histone acetyltransferase



Gene repressor recruits histone deacetylase



gure 2. Histone-modifying Enzymes Are Recruited to Promot-
s by DNA-binding Transcription Factors

Transmissió de les marques epigenètiques a les cèl·lules filles

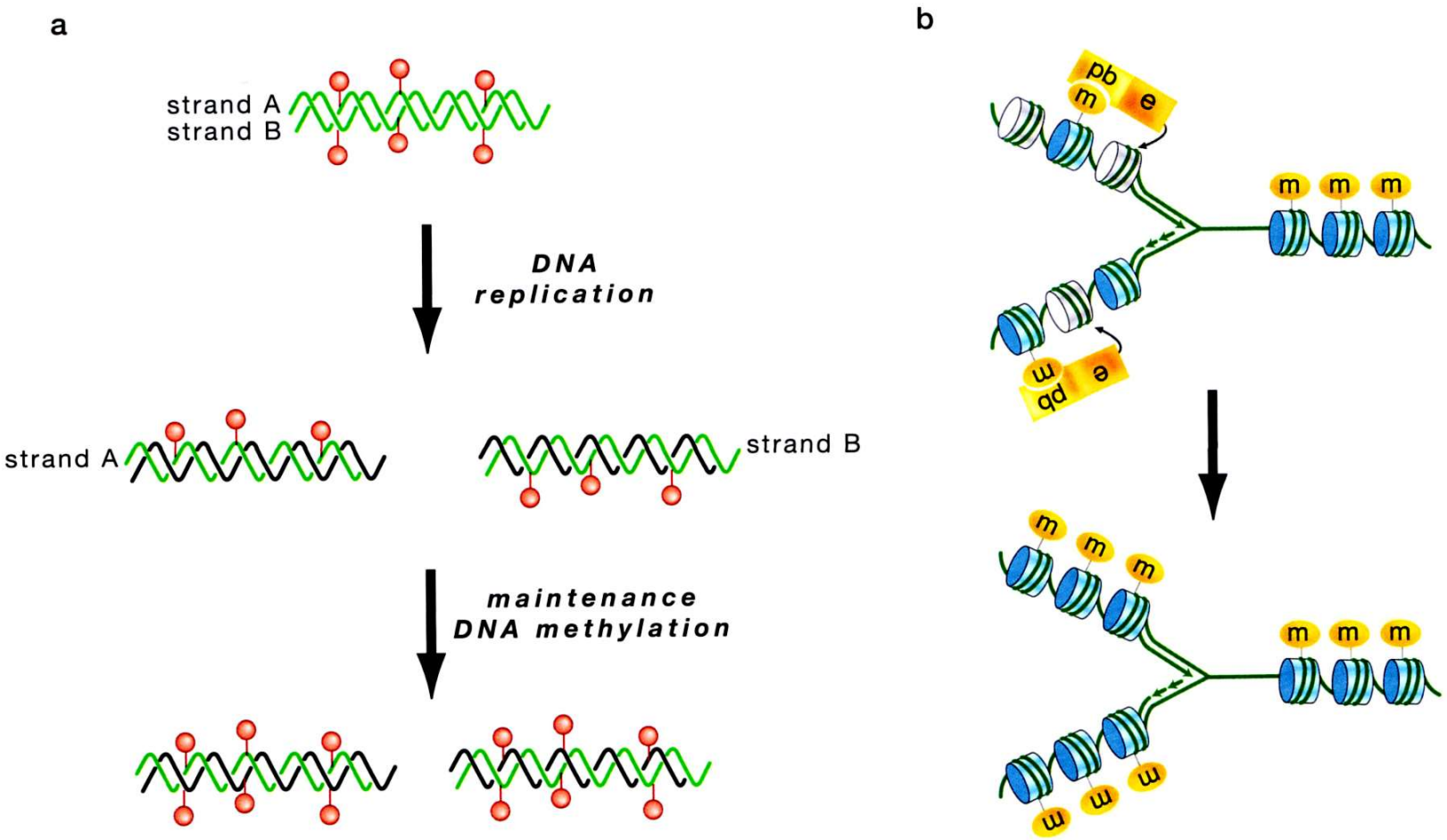


Figure 1. Mechanisms for Maintaining a Pattern of DNA Methylation and a Histone Modification during DNA Replication

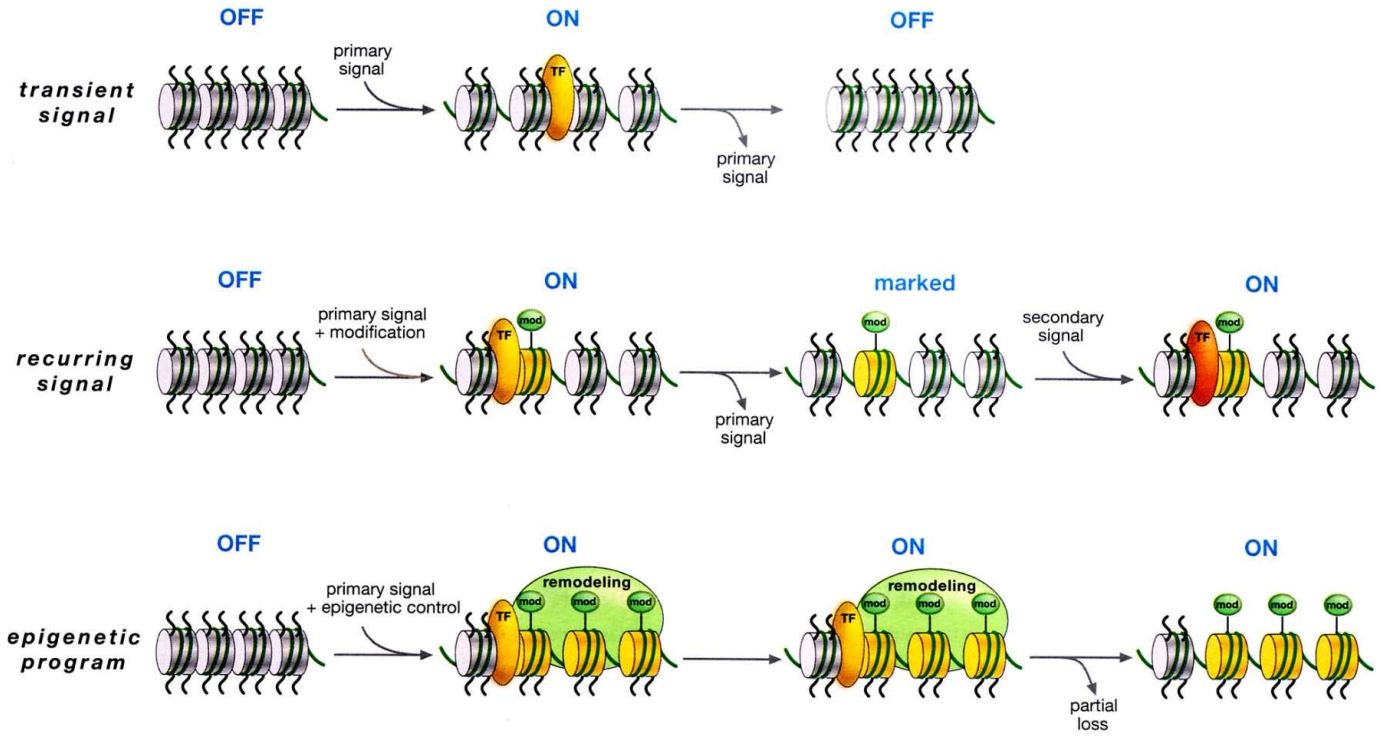
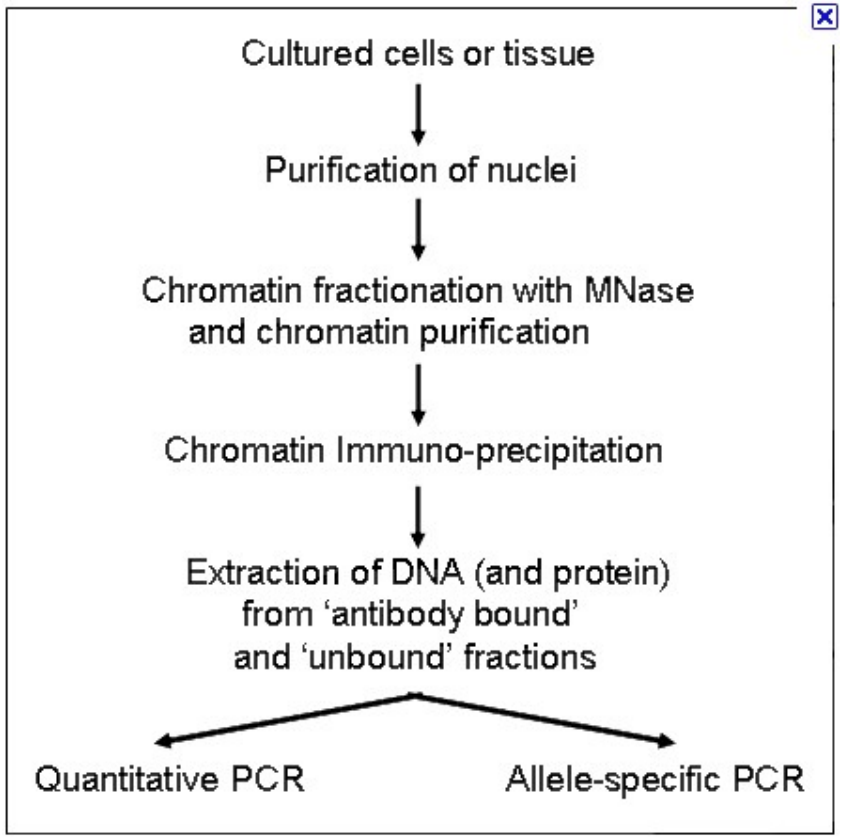
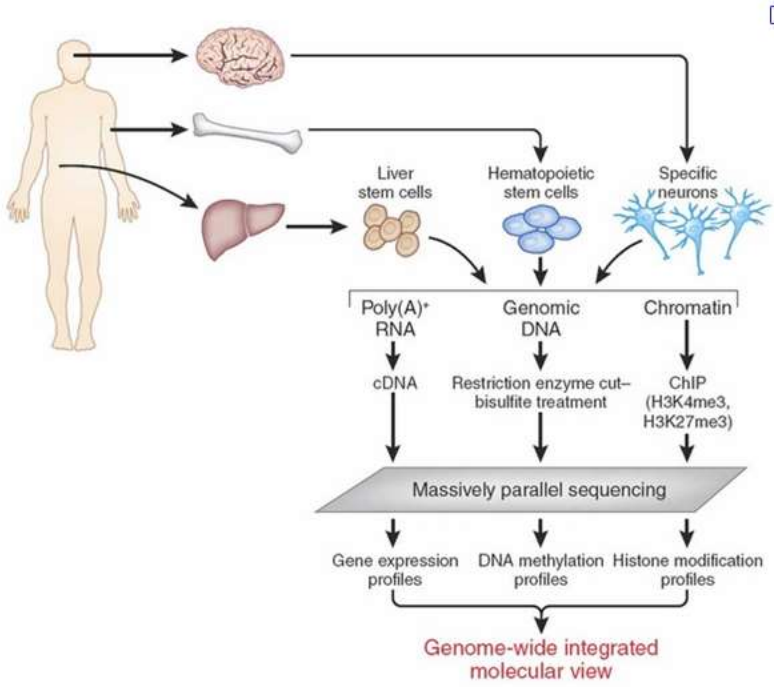


