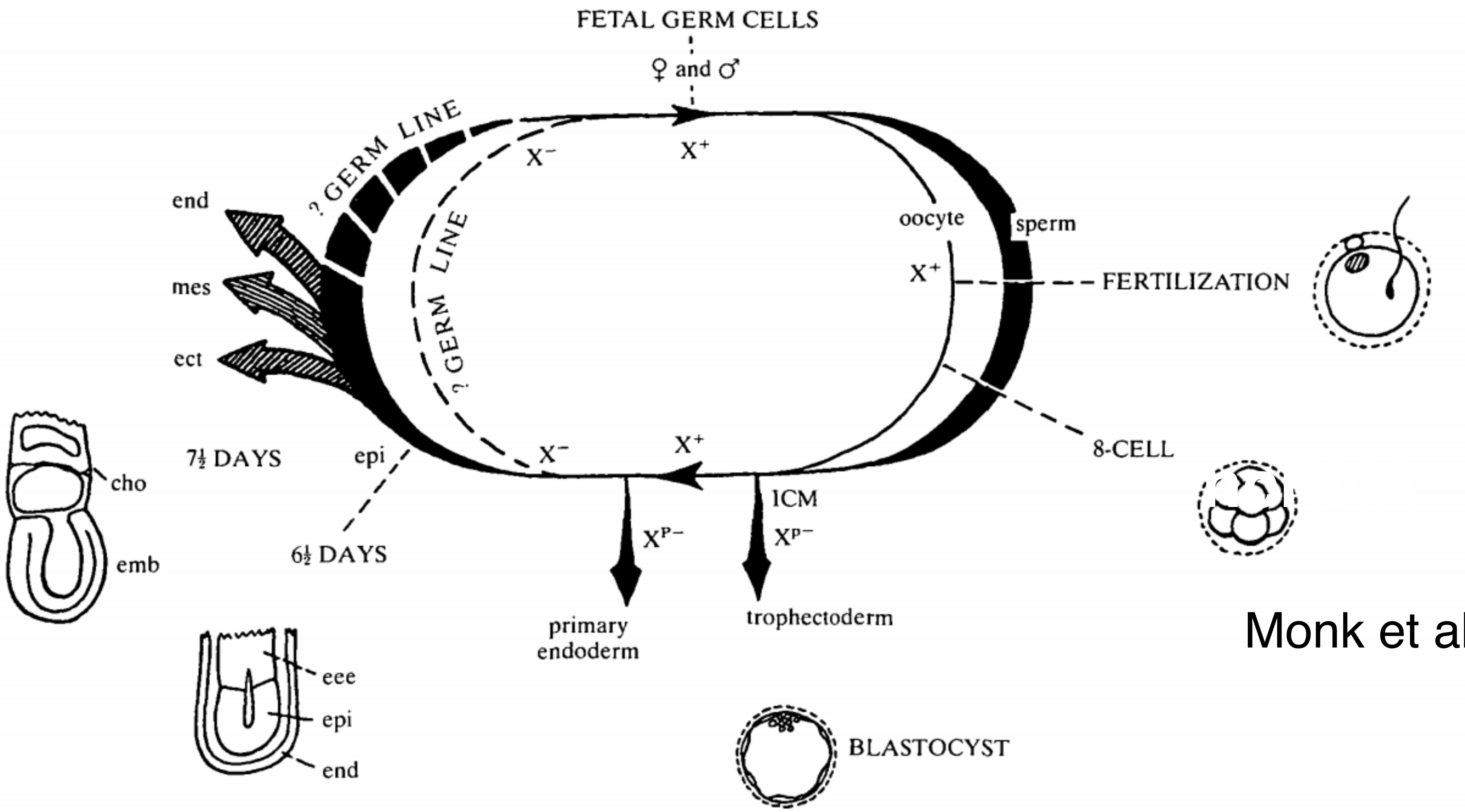
Epigenetic Reprogramming in Mammals

GenE2 – Université Paris-Saclay "Non-coding RNAs and Epigenetics"

October 6, 2025



Monk et al., *Development* 1987

Maxim Greenberg

Group Leader: "Chromatin Dynamics in Mammalian Development"
Institut Jacques Monod, Paris

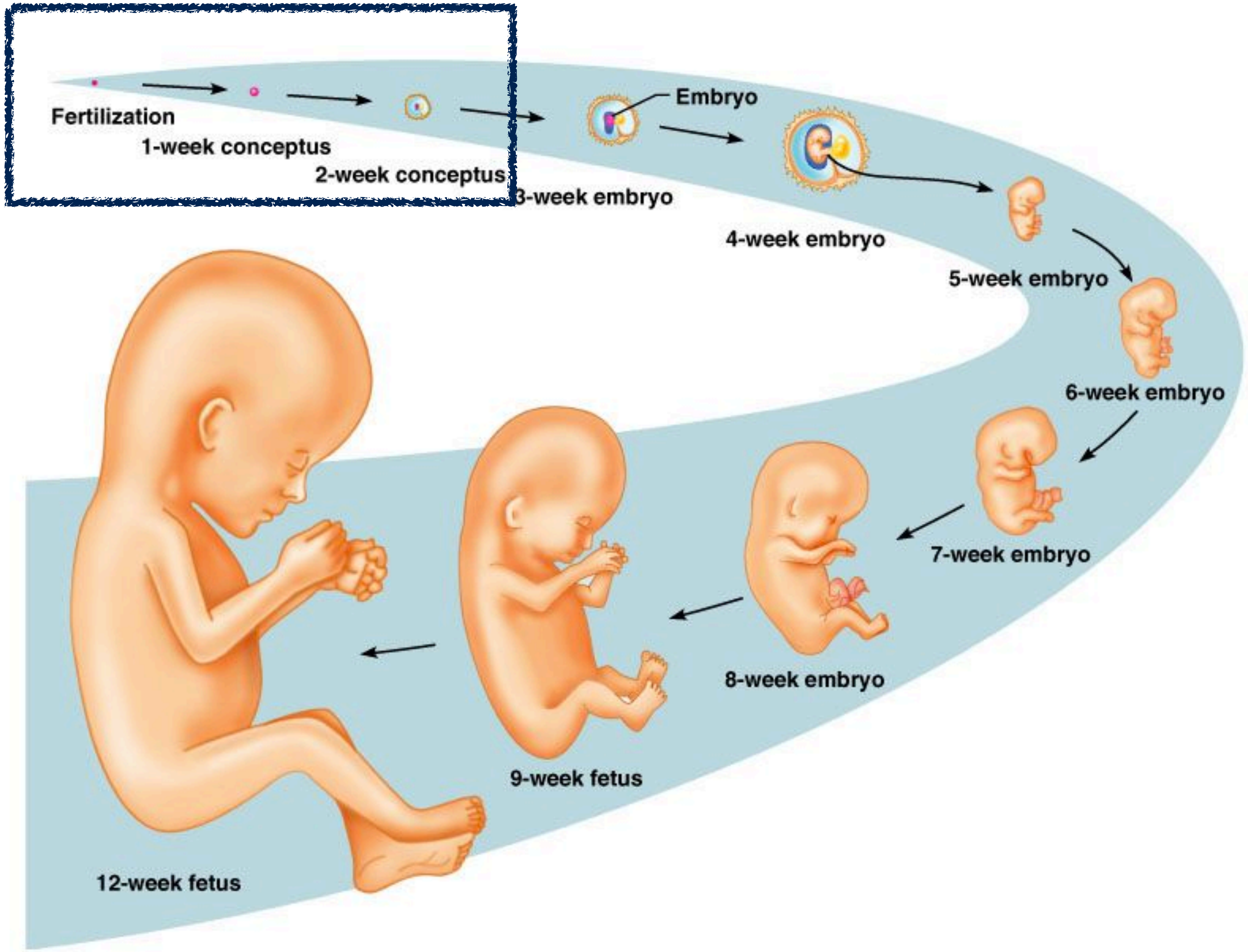
Epigenetics

Definition (for this particular course):

Changes to the chromatin state that can be faithfully inherited across cell division

Epigenetic reprogramming occurs in very early development

Mouse: First week
Human: First two weeks



Questions We Will Address Today

I. What is epigenetic reprogamming?

II. How does epigenetic reprogramming occur?

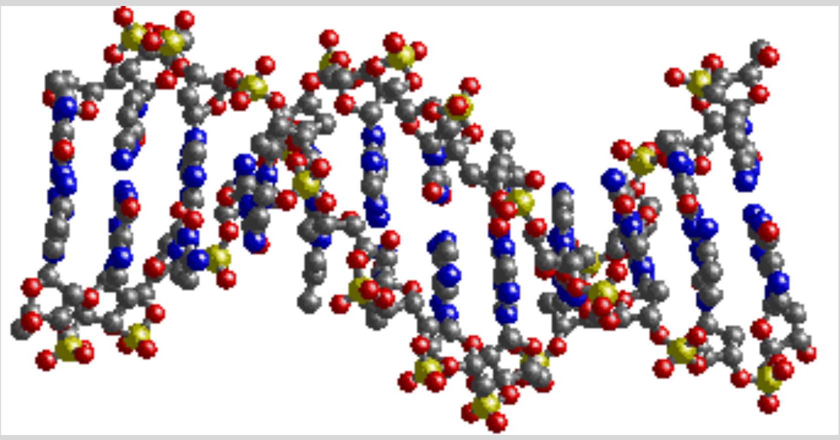
III. Can any regions of the genome escape reprogramming?

IV. **Why** does epigenetic reprogramming occur????

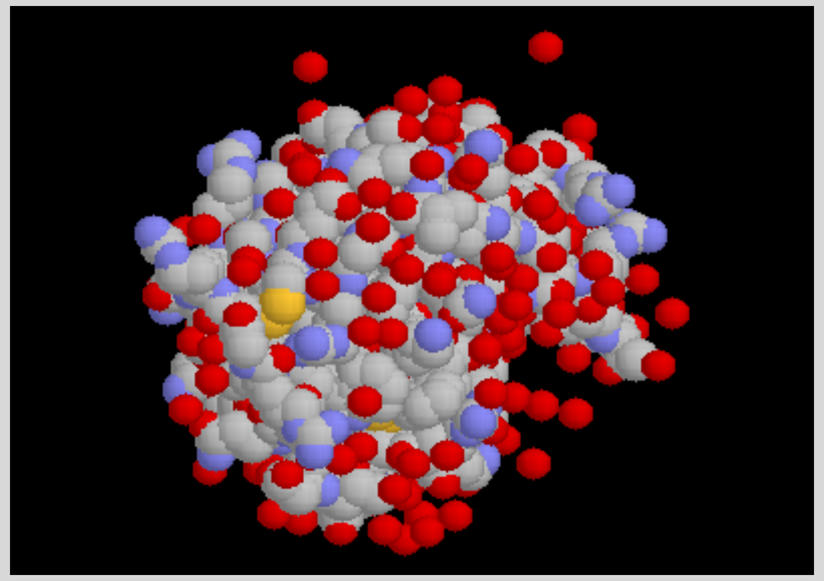
Questions We Will Address Today

I. What is epigenetic reprogamming?

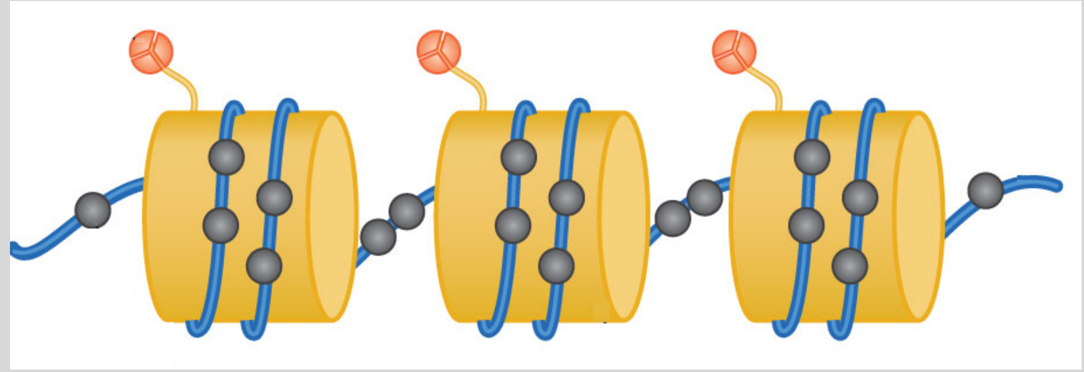

 



GENETIC INFORMATION



CYTOPLASMIC FACTORS



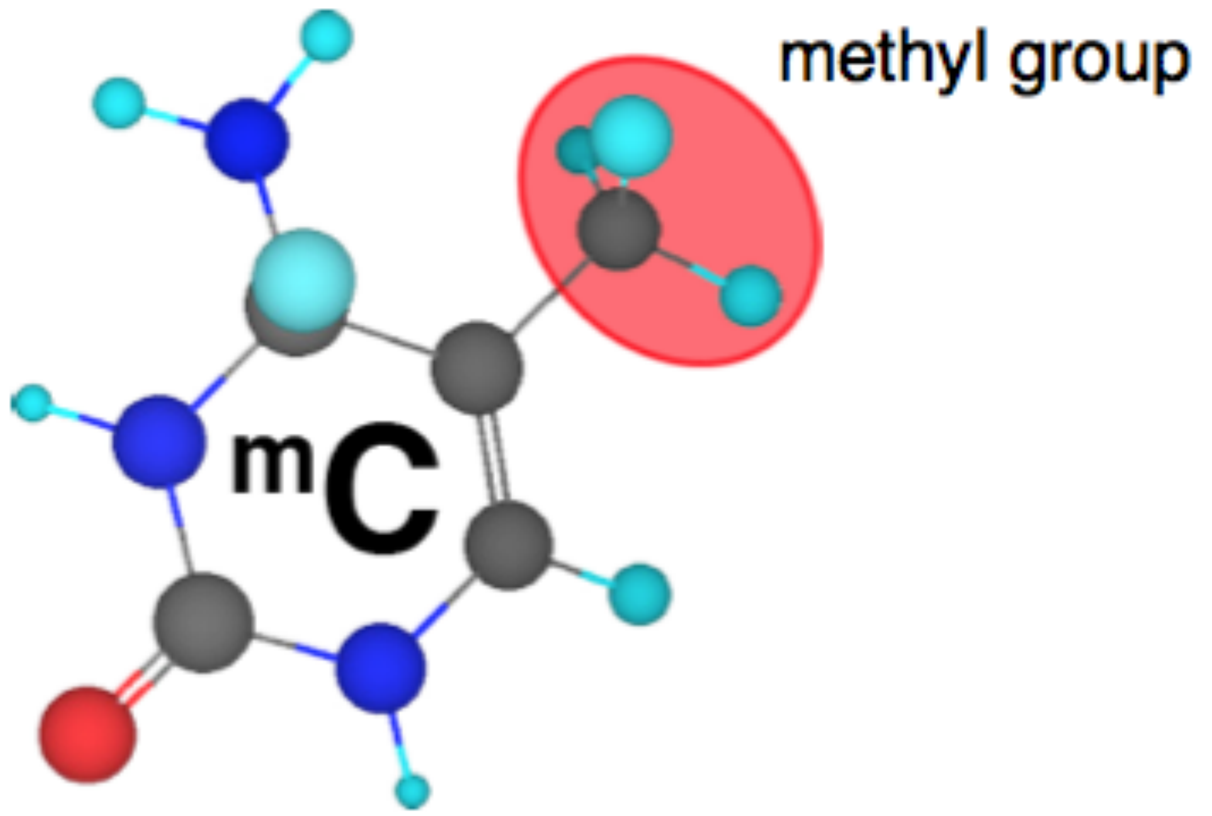
EPIGENETIC INFORMATION

Embryo: “Thanks, but no thanks”

DNA METHYLATION

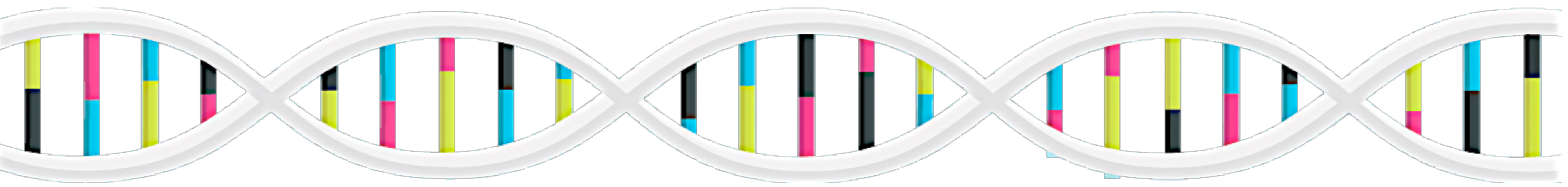
DNA METHYLATION

Covalent modifications of DNA: cytosine methylation



methyl cytosine

in mammals:

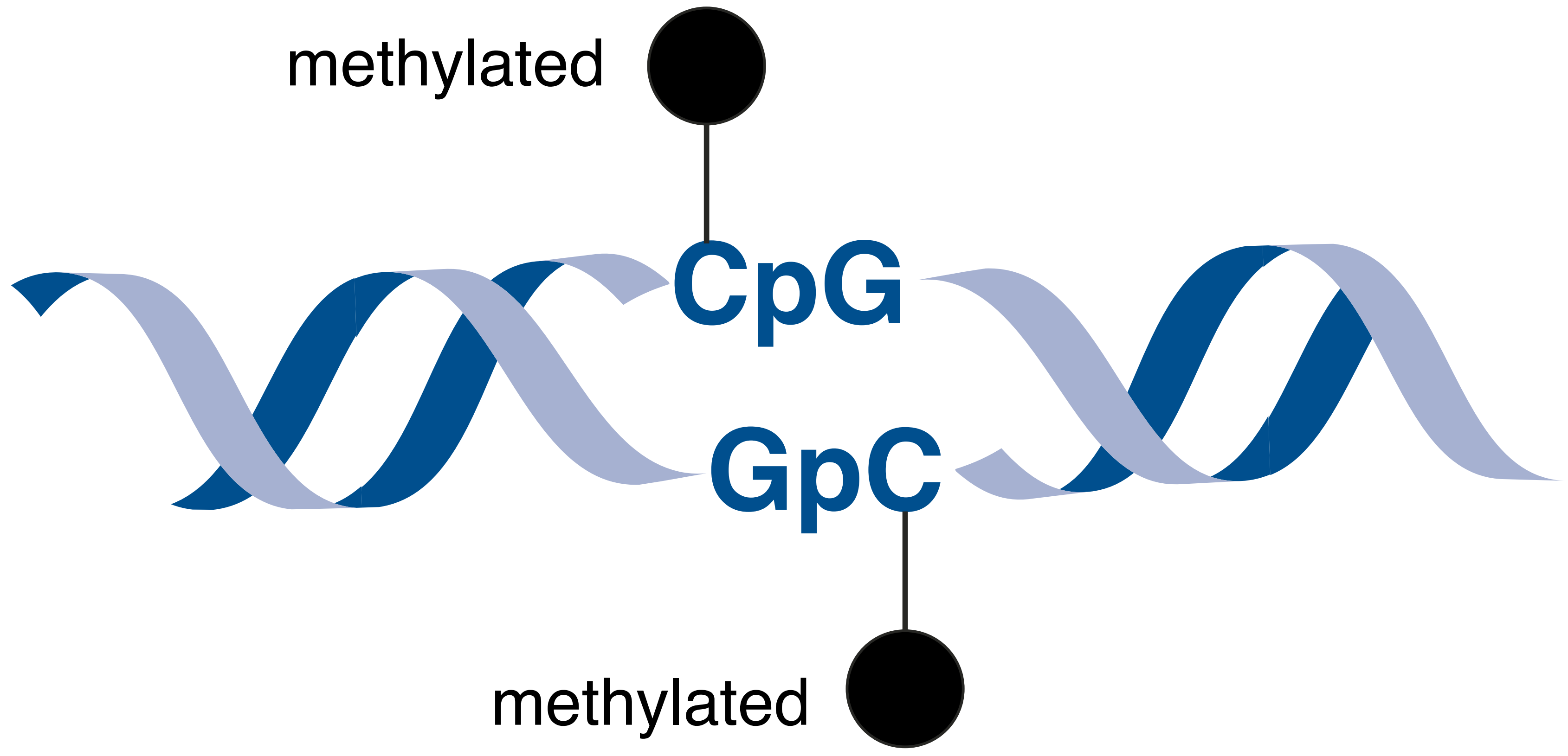


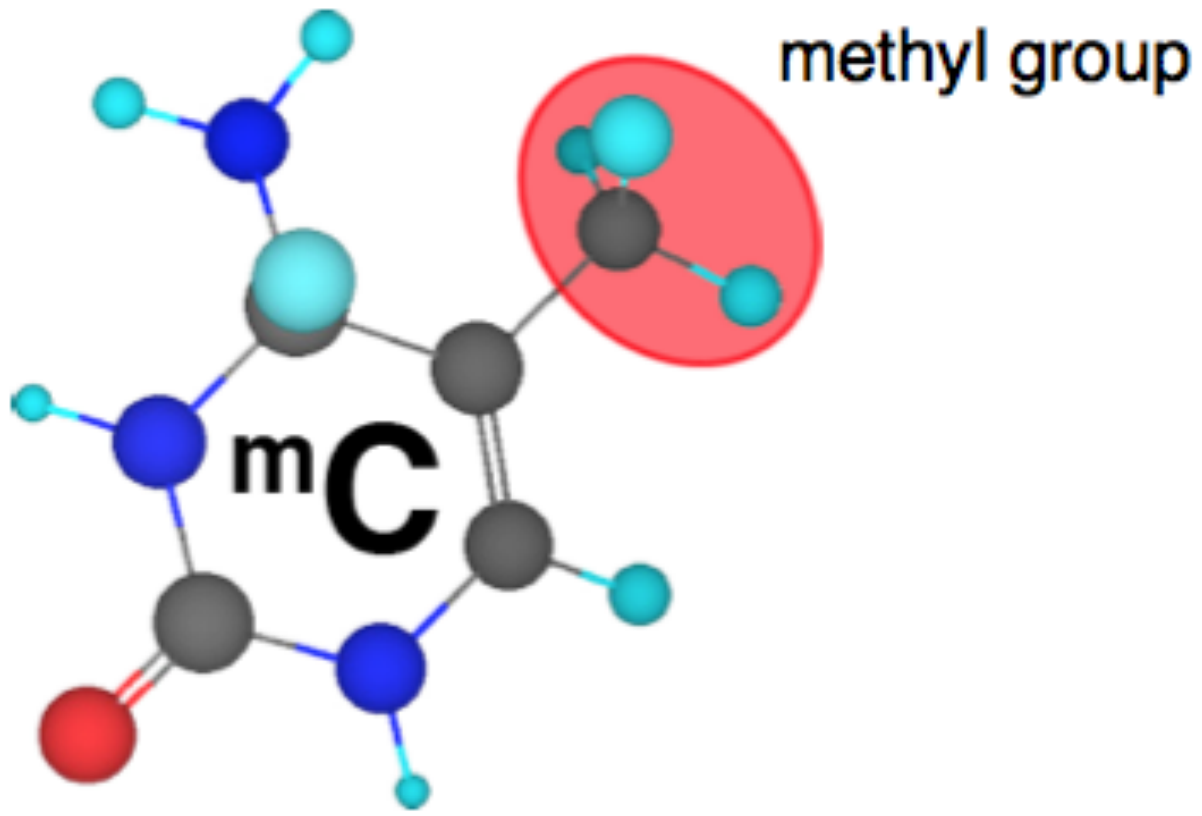
A T G A ^{m}C G A T T C A A G C C

T A C T G C_{m} T A A G T T C G G

DNA METHYLATION

Covalent modifications of DNA: cytosine methylation



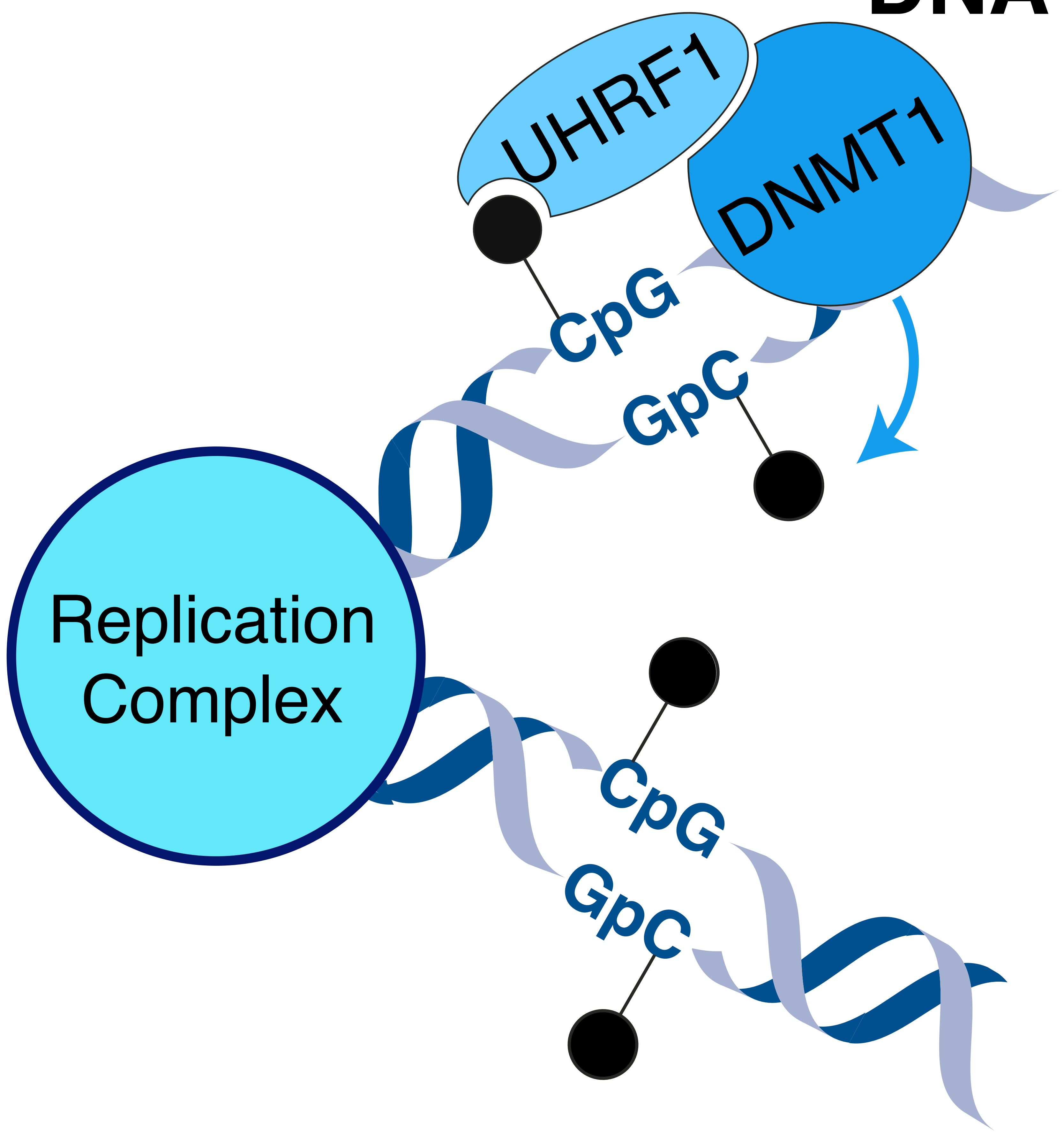


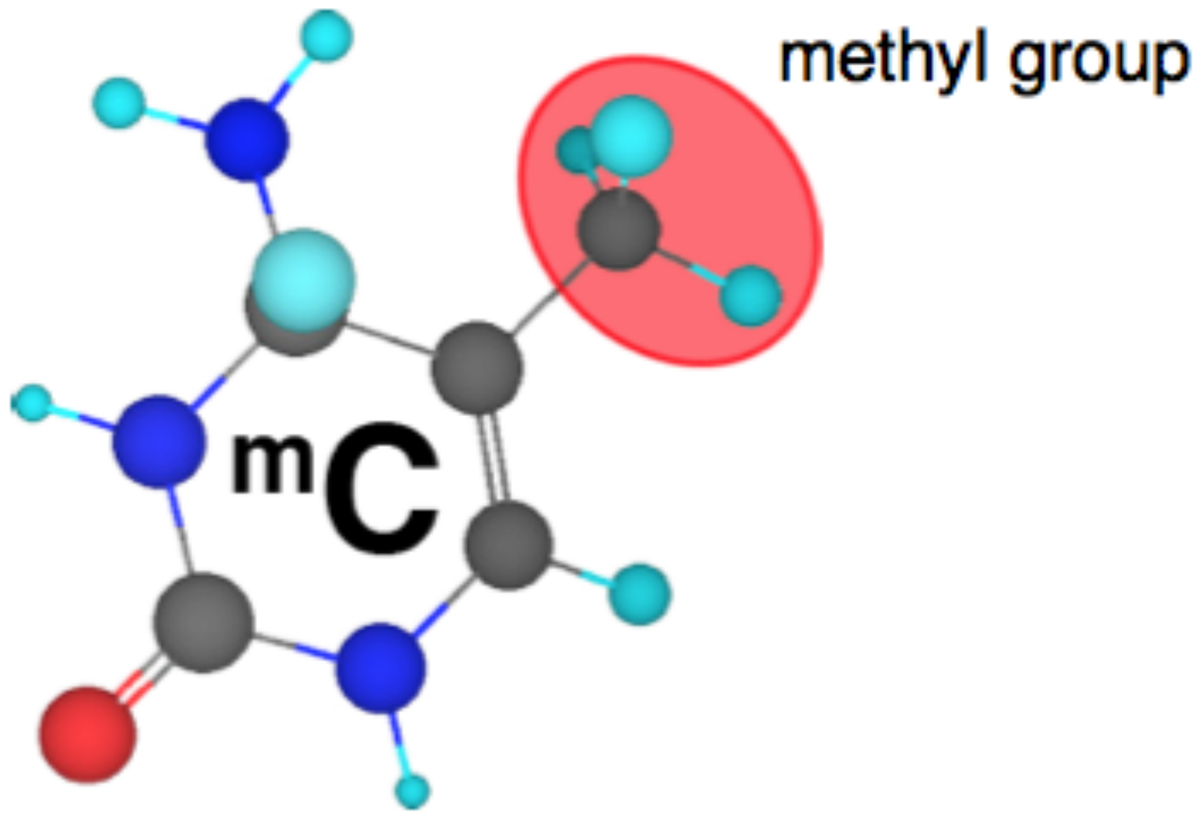
methyl cytosine

DNA METHYLATION

Covalent modifications of DNA: cytosine methylation

DNA Methyltransferase 1

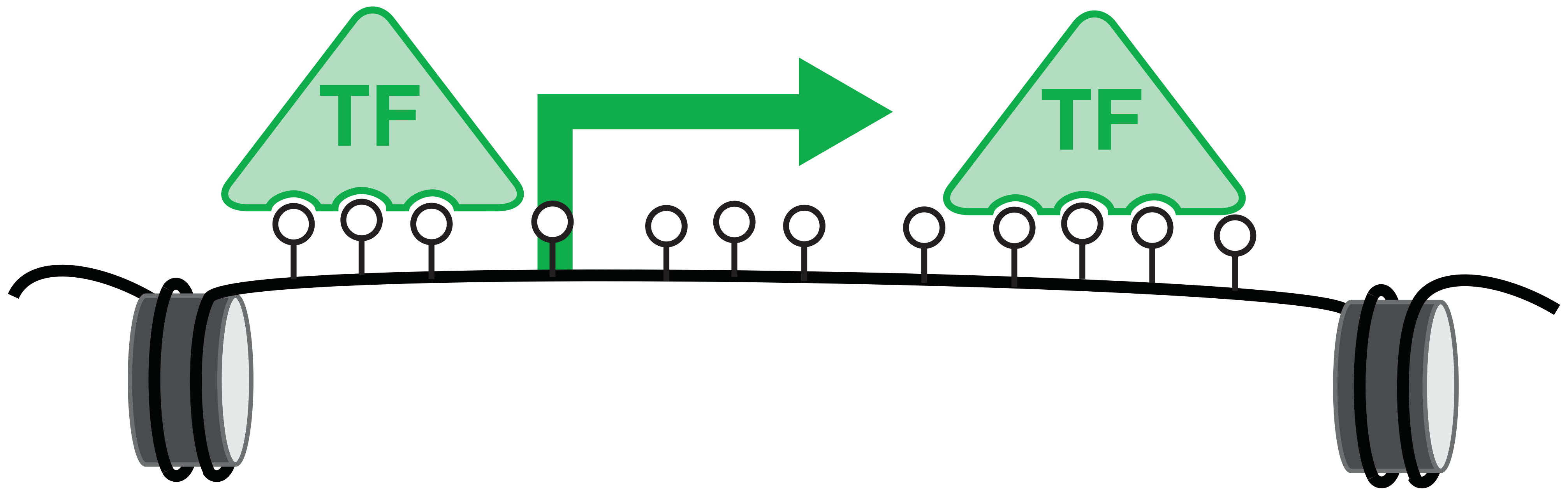




methyl cytosine

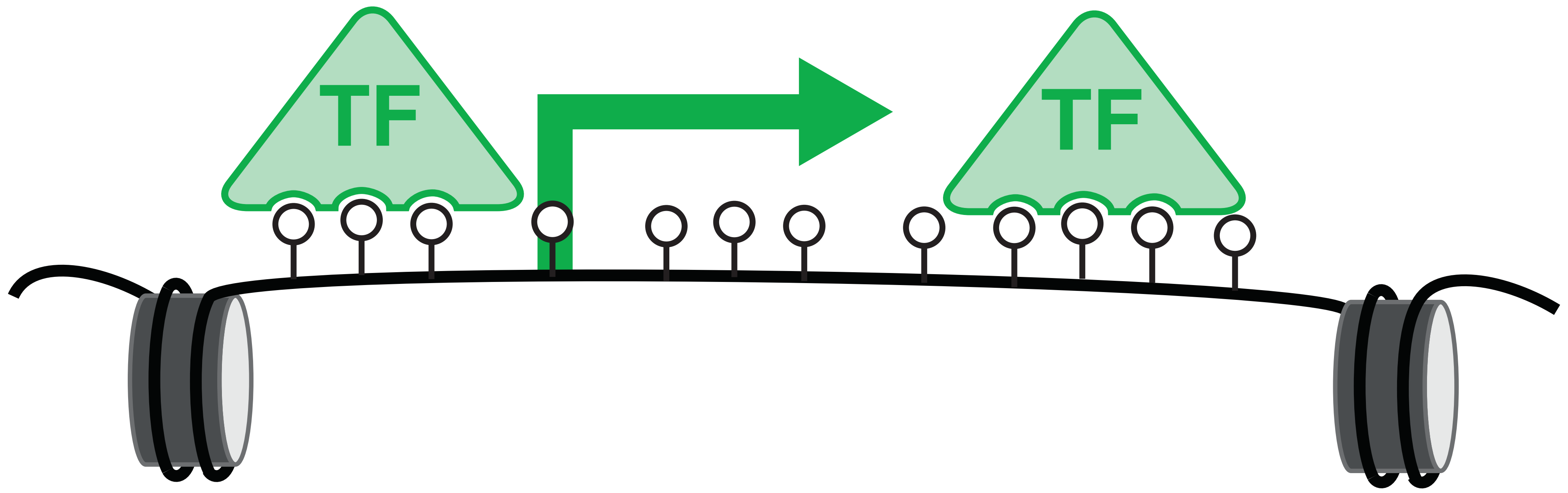
Mechanism for faithful epigenetic inheritance

DNA METHYLATION



Unmethylated promoter potentially active
(TF = Transcription Factor)

DNA METHYLATION



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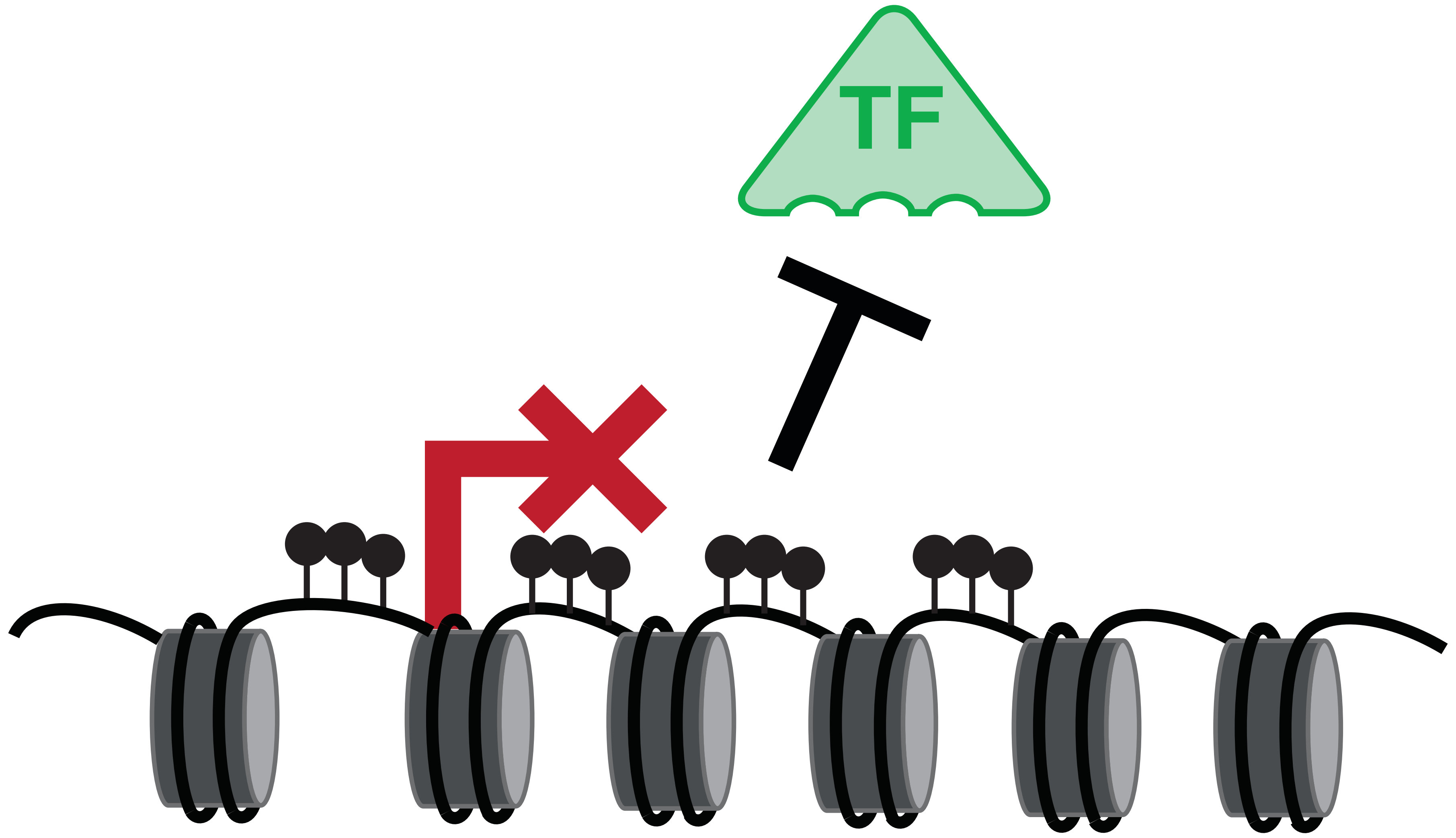
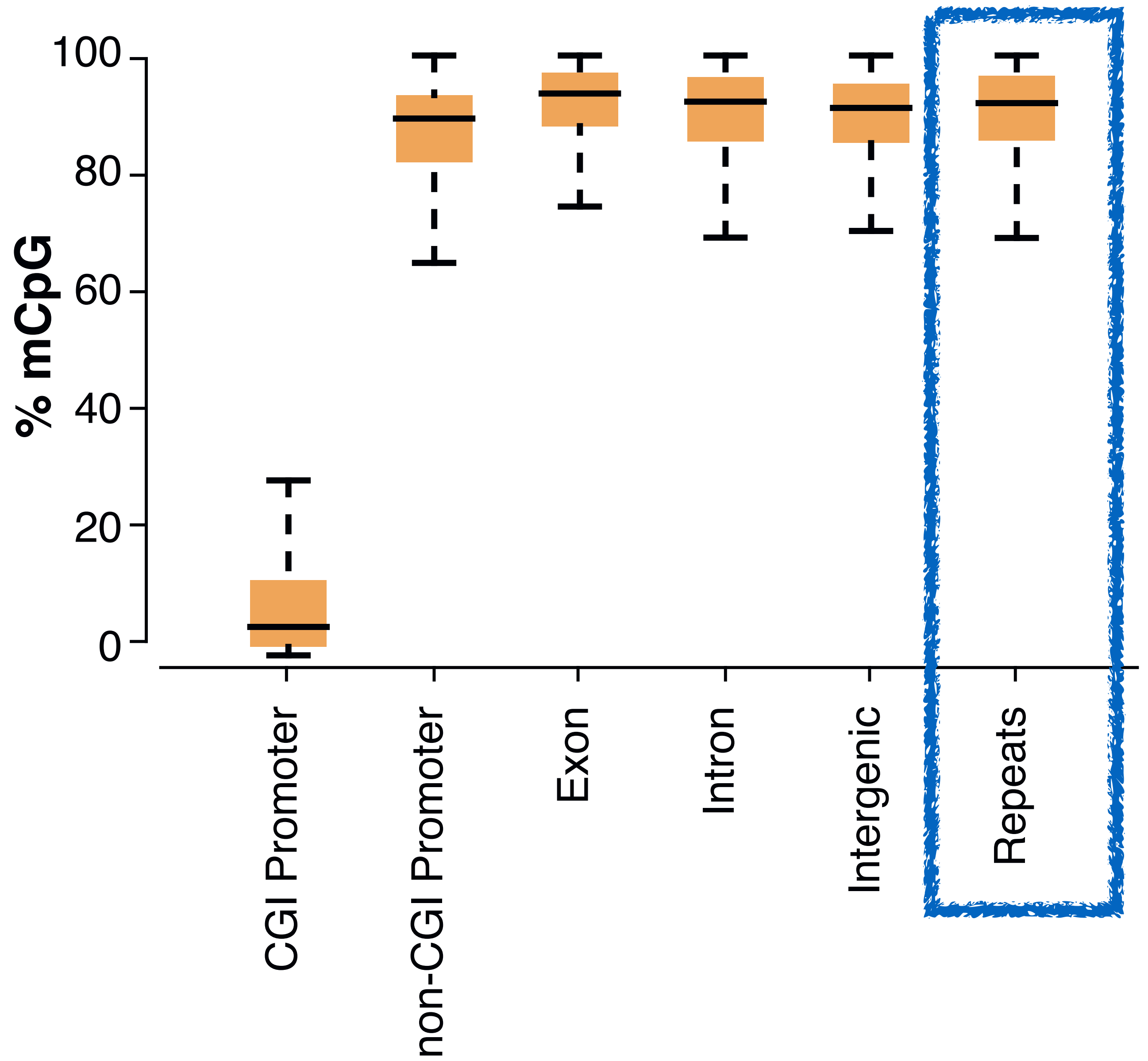