Figure 20. Epigenetic Potentiation of a Primary Signal (Memory/Inheritance)

Classic genetics predicts that gene expression is dependent on the availability and binding of the appropriate panel of transcription factors (TF). Removal of such factors (i.e., a primary signal) results in the loss of gene expression, and thus constitutes a transient activating signal (*top*). Chromatin structure contributes to gene expression, where some conformations are repressive and others active. The activation of a locus may therefore occur through a primary signal and result in the downstream change in chromatin structure, involving active covalent histone marks (mod) and the replacement of core histones with variants (e.g., H3.3). Through cell division, this chromatin structure may only be reestablished in the presence of an activating signal (denoted "recurring signal"). Epigenetic memory results in the maintenance of a chromatin state through cell division, even in the absence of the primary activating signal. Such a memory system is not absolute, but involves multiple levels of epigenetic regulation for remodeling chromatin structure. The dynamic nature of chromatin means that although a chromatin state may be mitotically stable, it is nonetheless prone to change, hence affecting the longevity of epigenetic memory.

Com se “seqüenciem” les marques epigenètiques a les histones?





Els jugadors de l'epigenoma

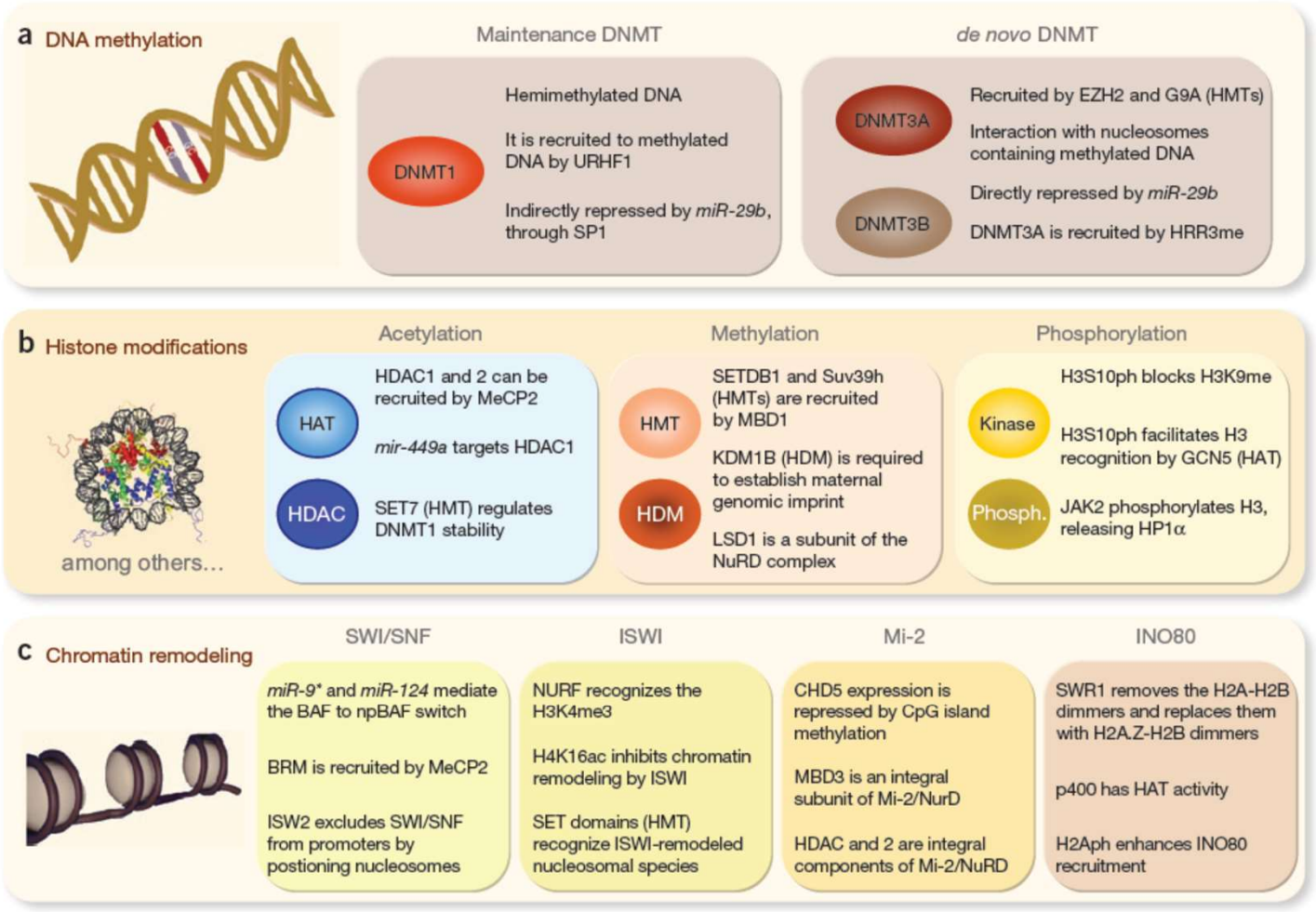


Figure 2 Epigenetic machinery and interplay among epigenetic factors. Epigenetic marks are catalyzed by different epigenetic complexes, whose principal families are illustrated here. (**a–c**) Epigenetic regulation depends on the interplay among the different players: DNA methylation (**a**), histone marks (**b**) and nucleosome positioning (**c**). The interaction among the different factors brings about the final outcome. This figure illustrates selected examples of the possible interrelations among the various epigenetic players.

Efectes de les marques epigenètiques
EUCROMATIONA I HETEROCROMATINA

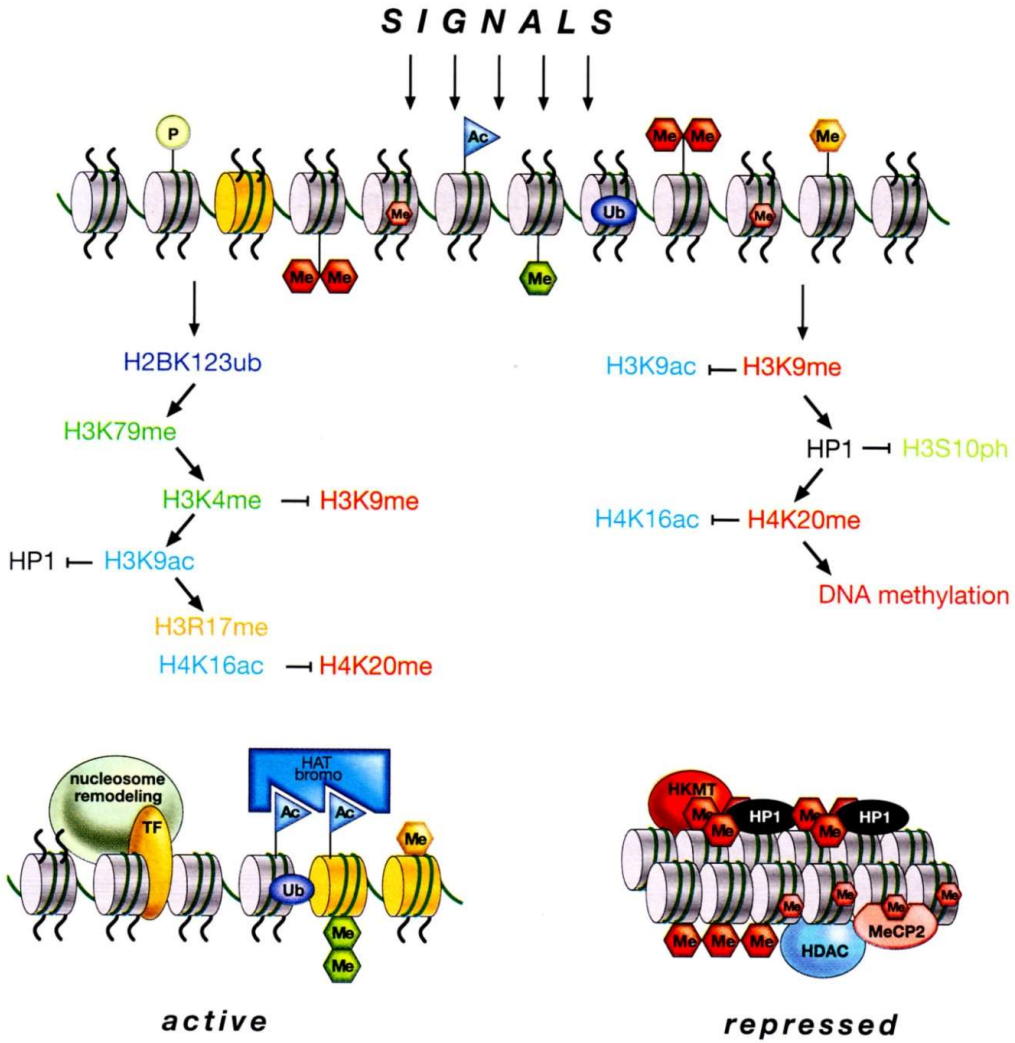
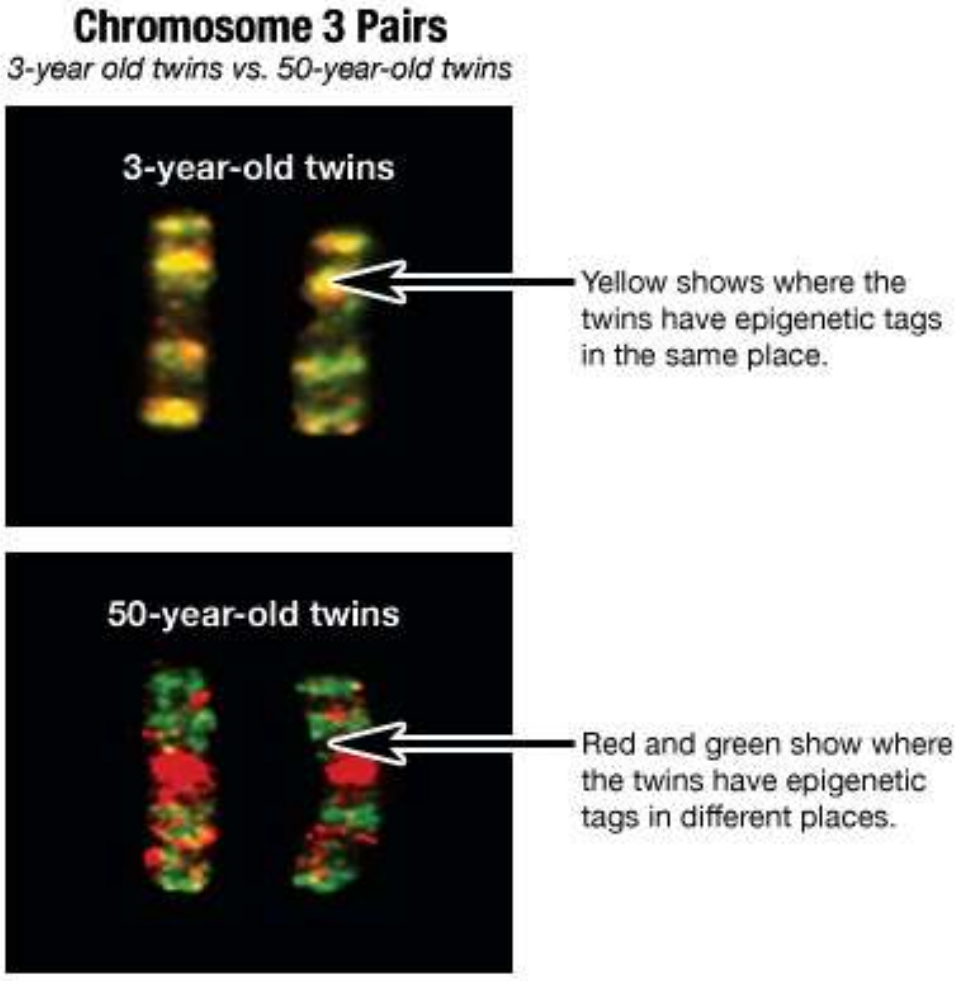