TFs can not bind methylated promoter

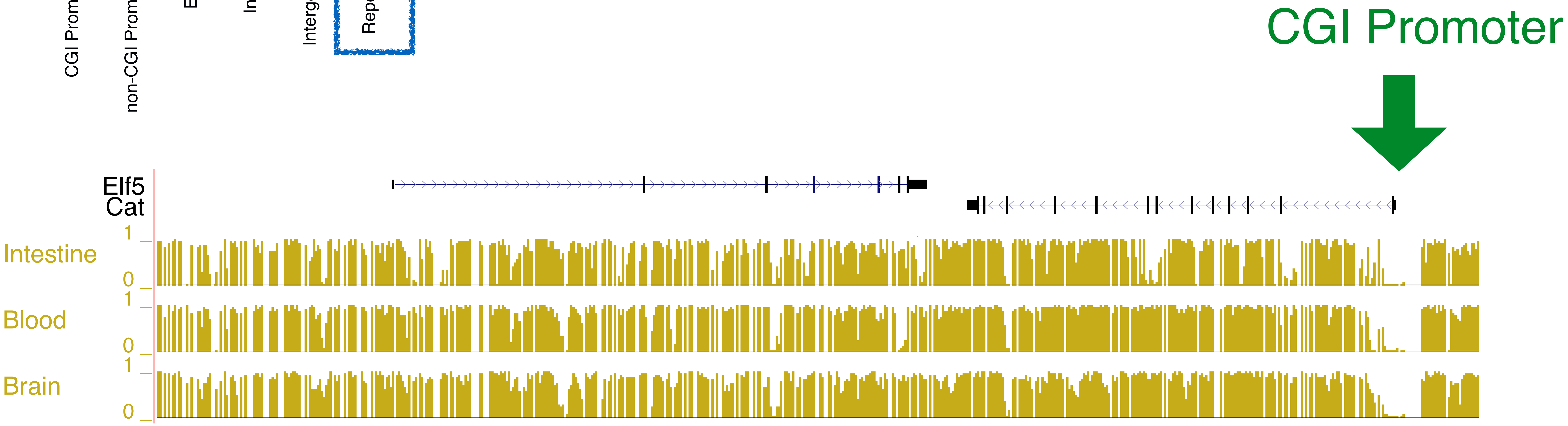
DNA methylation = Mark of transcriptional silencing

DNA METHYLATION



DNA methylation is actually regulating few gene promoters

CGI = CpG Island, (Very CpG-rich)



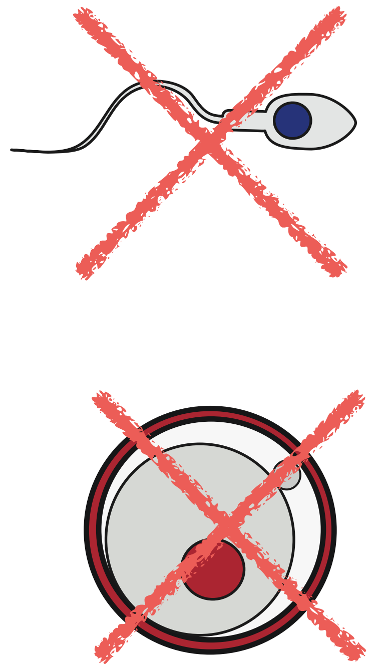
CpG Methylation basepair resolution

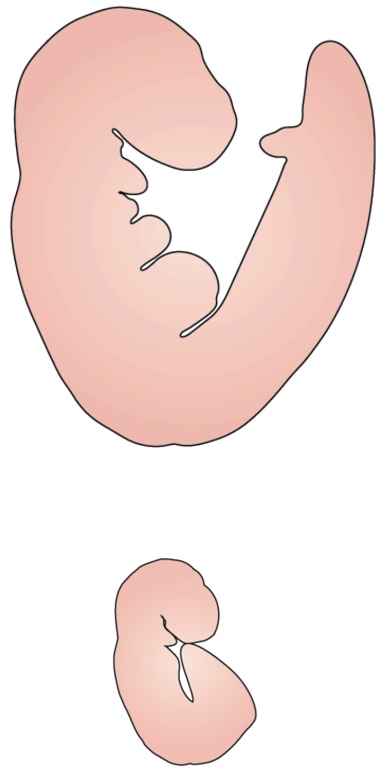
DNA METHYLATION

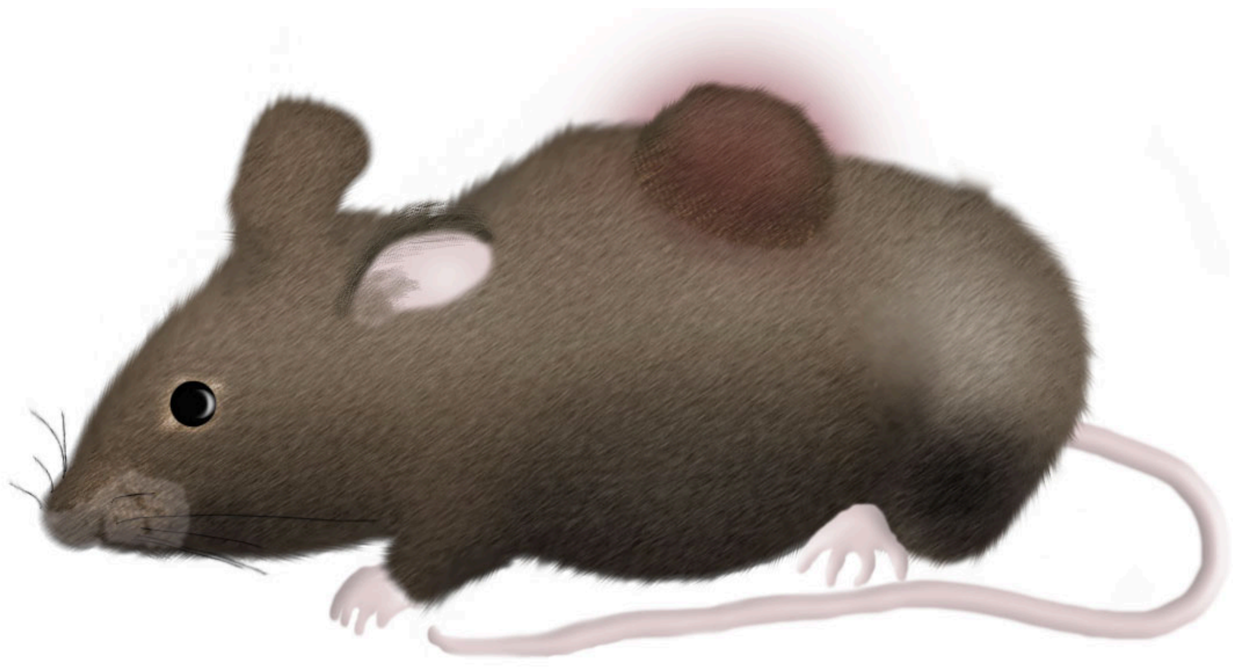
Gene dosage control
Cell fate/differentiation
Genomic stability (transposon control)

Germline **Embryo** **Soma**

DNA methylation defects







Sterility

Lethality
Anomalies

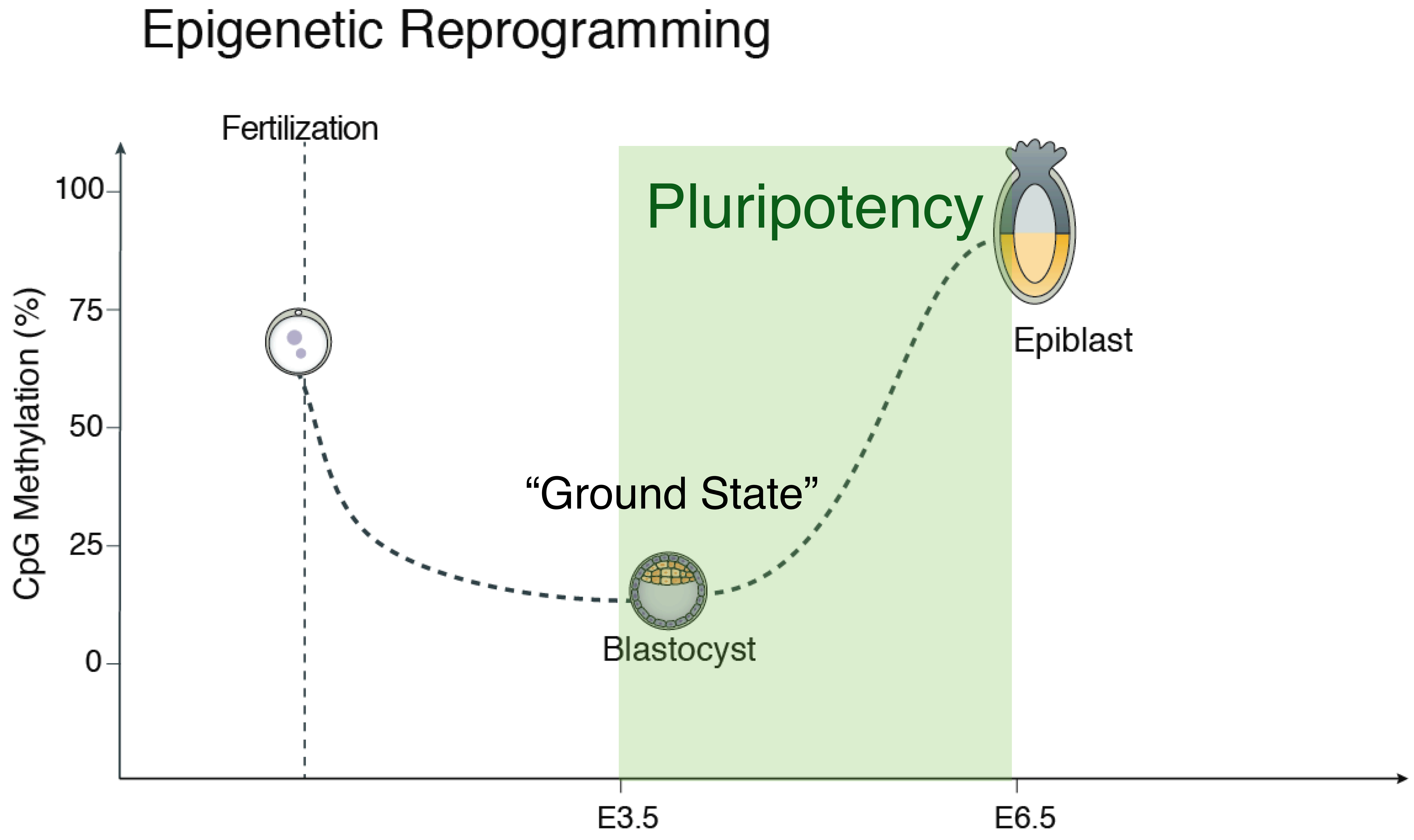
Cancer

DNA METHYLATION

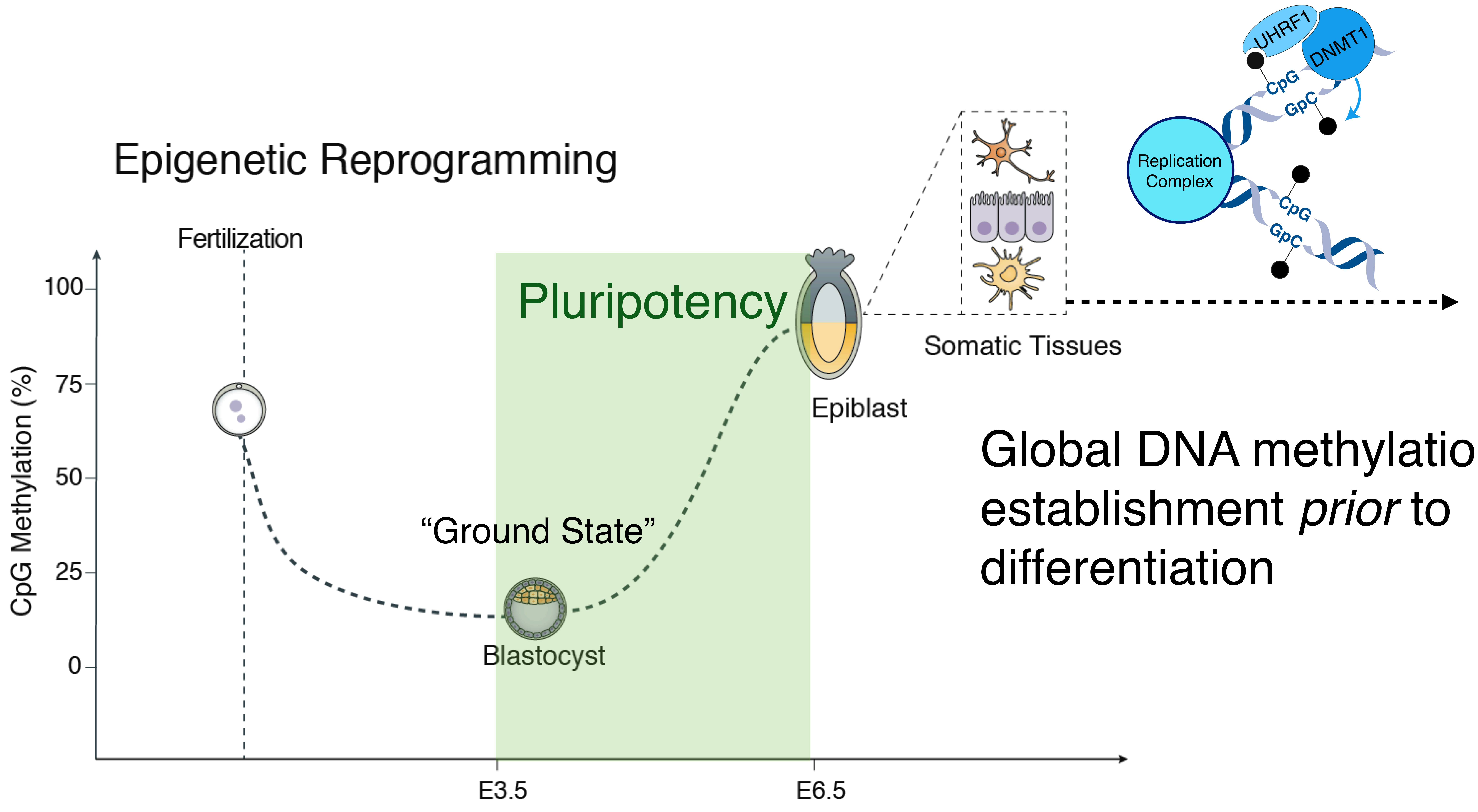
Gene dosage control
Cell fate/differentiation
Genomic stability (transposon control)

DNA Methylation is important!

DNA Methylation Highly Dynamic in Embryo

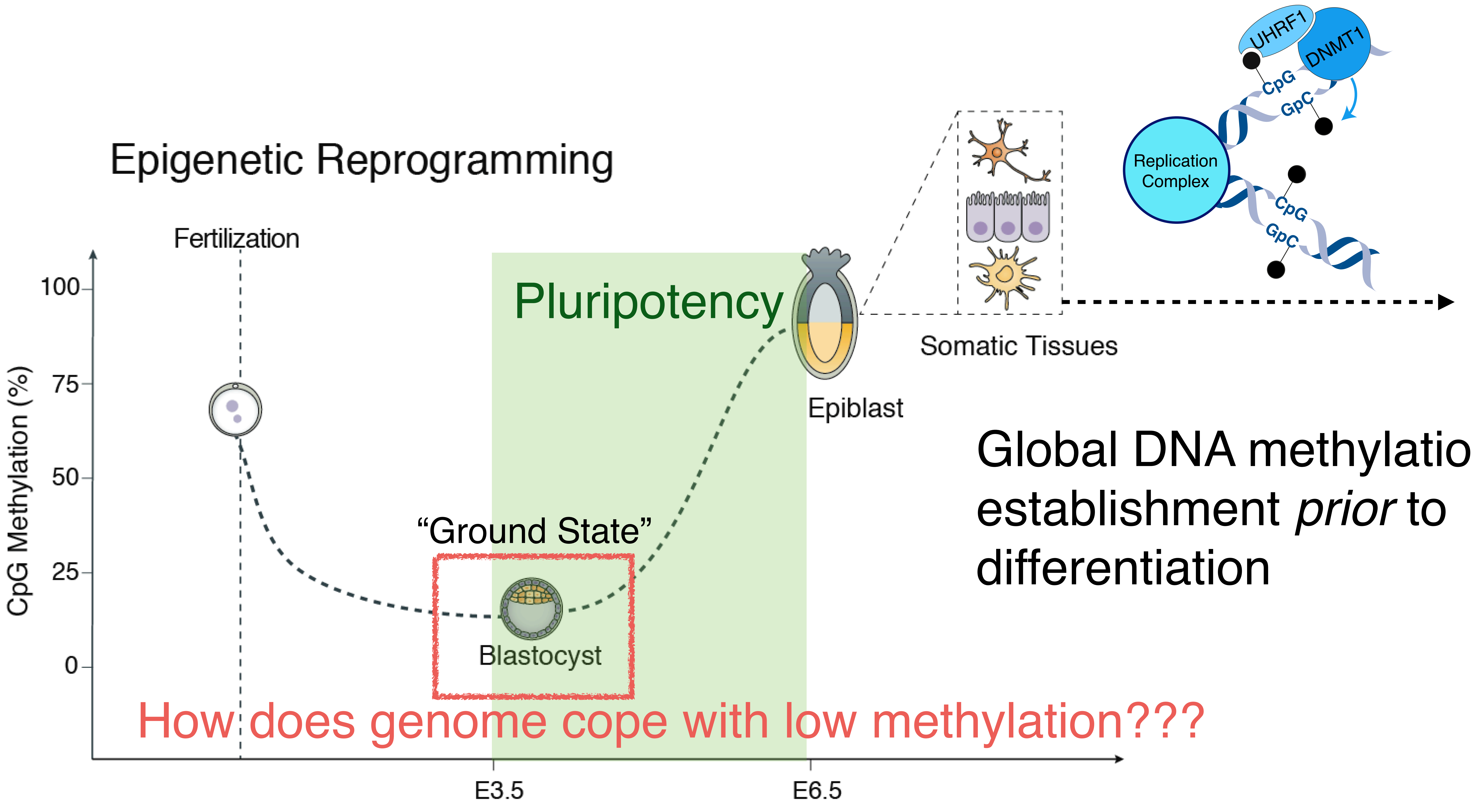


DNA Methylation Highly Dynamic in Embryo



Global DNA methylation establishment *prior* to differentiation

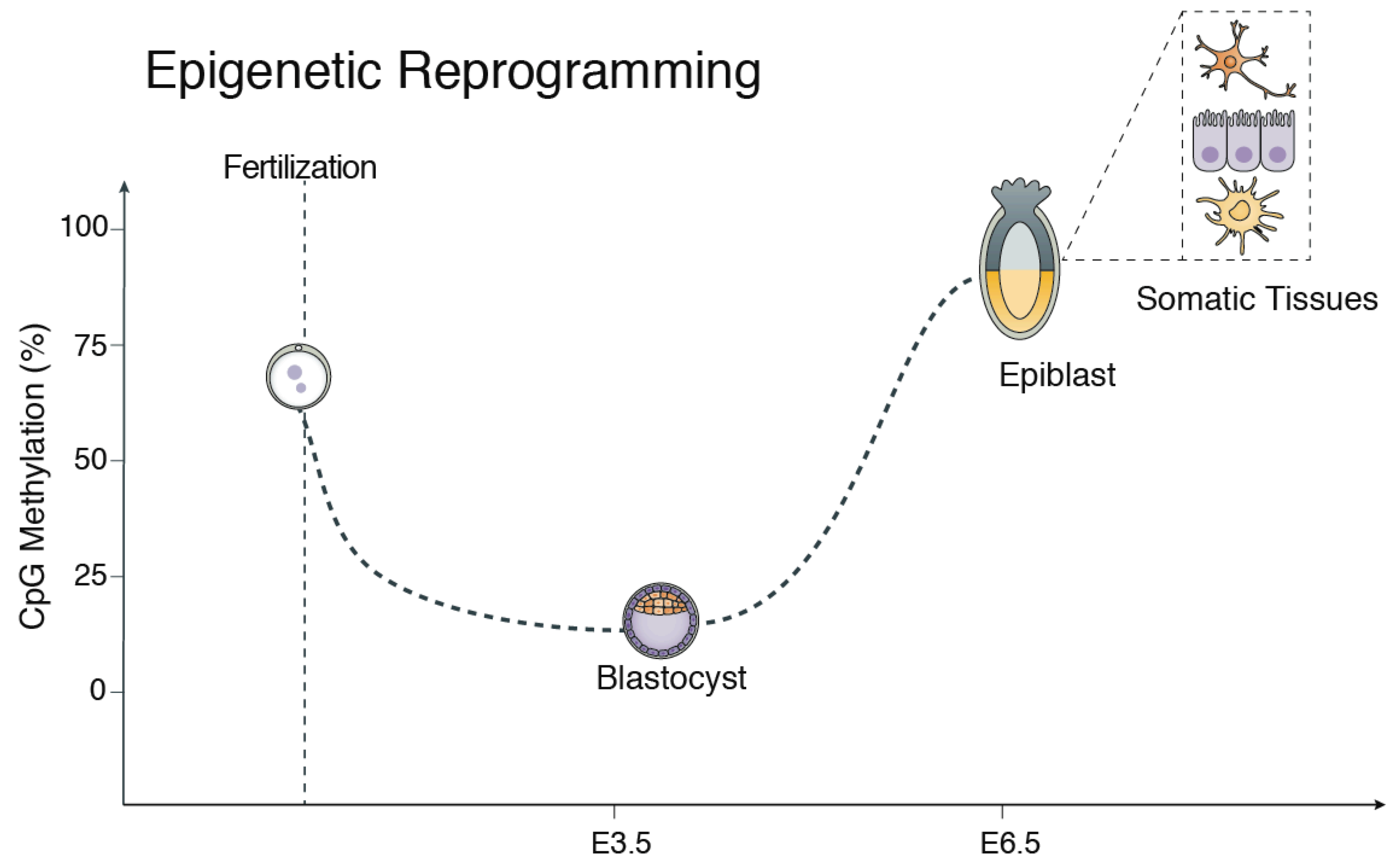
DNA Methylation Highly Dynamic in Embryo



Global DNA methylation establishment *prior* to differentiation

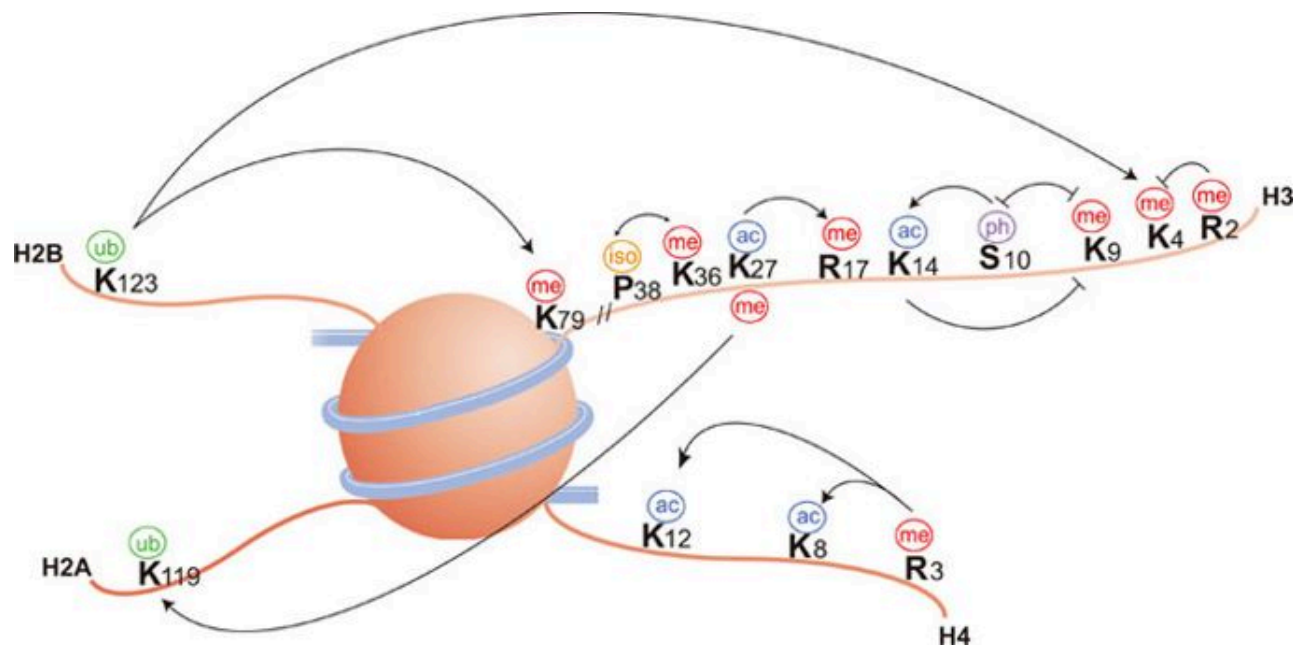
How does genome cope with low methylation???

DNA Methylation Highly Dynamic in Embryo

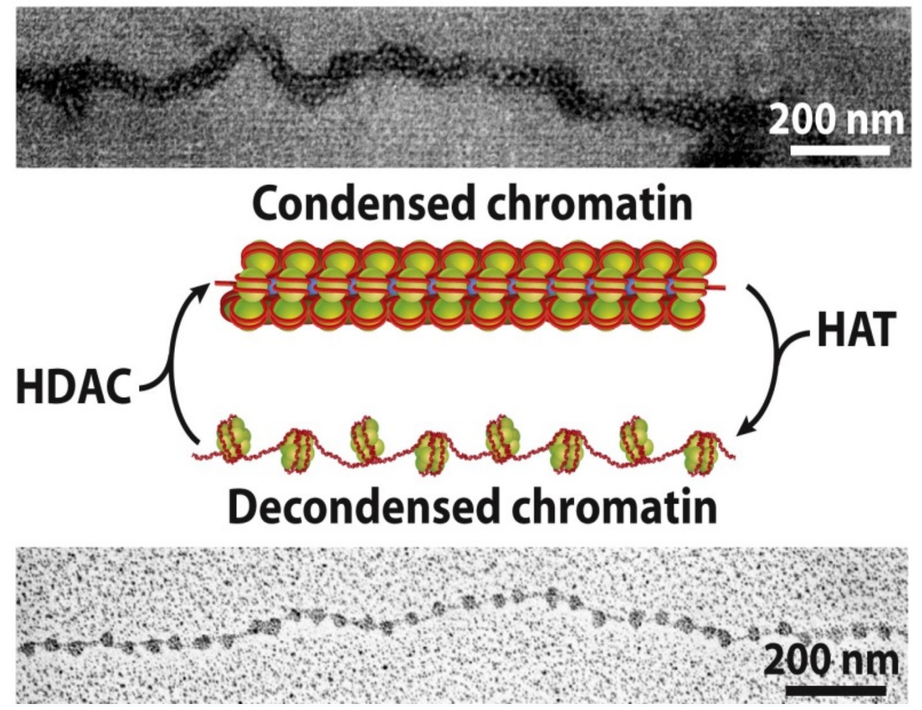


What about other aspects of chromatin?

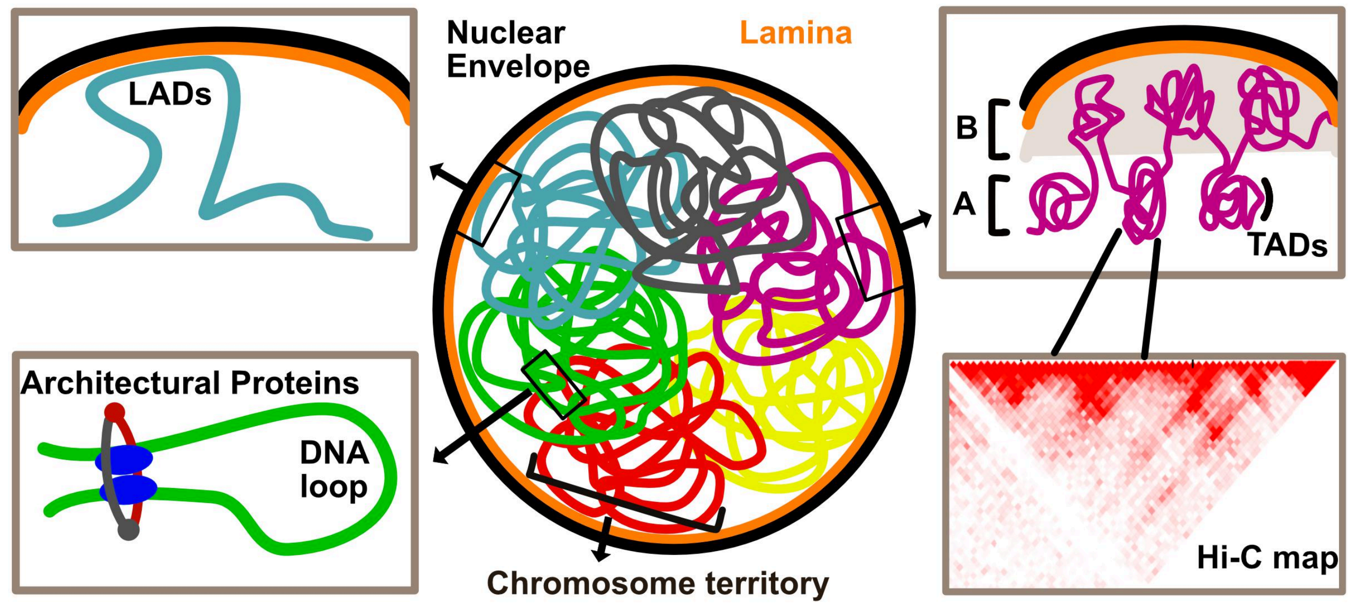
Histone Modifications



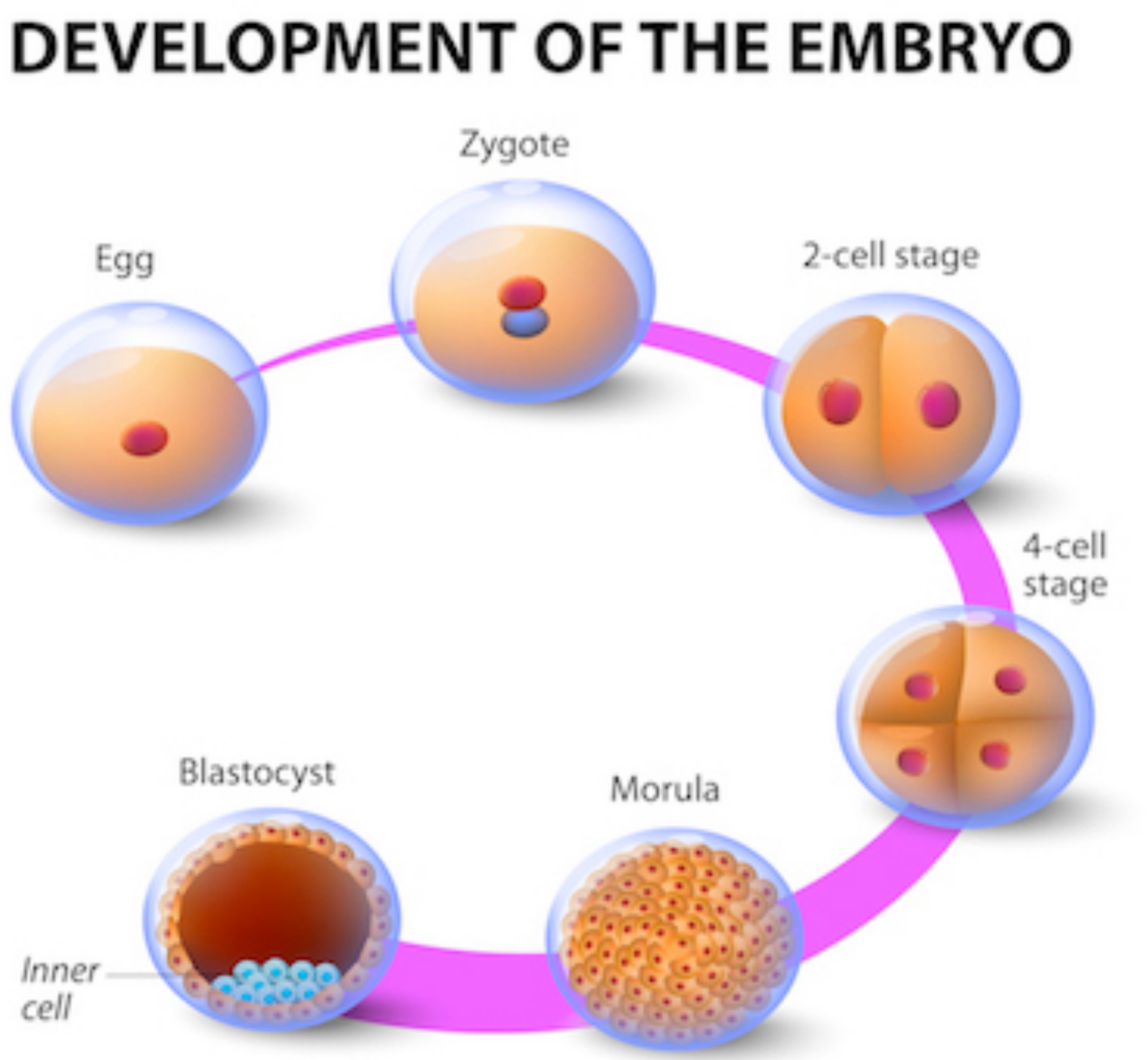
Chromatin Accessibility



Nuclear Architecture

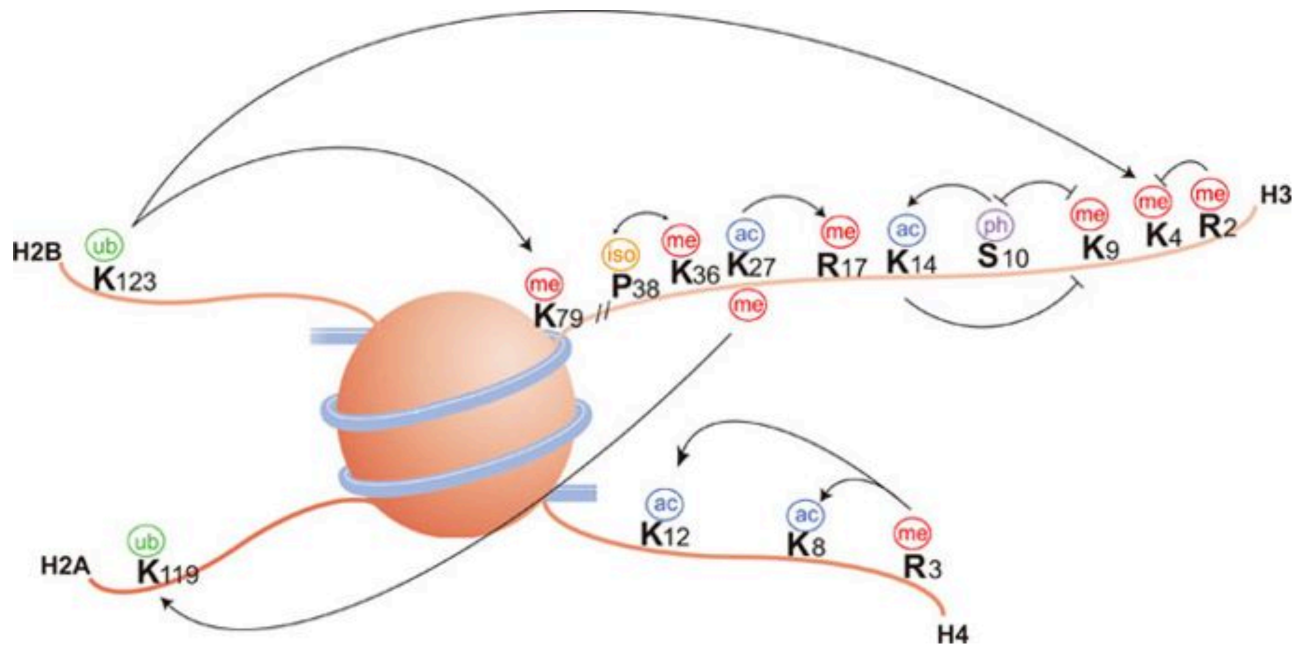


Problem



- Precocious embryos are tedious to collect in large numbers (and obvious ethical issues for human embryos)
- Early embryos have limited number of cells
- Chromatin assays historically required a lot of tissue (ChIP, 3C, etc)