Figure 11. Coordinated Modification of Chromatin

The transition of a naïve chromatin template to active euchromatin (*left*) or the establishment of repressive heterochromatin (*right*), involving a series of coordinated chromatin modifications. In the case of transcriptional activation, this is accompanied by the action of nucleosome-remodeling complexes and the replacement of core histones with histone variants (*yellow*, namely H3.3).

Efectes de les marques epigenètiques
BESSONS IDÈNTICS



Efectes de les marques epigenètiques
IMPROMPTA GENÈTICA

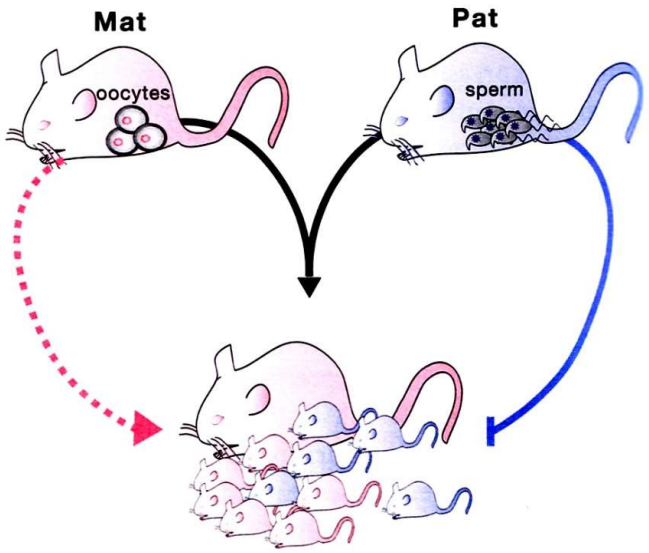
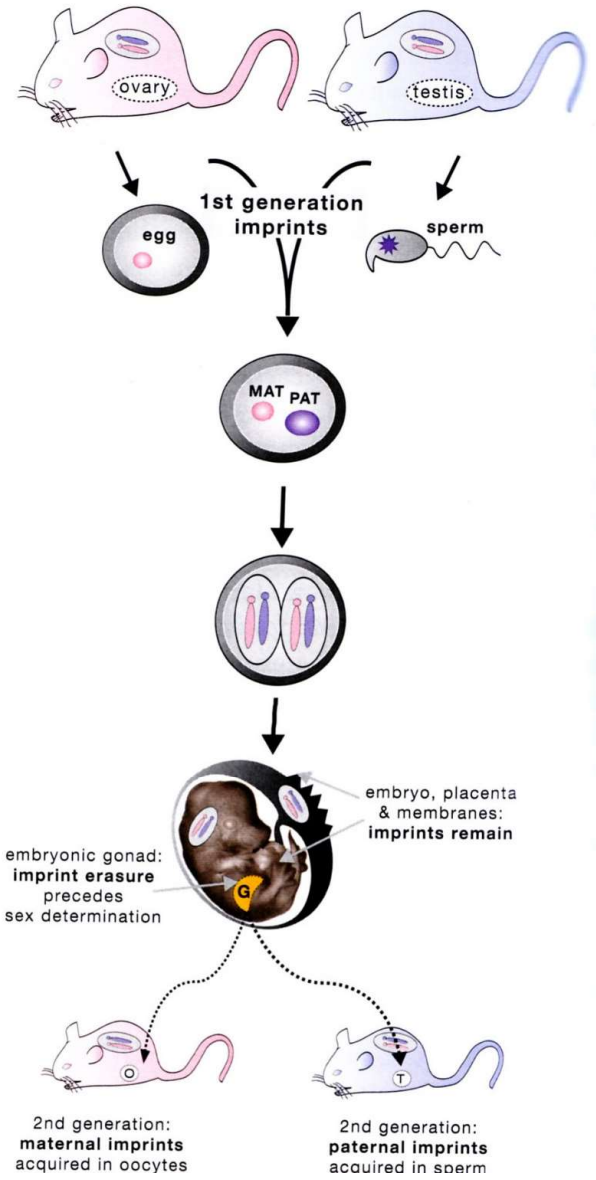


Figure 4. Imprinted Genes Play a Role in Mammalian Reproduction

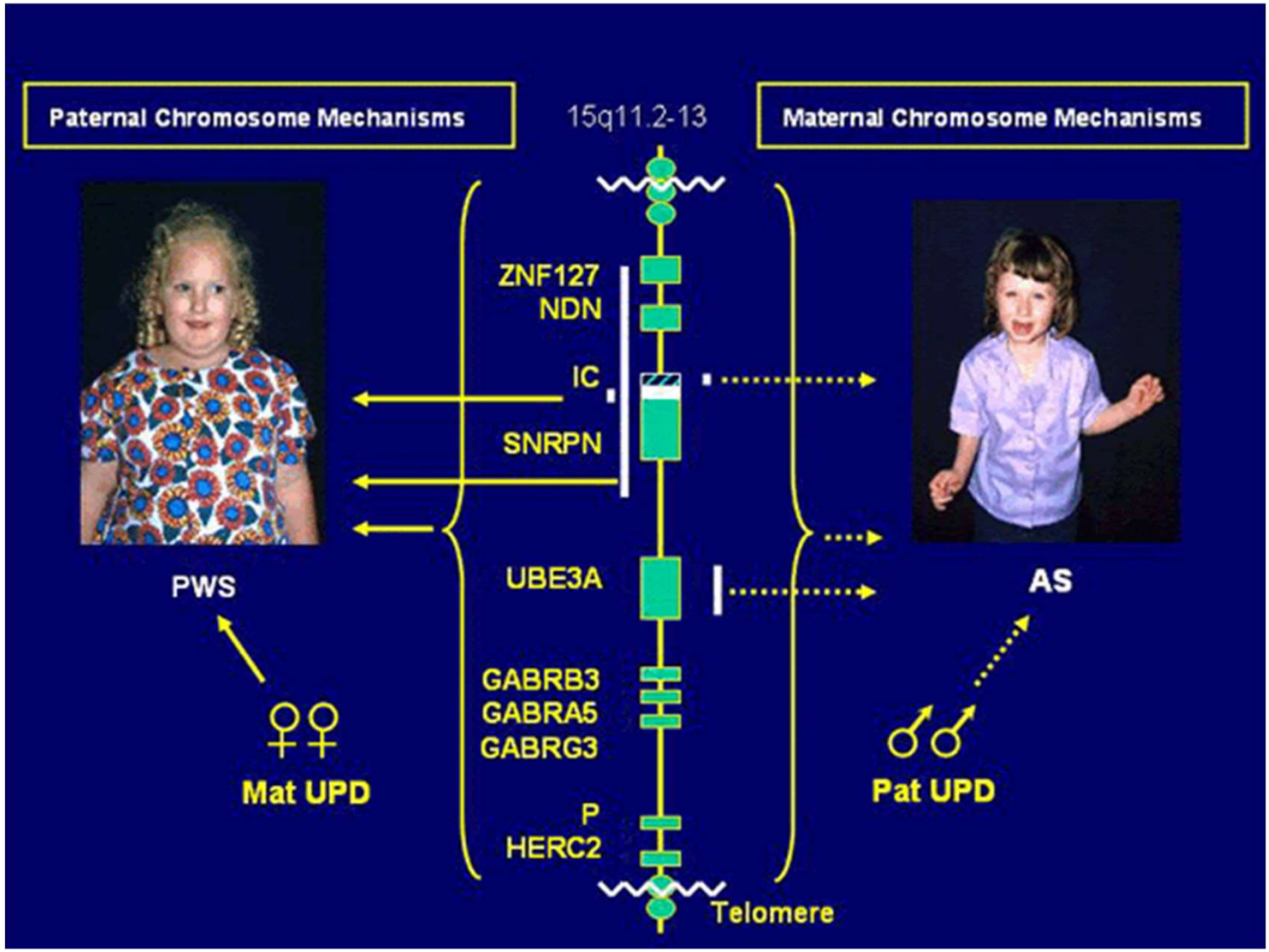
Mammals are diploid, and reproduction requires fertilization of a haploid female egg by a haploid male sperm to recreate a diploid embryo. Only females are anatomically equipped for reproduction, but they cannot use parthenogenesis to reproduce because essential imprinted genes needed for fetal growth are imprinted and silenced on maternal chromosomes. These genes are expressed only from paternal chromosomes; thus, both parental genomes are needed for reproduction in mammals. Parthenogenesis is the production of diploid offspring from two copies of the same maternal genome.



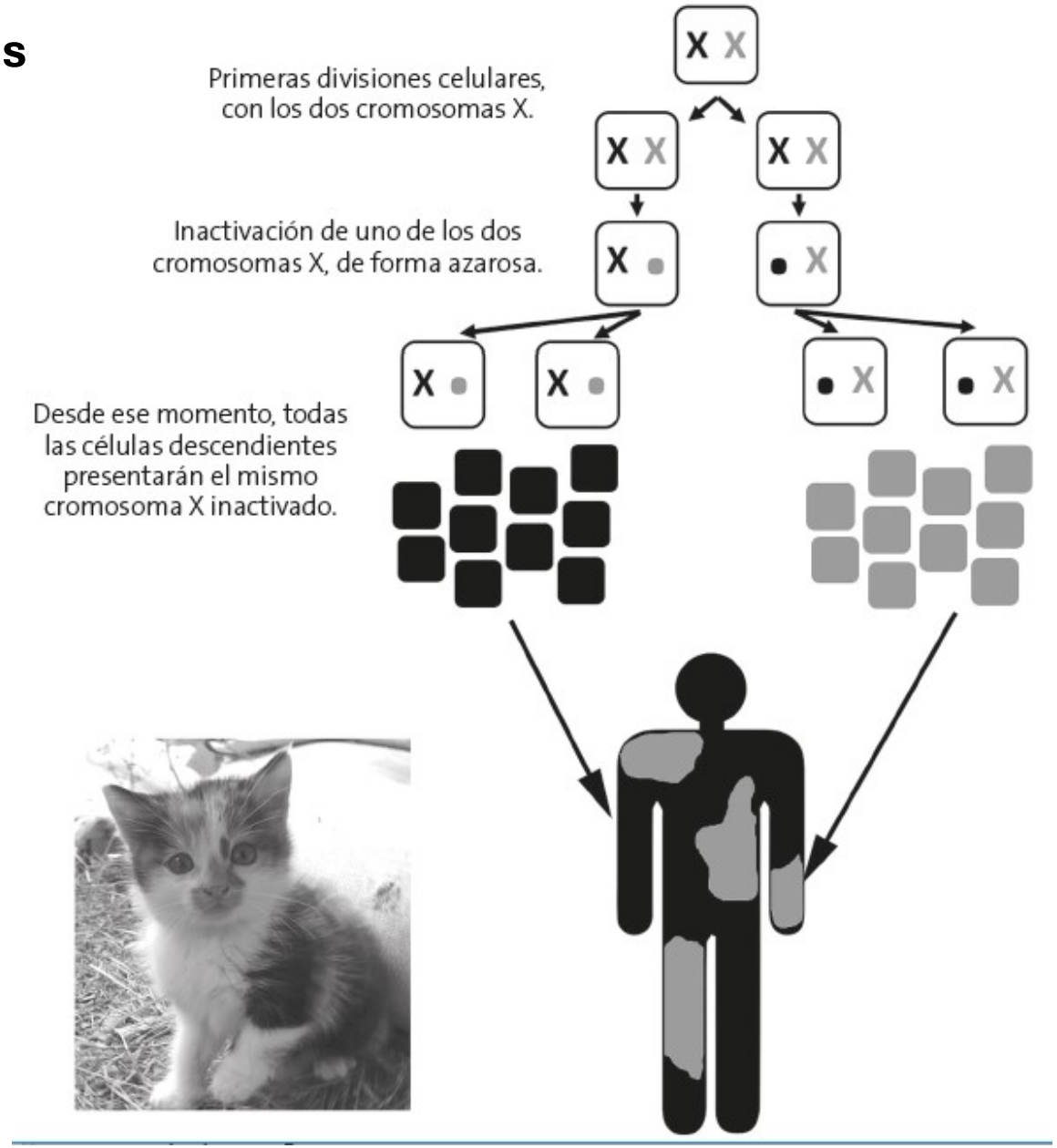
p362

Efectes de les marques epigenètiques
IMPROMPTA GENÈTICA

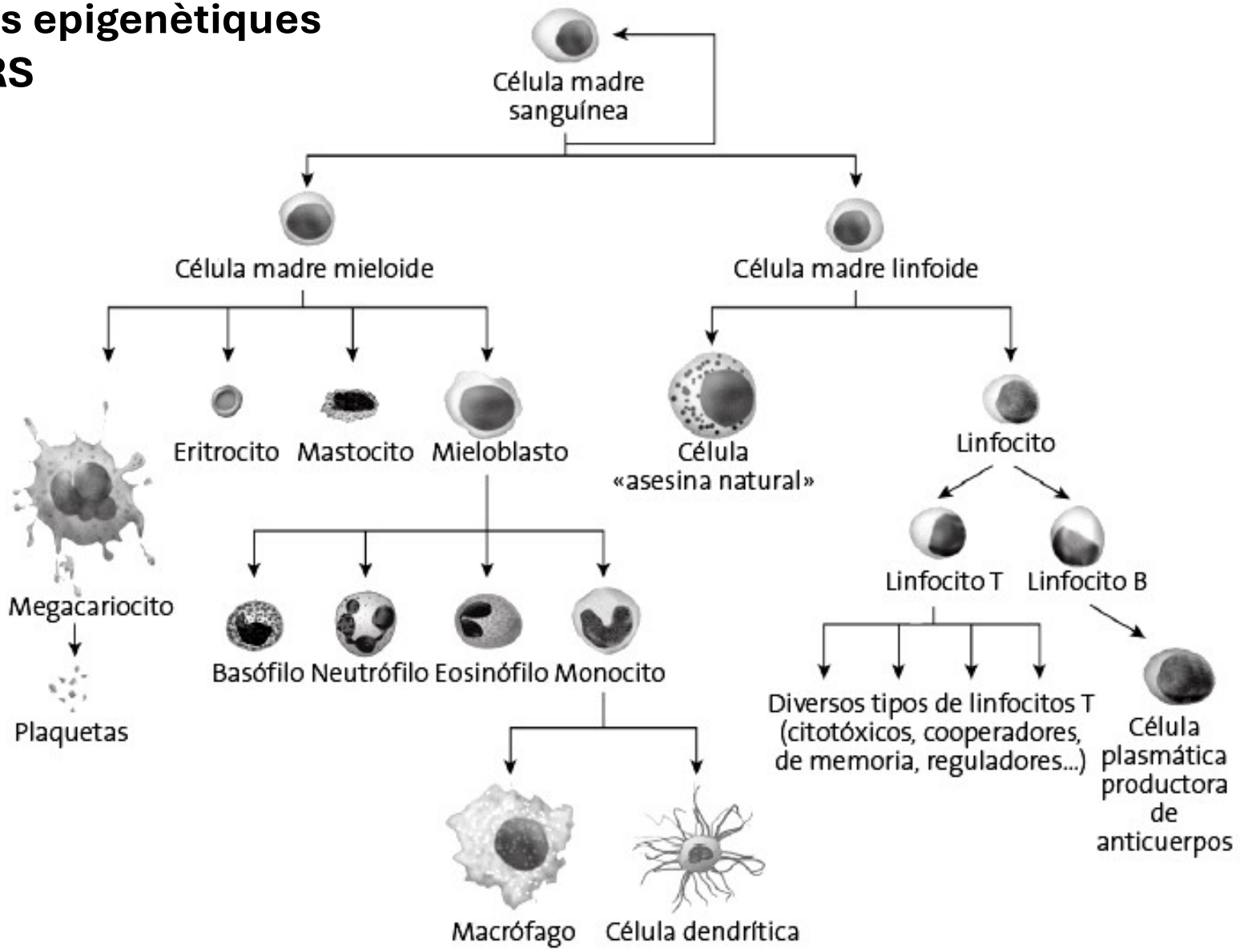
Síndrome de Prader-Willi
Vs
Síndrome d'Angelman



Efectes de les marques epigenètiques
INACTIVACIÓ ALEATÒRIA
DURANT EL DESENVOLUPAMENT