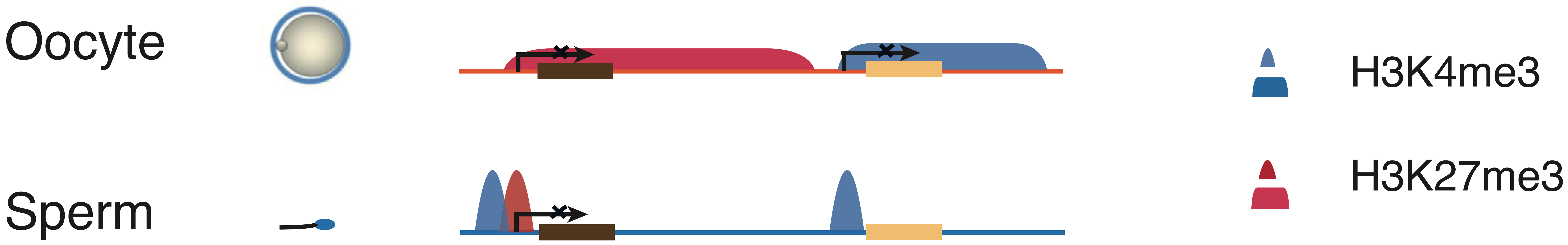
Solution: the rise of low input sequencing technologies (exploding field)

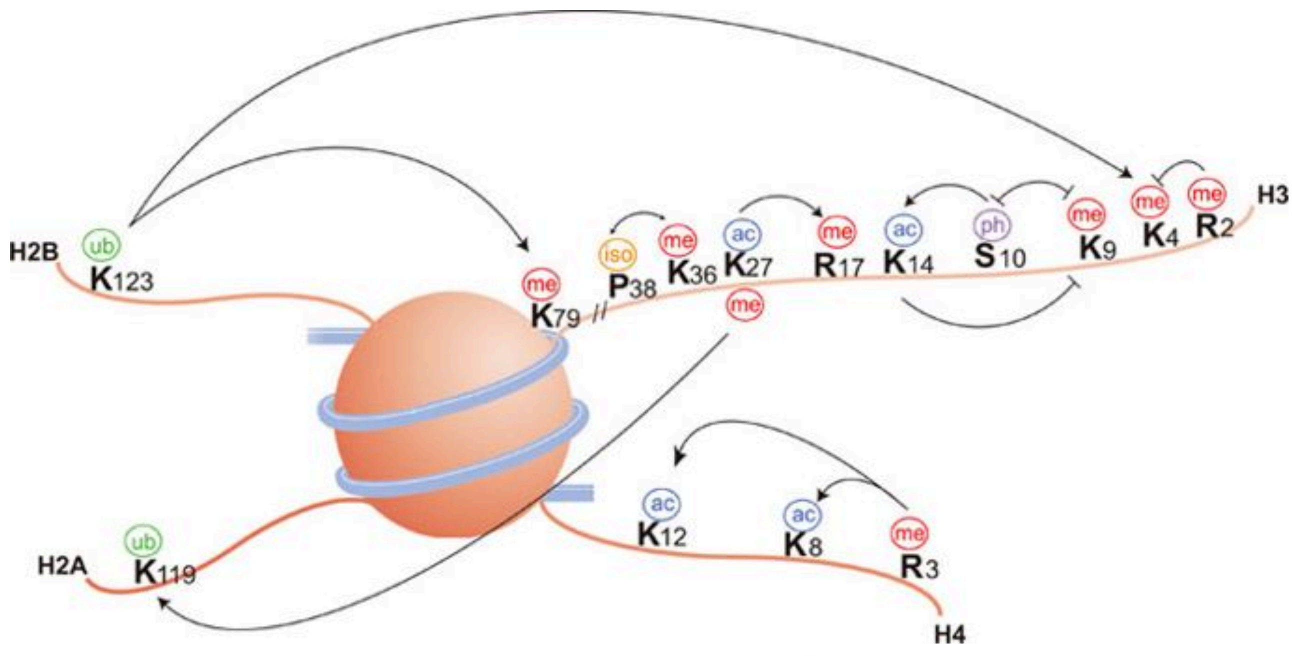


Histone Modifications

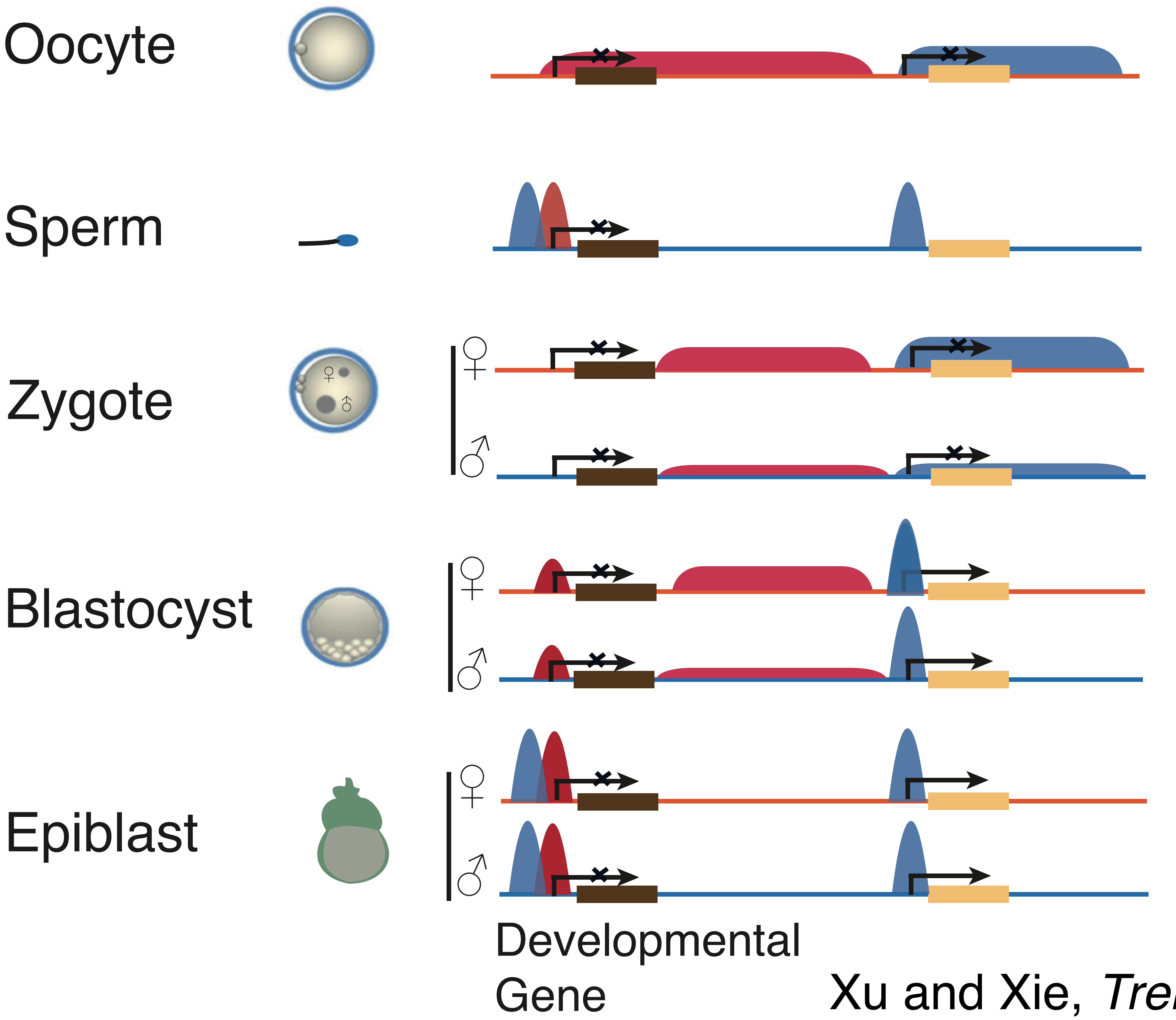
H3K4me3: Associated with active/poised promoters

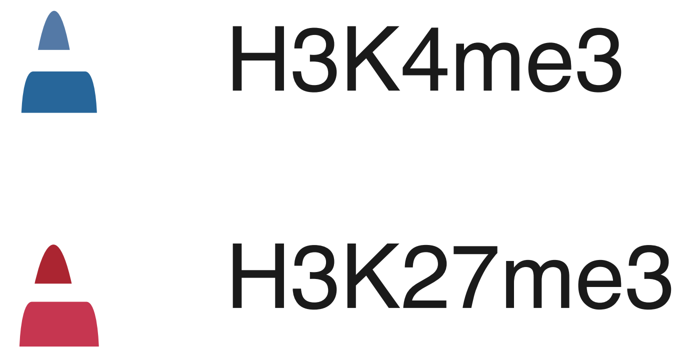
H3K27me3: Associated with silent/poised promoters





Histone Modifications

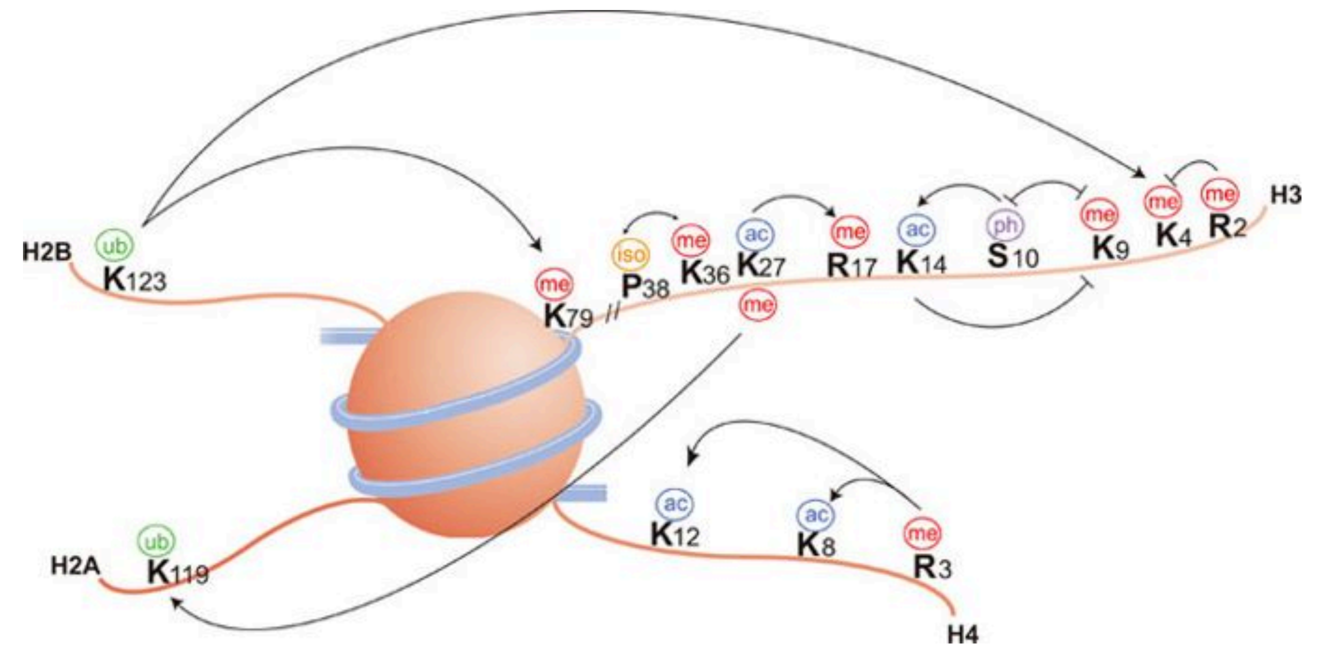




Rapid remodeling of paternal marks

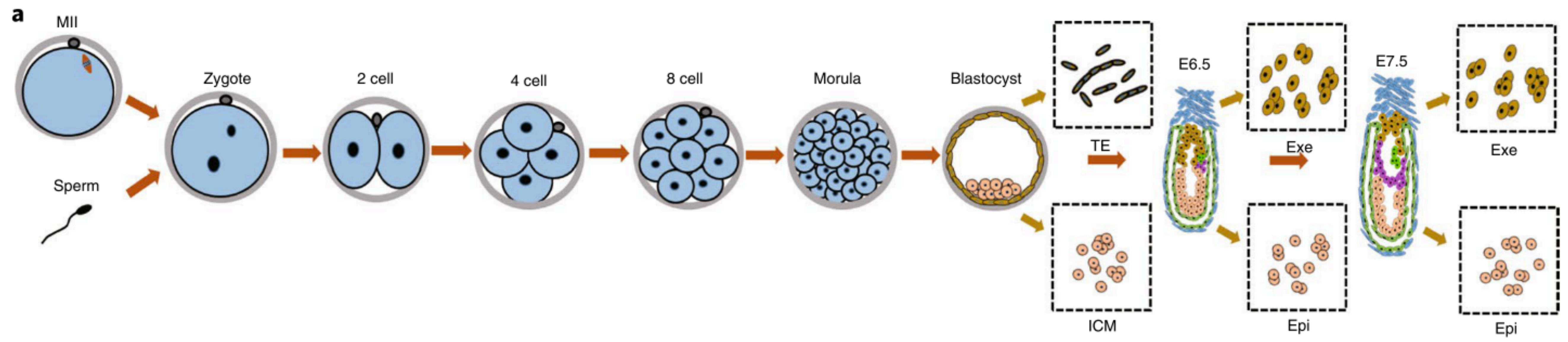
H3K27me3 restricted when DNA methylation is high

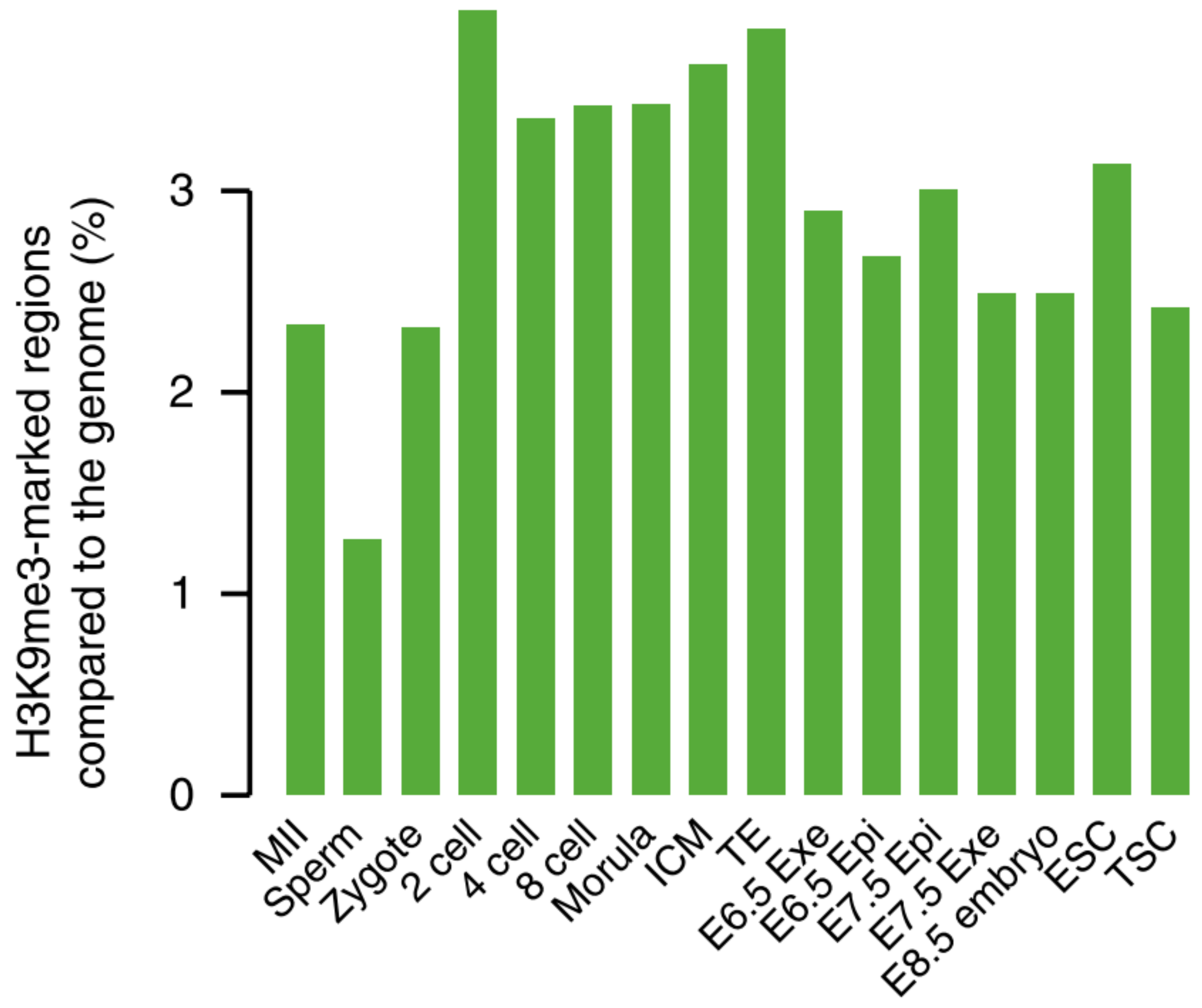
Xu and Xie, *Trends in Cell Biology*, 2018



Histone Modifications

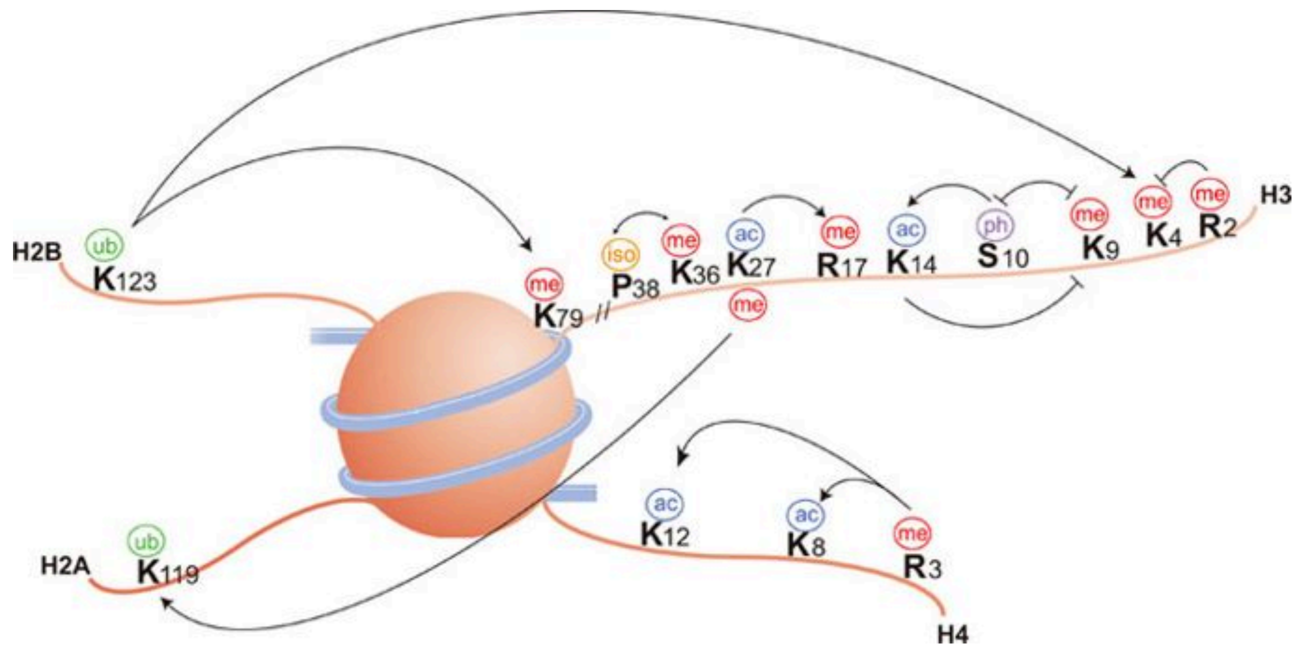
H3K9me3: Associated with constitutive heterochromatin





High H3K9me3 levels throughout pre-implantation development

Wang et al., *NCB* 2018



Histone Modifications

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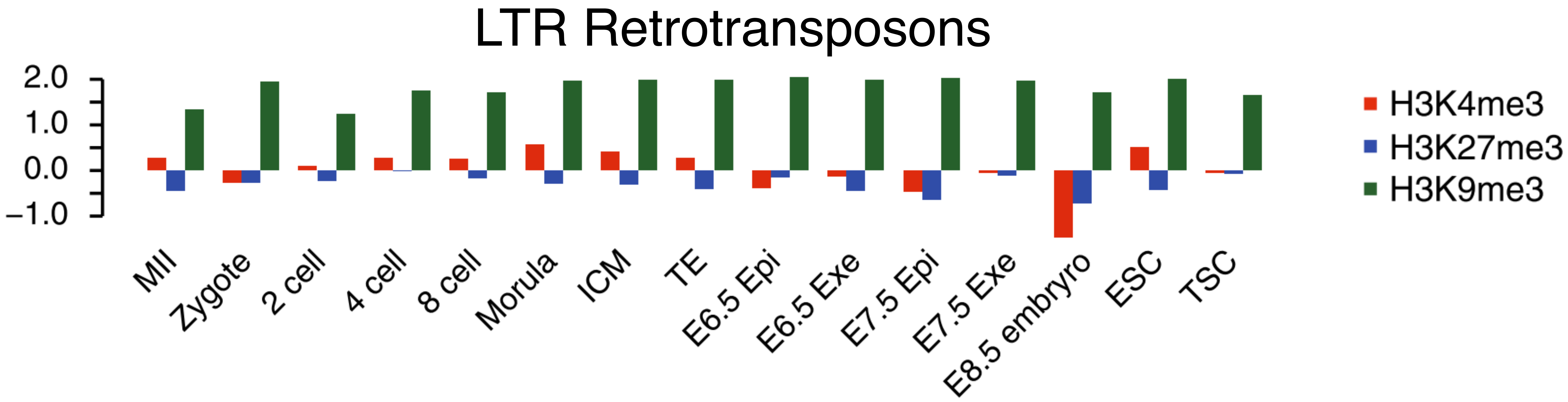
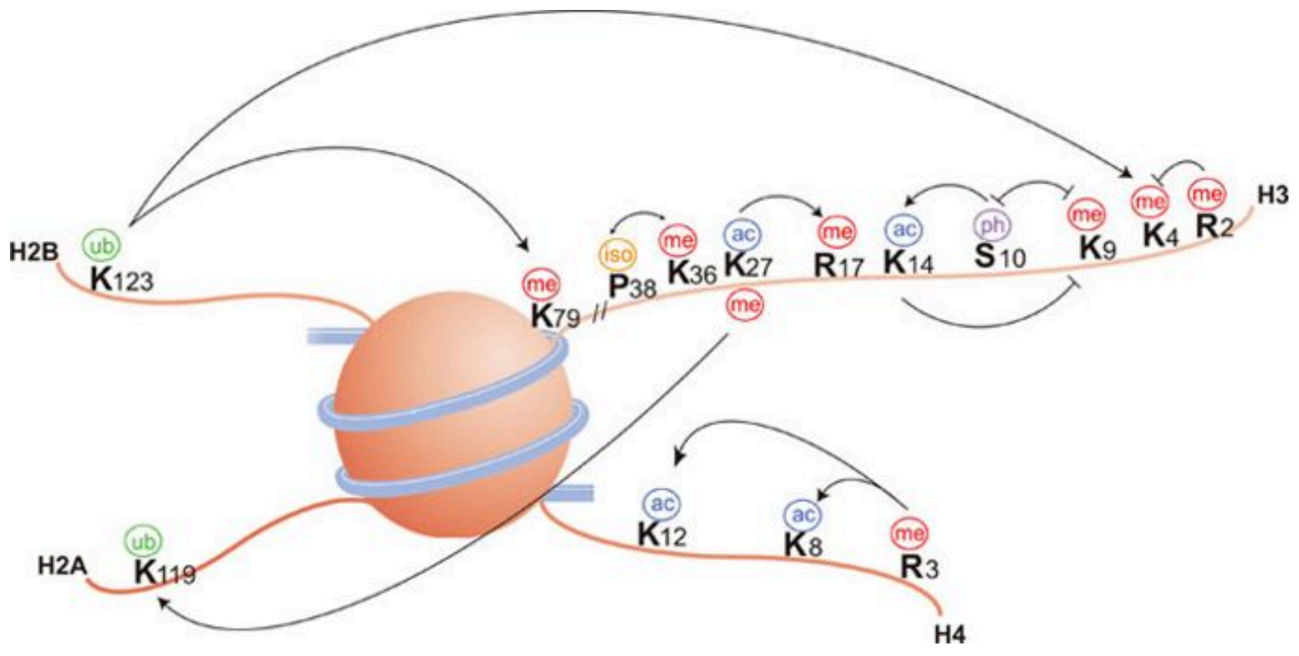


Virtually no global reprogramming

Or is there??

Wang et al., *NCB* 2018



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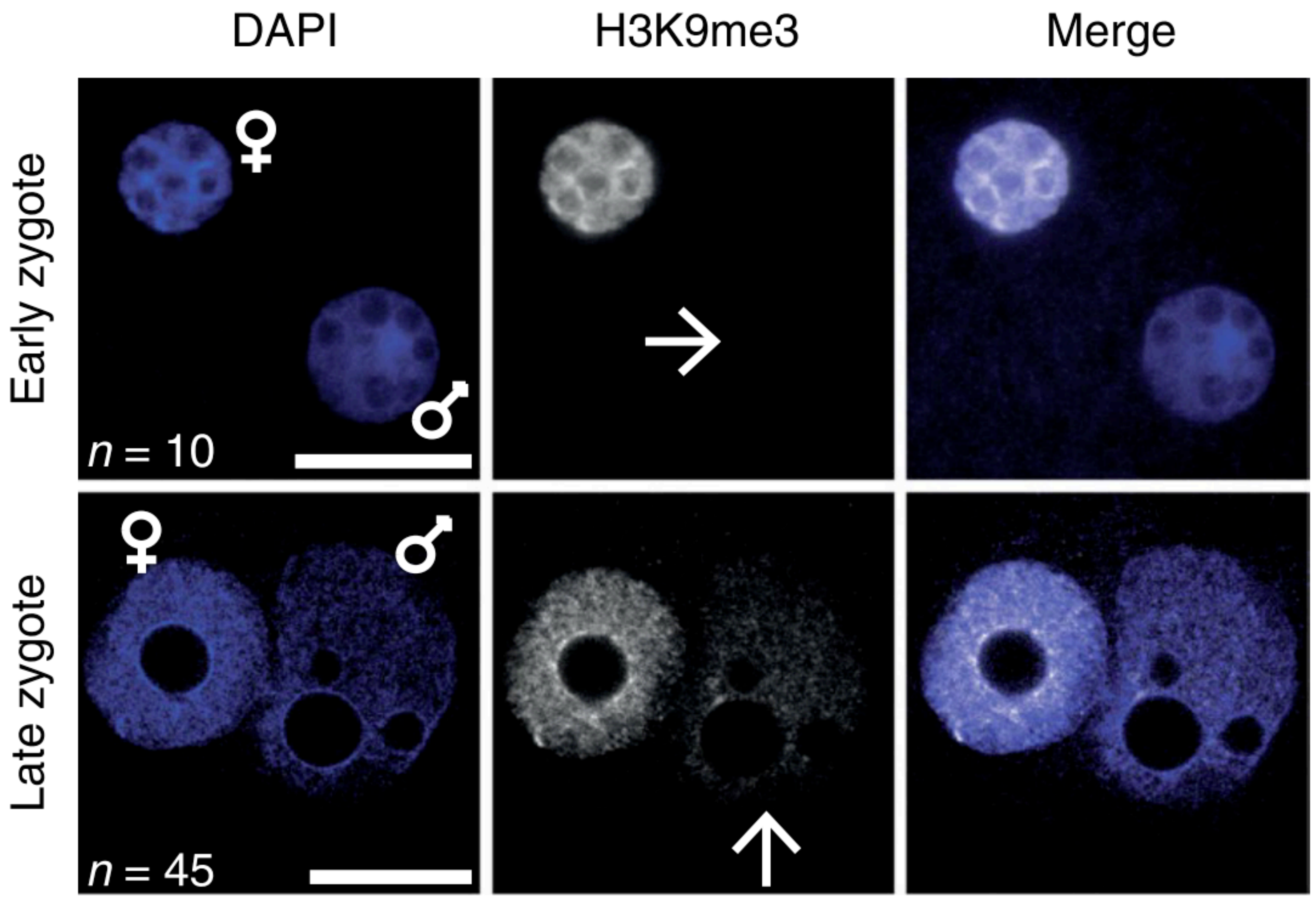
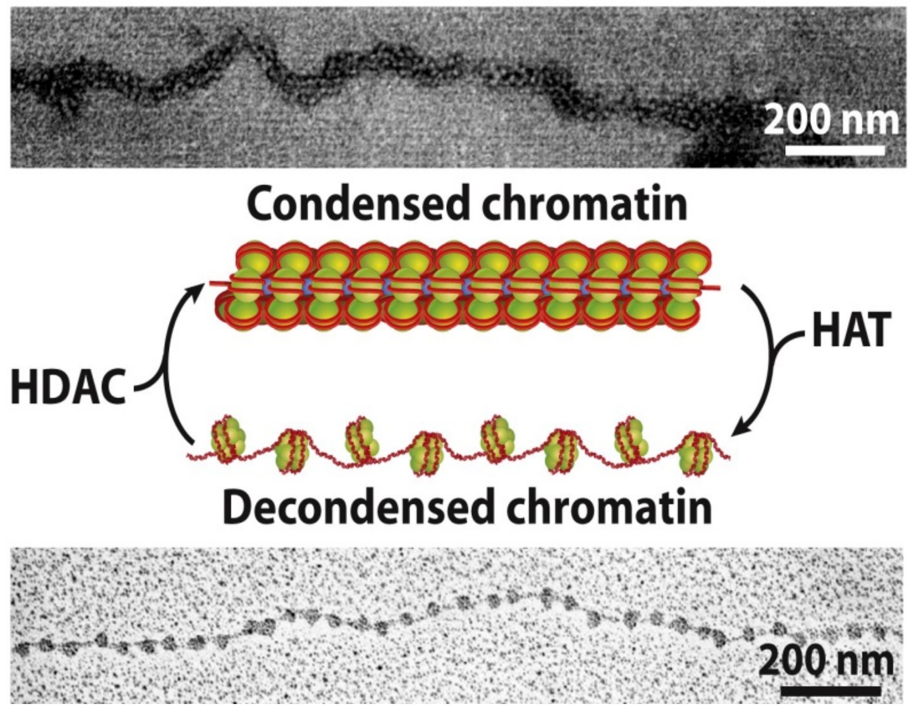


Zygotic deposition of H3K9me3 in paternal pronucleus

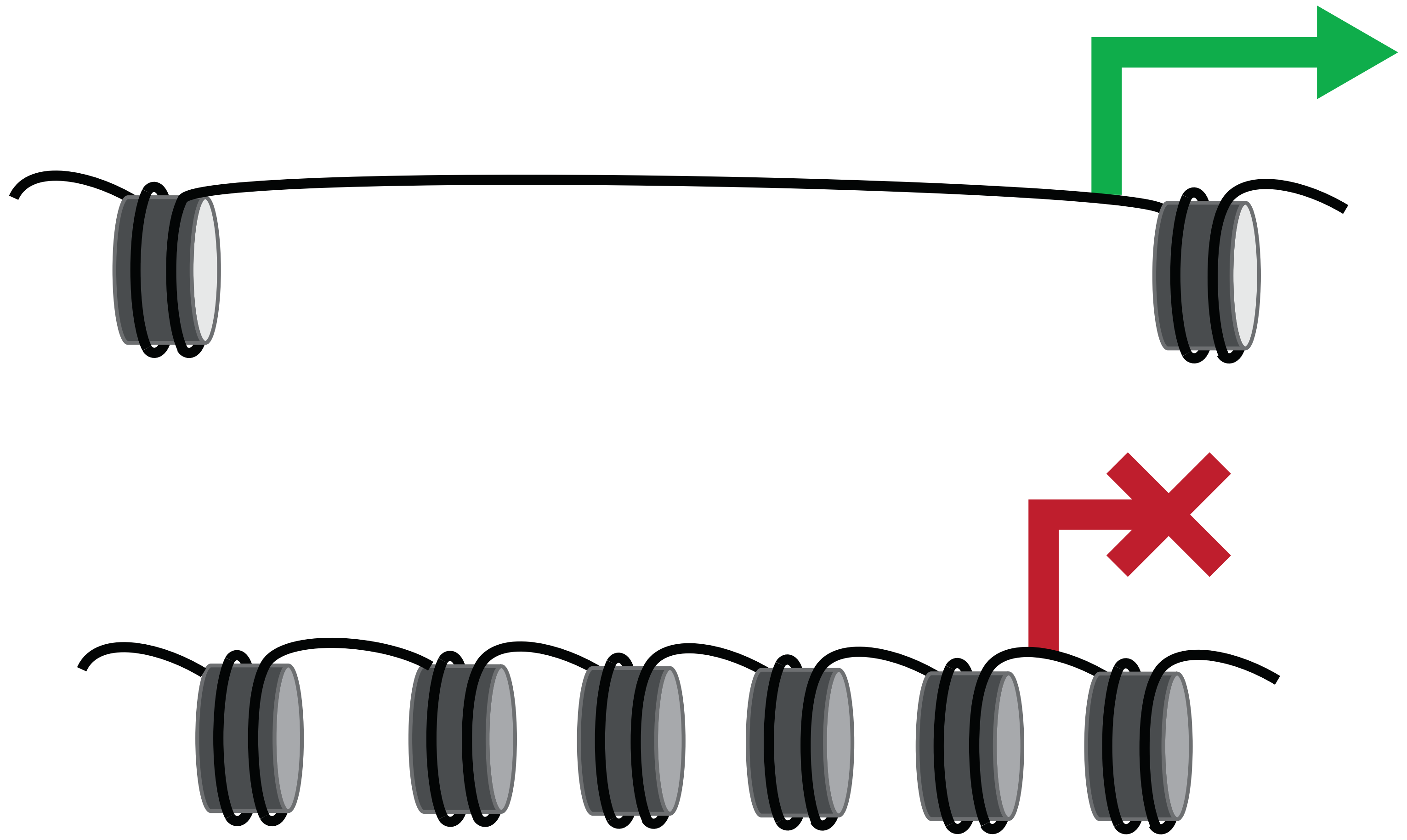
Asymmetric embryonic H3K9me3 patterns until blastocyst

Burton et al., *NCB* 2020



Chromatin Accessibility

A measure of how “open” (ie, permissive for transcription) or “closed” nucleosomes are positioned relative to each other: **ie, poised/active enhancers and promoters**

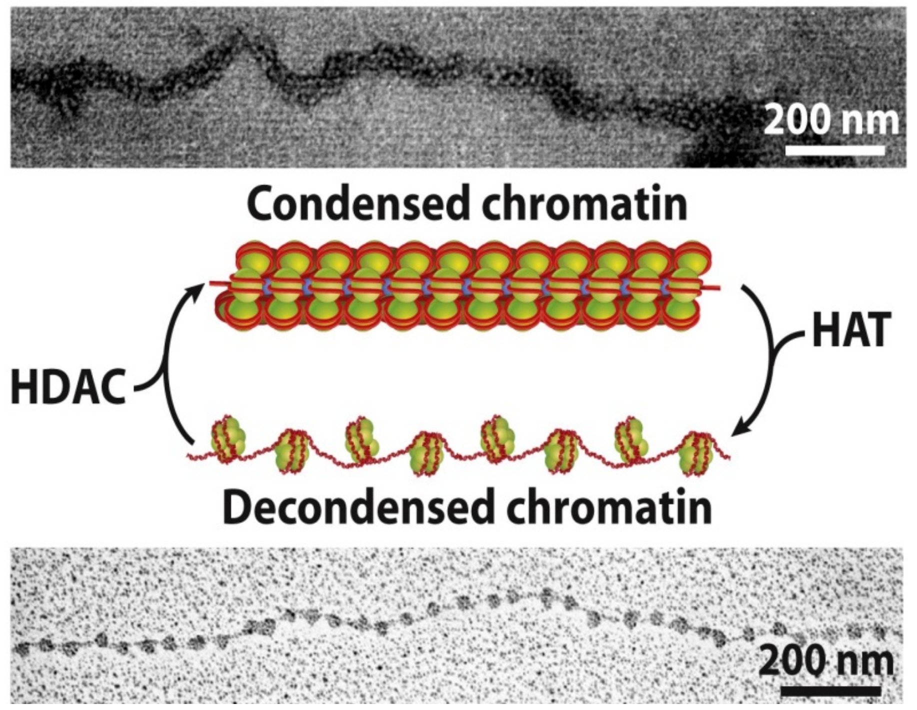


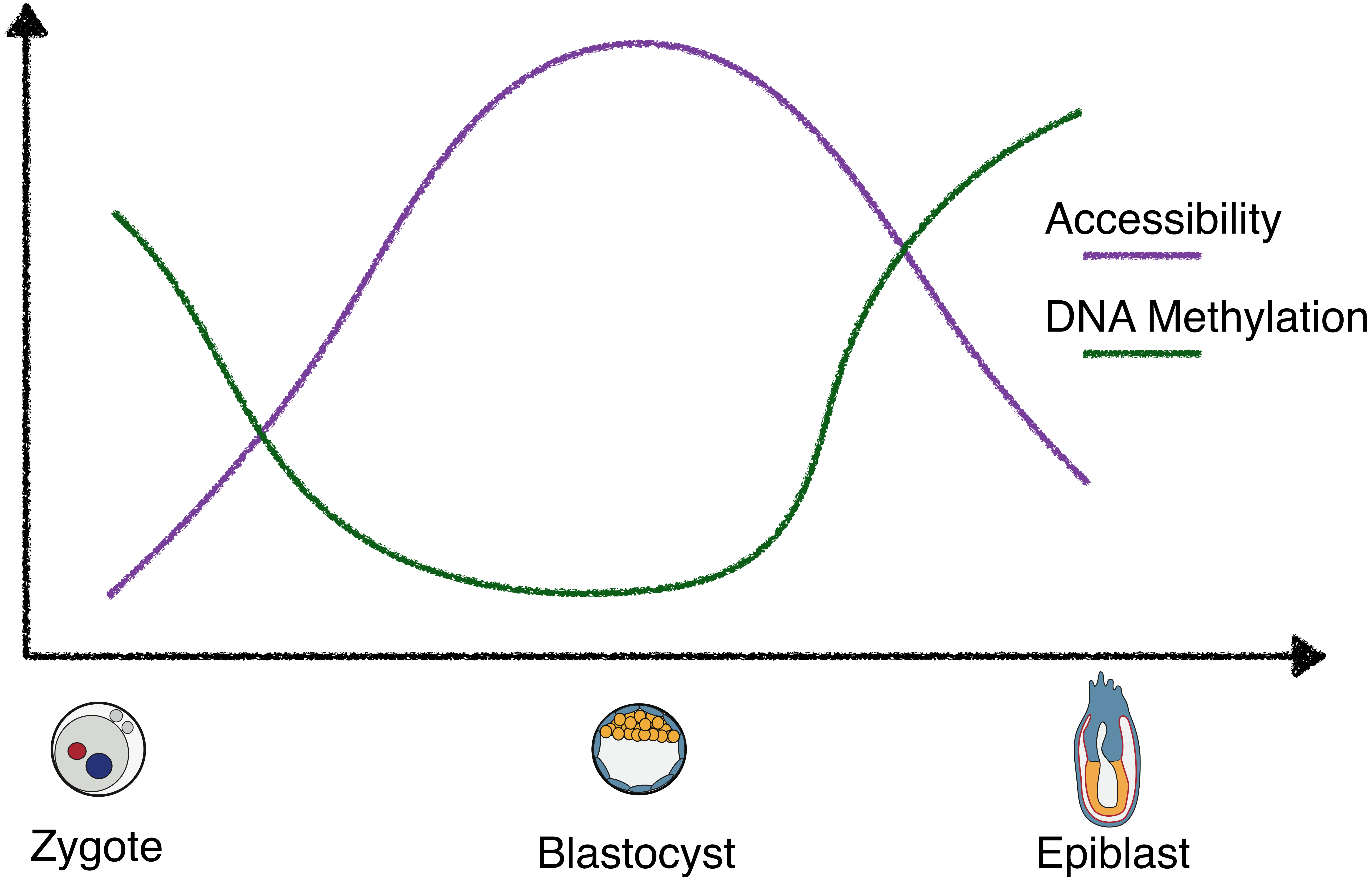
DNAse I Hypersensitivity-Seq

ATAC-Seq (Transposase mediated)

NOMe-Seq (GC methyltransferase mediated)

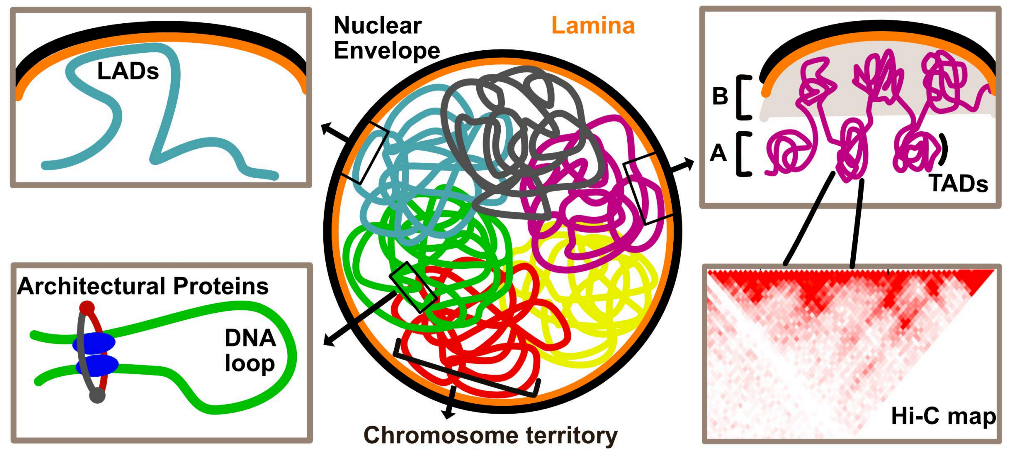
Chromatin Accessibility



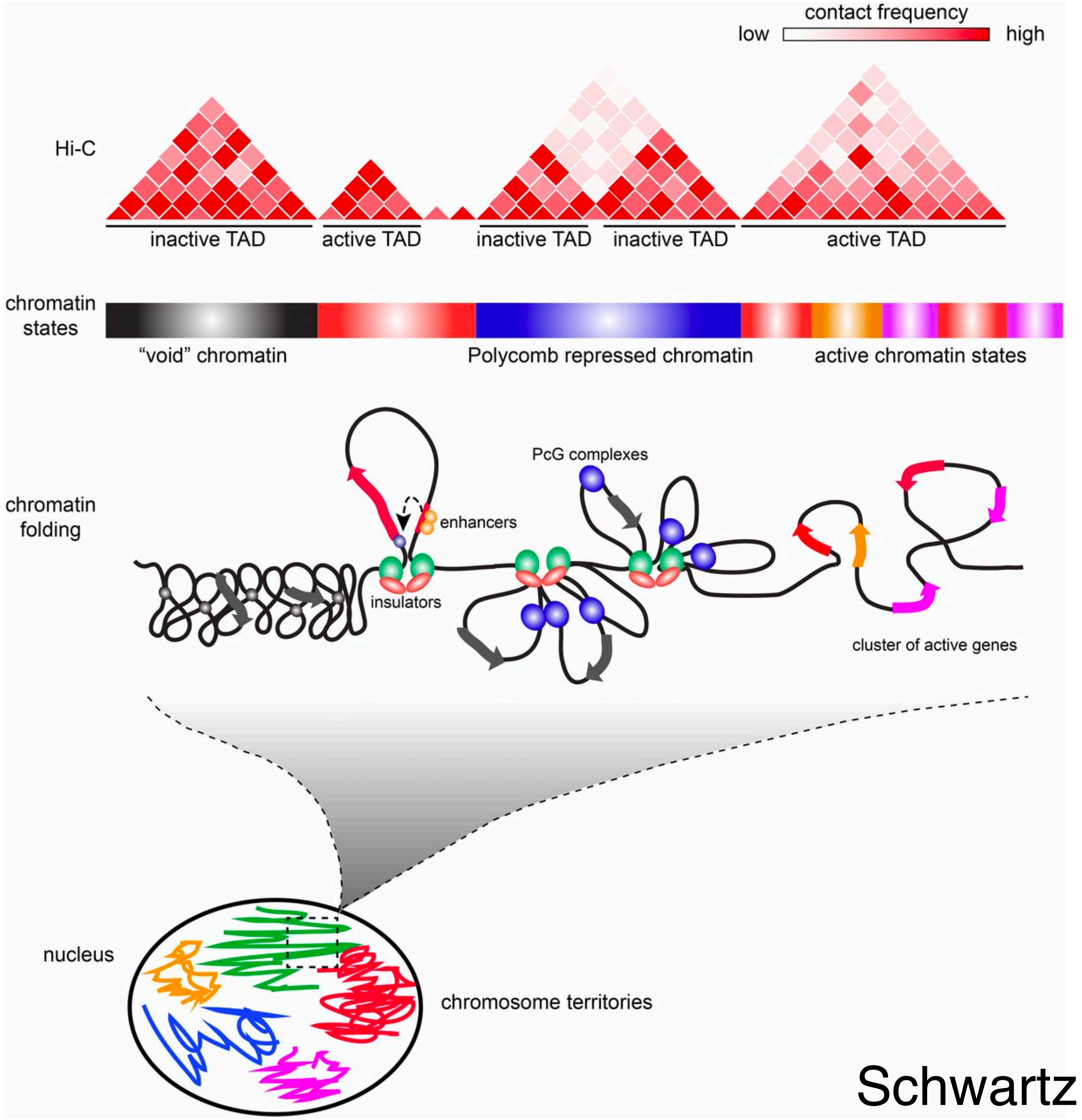


Mirror graphs

Chromatin becomes more restricted as embryo approaches lineage specification

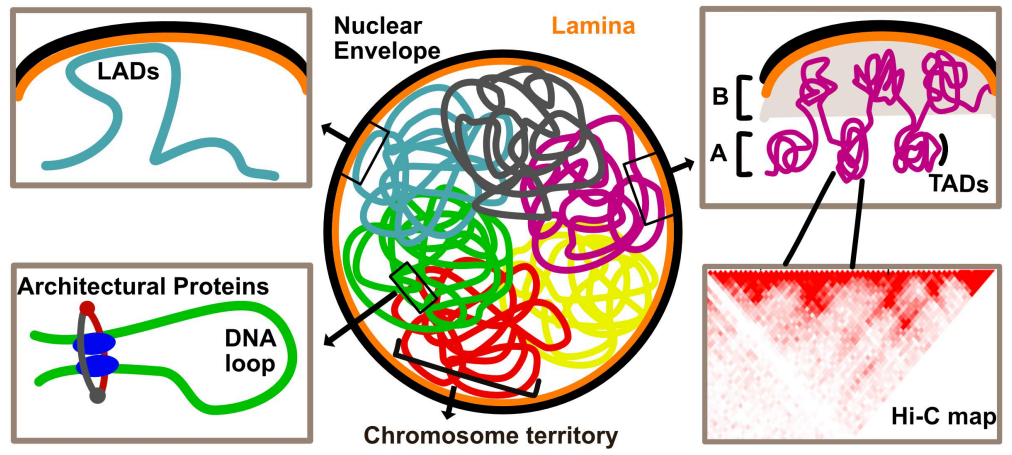


Nuclear Architecture



DNA is organized in regulatory neighborhoods called "Topologically Associated Domains (TADs)"

Schwartz and Cavalli, *Genetics*, 2017



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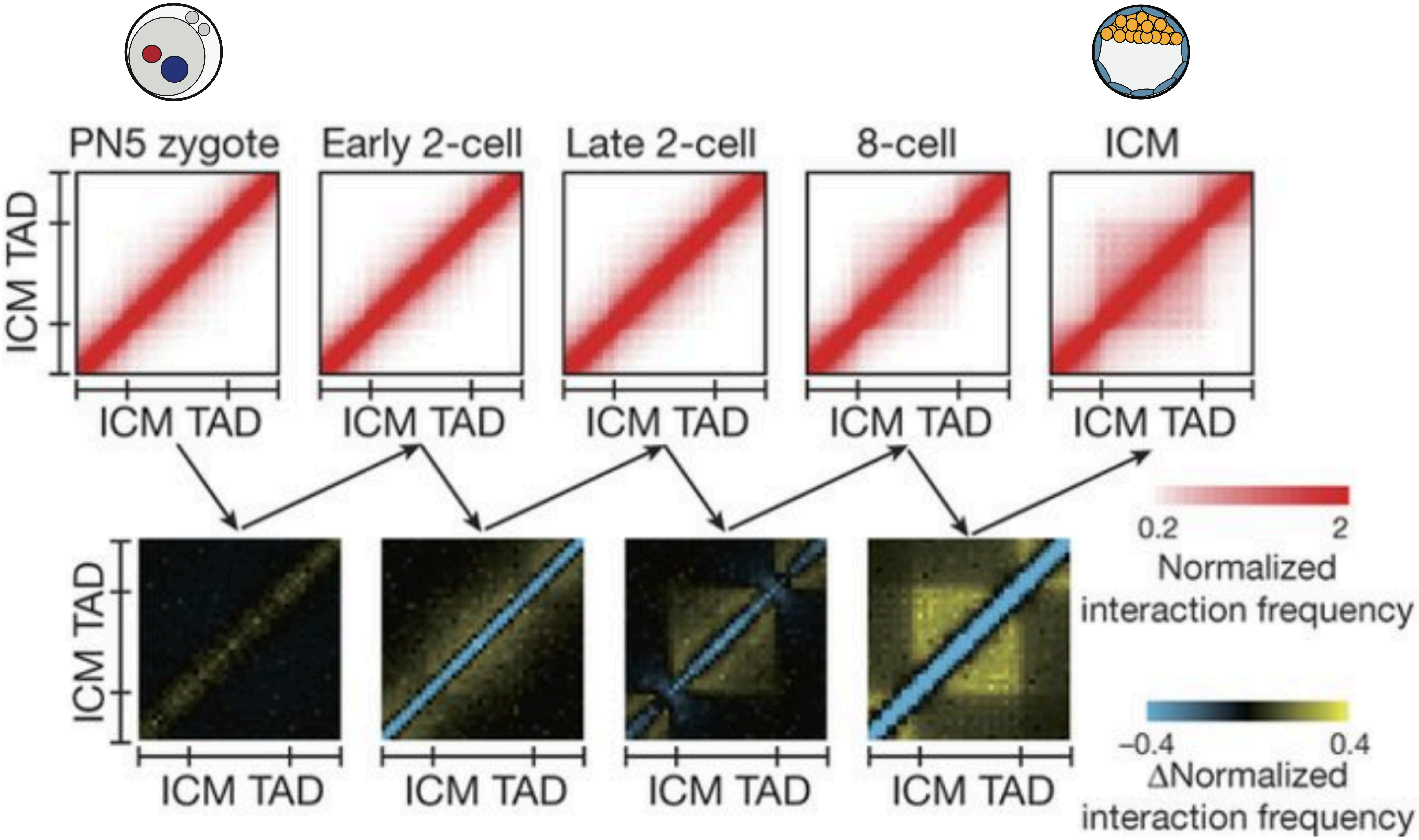
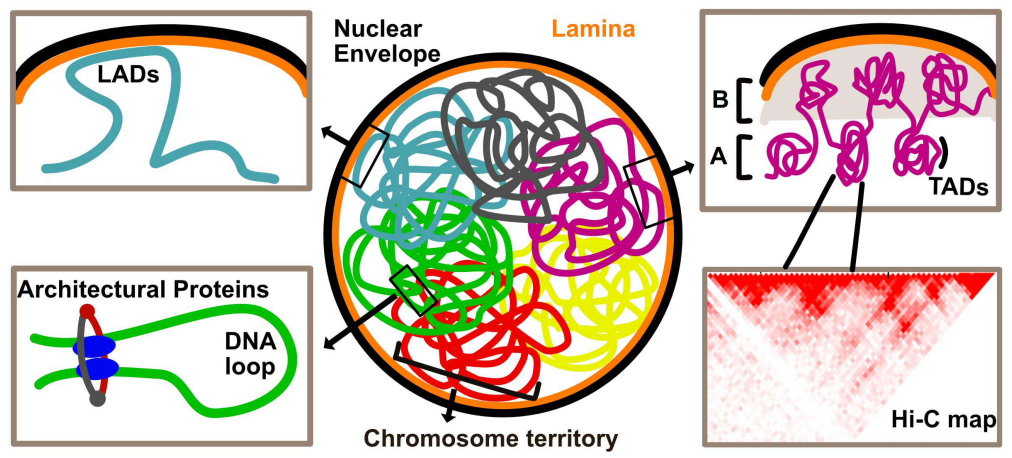


Du et al, *Nature*, 2017



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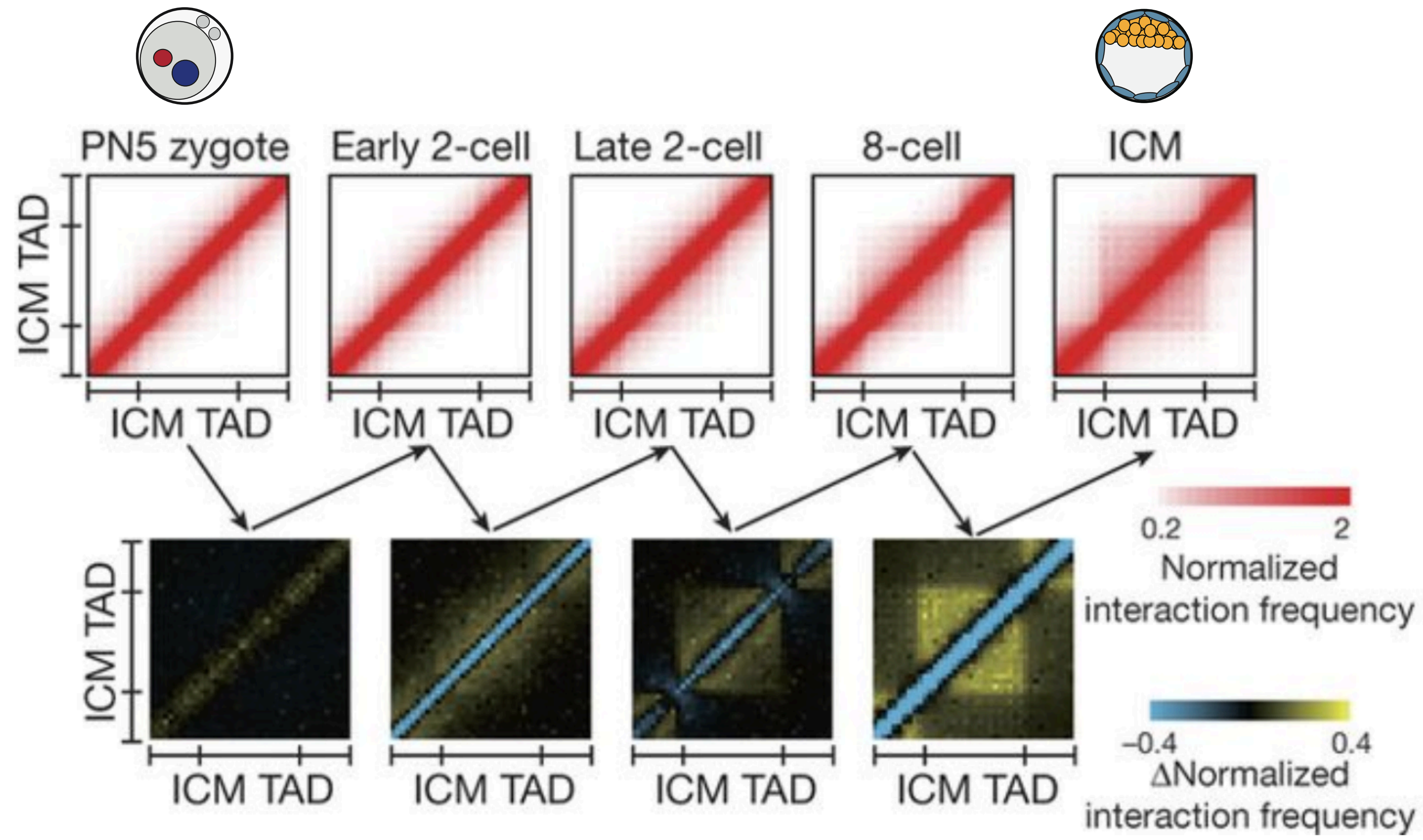
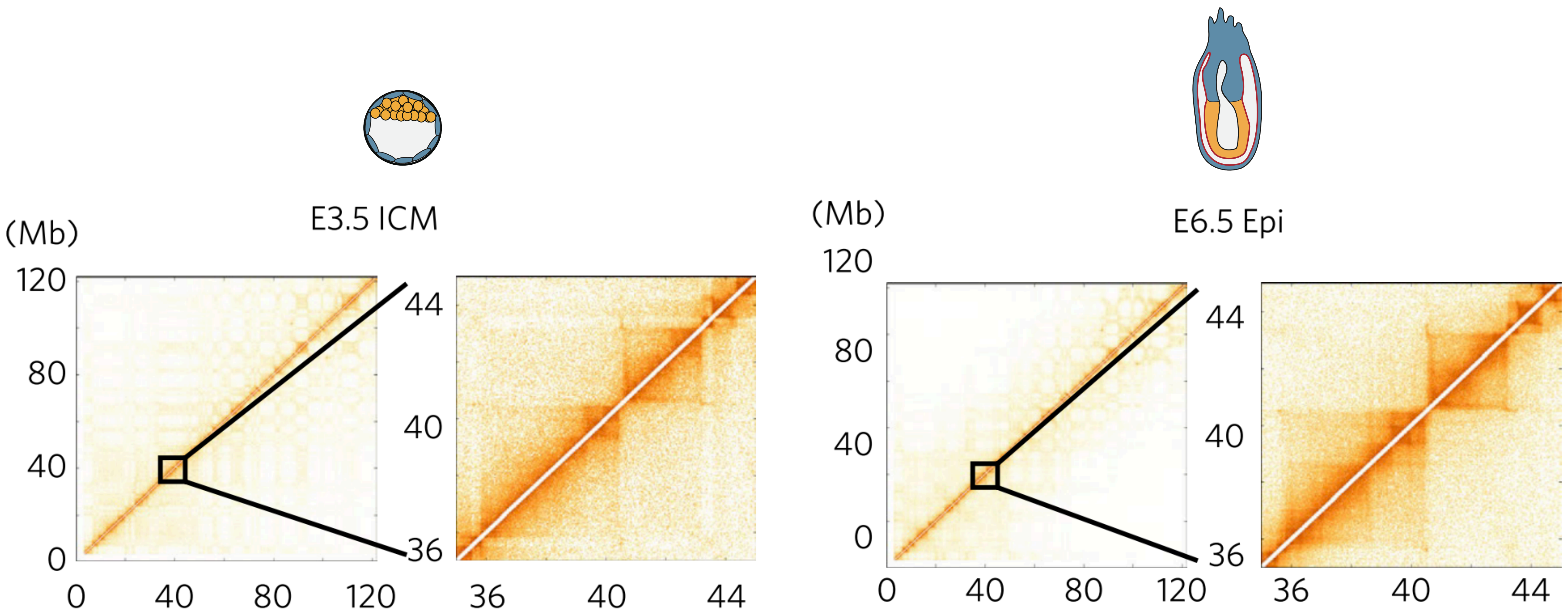


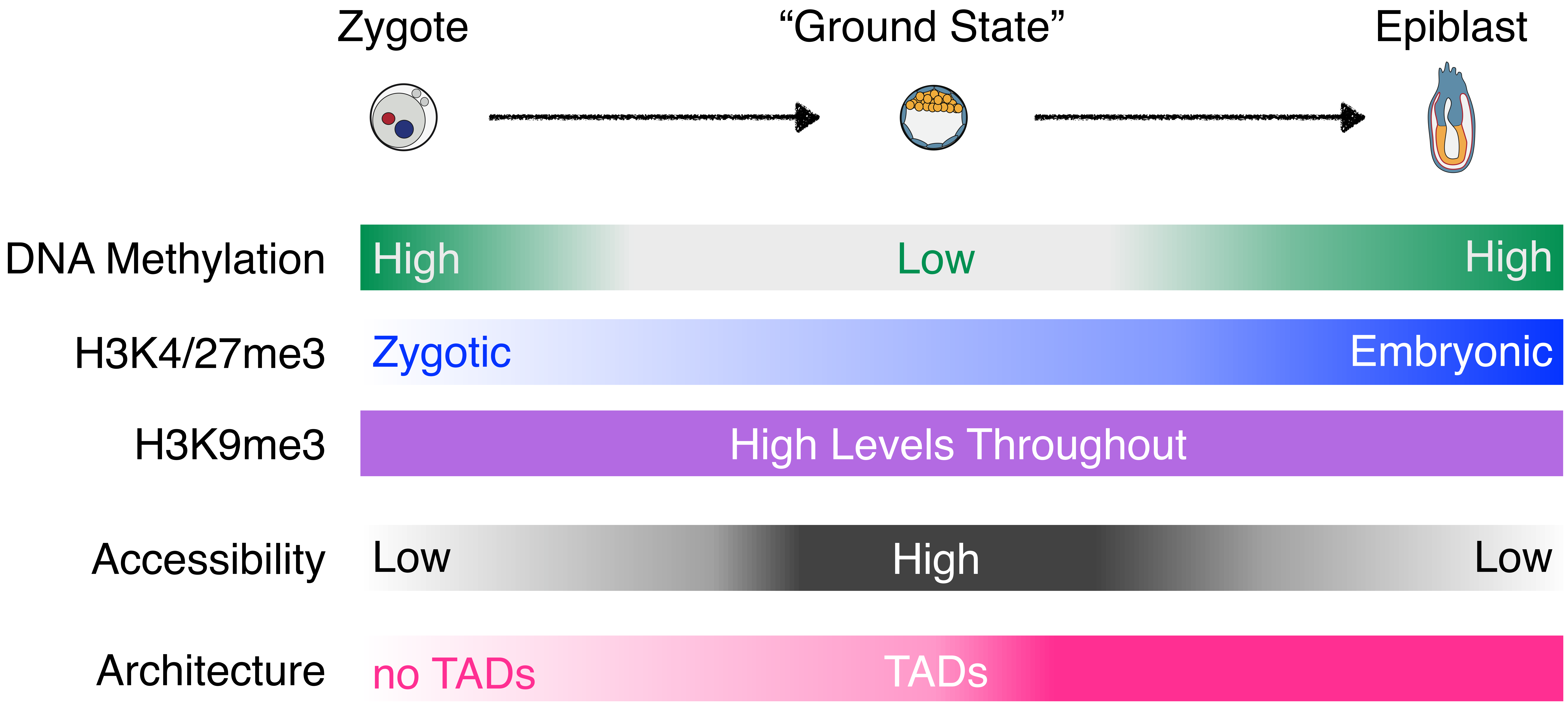


TAD structure basically established by ground-state

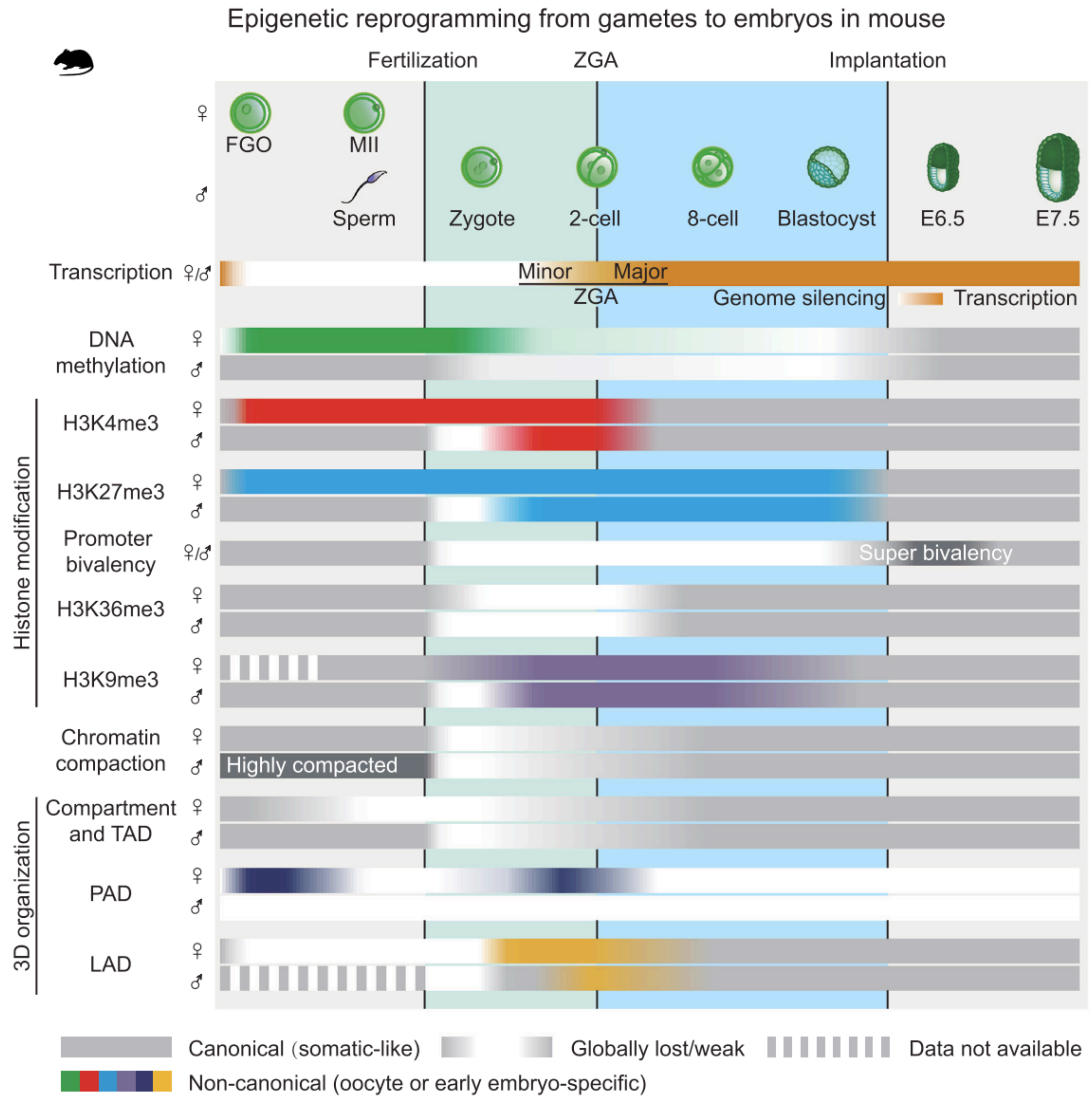
Implies DNA methylation plays minimal role in nuclear architecture at this level

Zhang et al, *Nat Gen*, 2018

Summary: Epigenetic Reprogramming



Further Reading

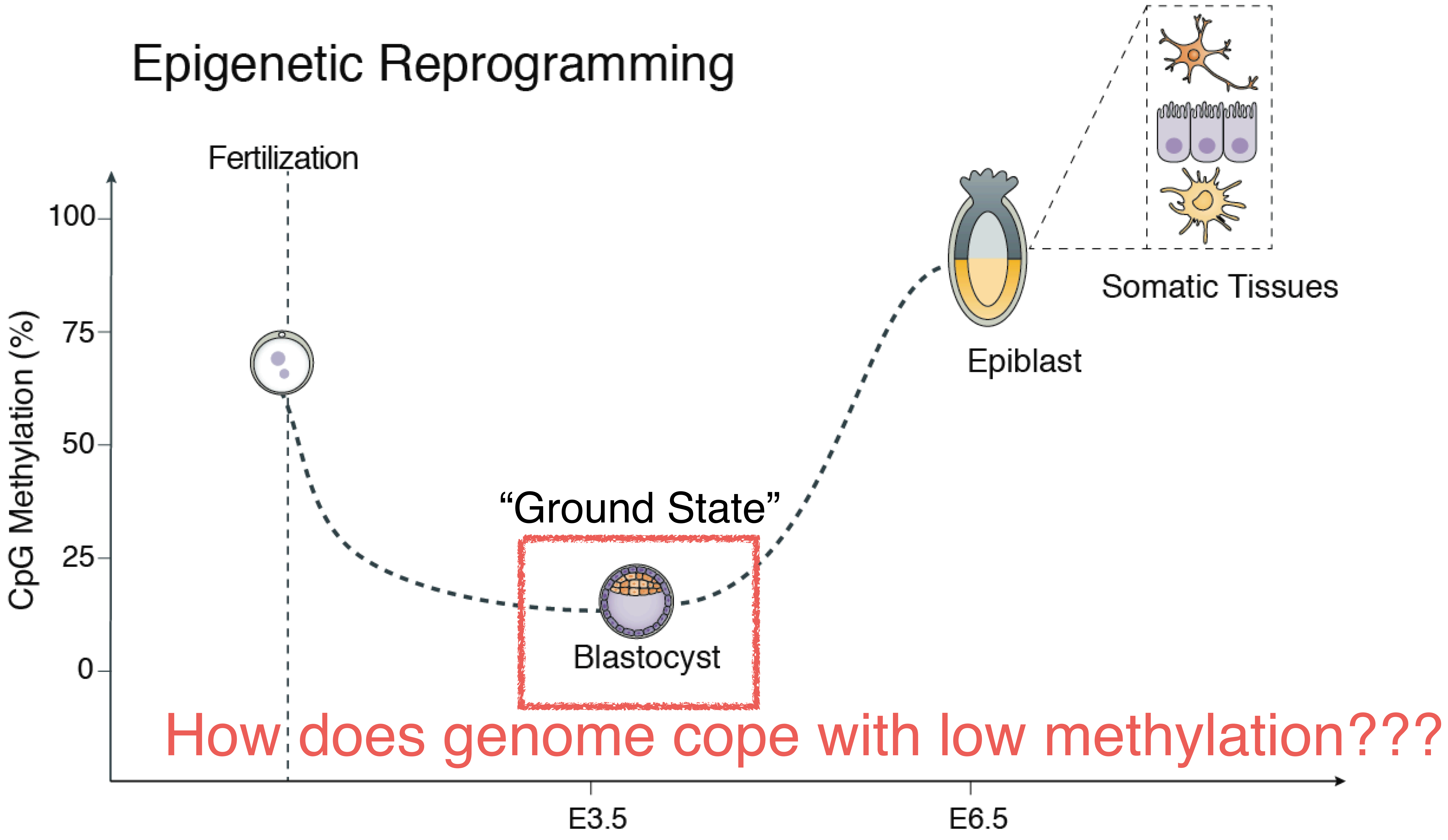


Rebooting the Epigenomes during Mammalian Early Embryogenesis (Review)

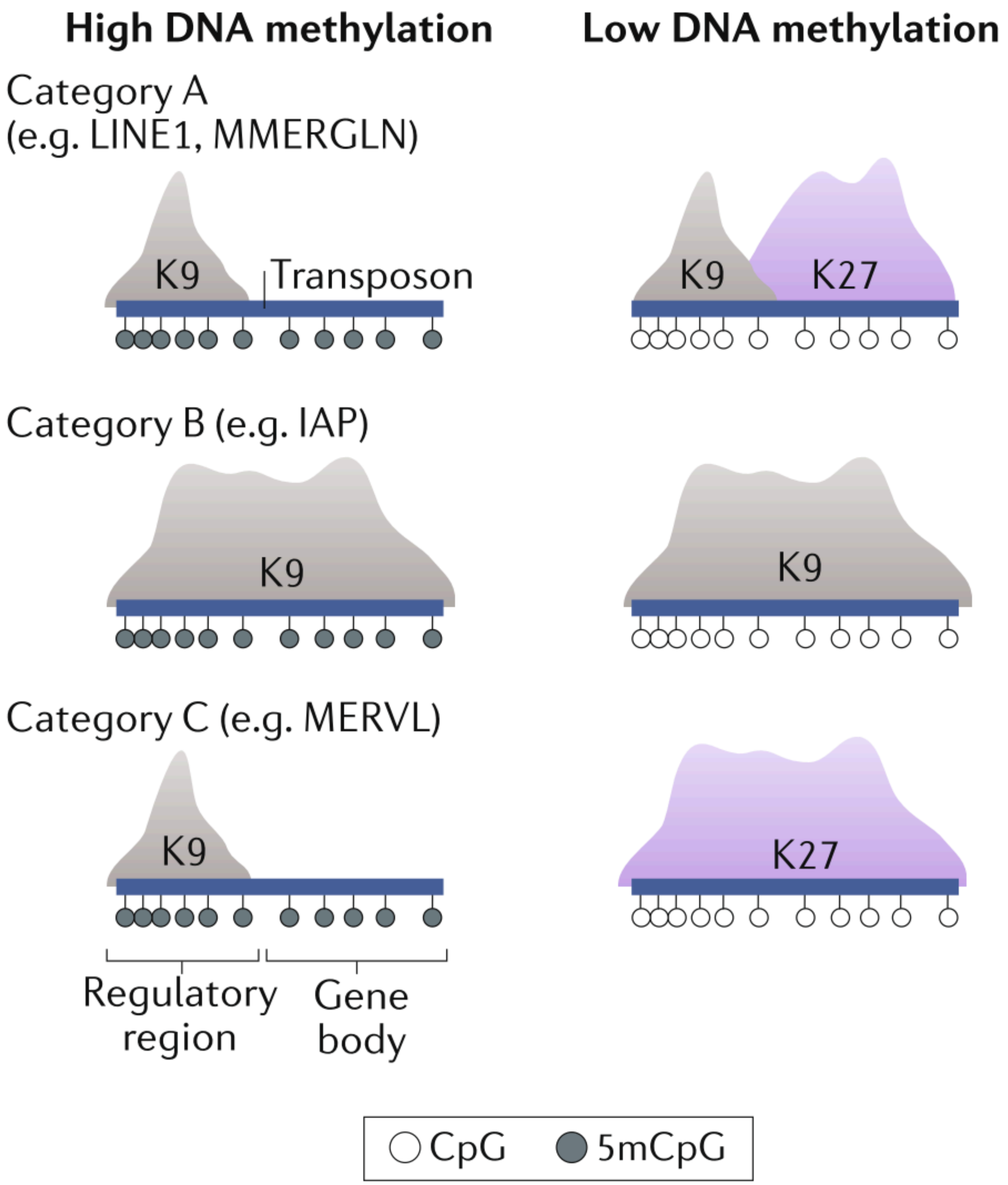
Weikun Xia and Wei Xie

Stem Cell Reports, October 2020

DNA Methylation Highly Dynamic in Embryo



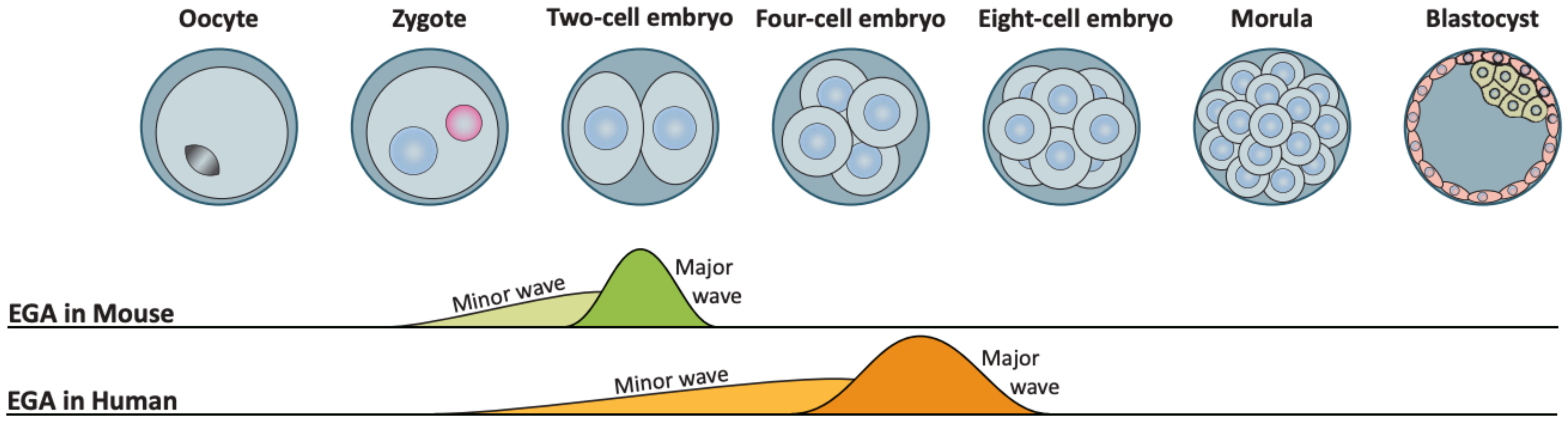
Compensatory Mechanisms



In absence of DNA methylation, chromatin pathways ensure transposon silencing

Walter et al, *eLife*, 2016

Transposon expression is important!



EGA = Embryonic Genome Activation

LINE1 Elements — Mouse

ERV3 Elements — Mouse, Human

TE expression helps "open" chromatin

ERV3 elements drive embryonic genome awakening

Is DNA methylation loss required for attaining pluripotency??