Efectes de les marques epigenètiques
LLINATGES CEL·LULARS



Efectes de les marques epigenètiques
MALALTIES EPIGENÈTIQUES

Table 1 Epigenetic modifications in human diseases

Aberrant epigenetic mark	Alteration	Consequences	Examples of genes affected and/or resulting disease
Cancer			
DNA methylation	CpG island hypermethylation	Transcription repression	*MLH1* (colon, endometrium, stomach[11]), *BRCA1* (breast, ovary[11]), *MGMT* (several tumor types[11]), *p16^{INK4a}* (colon[11])
	CpG island hypomethylation	Transcription activation	*MASPIN* (pancreas[92]), *S100P* (pancreas[92]), *SNCG* (breast and ovary[92]), *MAGE* (melanomas[92])
	CpG island shore hypermethylation	Transcription repression	*HOXA2* (colon[20]), *GATA2* (colon[20])
	Repetitive sequences hypomethylation	Transposition, recombination genomic instability	*L1* (ref. 11), *IAP*[11], *Sat2* (ref. 107)
Histone modification	Loss of H3 and H4 acetylation	Transcription repression	*p21^{WAF1}* (also known as *CDKN1A*)[11]
	Loss of H3K4me3	Transcription repression	*HOX* genes
	Loss of H4K20me3	Loss of heterochromatic structure	*Sat2*, *D4Z4* (ref. 107)
	Gain of H3K9me and H3K27me3	Transcription repression	*CDKN2A, RASSF1* (refs. 115–116)
Nucleosome positioning	Silencing and/or mutation of remodeler subunits	Diverse, leading to oncogenic transformation	*BRG1, CHD5* (refs. 127–131)
	Aberrant recruitment of remodelers	Transcription repression	*PLM-RARa*[103] recruits NuRD
	Histone variants replacement	Diverse (promotion cell cycle/destabilization of chromosomal boundaries)	H2A.Z overexpression/loss
Neurological disorders			
DNA methylation	CpG island hypermethylation	Transcription repression	Alzheimer's disease (*NEP*)[135]
	CpG island hypomethylation	Transcription activation	Multiple sclerosis (*PADI2*)[135]
	Repetitive sequences aberrant methylation	Transposition, recombination genomic instability	ATRX syndrome (subtelomeric repeats)[135,143]
Histone modification	Aberrant acetylation	Diverse	Parkinson's and Huntington's diseases[135]
	Aberrant methylation	Diverse	Huntington's disease and Friedreich's ataxia[135]
	Aberrant phosphorylation	Diverse	Alzheimer's disease[135]
Nucleosome positioning	Misposition in trinucleotide repeats	Creation of a 'closed' chromatin domain	Congenital myotonic dystrophy[151]
Autoimmune diseases			
DNA methylation	CpG island hypermethylation	Transcription repression	Rheumatoid arthritis (*DR3*)[154,155]
	CpG island hypomethylation	Transcription activation	SLE (*PRF1*, *CD70*, *CD154*, *AIM2*)[6]
	Repetitive sequences aberrant methylation	Transposition, recombination genomic instability	ICF (*Sat2*, *Sat3*), rheumatoid arthritis (*L1*)[152,155]
Histone modification	Aberrant acetylation	Diverse	SLE (*CD154*, *IL10*, IFN-γ)[6]
	Aberrant methylation	Diverse	Diabetes type 1 (*CLTA4*, *IL6*)[159]
	Aberrant phosphorylation	Diverse	SLE (NF-κB targets)
Nucleosome positioning	SNPs in the 17q12-q21 region	Allele-specific differences in nucleosome distribution	Diabetes type 1 (*CLTA4*, *IL6*)
	Histone variants replacement	Interferes with proper remodeling	Rheumatoid arthritis (histone variant macroH2A at NF-κB targets)[157]

Efectes de les marques epigenètiques
MALALTIES EPIGENÈTIQUES

EJN fens EUROPEAN JOURNAL OF NEUROSCIENCE

European Journal of Neuroscience, Vol. 30, pp. 1–8, 2009 doi:10.1111/j.1460-9568.2009.06787.x

REVIEW ARTICLE

Epigenetic dysregulation in cognitive disorders

Johannes Gräff and Isabelle M. Mansuy
Brain Research Institute, Medical Faculty of the University of Zürich, and Department of Biology, Swiss Federal Institute of Technology, Winterthurerstrasse 190, Zürich, Switzerland

TABLE 1. Epigenetic mechanisms in neurodegenerative disorders and potential epigenetic treatment

Disorder	Epigenetic alteration	Gene involved	Model organism	Epigenetic-related treatment	References
Alzheimer's disease	Histone acetylation ↑	*APP*	Human cell culture	None	Cao & Sudhof, 2001
		PS1	Human cell/murine neuronal culture	Substitution of PS1-mediated enzymatic activity	Marambaud *et al.*, 2003
		None specific	*p25*/Cdk5 mouse model	HDAC SIRT1	Kim *et al.*, 2007
	Histone acetylation ↓	*APP*	Murine neuronal culture	None	Rouaux *et al.*, 2003
		PS1	*PS1* mouse model	None	Saura *et al.*, 2004
		None specific	*p25*/Cdk5 mouse model	HDACi sodium butyrate	Fischer *et al.*, 2007
	DNA methylation ↓	*APP*	Aged monkey	None	Wu *et al.*, 2008
		PS1	Human cell culture	Methyl-donor SAM	Scarpa *et al.*, 2003
			Human *post mortem* tissue	None	Wang *et al.*, 2008
Huntington's disease	Histone acetylation ↓	*Htt*	Human cell culture/ *Drosophila* model	HDACi SAHA and sodium butyrate	Steffan *et al.*, 2001
		Htt	Human cell culture	None	Sugars *et al.*, 2004
		Htt	R6/2 and 82Q mouse models	HDACi sodium butyrate, SAHA, phenylbutyrate, anthracycline	Ferrante *et al.*, 2003; Hockly *et al.*, 2003; Gardian *et al.*, 2005; Stack *et al.*, 2007
	Histone methylation H3K9 ↑	*Htt*	R6/2 and 82Q mouse models	Anthracycline	Stack *et al.*, 2007

APP, amyloid precursor protein; HDAC, histone deacetylase; HDACi, histone deacetylase inhibitor; Htt, huntingtin; PS1, presenilin 1; SAHA, suberoylanilide hydroxamic acid; SAM, S-adenosylmethionine; SIRT1, silent information regulator homologue 1.

Efectes de les marques epigenètiques
MALALTIES EPIGENÈTIQUES

TABLE 2. Epigenetic mechanisms in neurodevelopmental disorders and potential epigenetic treatment

Disorder, epigenetic alteration and gene involved	Model organism	Epigenetic-related treatment	References
Rett syndrome			
DNA methylation ↓			
MeCP2	Human patients	None	Amir *et al.*, 1999; Chen *et al.*, 2001; Guy *et al.*, 2001
MeCP2	*MeCP2* mouse models	None	Chen *et al.*, 2001; Guy *et al.*, 2001; Shahbazian *et al.*, 2002
DNA methylation ↓/Histone acetylation ↑			
MeCP2	*MeCP2* mouse models	None	Shahbazian *et al.*, 2002
DNA methylation ↓/Histone acetylation ↑/Histone methylation H3K9 ↓/H3K4 ↑			
MeCP2	Murine cell culture	None	Martinowich *et al.*, 2003
Rubinstein–Taybi syndrome			
Histone acetylation ↓			
CBP	*CBP* mouse models	HDACi SAHA and TSA	Alarcon *et al.*, 2004; Korzus *et al.*, 2004; Roelfsema *et al.*, 2005
EP300	Human patients	None	Roelfsema *et al.*, 2005; Bartholdi *et al.*, 2007
Fragile X syndrome			
DNA methylation ↑/Histone acetylation ↓			
FMR1	Human patient-derived cell lines	5-aza, HDACi sodium butyrate, SAHA and TSA	Chiurazzi *et al.*, 1998, 1999
DNA methylation ↑/Histone acetylation ↓/Histone methylation H3K9 ↑/H3K4 ↓			
FMR1	Human patient-derived cell lines	None	Tabolacci *et al.*, 2005, 2008
Schizophrenia			
DNA methylation ↑			
RELN	Human patients; reeler mouse model; cell culture	5-aza, HDACi sodium butyrate, and valproic acid	Chen *et al.*, 2002; Costa *et al.*, 2002
Histone methylation H3K27 ↑/H3K4 ↓			
GAD1	Human patients; neural stem cells	Antipsychotic clozapine	Huang & Akbarian, 2007; Huang *et al.*, 2007

CBP, Creb-binding protein; EP300, E1A binding protein p300; FMR, fragile X mental retardation; H, histone; HAT, histone acetyltransferase; HDACi, histone deacetylase inhibitor; K, lysine; MeCP2, methyl-CpG binding protein 2; SAHA, suberoylanilide hydroxamic acid; TSA, trichostatin A; 5-aza, 5-aza-2-deoxycytidine.