Rodriguez-Terrones and Torres-Padilla, *Trends in Genetics*, 2018

Questions We Will Address Today

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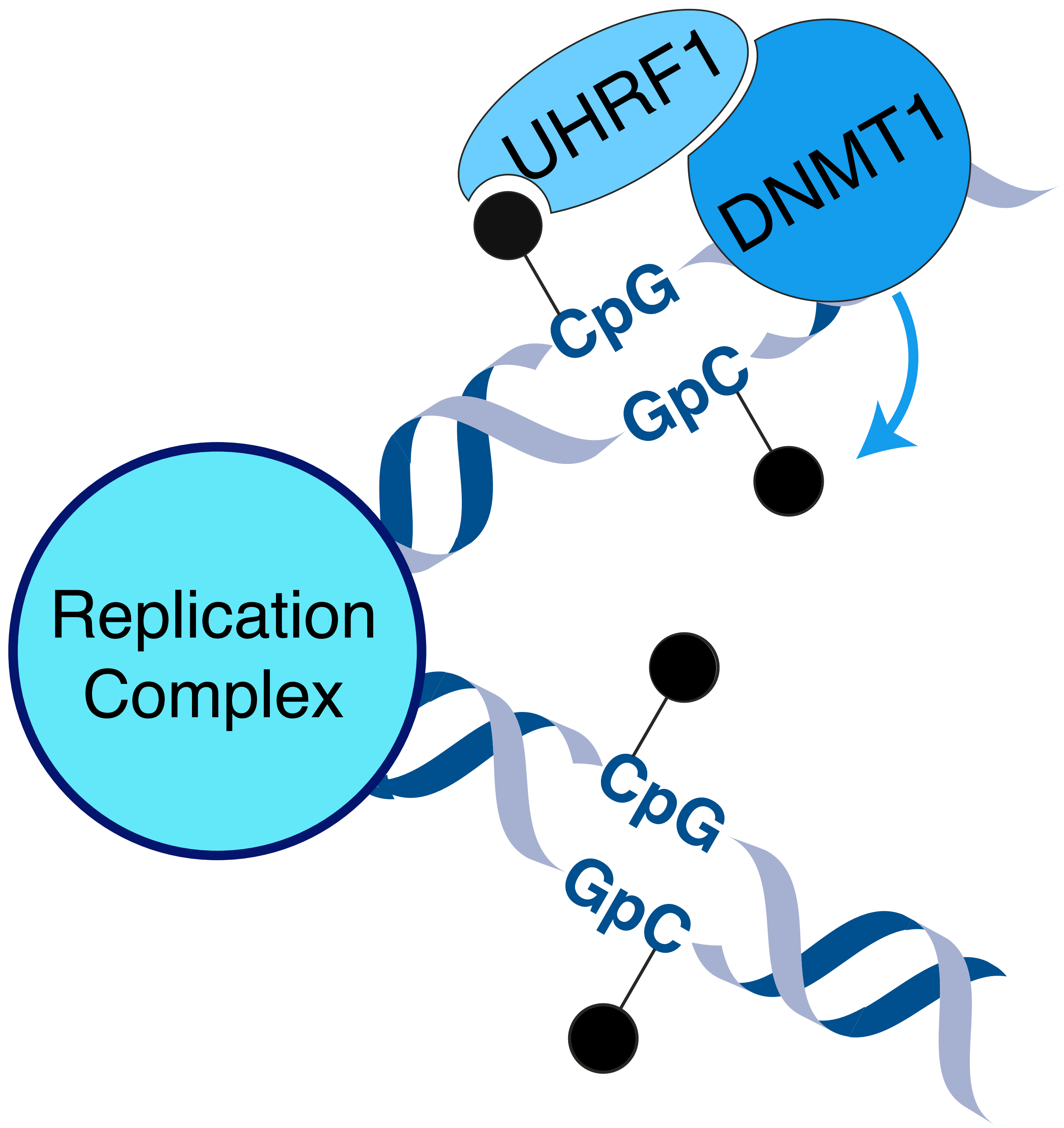
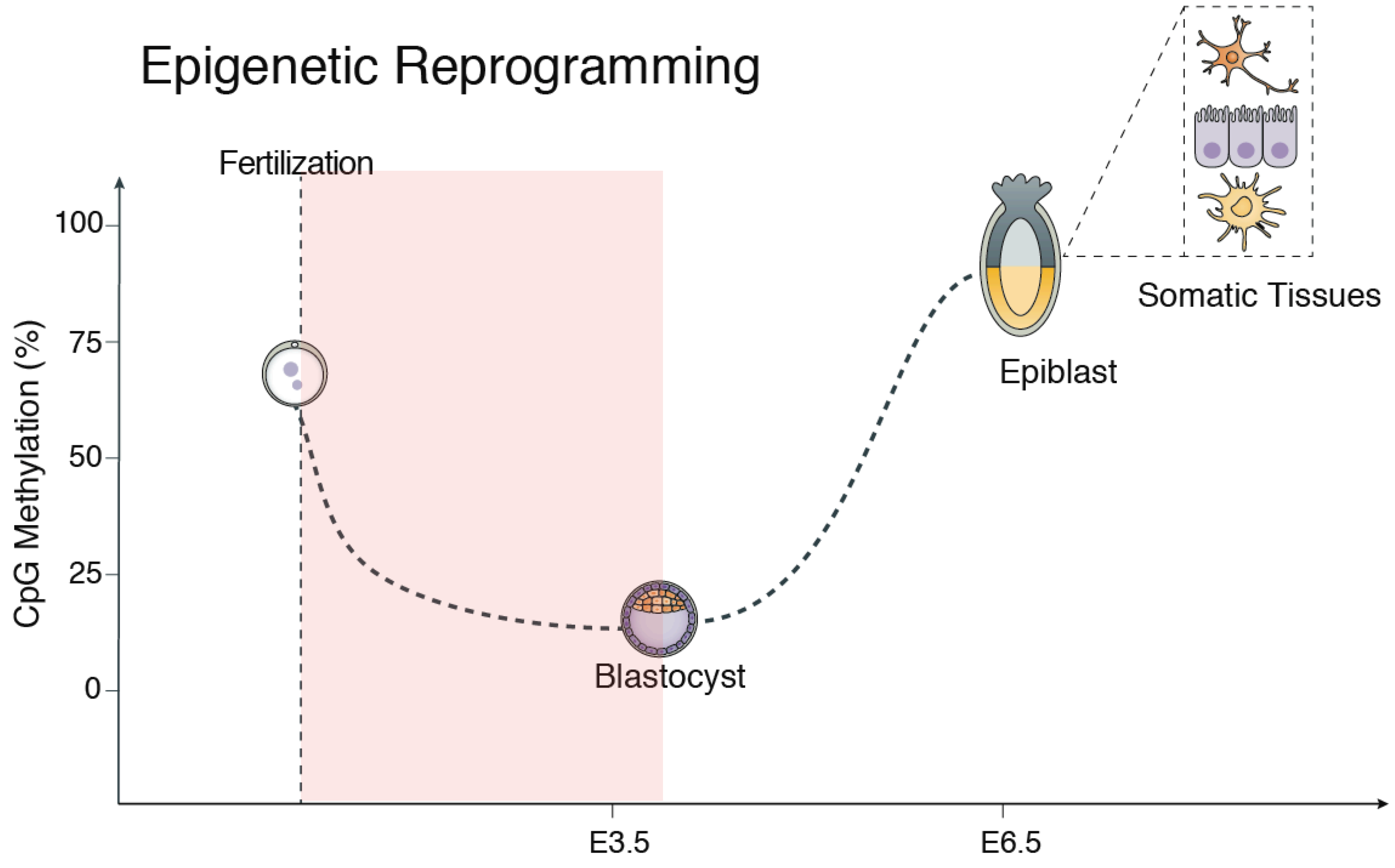
II. How does epigenetic reprogramming occur?

III. Can any regions of the genome escape reprogramming?

IV. **Why** does epigenetic reprogramming occur????

Mechanism for Reprogramming



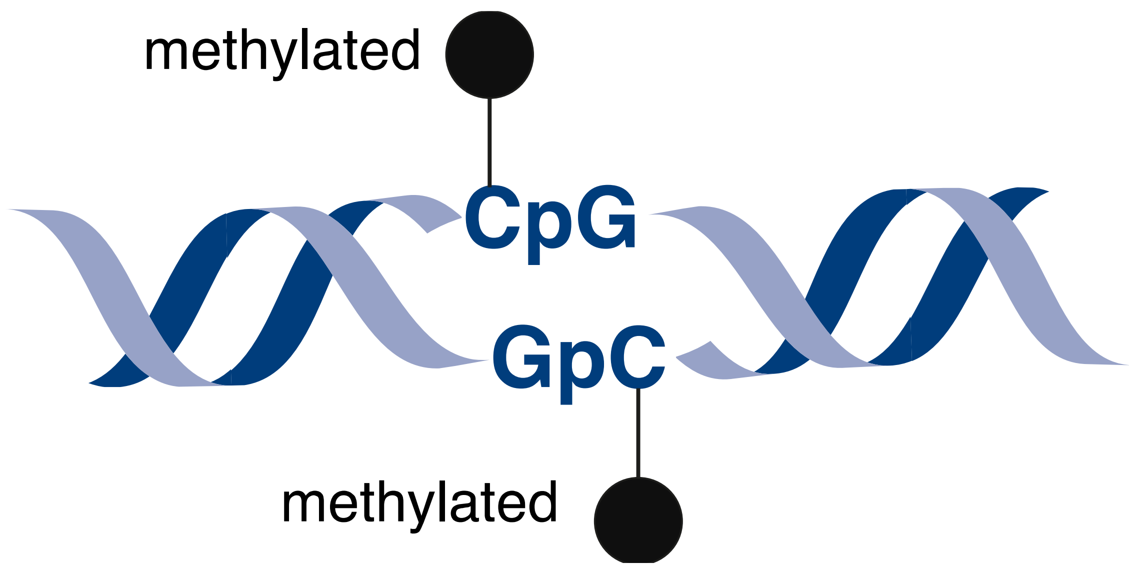


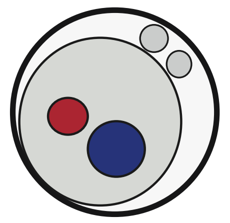
Stable maintenance mechanism

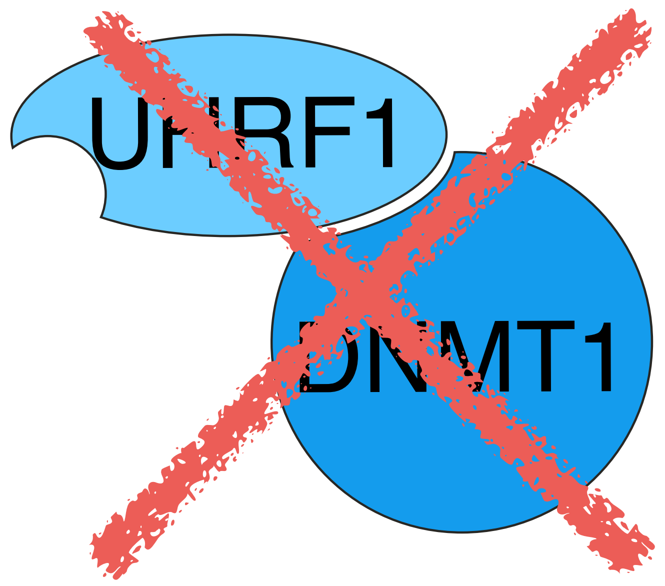
Rapid loss of DNA methylation

How??

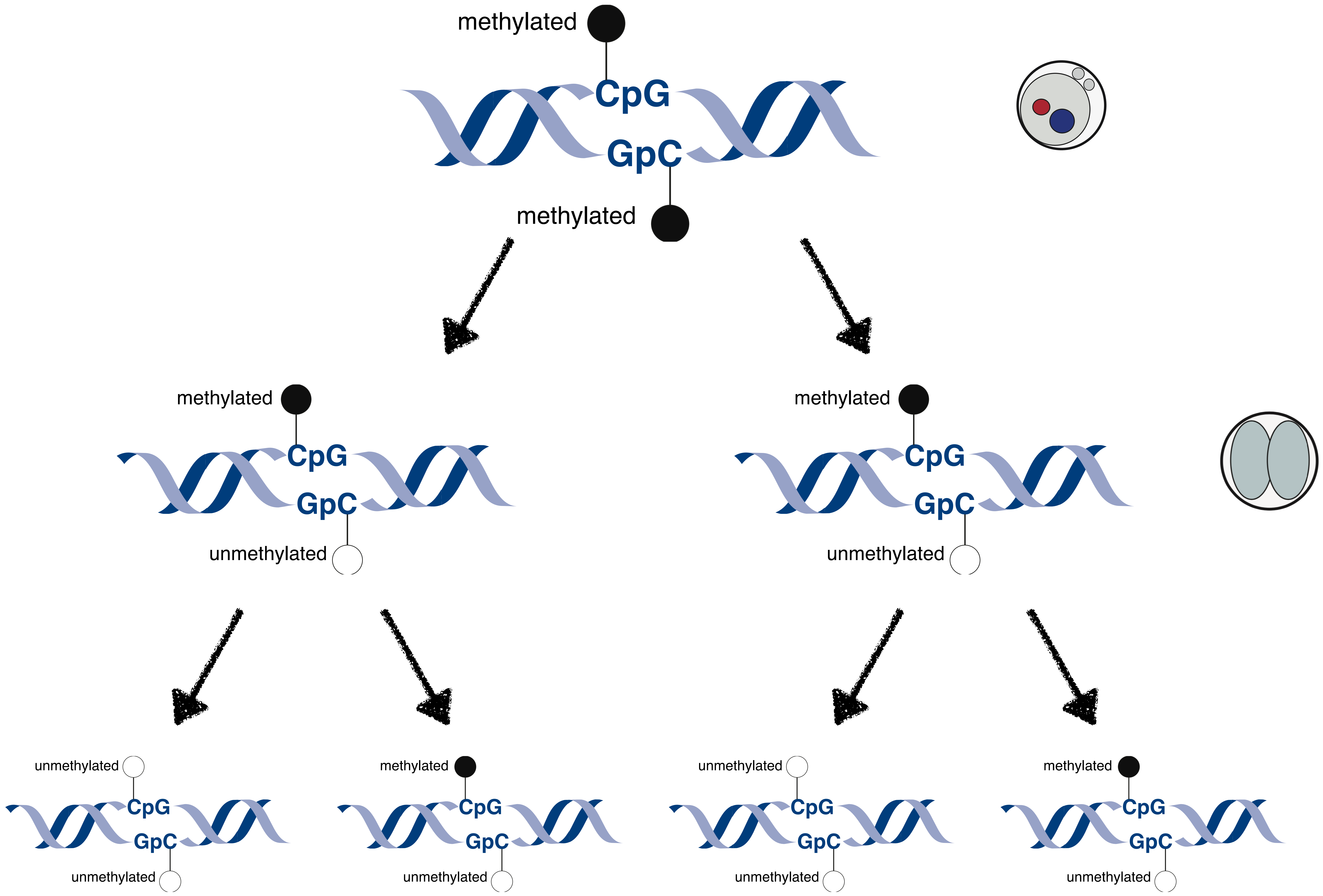
Passive Dilution

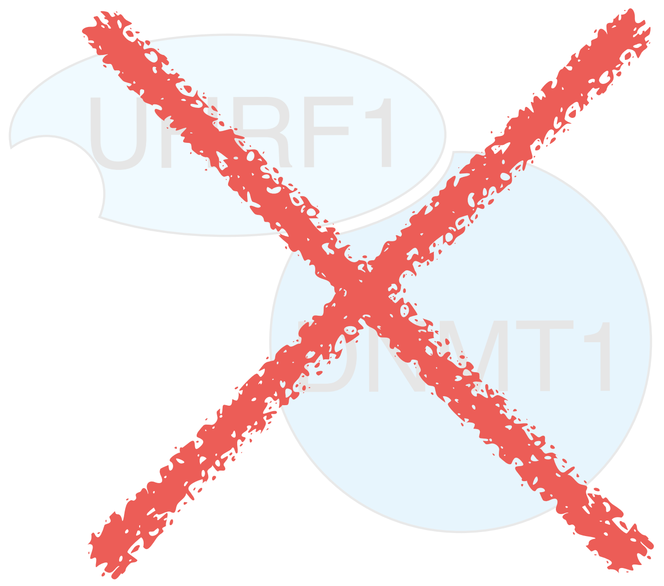


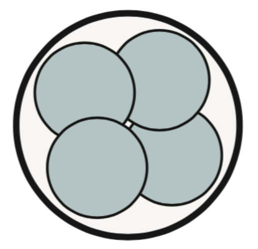




Passive Dilution

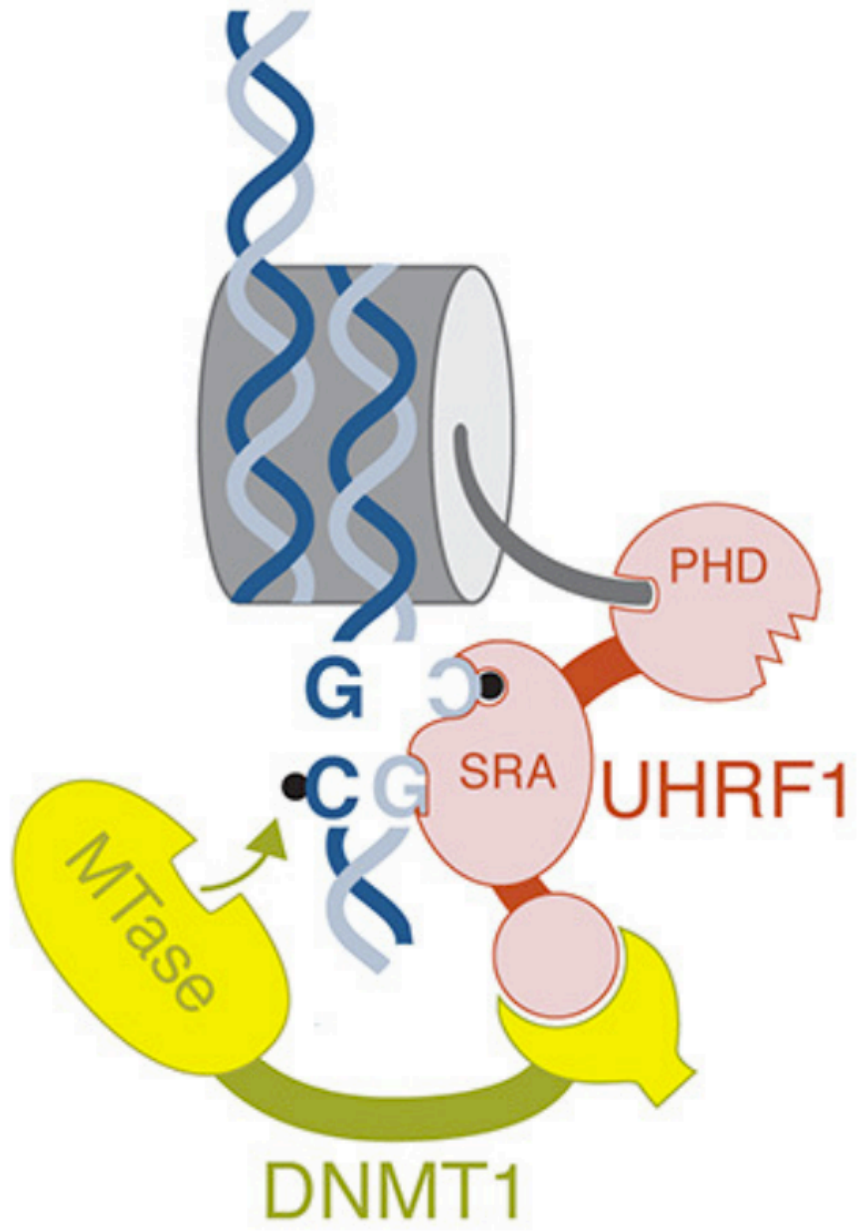






Exponential loss of DNA methylation

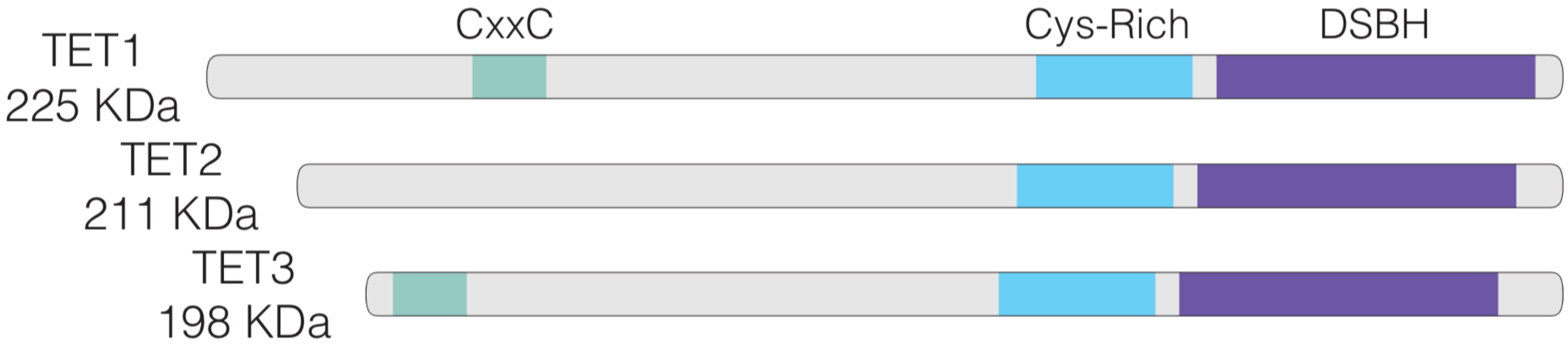
Passive Dilution



○ unmethylated CpG

● methylated CpG

Active Demethylation

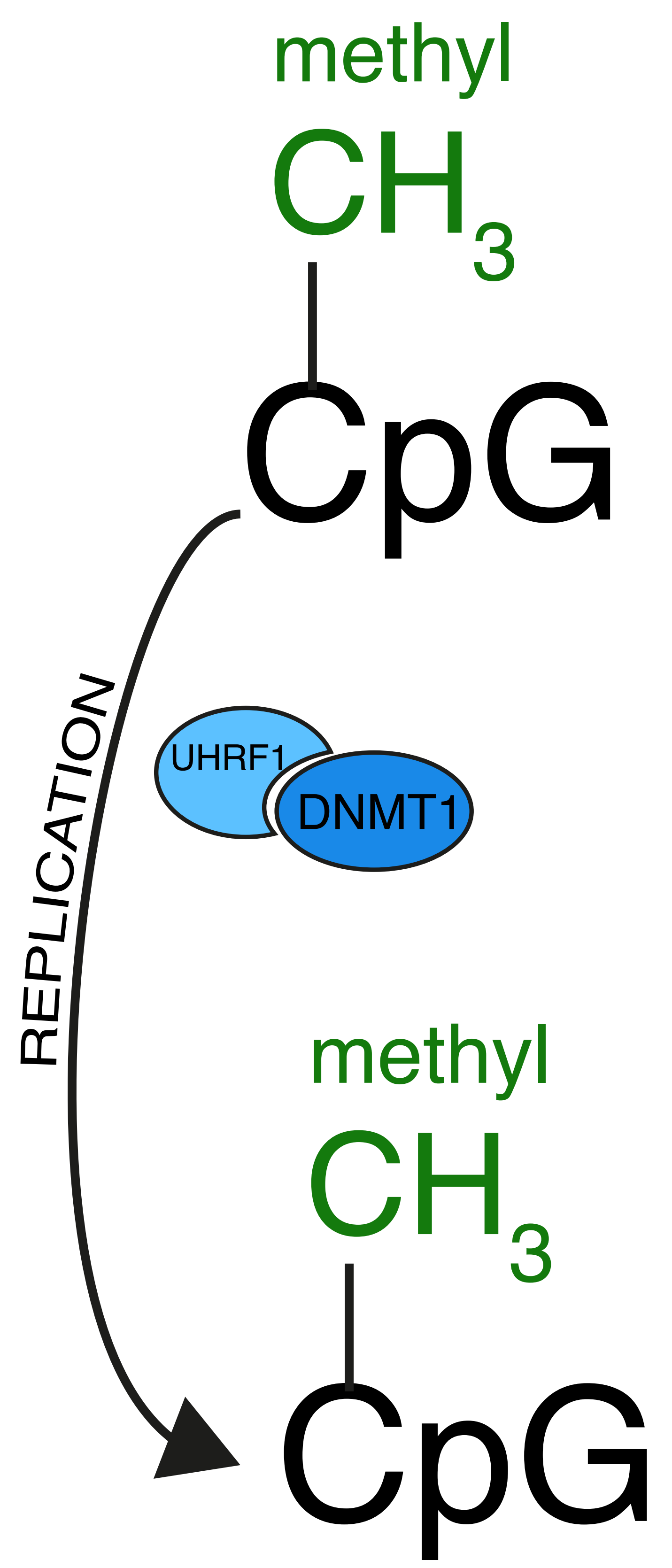


Ten-eleven translocation enzymes

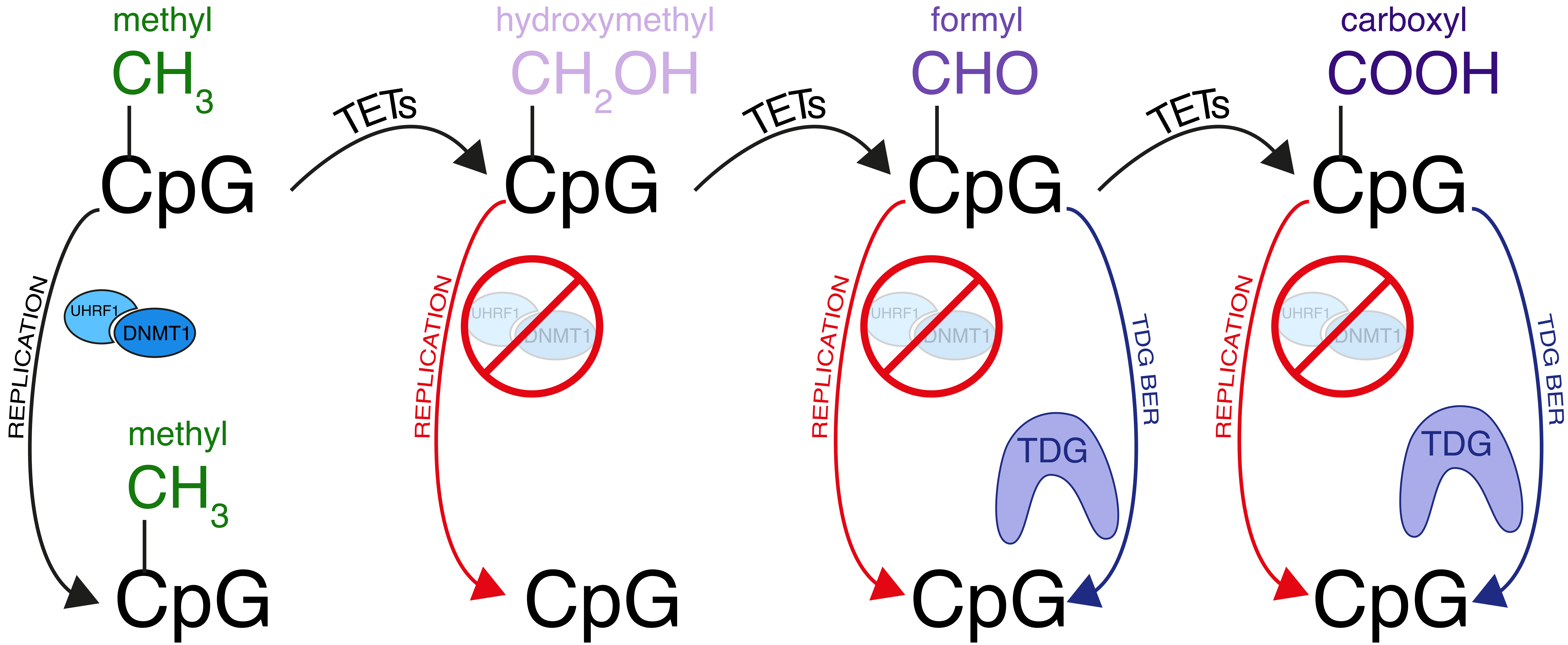
CxxC domain binds to unmethylated CpG rich regions

Cysteine-Rich and **Double stranded β-helix** domains confer catalytic activity

Active Demethylation



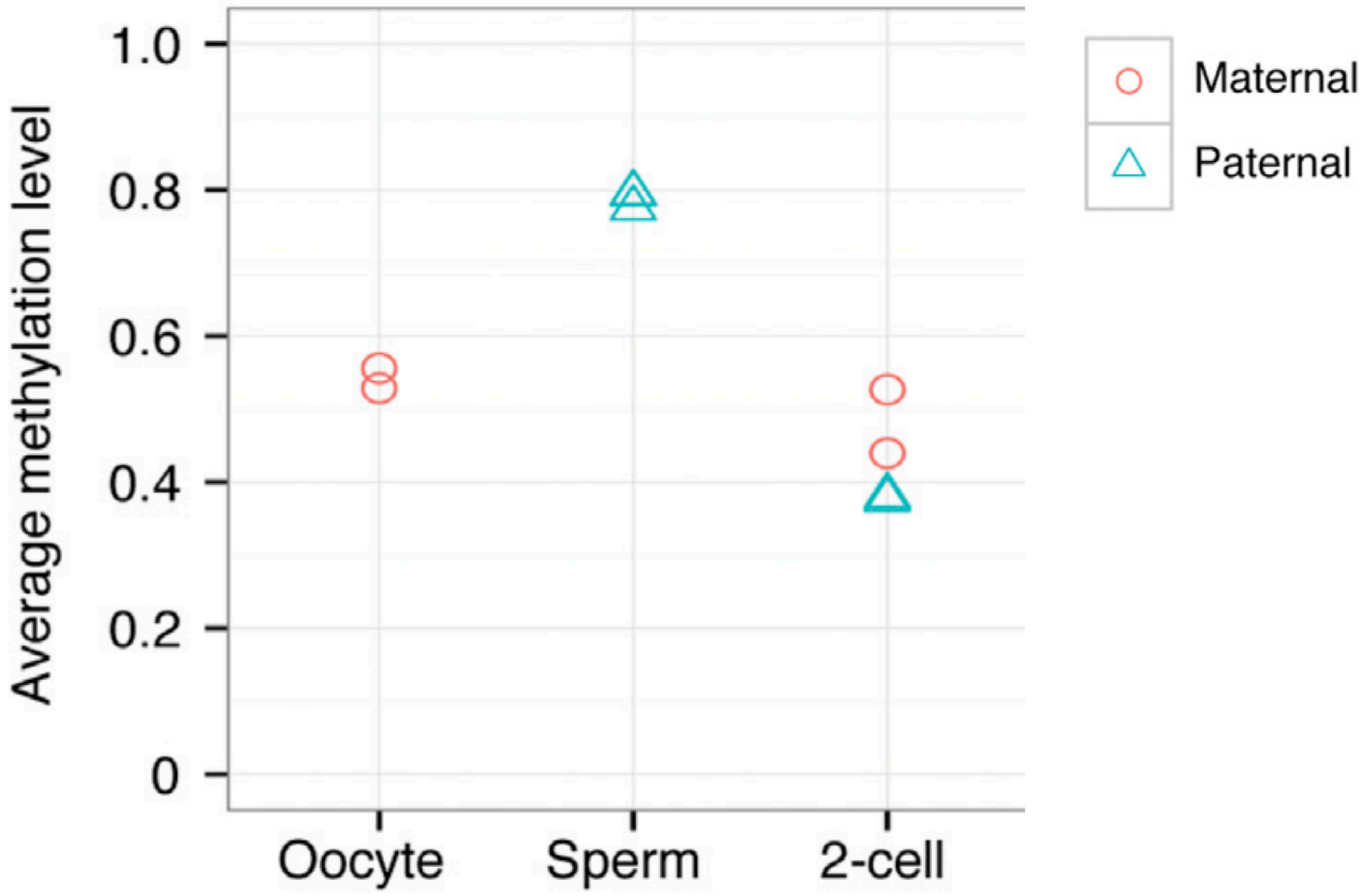
Active Demethylation



Progressive oxidation leads to demethylation:

1) Inhibiting maintenance and 2) base excision repair

Asymmetric Demethylation



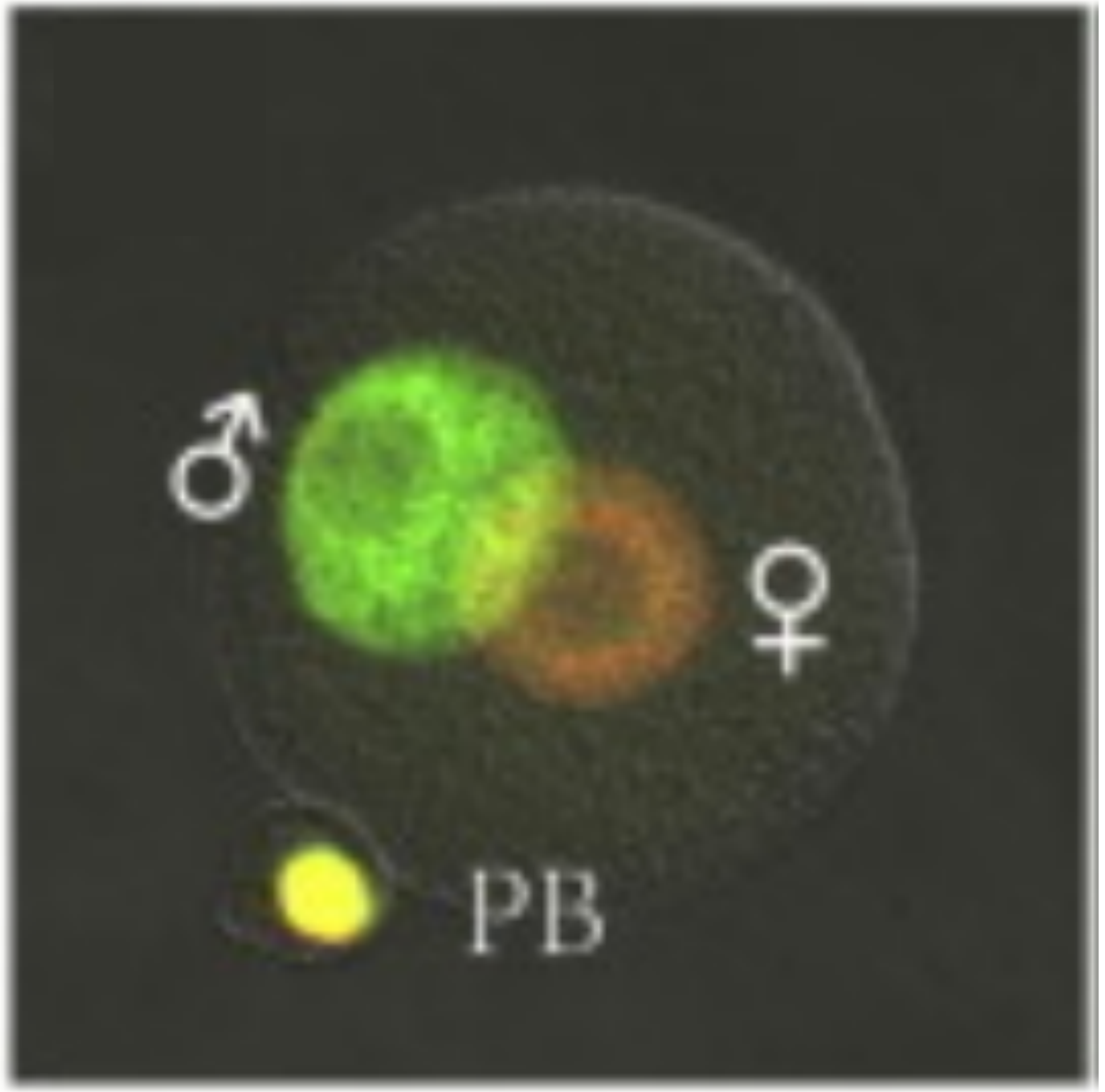
Wang et al., *Cell* 2014

Sperm arrives more methylated than oocyte

At two still stage, paternal genome is **less** methylated than maternal

Asymmetric Demethylation

Zygote



Liu et al., *Cell Reports*, 2014

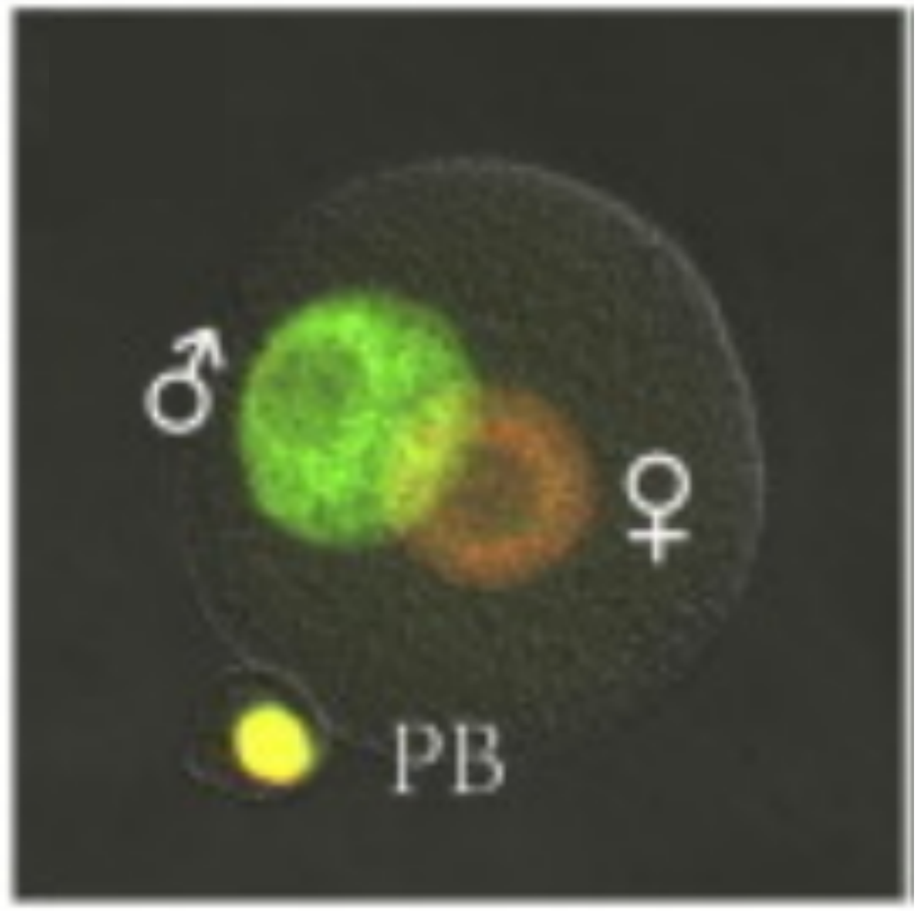
Red = DNA methylation
Green = DNA hydroxymethylation

Model:

Paternal genome is actively demethylated by TET3

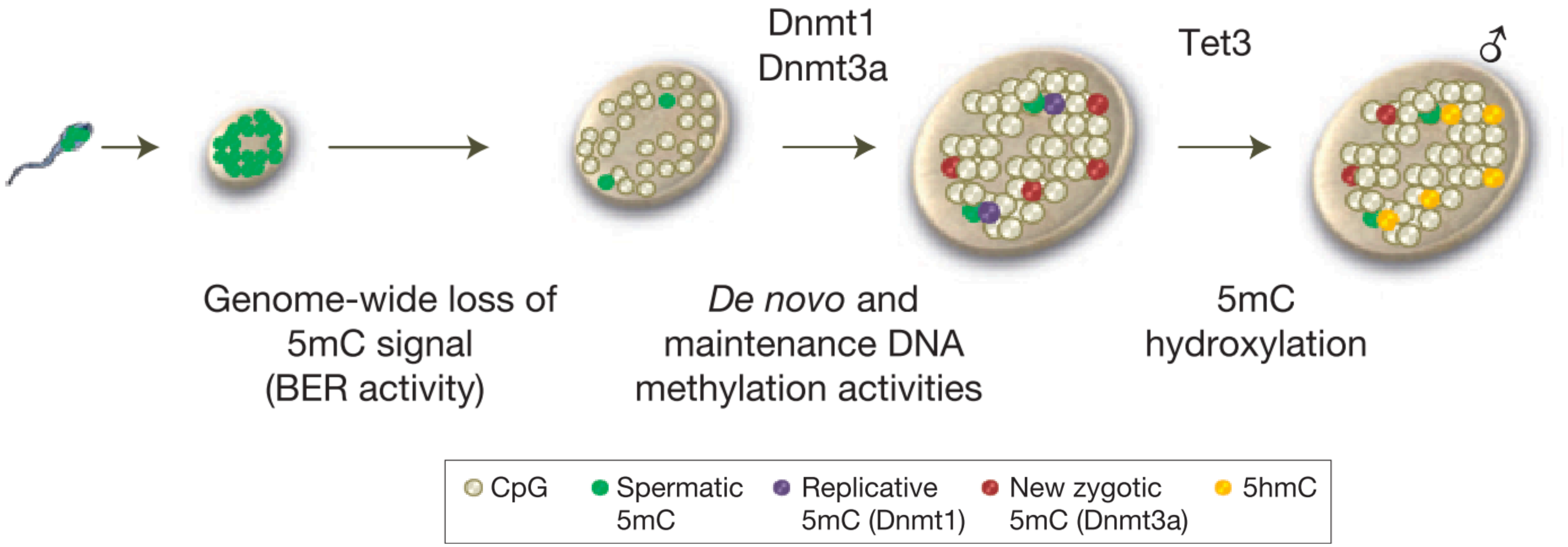
Maternal genome is passively demethylated

Asymmetric Demethylation

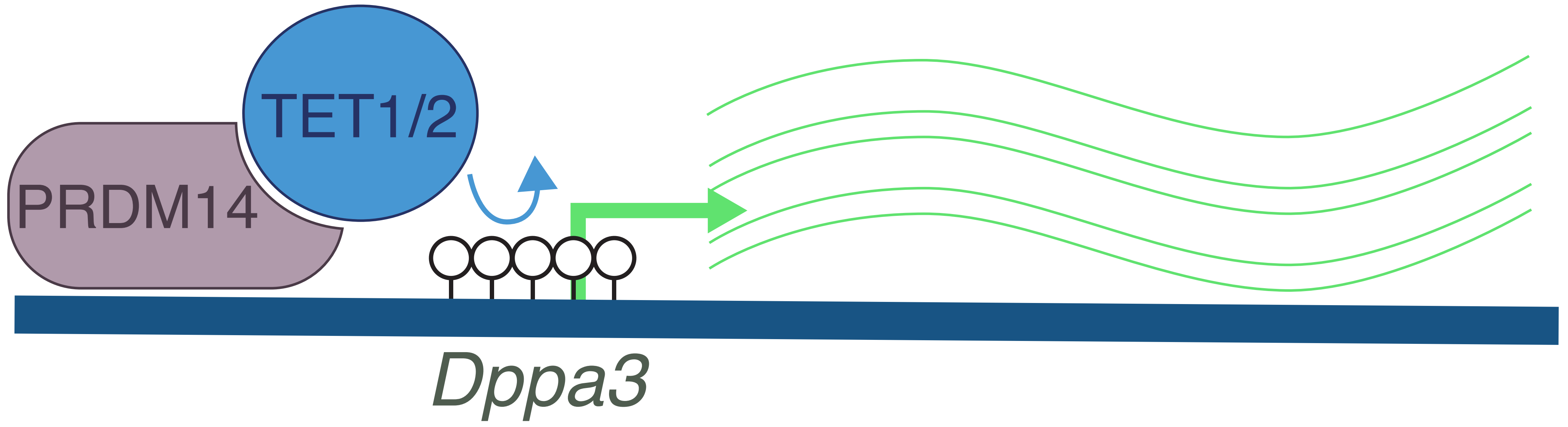


Controversial field:

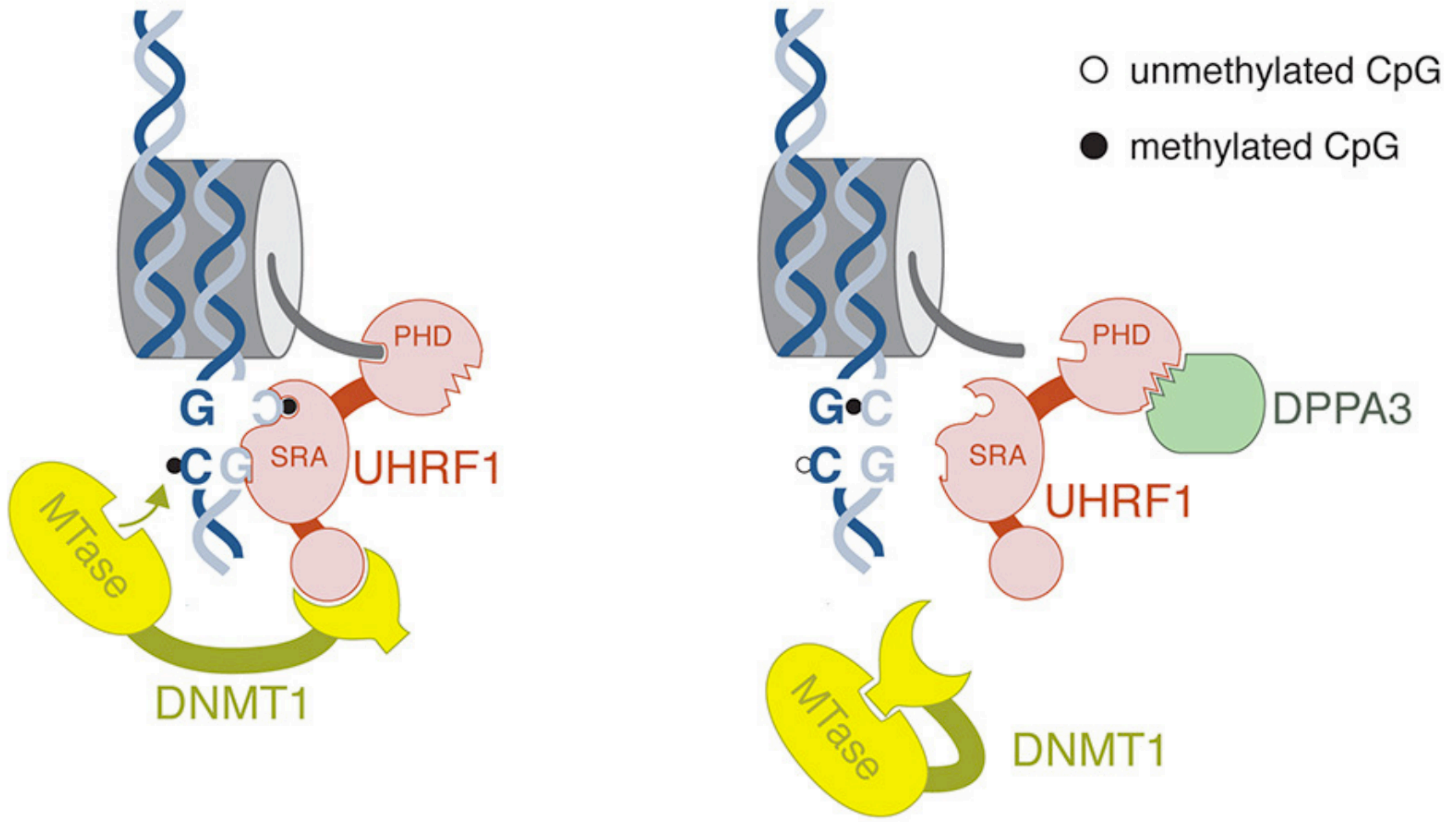
Hajkova lab claims that paternal DNA methylation is lost independently of TET3



Active Demethylation



TET proteins required to demethylate *Dppa3* gene

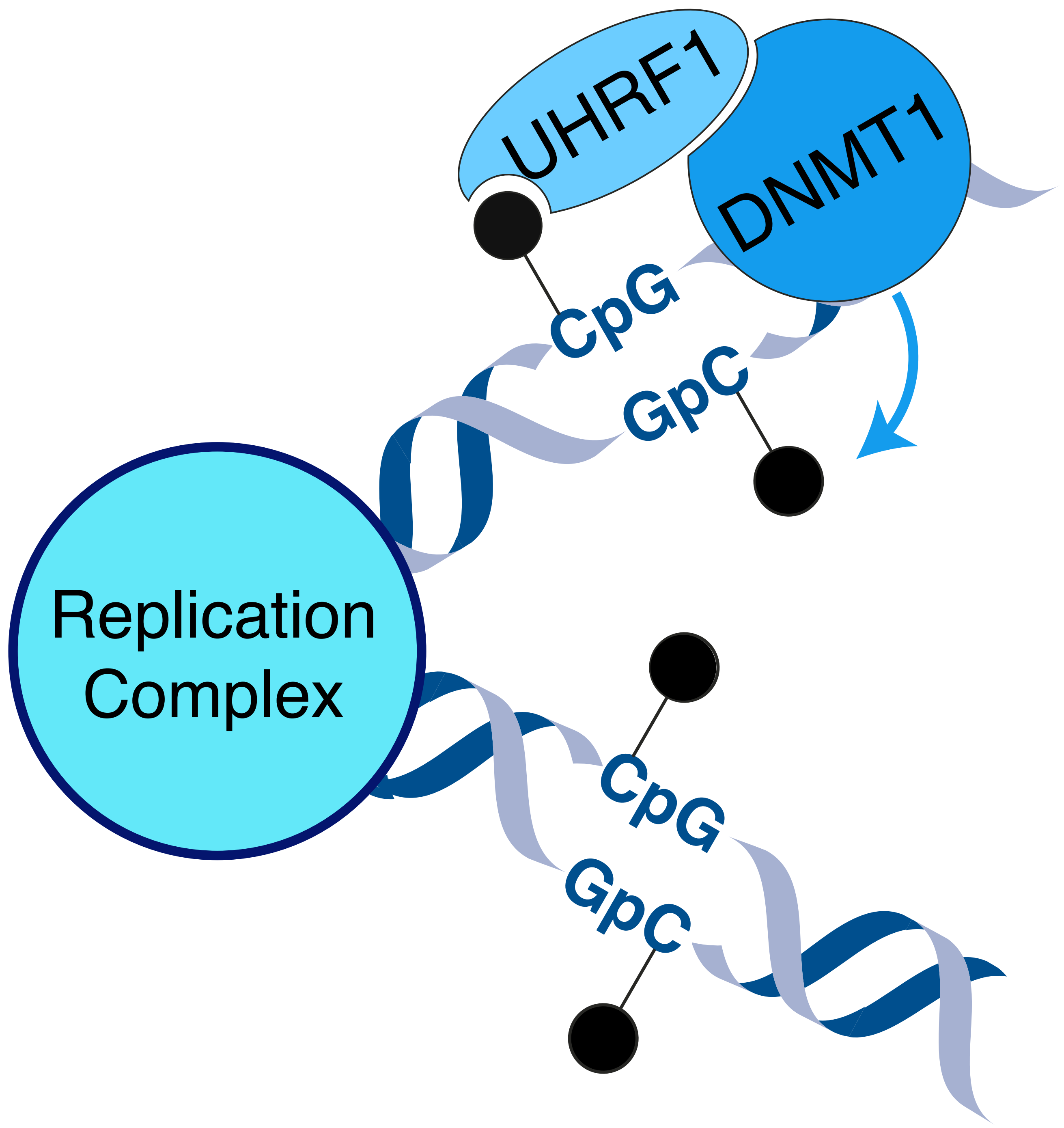


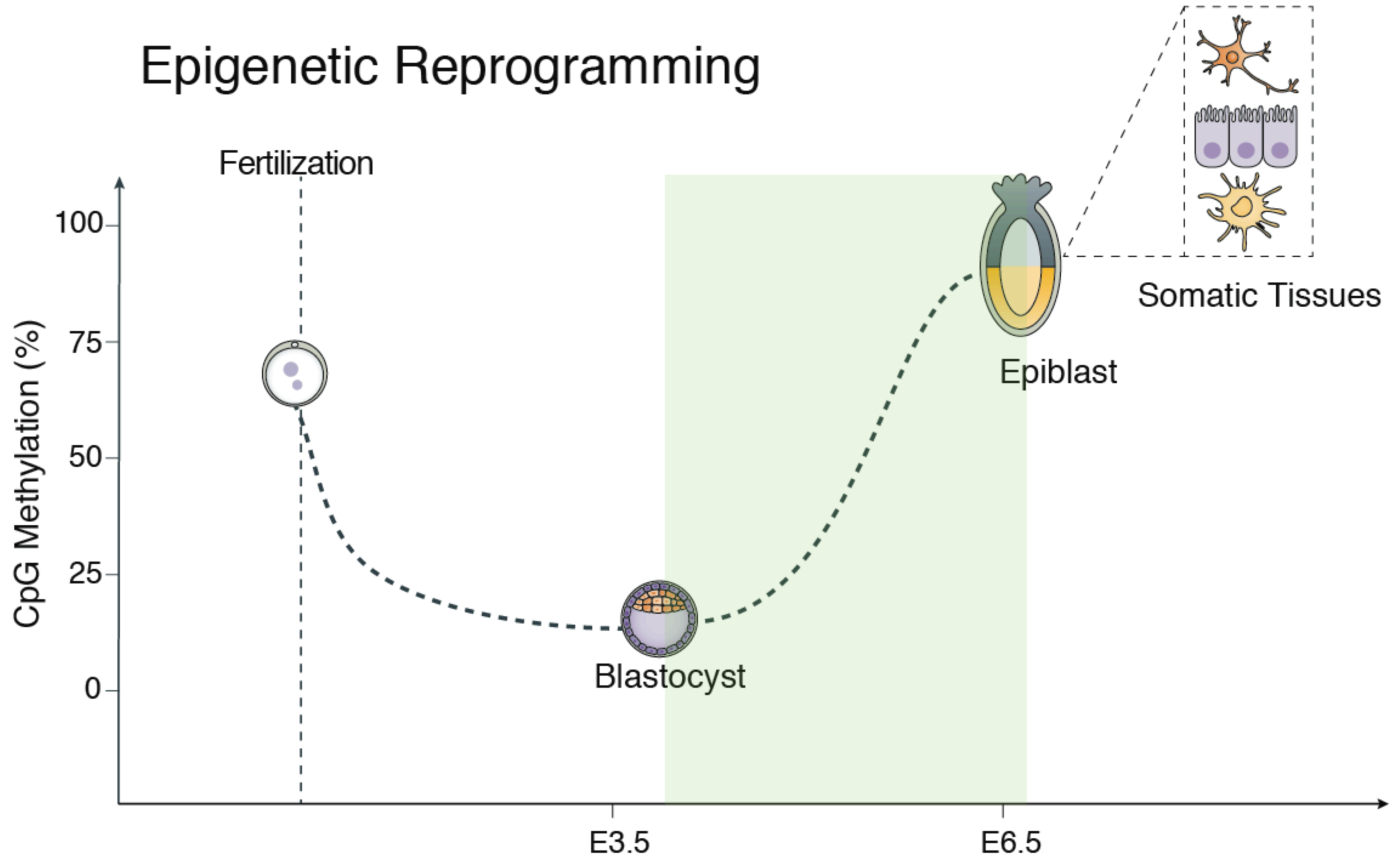
Leading to global demethylation!

NB: Not validated *in vivo*

Mulholland et al., *Nature Comms*, 2020

Mechanism for Reprogramming



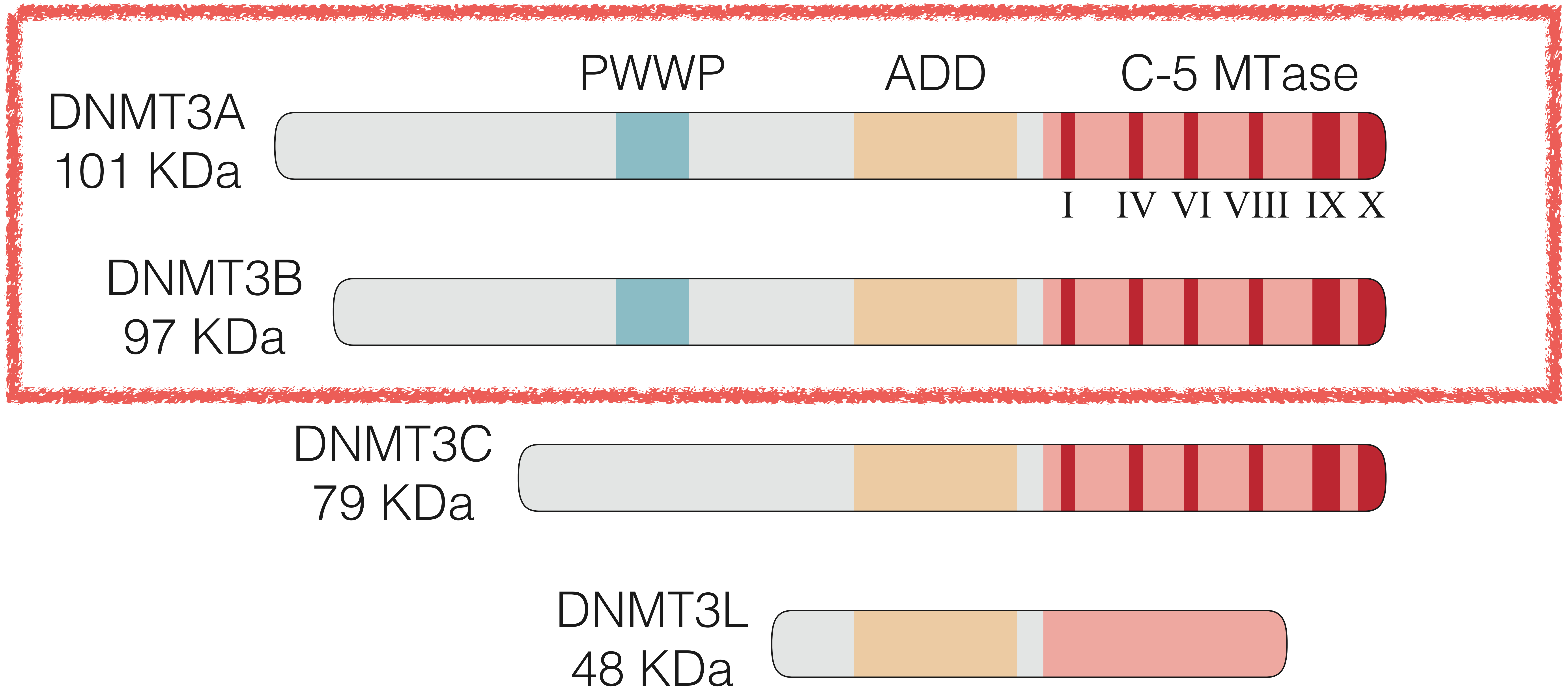


Stable maintenance mechanism

Rapid gain of DNA methylation

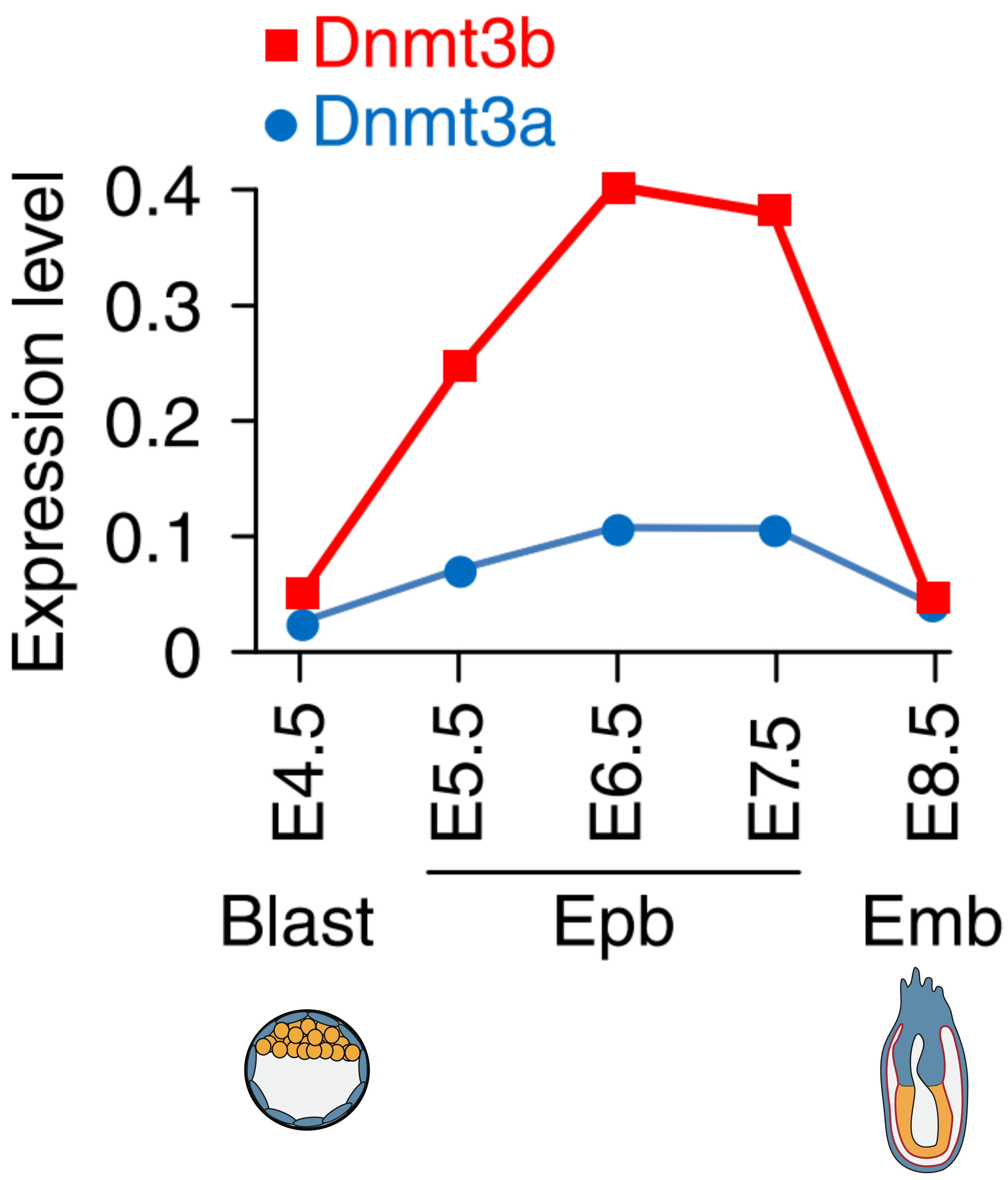
How??

De novo Methylation Program



DNMT3A and DNMT3B are the major embryonic *de novo* methyltransferases

DNMT3A/B Expression Dynamics

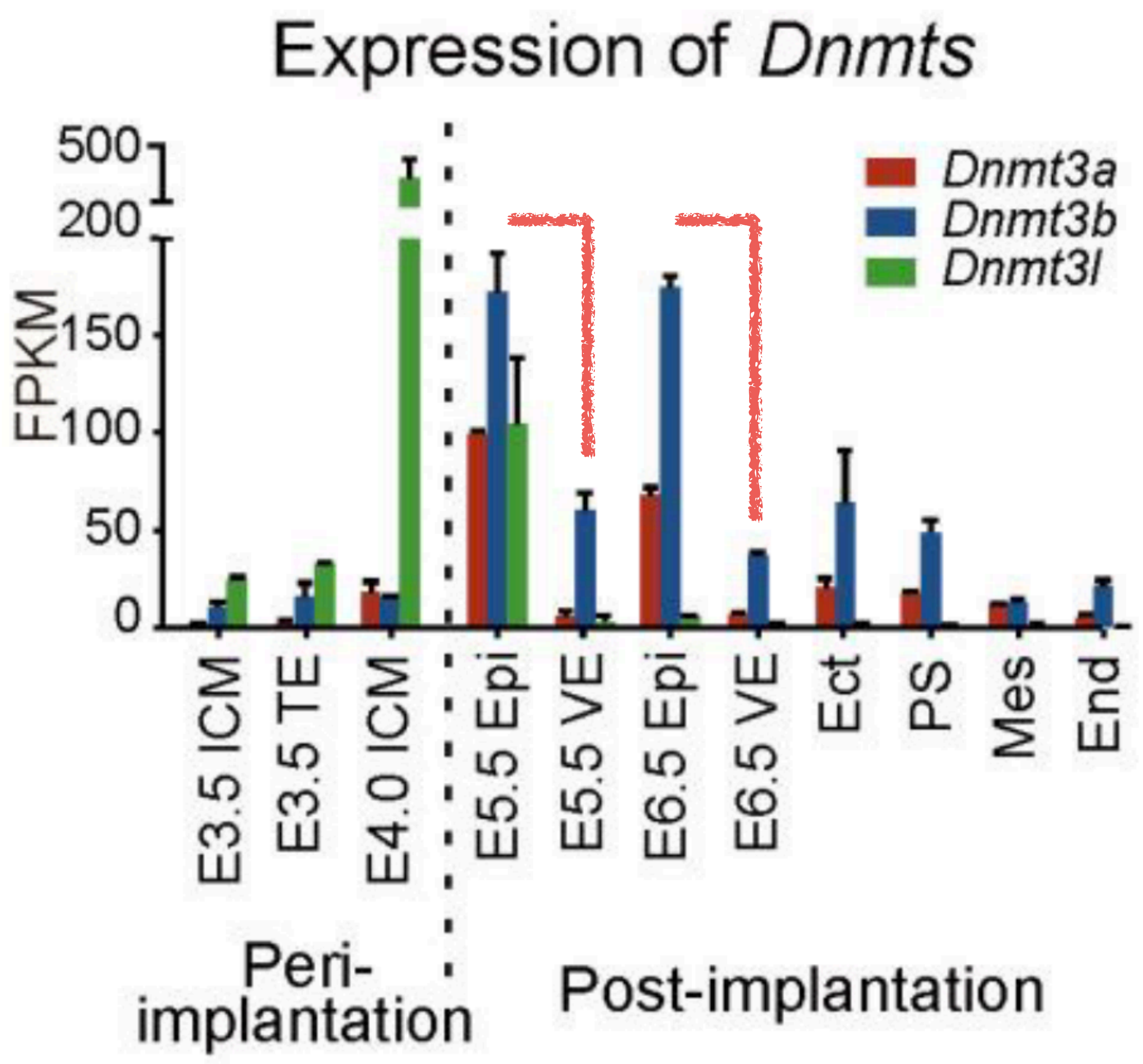


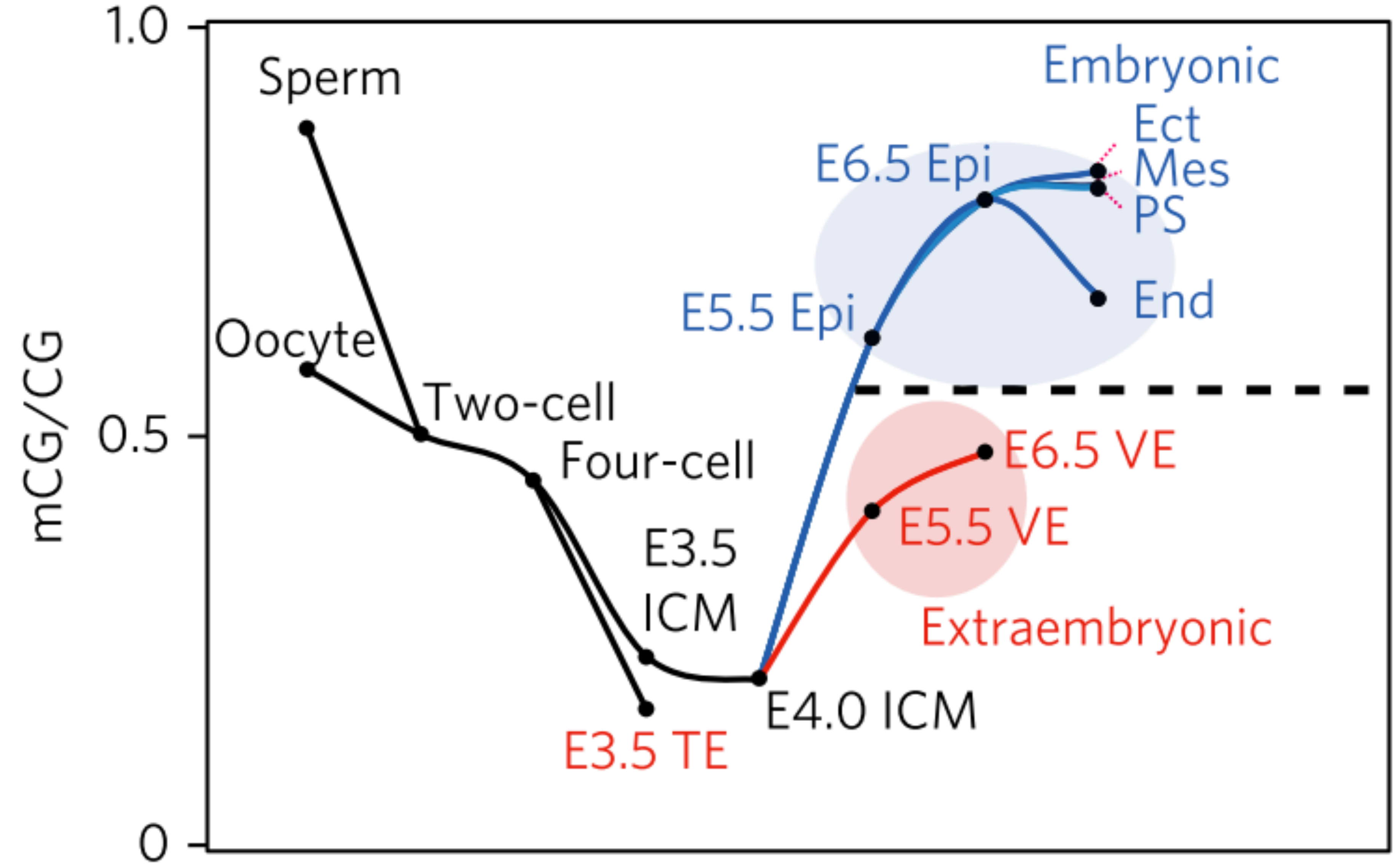
When embryo implants, expression of *de novo* MTases are upregulated

After ~E7.5 global remethylation complete

Auclair et al, *Genome Biology*, 2014

DNMT3A/B Expression Dynamics





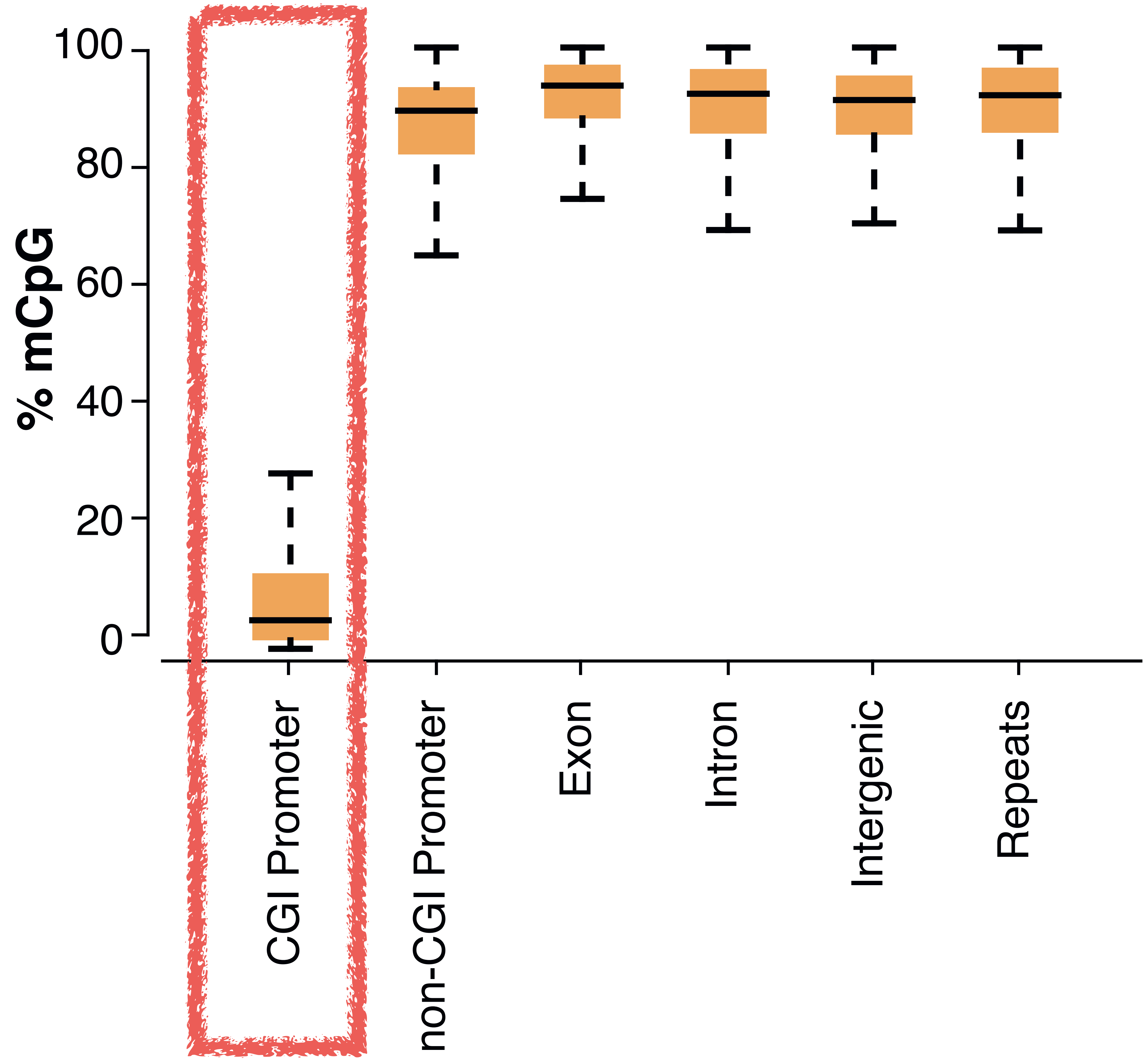
Zhang et al, *Nat Gen*, 2018

Extra-embryonic tissue exhibit less *Dnmt3* expression and methylation

Less need for stringent regulation in extra embryonic tissues?

Does TE expression contribute to placenta formation?

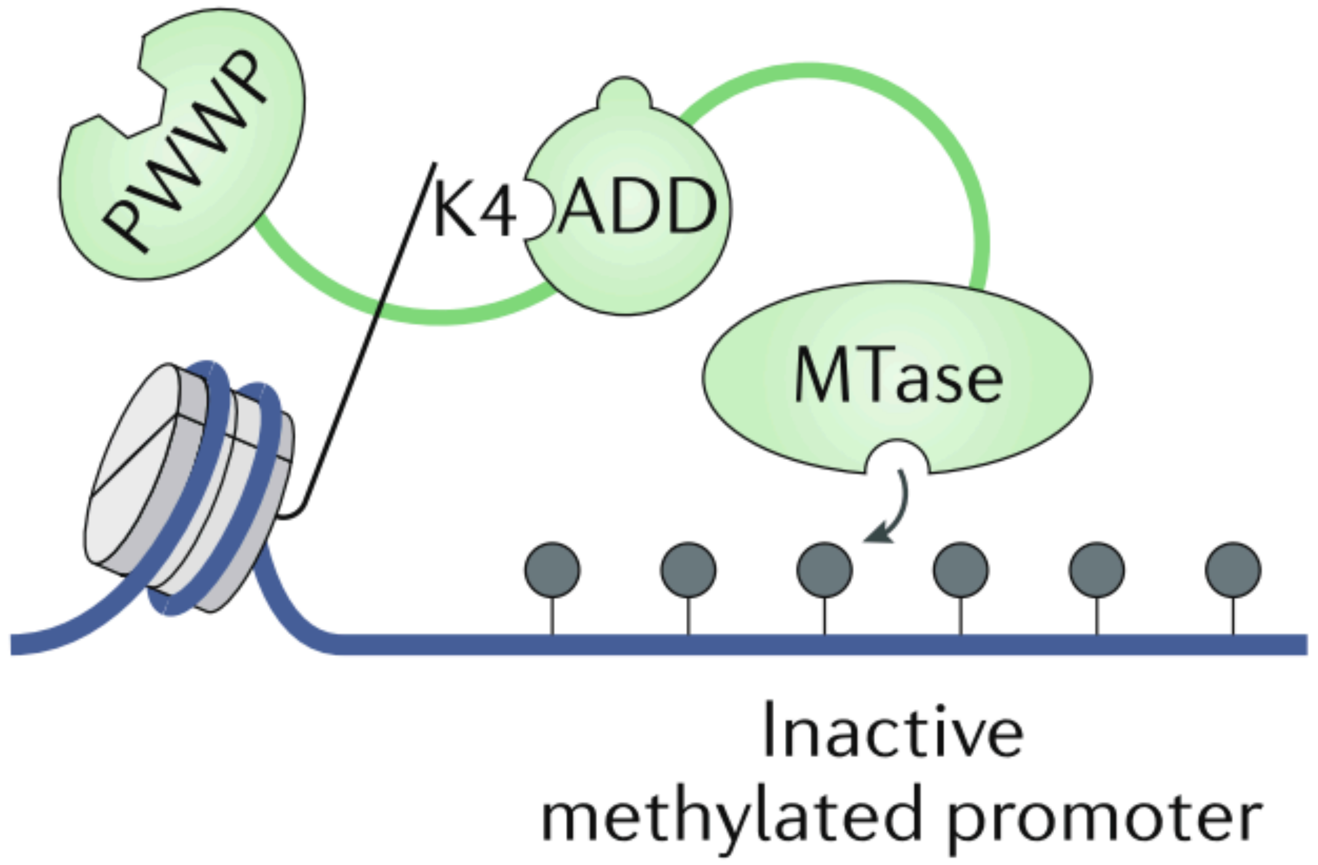
DNMT3A/B are (almost) indiscriminate

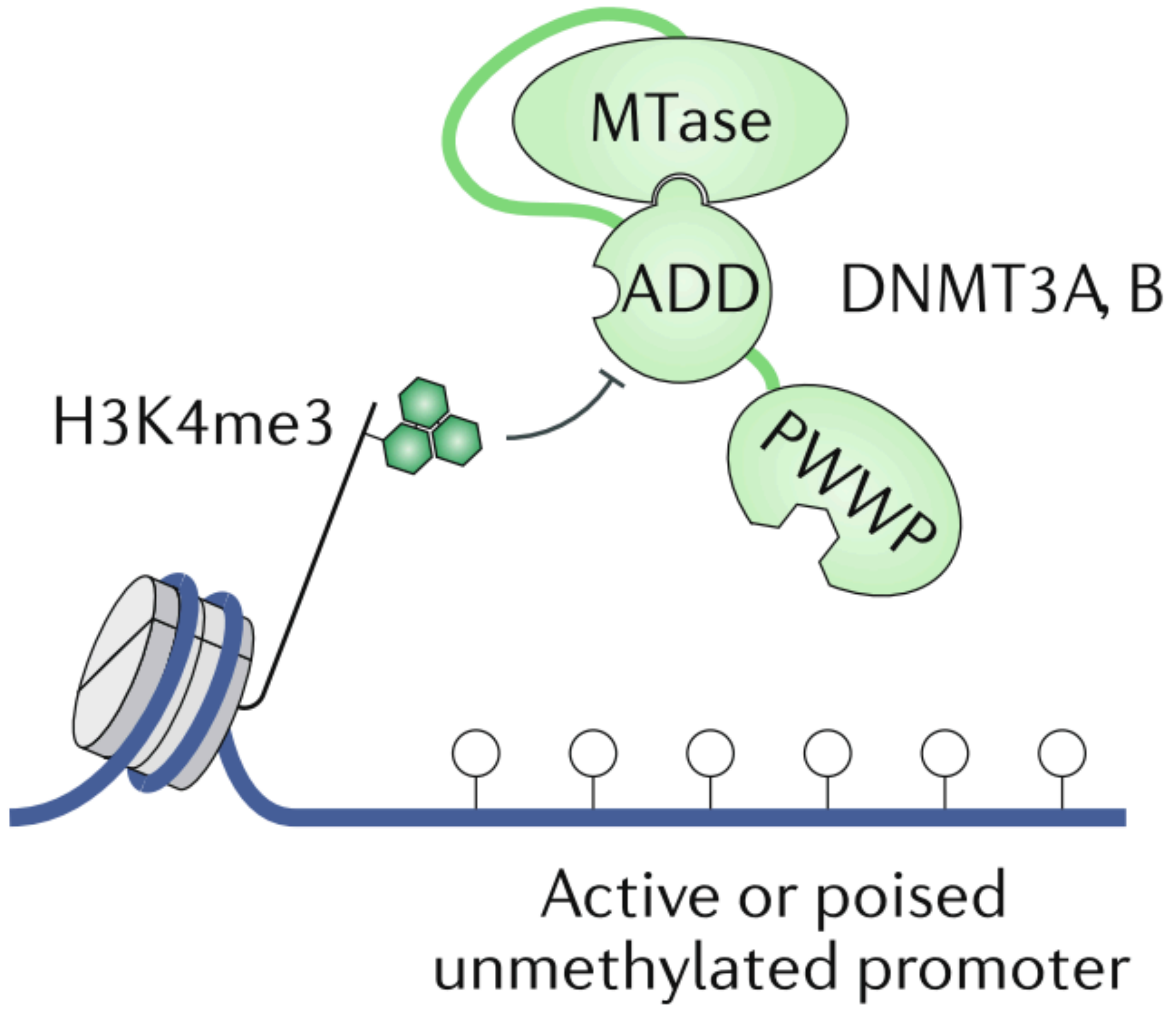


DNA methylation confers stable silencing, so…

How do promoters stay DNA methylation free?