L'ambient també influeix la formació de marques epigenètiques

ELSEVIER

Progress in Molecular Biology and Translational Science

Volume 108, 2012, Pages 427-446

Nutrition and the Epigenome

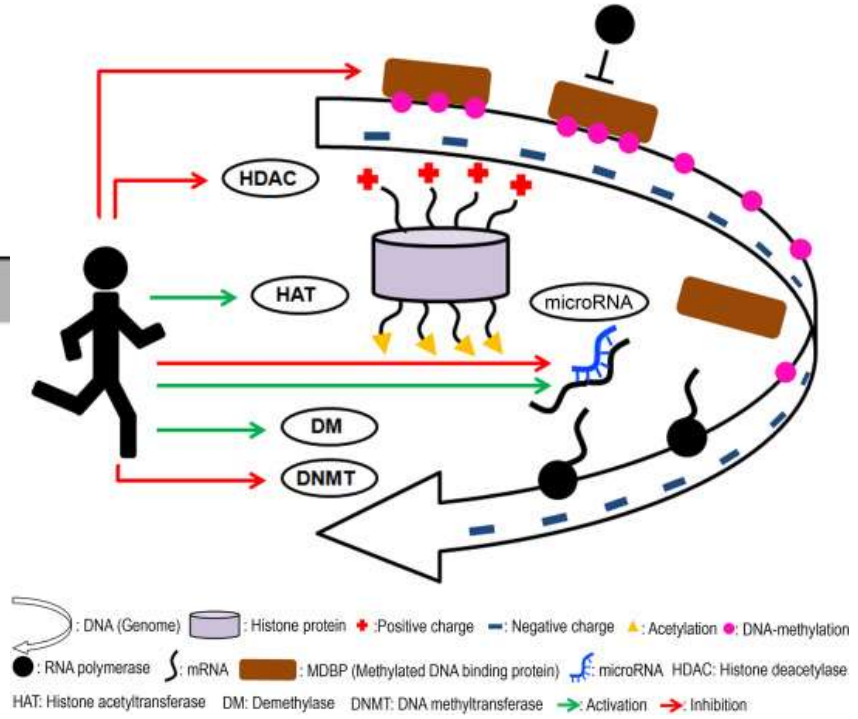
Paul Haggarty

Sports Med (2013) 43:93–110
DOI 10.1007/s40279-012-0012-y

REVIEW ARTICLE

Epigenetics in Sports

Tobias Ehlert · Perikles Simon · Dirk A. Moser



L'ambient també influeix la formació de marques epigenètiques

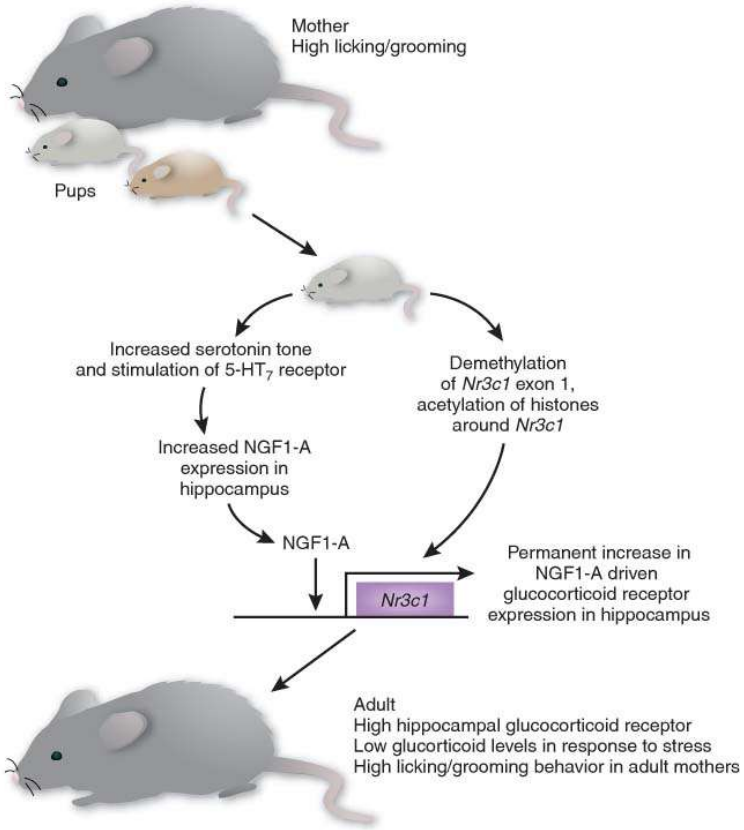
Dev Psychopathol. Author manuscript; available in PMC 2013 Nov 1.
Published in final edited form as:
Dev Psychopathol. 2012 Nov; 24(4): 1377–1390.
doi: 10.1017/S0954579412000776

PMCID: PMC3581096
NIHMSID: NIHMS440117
PMID: 23062304

The Epigenetics of Maternal Cigarette Smoking During Pregnancy and Effects on Child Development

Valerie S. Knopik,[1,2,*] Matthew A. Maccani,[1,3,*] Sarah Francazio,[1,4] and John E. McGeary[5,1,2]

L'ambient també influeix la formació de marques epigenètiques



Psychoneuroendocrinology (2007) 32, 437–450

Available at www.sciencedirect.com

ScienceDirect

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ELSEVIER

Opposite effects of maternal separation on intermale and maternal aggression in C57BL/6 mice: Link to hypothalamic vasopressin and oxytocin immunoreactivity

Alexa H. Veenema*, Remco Bredewold, Inga D. Neumann

- Nounats de rata deprivats d'atenció materna, presenten una metilació al gen del receptor de glucocorticoids que provoca una major agressivitat entre mascles, i una manor atenció comunitària a les cries

- Semblant en el gen BDNF en mares estressades.

L'ambient també influeix la formació de marques epigenètiques

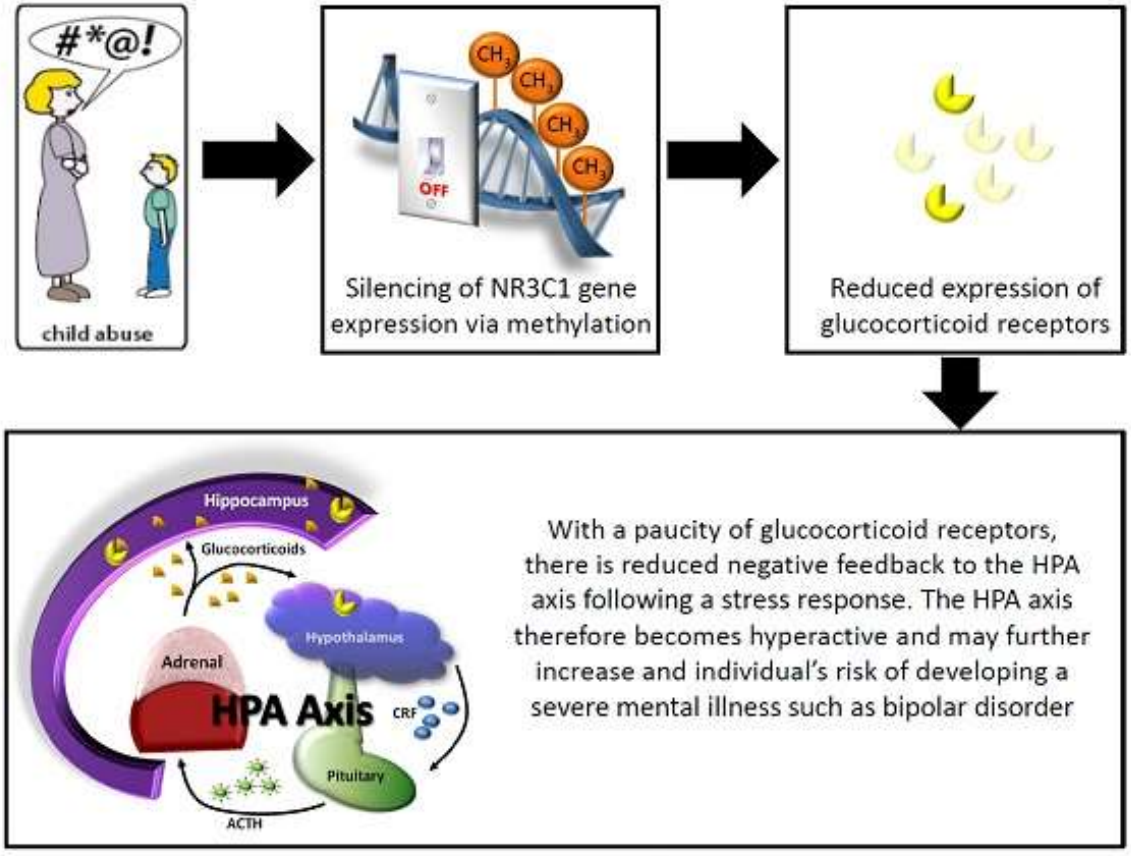
ARTICLES

nature neuroscience

Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse

Patrick O McGowan[1,2], Aya Sasaki[1,2], Ana C D'Alessio[3], Sergiy Dymov[3], Benoit Labonté[1,4], Moshe Szyf[2,3], Gustavo Turecki[1,4] & Michael J Meaney[1,2,5]

342 VOLUME 12 | NUMBER 3 | MARCH 2009 NATURE NEUROSCIENCE



- En infants que de petits han patit abusos s'observa una metilació epigenètica análoga al gen del receptor de glucocorticoids, que s'ha associat a una taxa elevada de suicidi

L'ambient també influeix la formació de marques epigenètiques

nature neuroscience

Resilience to social stress coincides with functional DNA methylation of the *Crf* gene in adult mice

Evan Elliott, Gili Ezra-Nevo, Limor Regev, Adi Neufeld-Cohen & Alon Chen

NATURE NEUROSCIENCE VOLUME 13 | NUMBER 11 | NOVEMBER 2010

L'ambient també influeix la formació de marques epigenètiques

Received: 26 March 2018 | Revised: 27 July 2018 | Accepted: 13 September 2018

DOI: 10.1002/dev.21789

Check for updates

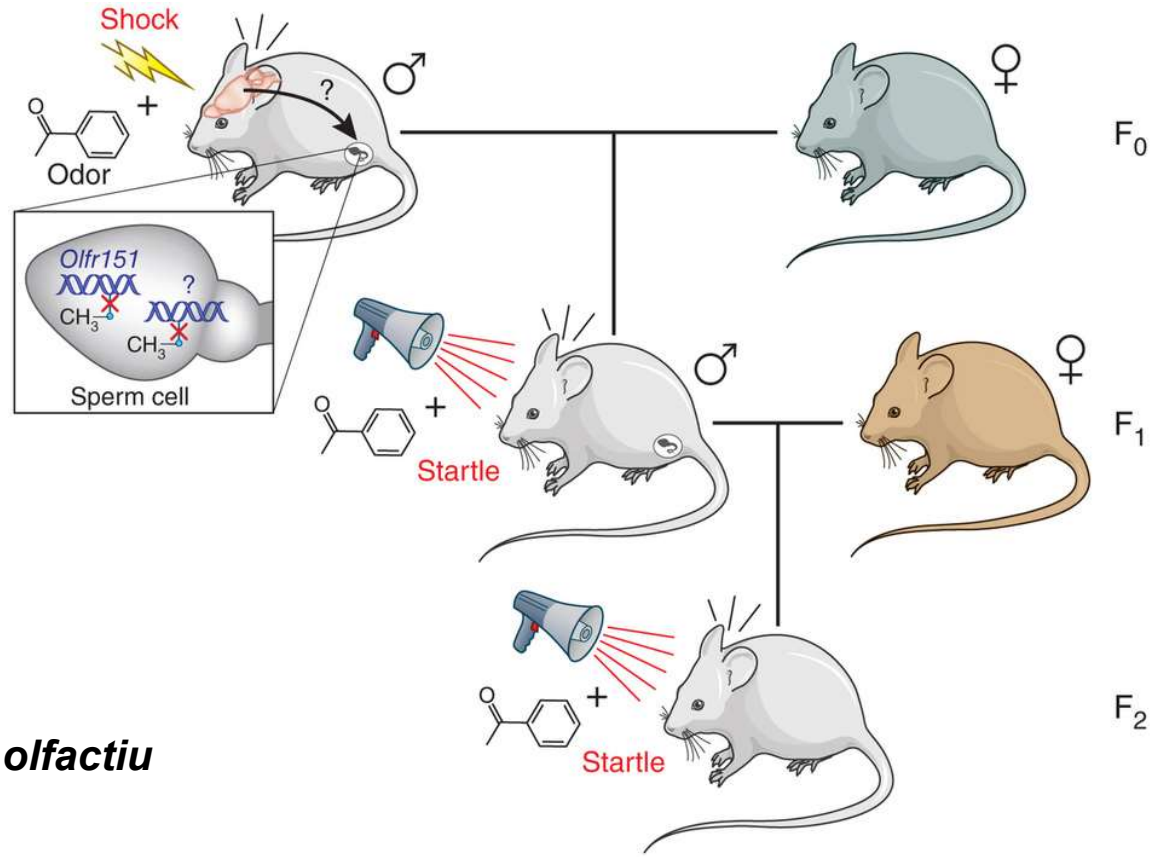
BRIEF REPORT

WILEY Developmental Psychobiology

Negative parenting modulates the association between mother's DNA methylation profiles and adult offspring depression

Sascha Hein[1*] | Tina Thomas[1*] | Oxana Yu. Naumova[1,2] | Suniya S. Luthar[3] | Elena L. Grigorenko[1,4]

Transmissió transgeneracional de marques epigenètiques



Desmetilació del gen d'un receptor olfactiu

A study shows that when mice are taught to fear an odor, both their offspring and the next generation are born fearing it. The gene for an olfactory receptor activated by the odor is specifically demethylated in the germ line and the olfactory circuits for detecting the odor are enhanced.