DNMT3A/B are (almost) indiscriminate



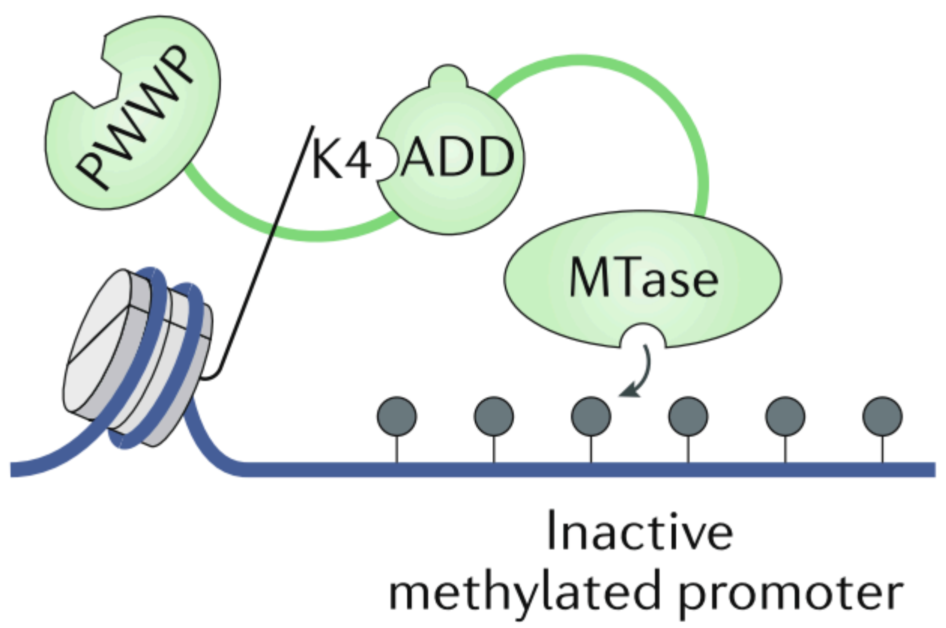


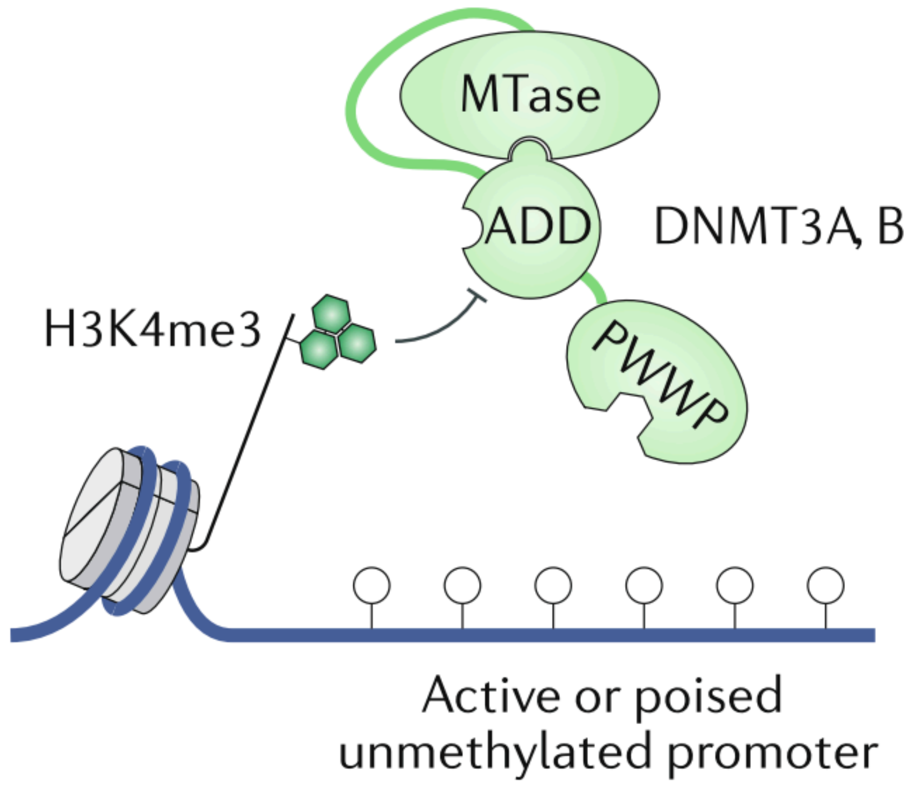
When H3K4 is **unmodified**, ADD domain binds

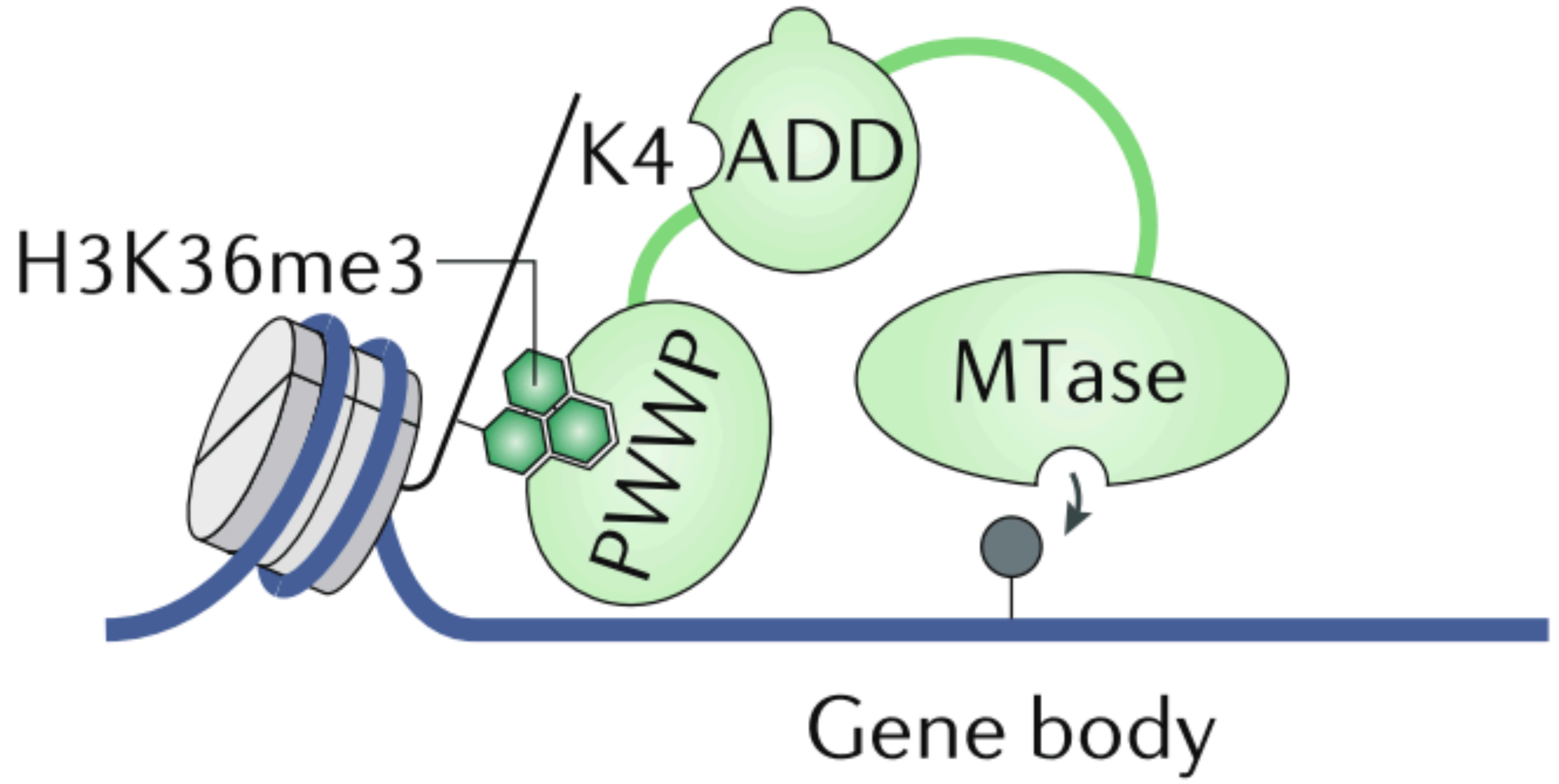
ADD domain is repelled by H3K4me3, and auto-inhibits catalytic domain.

H3K4me3 protects promoters from DNA methylation

DNMT3A/B are (almost) indiscriminate



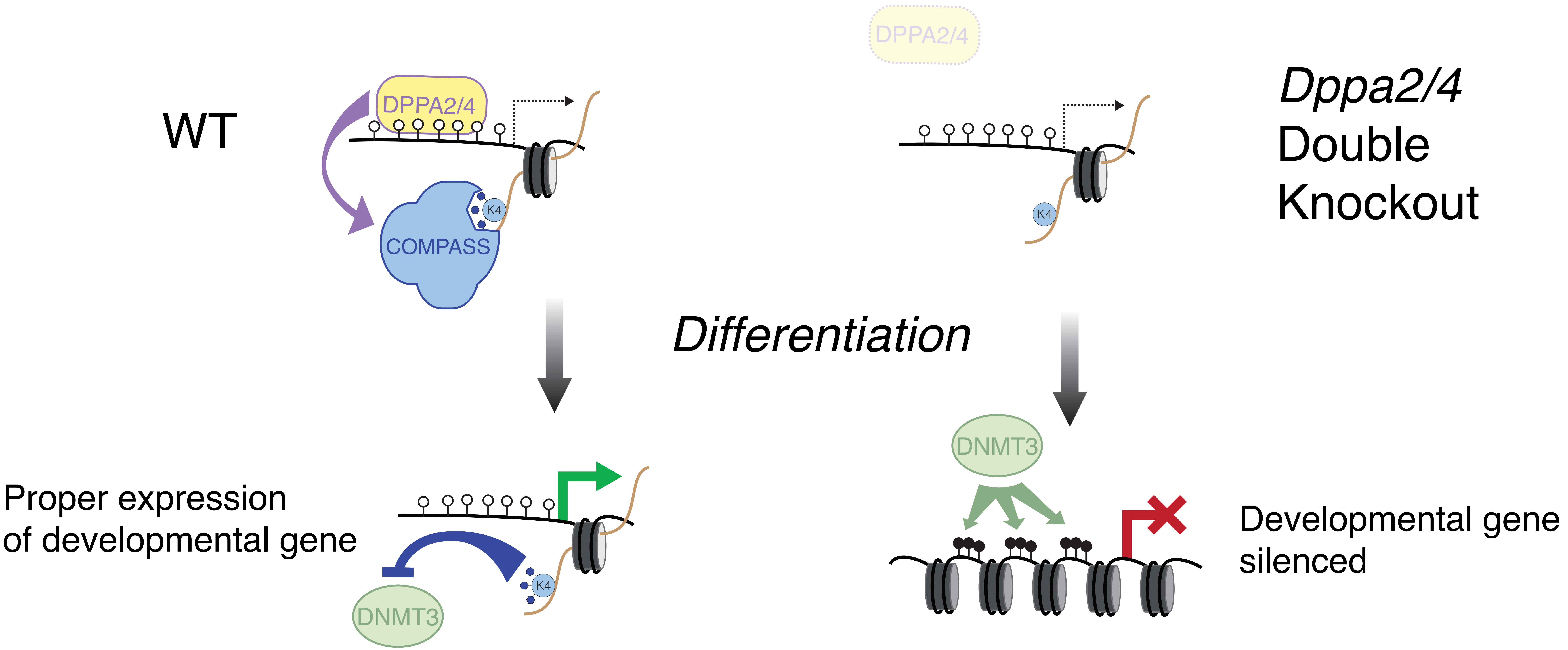




PWWP binds to H3K36me2/3

Abundant marks in many compartments but **NOT** promoters

What targets H3K4me3?



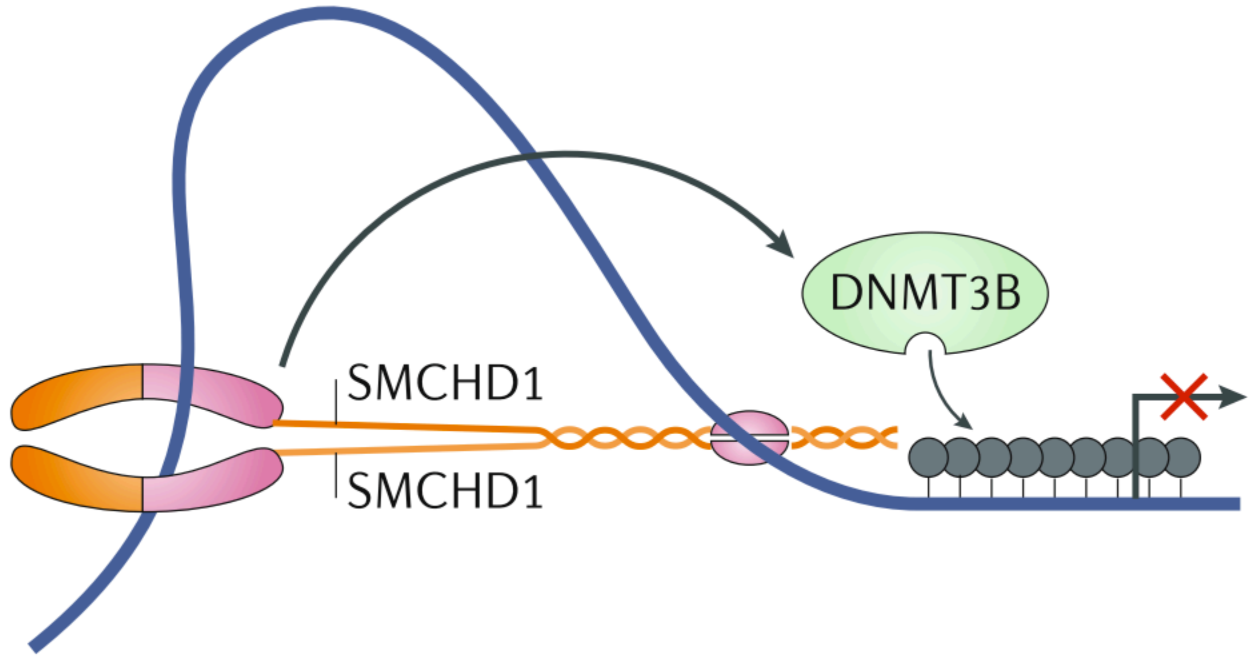
DPPA2 & DPPA4: transcription factors expressed in early development

Gretarsson & Hackett, *NSMB*, 2020
Eckersley-Maslin et al, *NSMB*, 2020

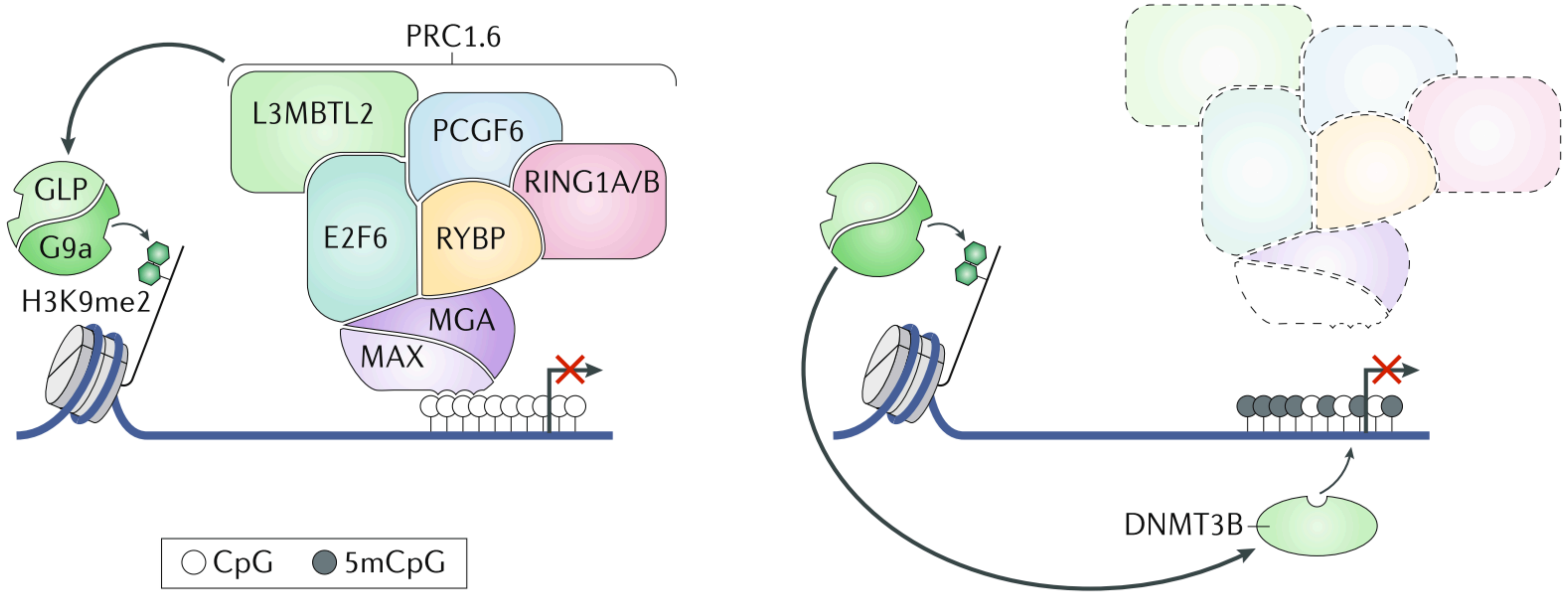
Subset of CGIs are Methylated

Which genes need to be silenced for life in somatic tissues?

Genes on the inactive X chromosome

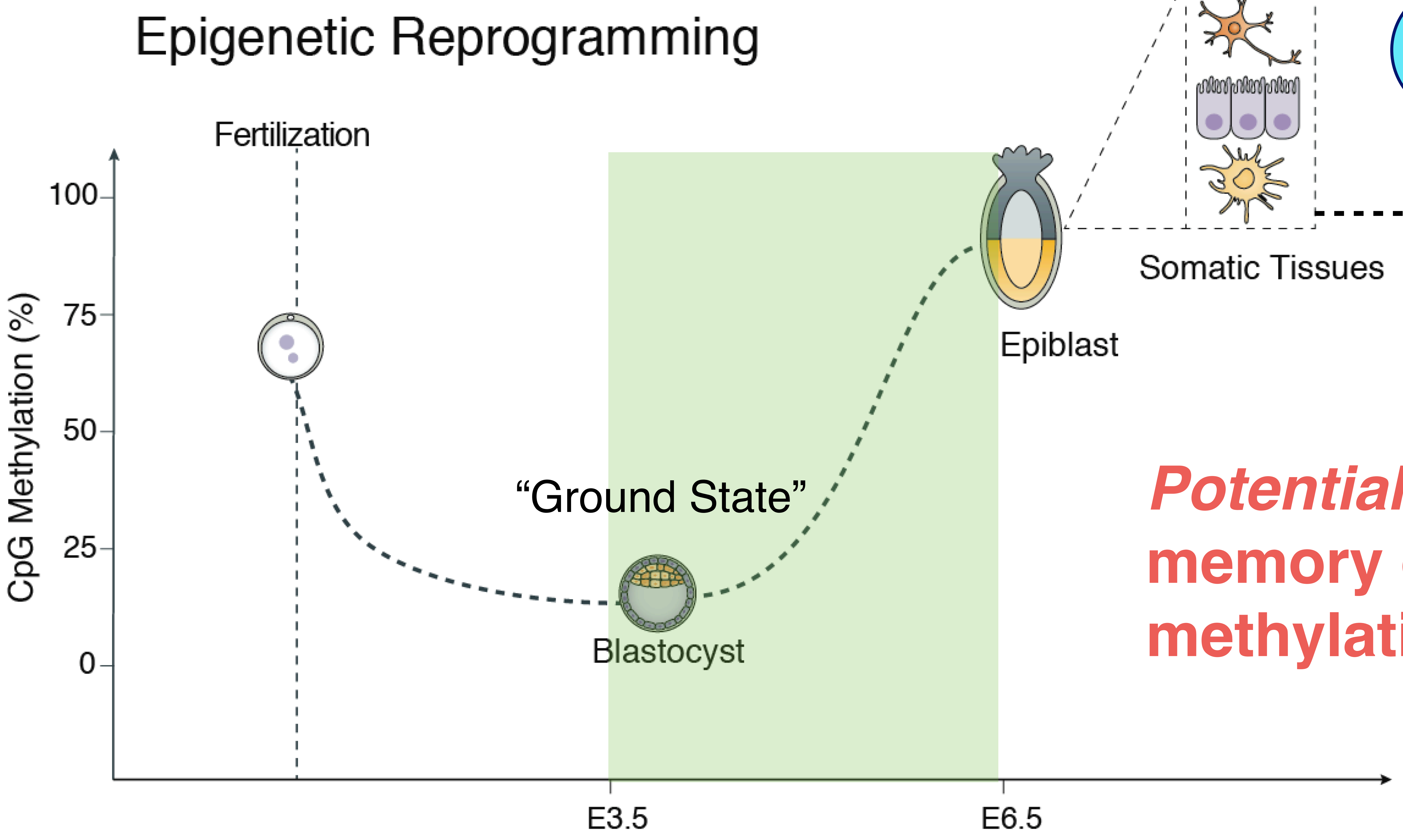


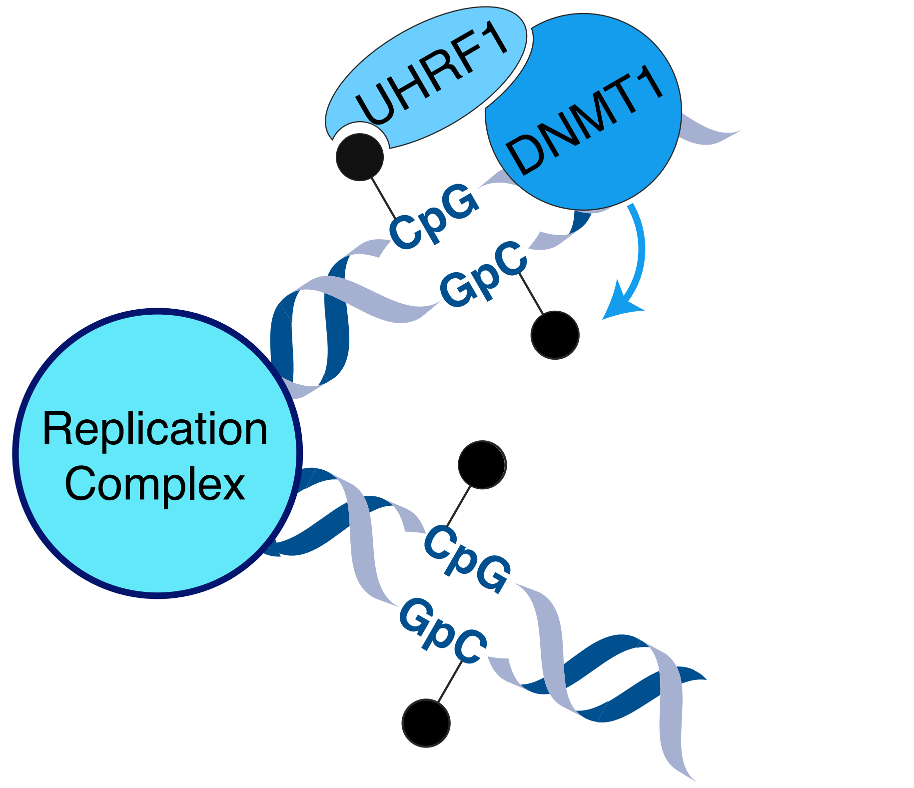
Germline-specific genes



Imprinted genes

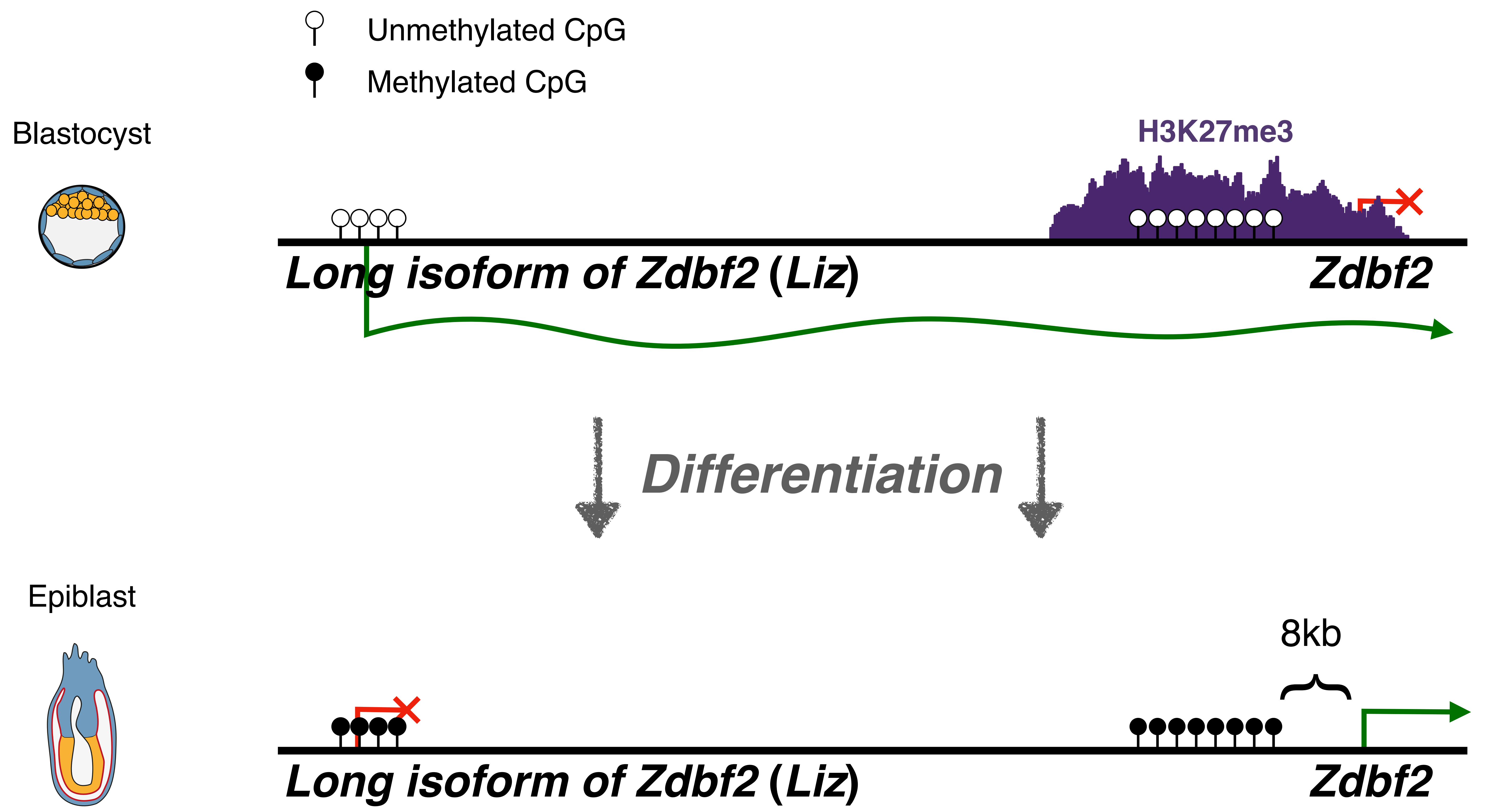
Life-long Consequences of Epigenetic Reprogramming





***Potential* for life-long memory of DNA methylation establishment**

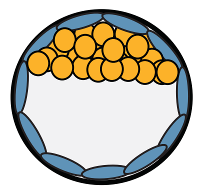
Zdbf2: A model for epigenetic memory



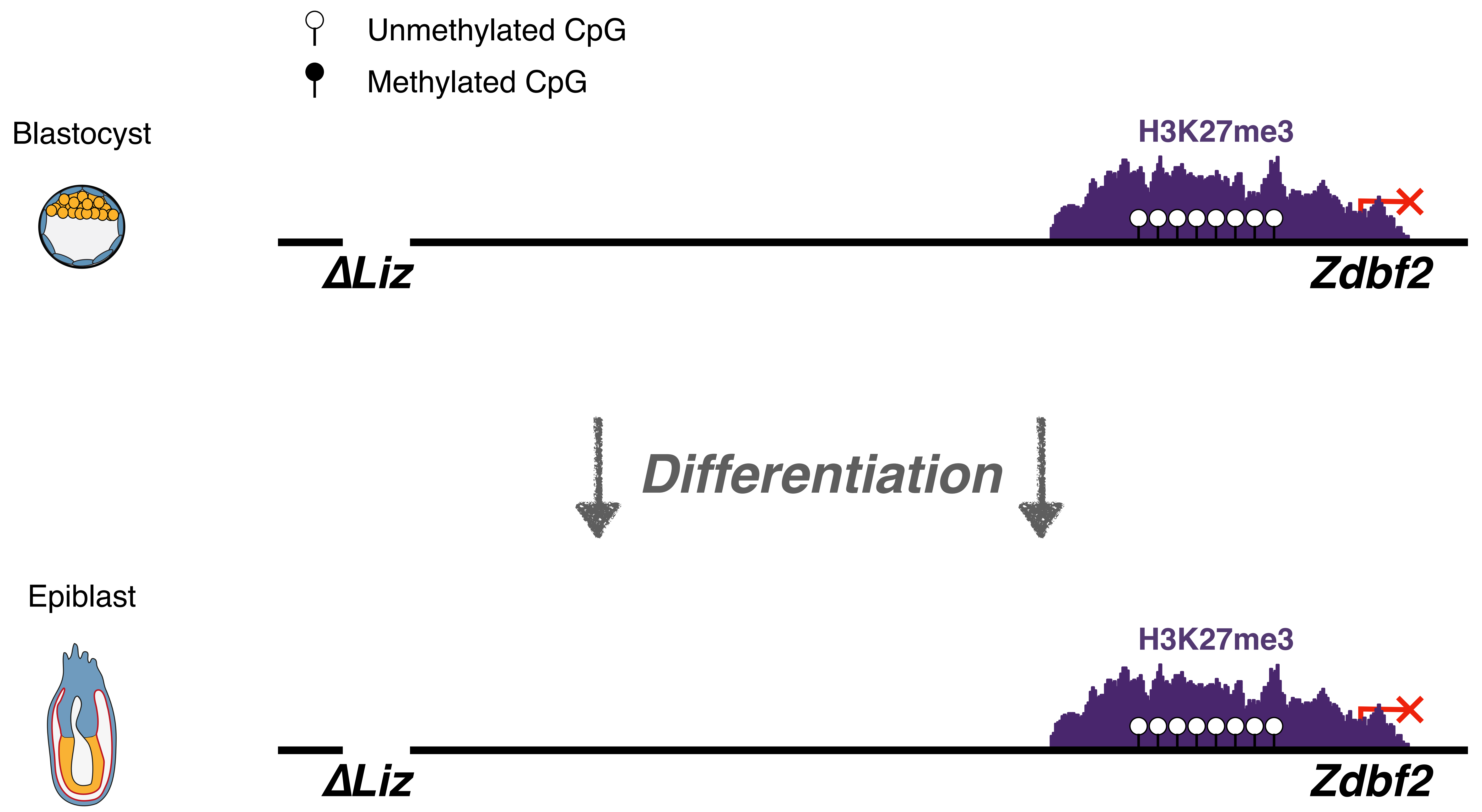
DNA methylation antagonizes polycomb-mediated silencing