Transmissió transgeneracional de marques epigenètiques

SCIENCE

The Famine Ended 70 Years Ago, but Dutch Genes Still Bear Scars

Babies born during the Dutch Hunger Winter became adults with higher rates of health problems. Now researchers may have found the genetic switches that made it happen.

Carl Zimmer
MATTER JAN. 31, 2018

A victim of starvation during the Dutch famine of 1944-45. Women pregnant during the period gave birth to babies who were affected by health problems throughout their lives. Hulton Archive/Getty Images

SCIENTIFIC AMERICAN MIND

MENTAL HEALTH

Descendants of Holocaust Survivors Have Altered Stress Hormones

Parents' traumatic experience may hamper their offspring's ability to bounce back from trauma

By Tori Rodriguez on March 1, 2015

Transmissió transgeneracional de marques epigenètiques

Biological Psychiatry
A Journal of Psychiatric Neuroscience and Therapeutics

Epigenetic Effects of Cannabis Exposure

Henrietta Szutorisz, Yasmin L. Hurd

International Journal of
Environmental Research and Public Health

MDPI

Review

Cannabis and Paternal Epigenetic Inheritance

Filomena Mazzeo [1,2] and Rosaria Meccariello [3,4,*]

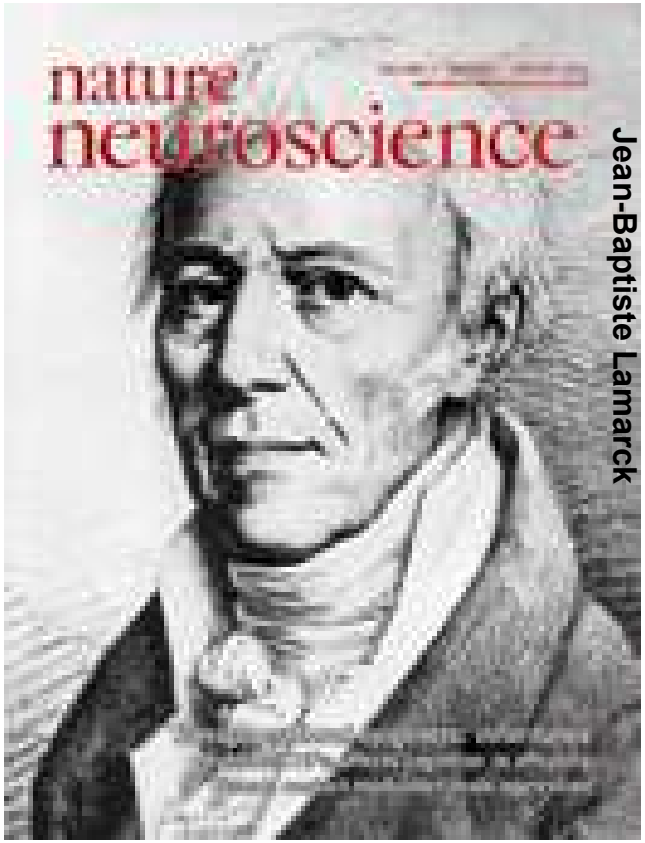
El lamarkisme ha ressucitat?

nature neuroscience

Lamarck revisited: epigenetic inheritance of ancestral odor fear conditioning

Moshe Szyf

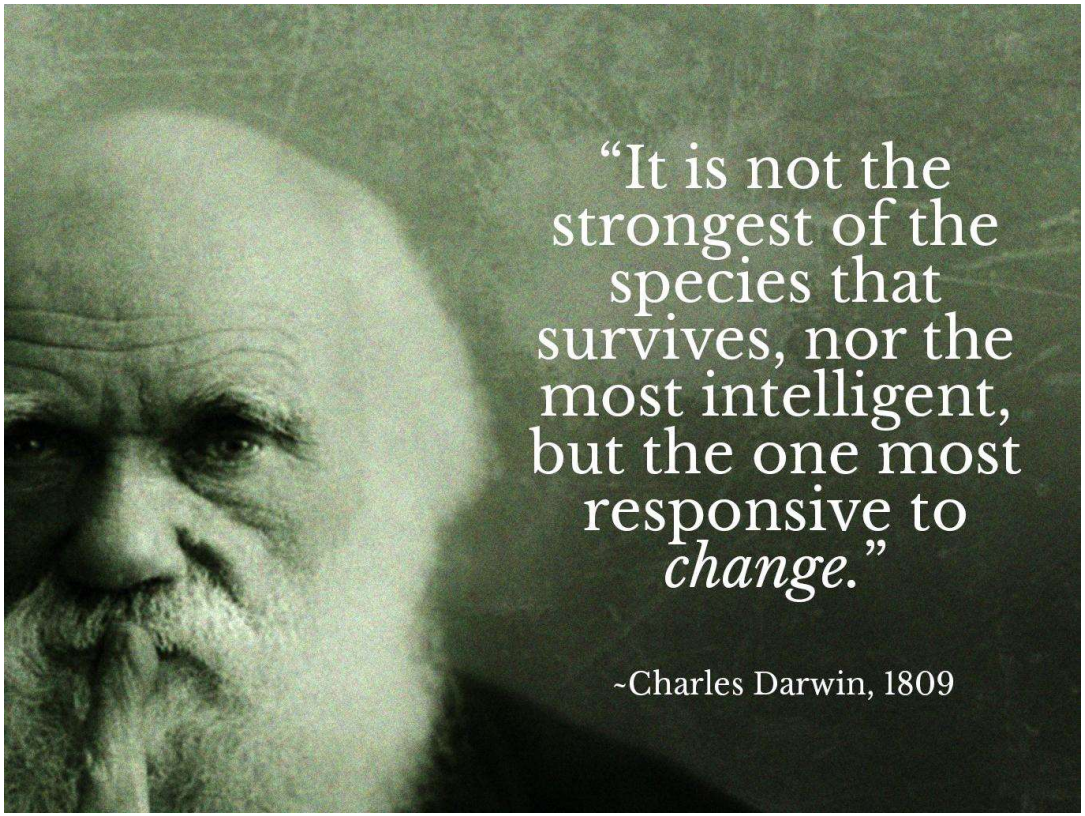
Nature Neuroscience **17**, 2–4 (2014) | doi:10.1038/nn.3603
Published online 26 December 2013

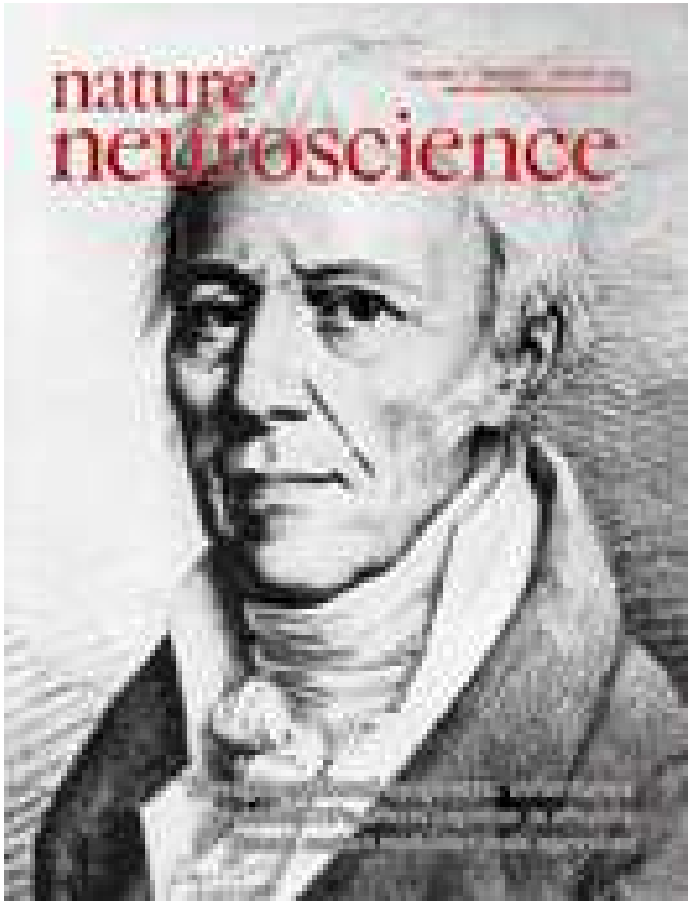


Jean-Baptiste Lamarck

El lamarkisme ha ressucitat?

LA NATURA ÉS COMPLEXA I ADPTABLE





Les preguntes que l'epigenètica encara té plantejades

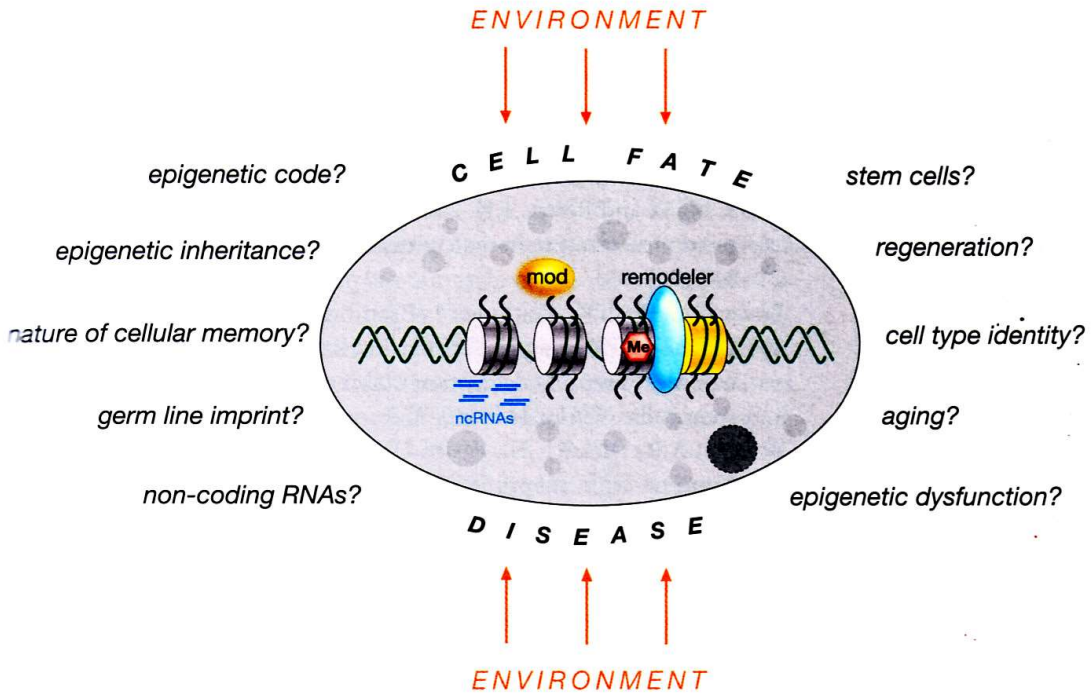
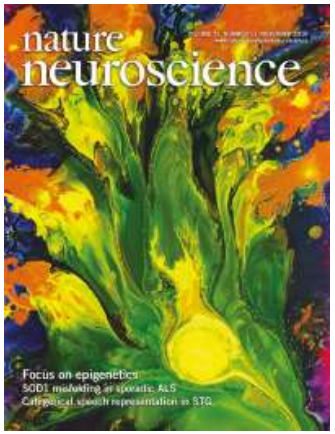
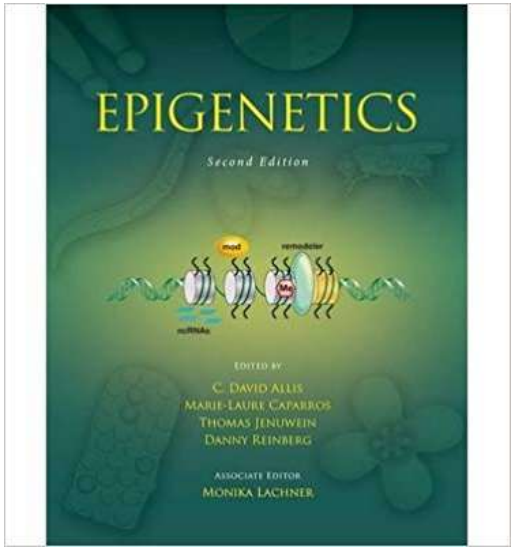


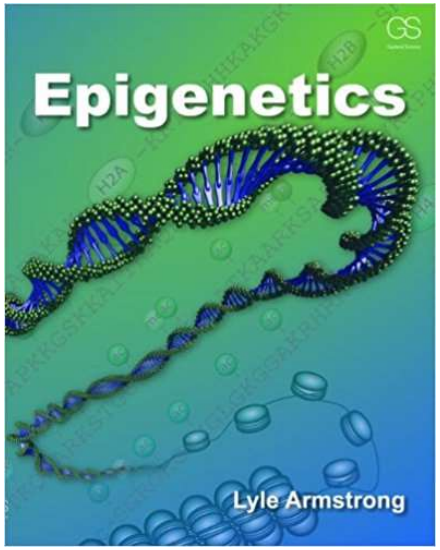
Figure 21. Big Questions in Epigenetic Research

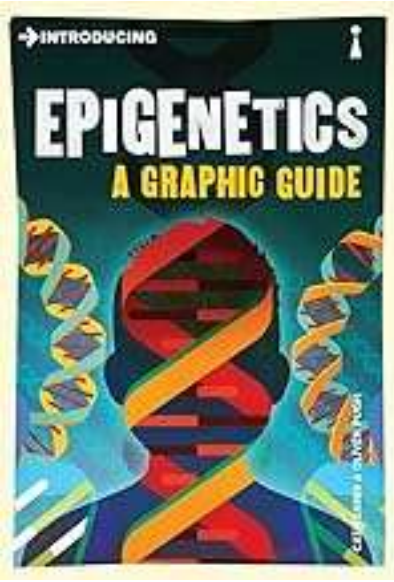
The many experimental systems used in epigenetic research have unveiled numerous pathways and novel insights into the mechanisms of epigenetic control. Many questions, as shown in the figure, still remain and require further elucidation or substantiation in new and existing model systems and methods.

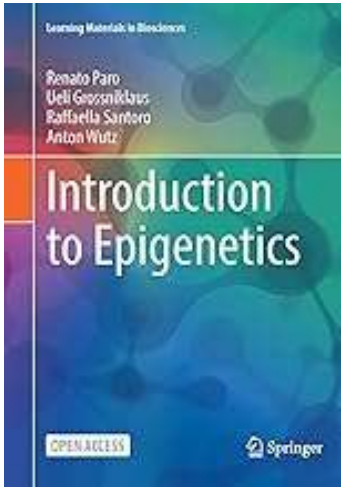
Focus on Epigenetics.
Nature Neurosciences 13(11)
November 2010.

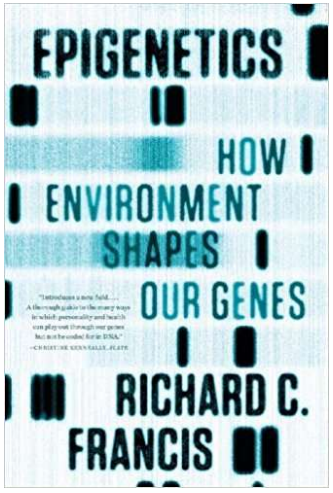


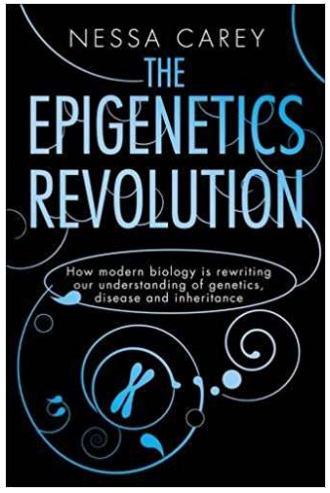


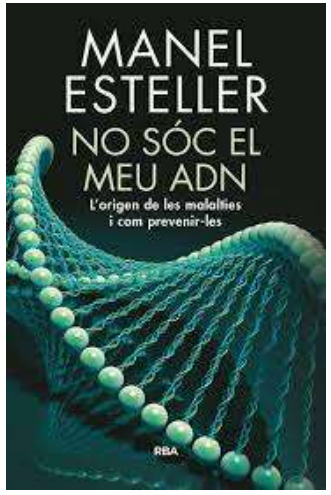


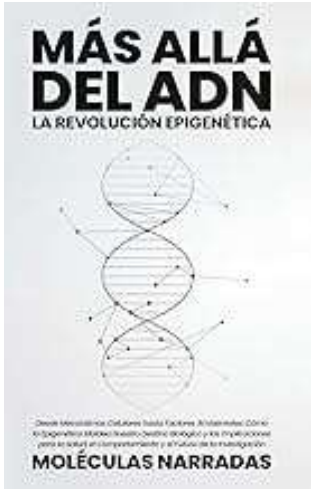


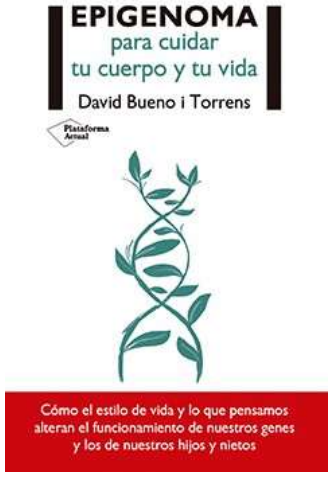






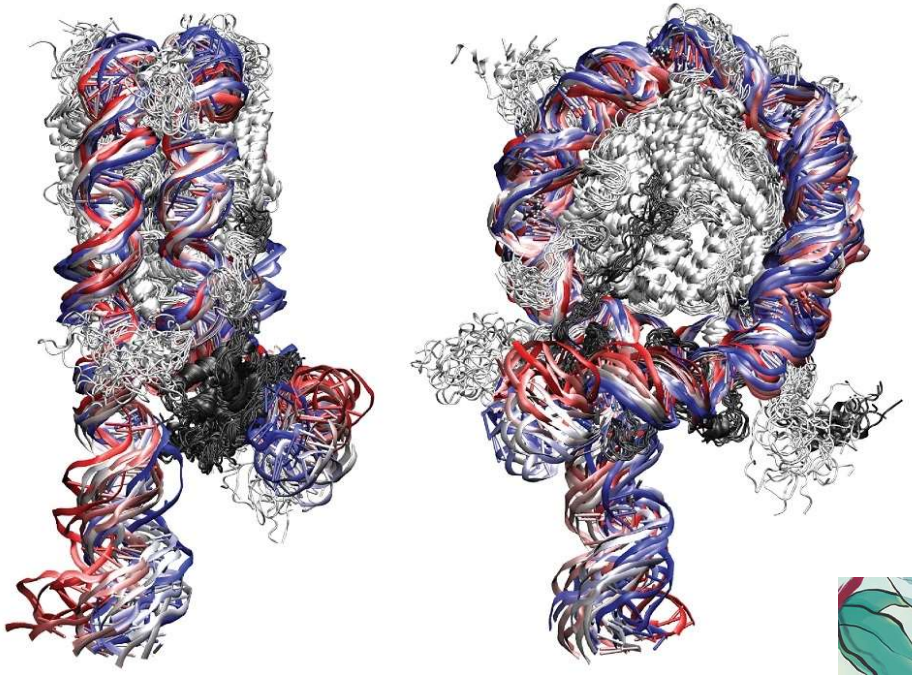






El negoci de l'epigenètica

L’epigenètica en l’art



Les pessigolles alteren l'expressió de 321 gens del nostre genoma: 136 s'expressen amb major intensitat, i 185 la disminueixen.

Contents lists available at ScienceDirect

Brain Behavior and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Social relationships and epigenetic aging in older adulthood: Results from the Health and Retirement Study

Kelly E. Rentscher [a,b,*], Eric T. Klopack [c], Eileen M. Crimmins [c], Teresa E. Seeman [d], Steve W. Cole [b], Judith E. Carroll [b]