Greenberg, Glaser et al, *Nat Gen* 2017

Zdbf2: A model for epigenetic memory

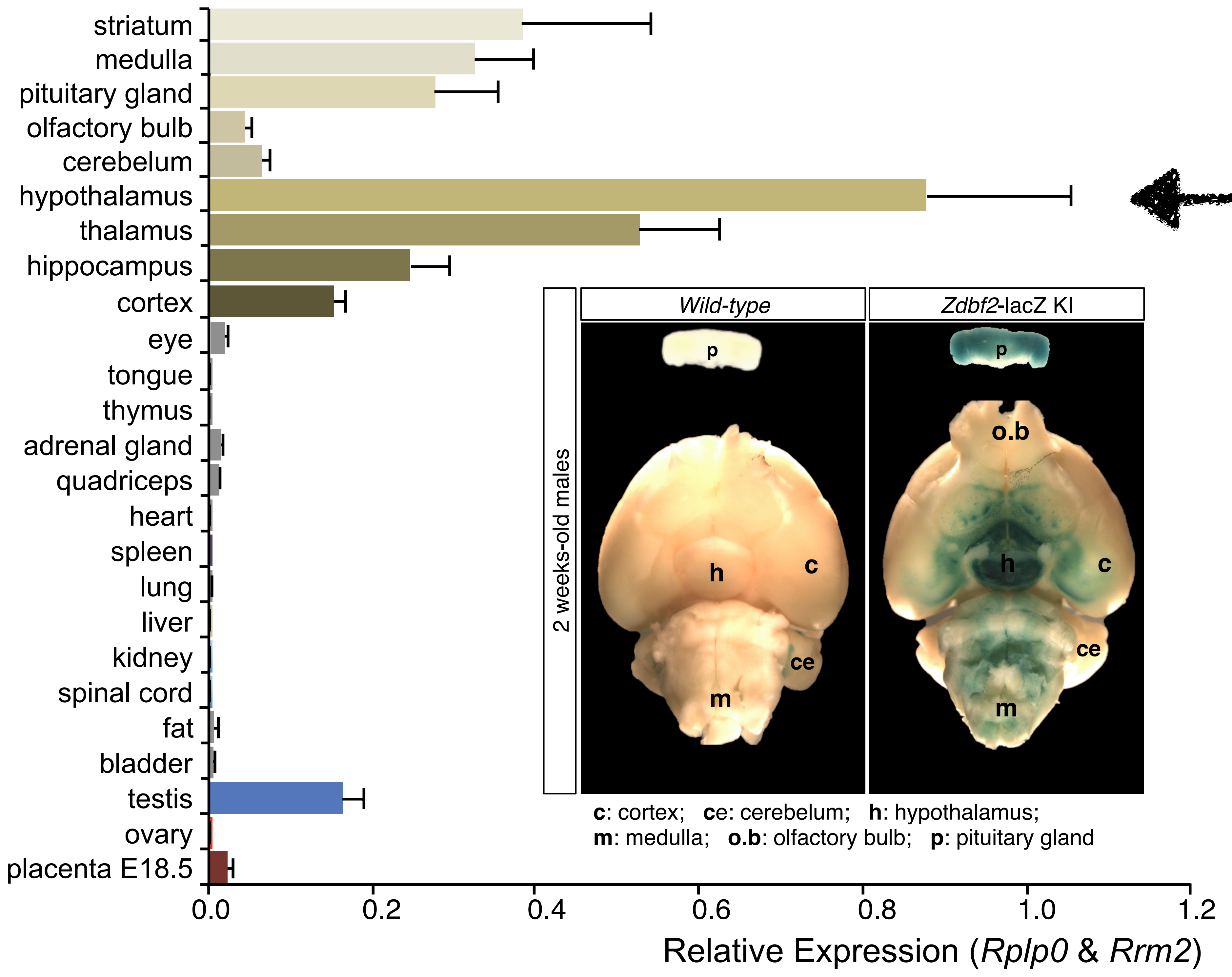


Zdbf2: A model for epigenetic memory



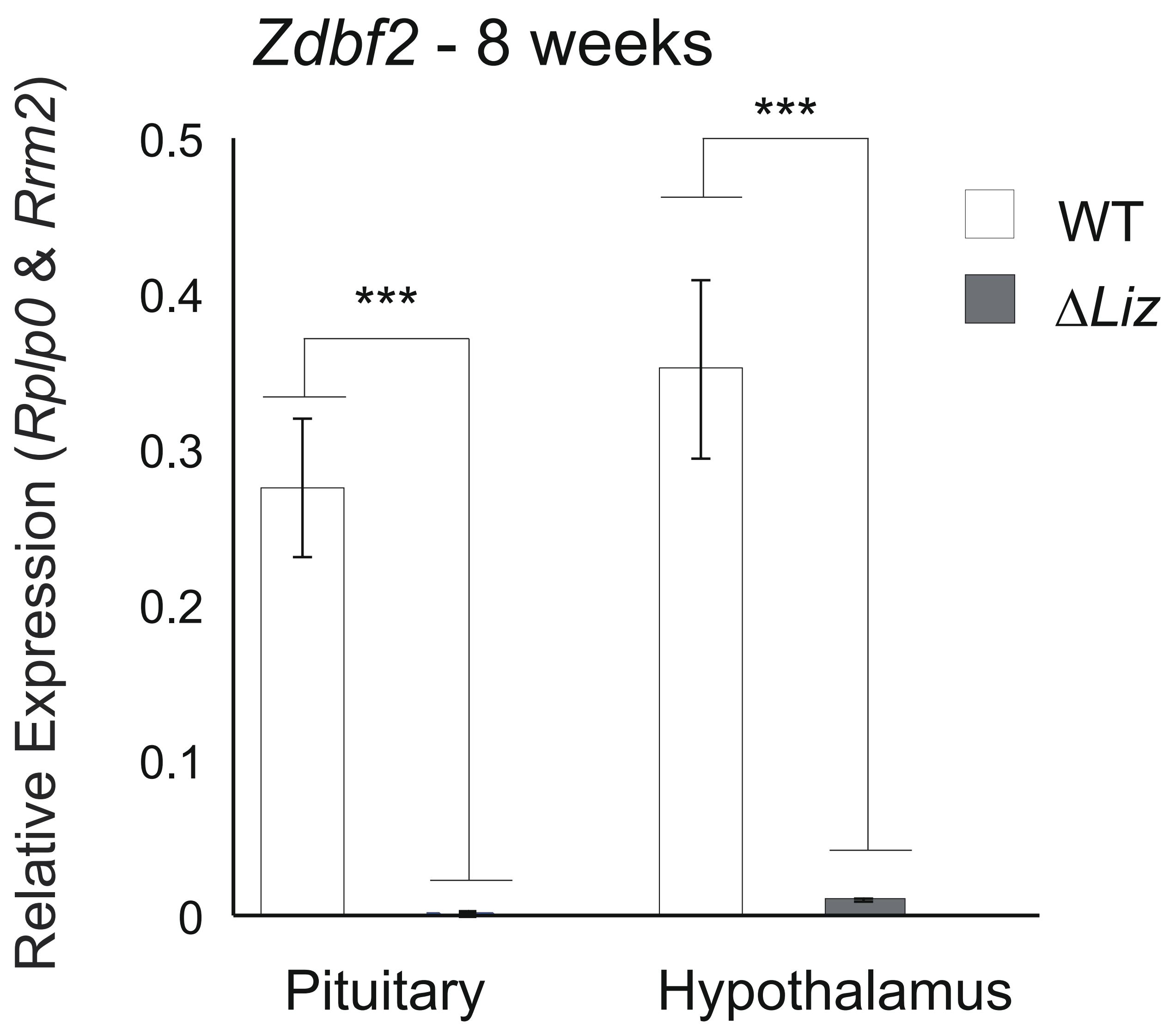
Zdbf2 **remains repressed**

Zdbf2: A model for epigenetic memory



Zdbf2 is most highly express in brain

Hypothalamus controls growth



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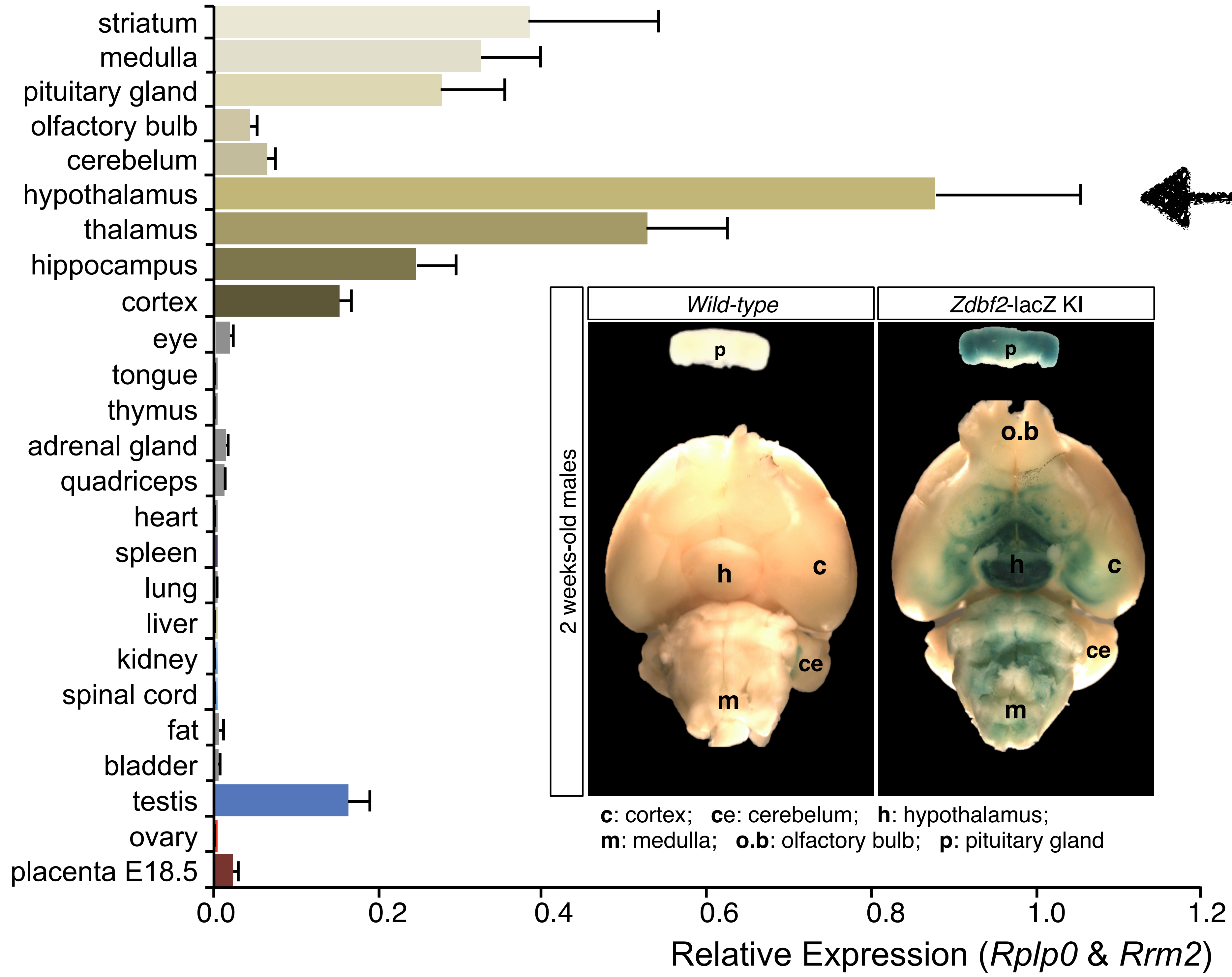
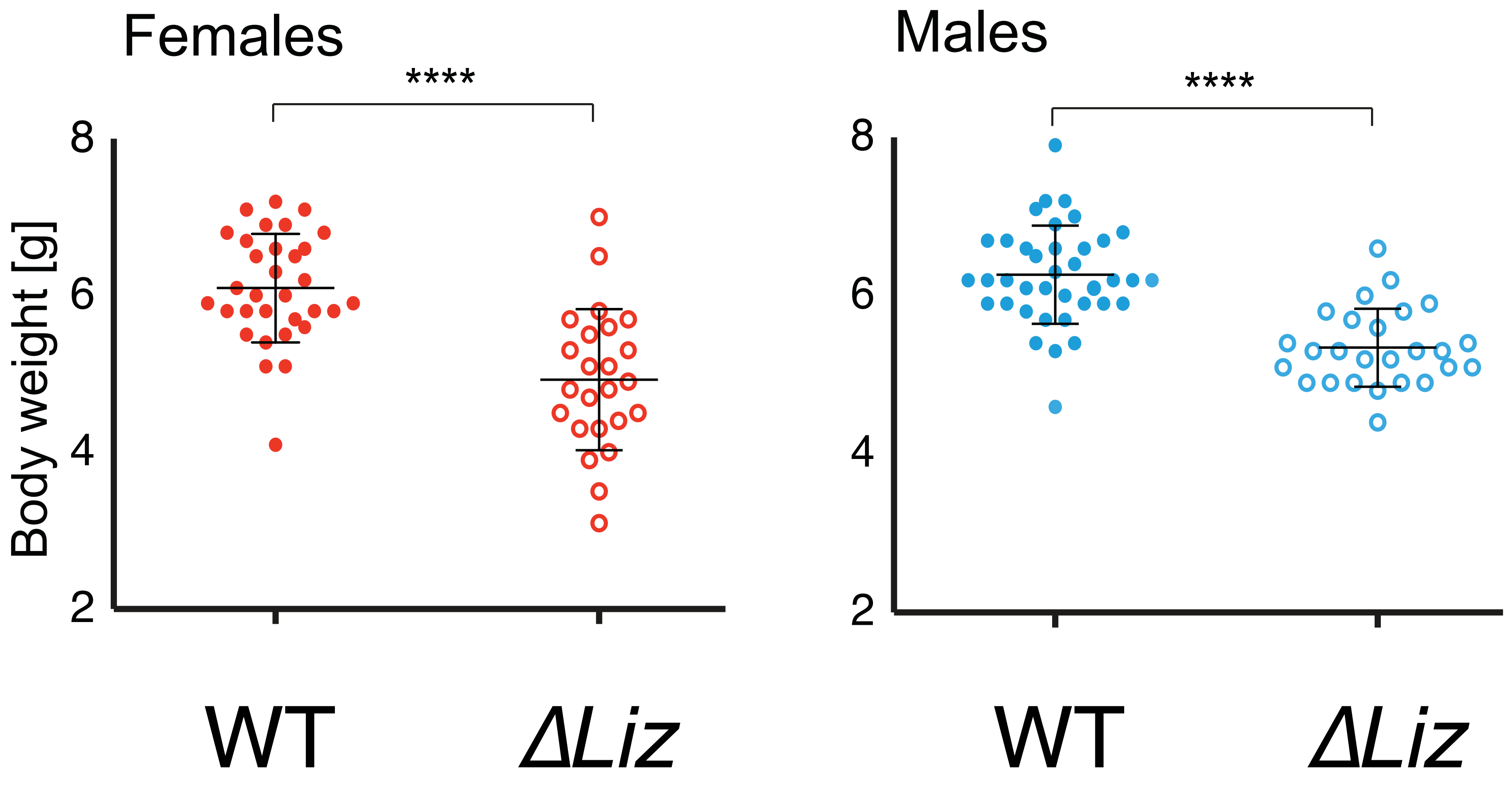
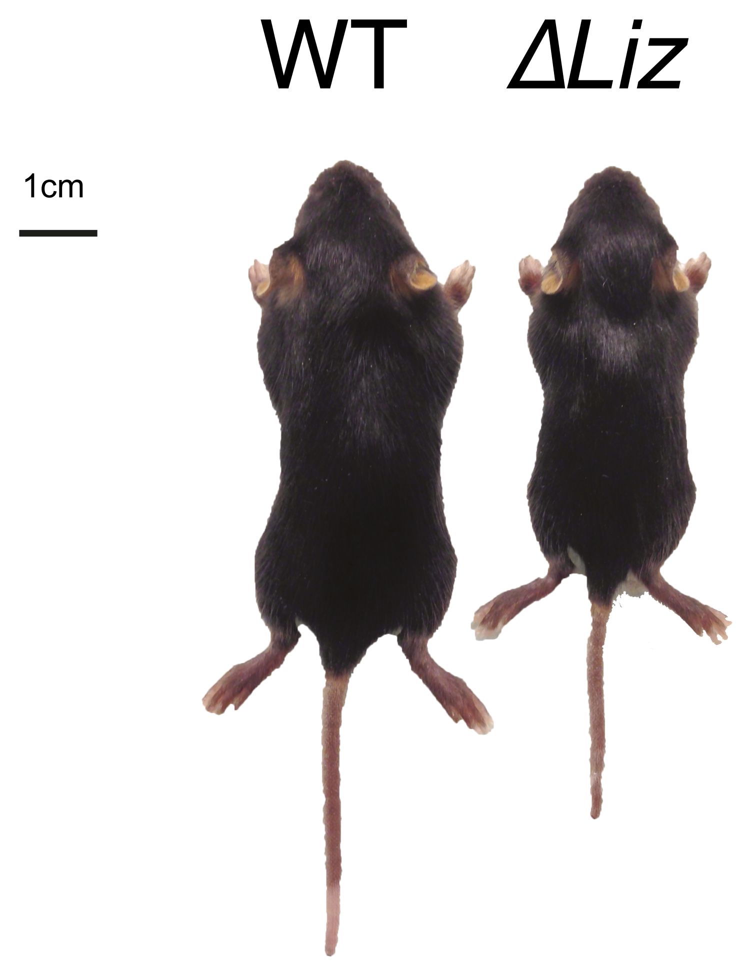


Zdbf2 is most highly express in brain

Hypothalamus controls growth

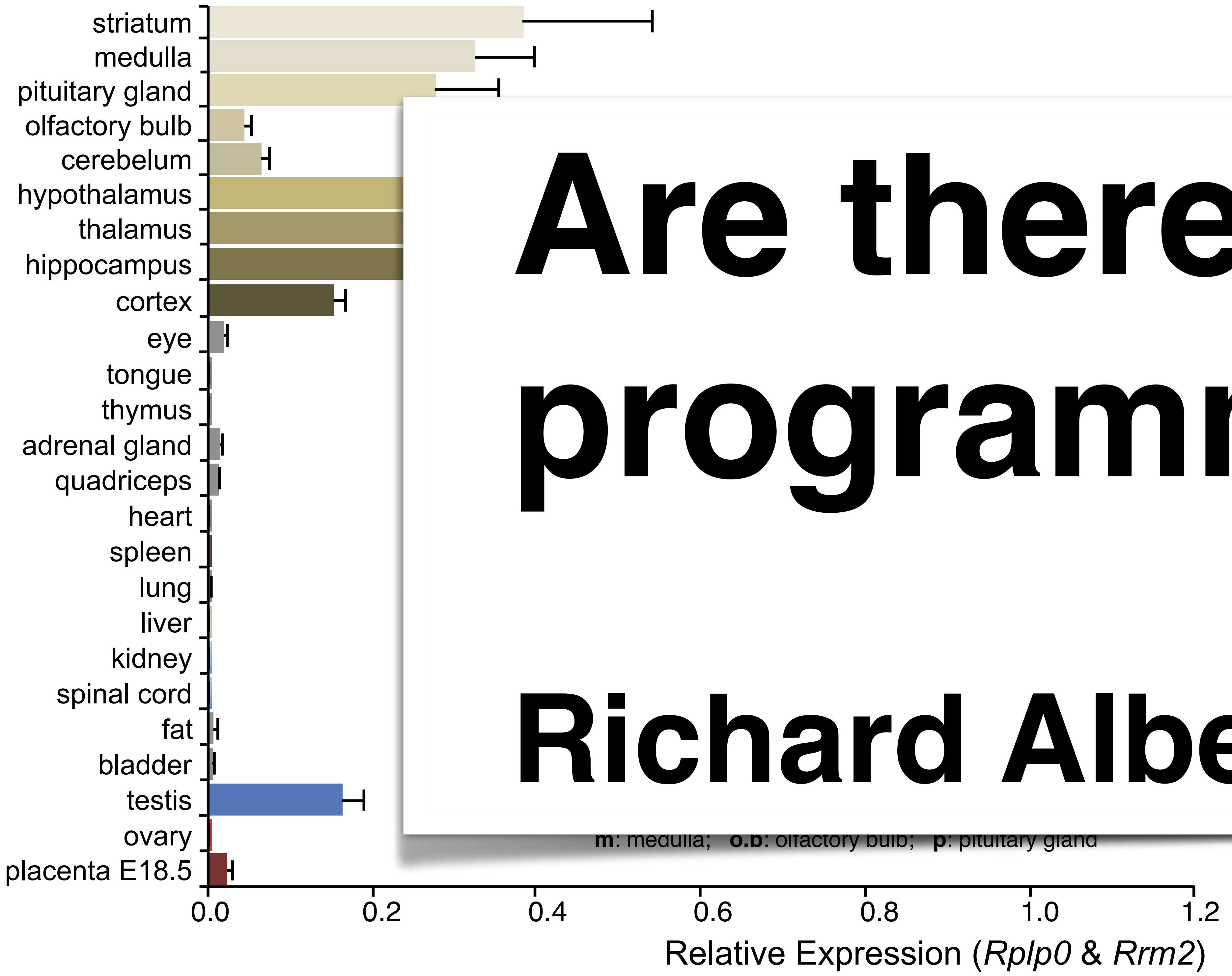




Early embryonic epigenetic programming of *Zdbf2* controls postnatal growth!!!

Zdbf2: A model for epigenetic memory

Zdbf2 is most highly express in brain



Are there other genes programmed like this???

Richard Albert et al., NSMB 2025

m: medulla; o.b: olfactory bulb; p: pituitary gland

WT *ΔLiz* WT *ΔLiz*

WT *ΔLiz*

Early embryonic epigenetic programming of *Zdbf2* controls postnatal growth!!!

Questions We Will Address Today

I. What is epigenetic reprogamming?

II. How does epigenetic reprogramming occur?

III. Can any regions of the genome escape reprogramming?

IV. **Why** does epigenetic reprogramming occur????

GENOMIC IMPRINTING

Mom vs Dad vs Embryo

GENOMIC IMPRINTING

Parent-allele-specific expression in eutherian mammals, dependent on the existence of **epigenetic differences between the two parental alleles** that allow them to be transcribed differently in the same nucleus.

Functional non-equivalence of parental genomes

Only difference between Tigon and Liger is origin of parental alleles

Lion x Tiger

Tiger x Lion

Only difference between Tigon and Liger is origin of parental alleles

on x Tiger

Asymmetry between parental alleles!

i.e., epigenetic

Tiger x Lion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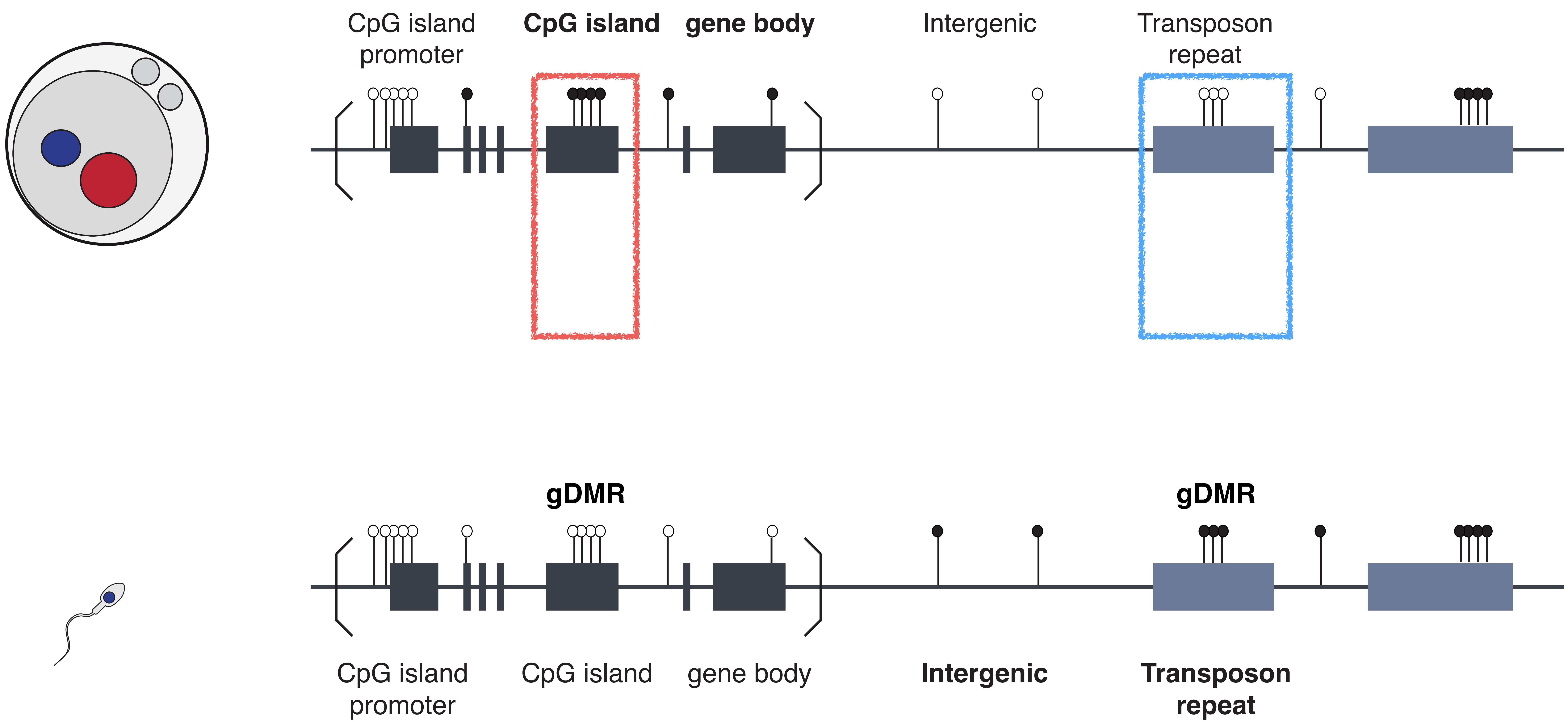
DNA methylation patterns: Fertilization



gDMR = germline Differentially Methylated Regions

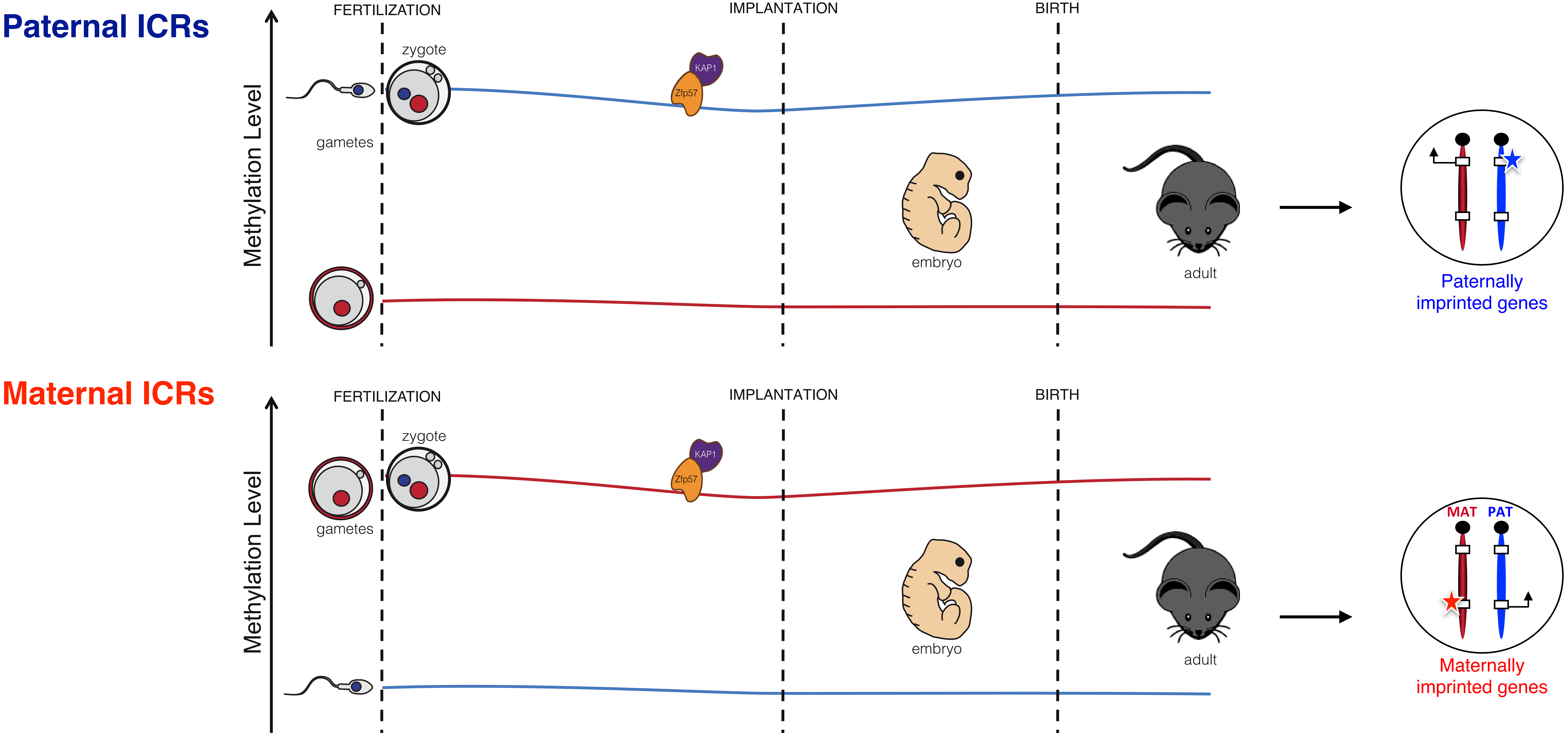
● Methylated CpG

○ Unmethylated CpG

Smallwood et al., 2011; Kobayashi et al., 2012; Smith et al., 2012

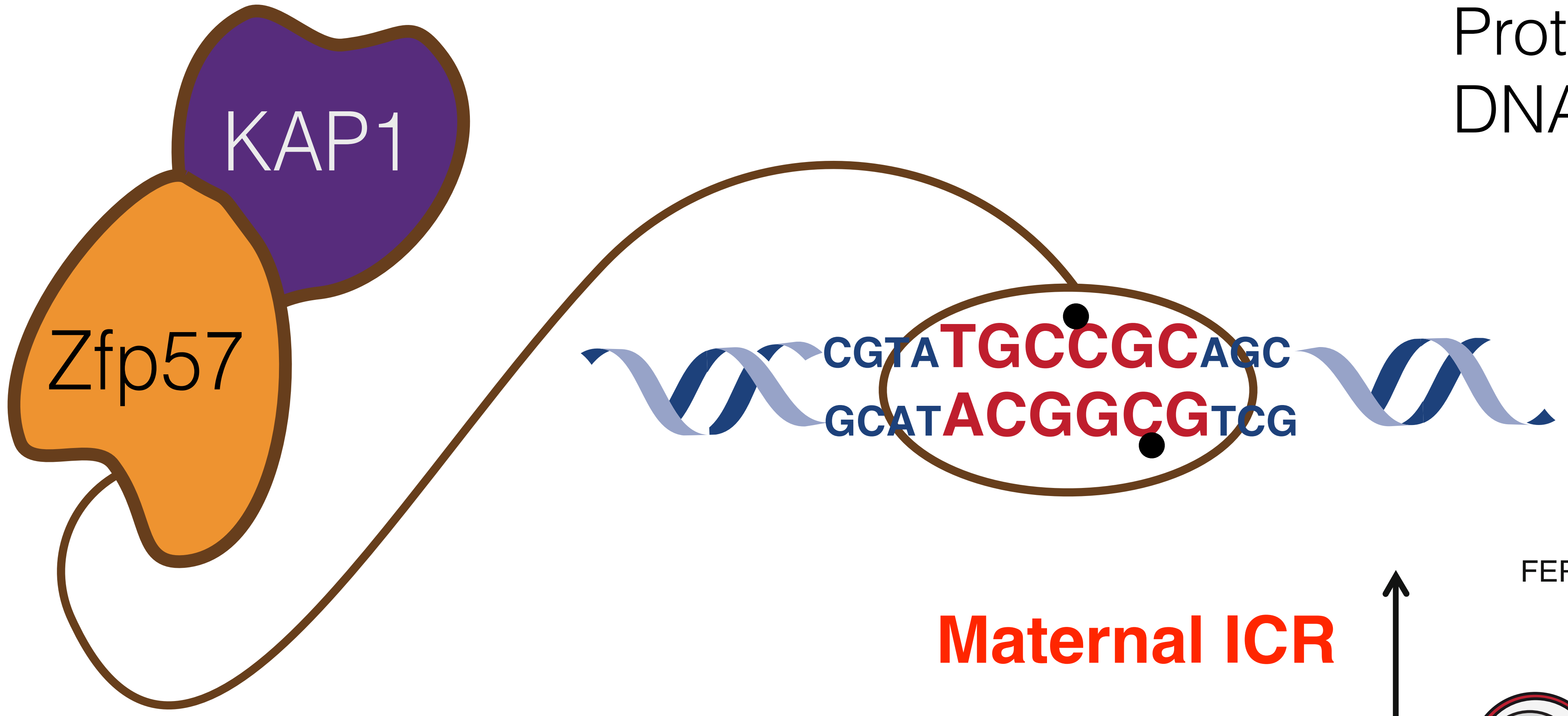
Life-long persistence of “special” gDMRS

Imprinting Control Regions = ICRs ~ DMRs that control allelic expression!



What makes ICRs so special?

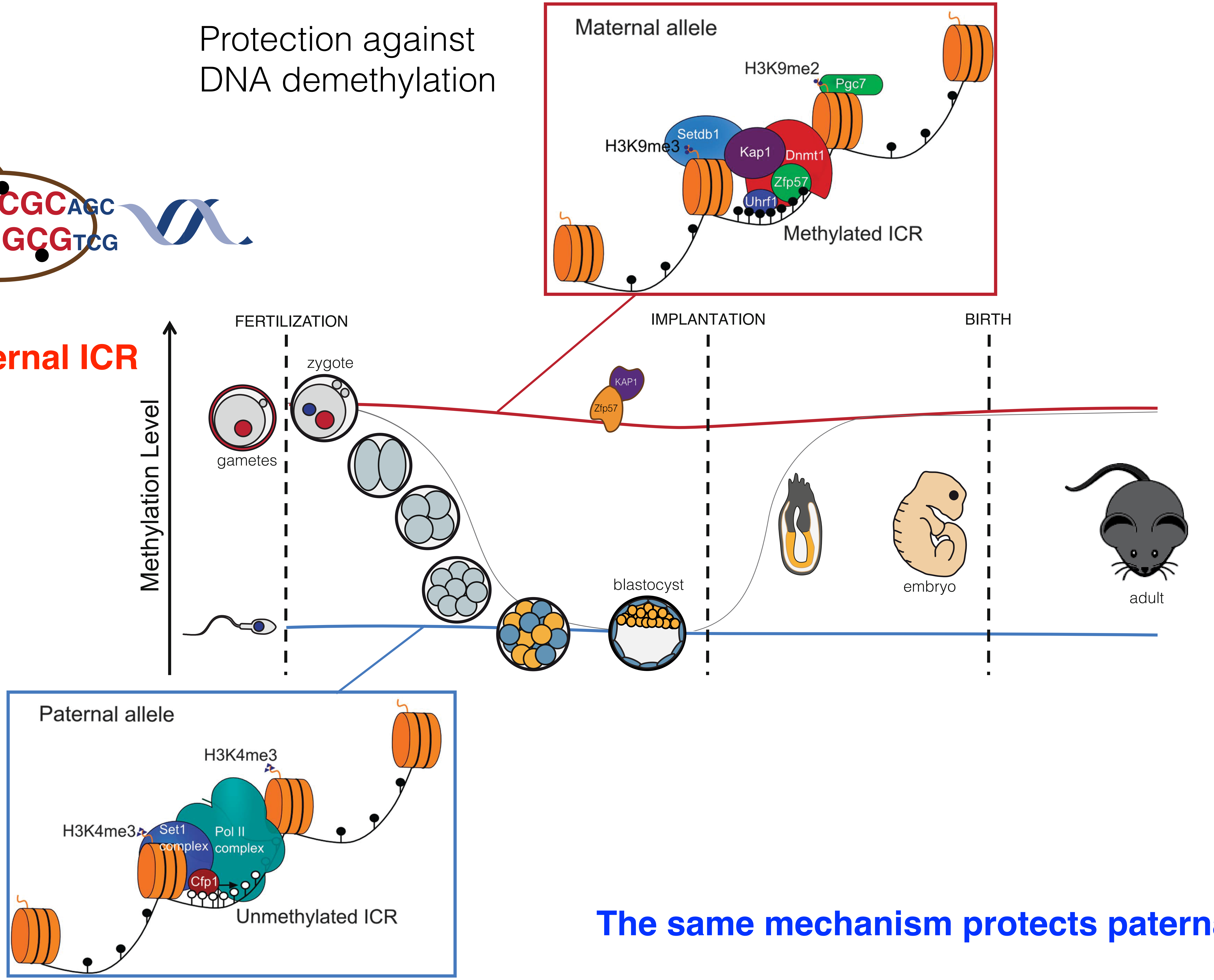
What makes ICRs so special?



Maternal ICR

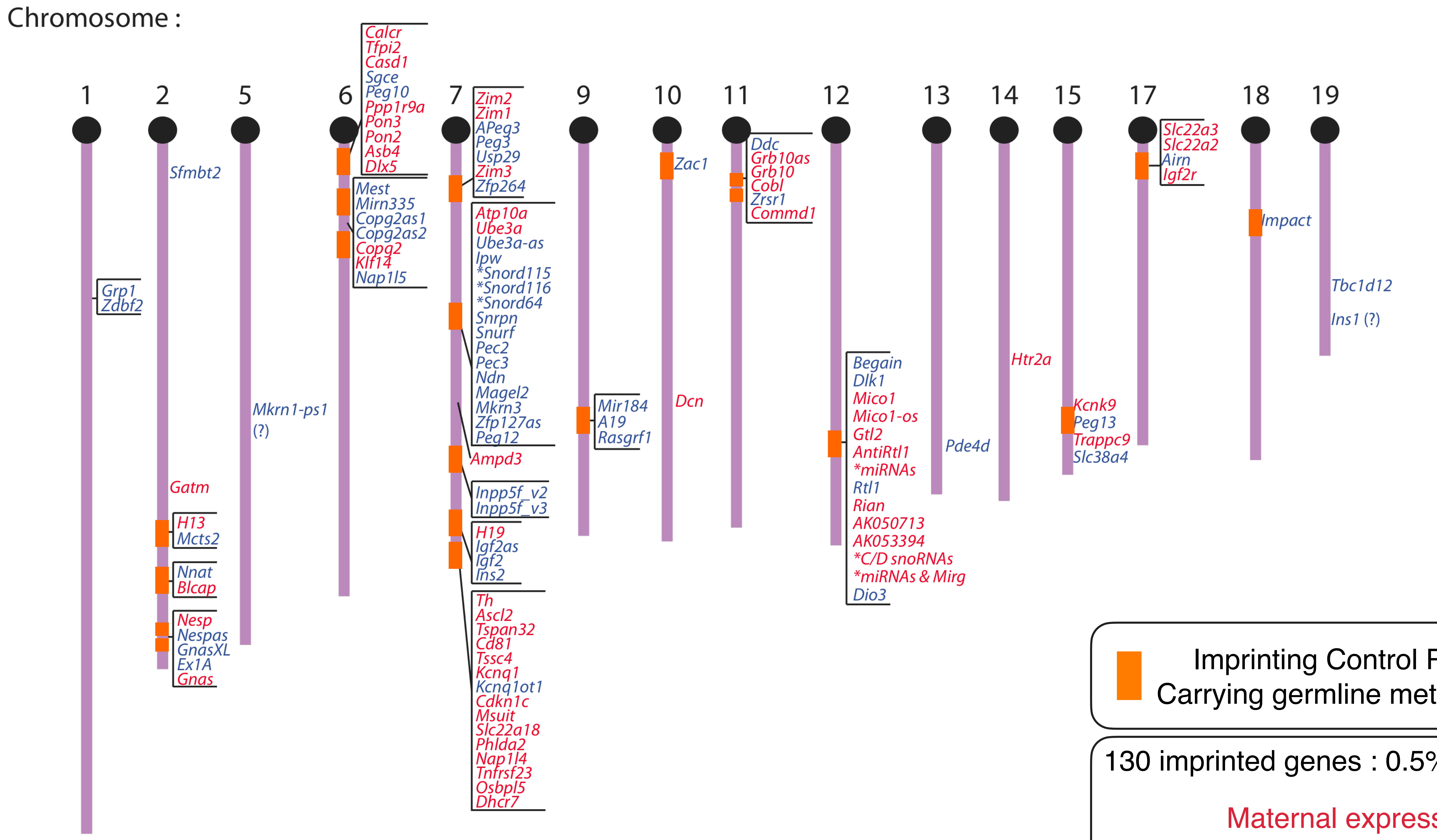
Genetic *and* Epigenetic information

Protection against DNA demethylation



The same mechanism protects paternal ICRs

20 known ICRs



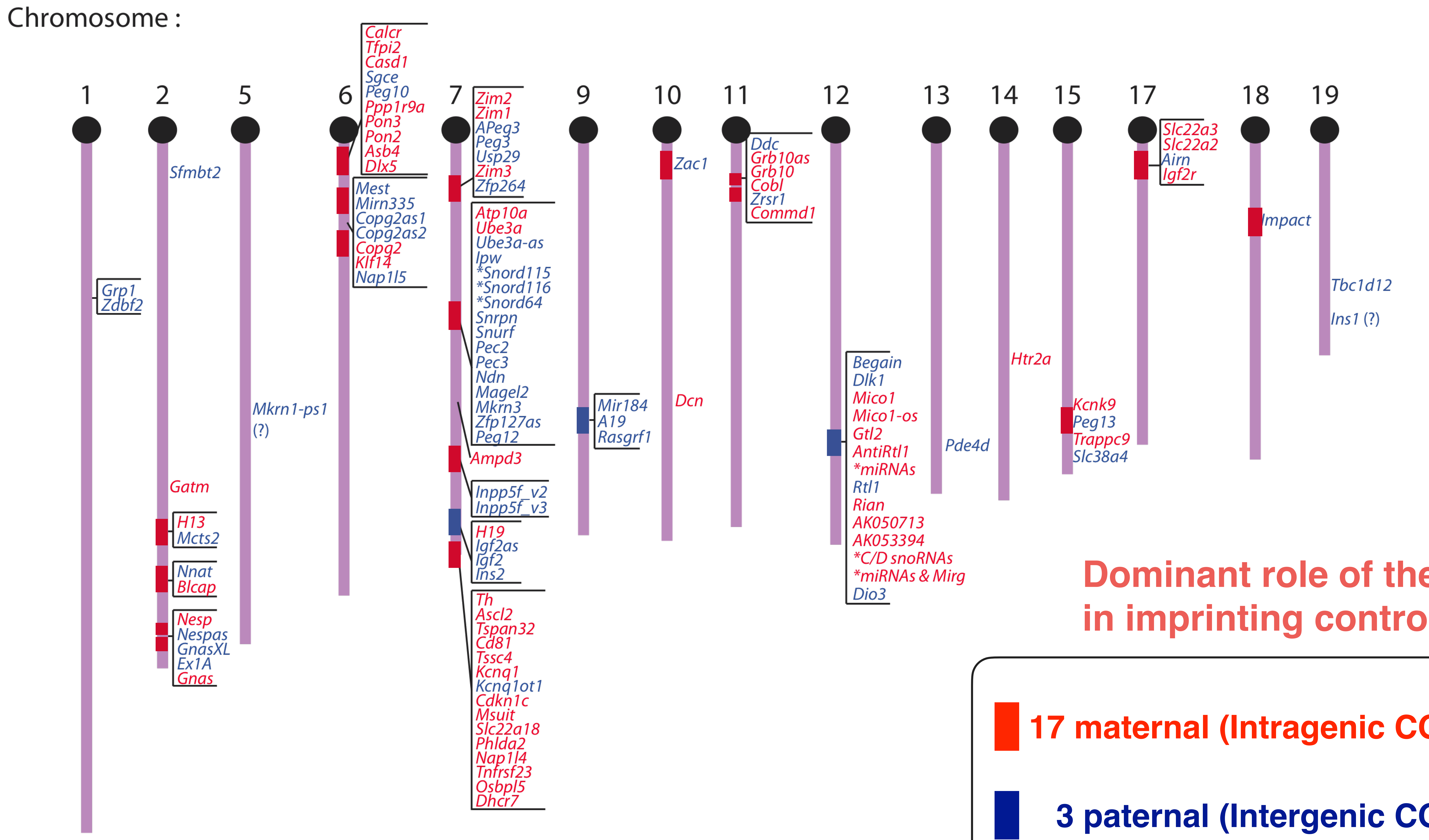
Imprinting Control Regions (ICRs)
Carrying germline methylation marks

130 imprinted genes : 0.5% of the genes

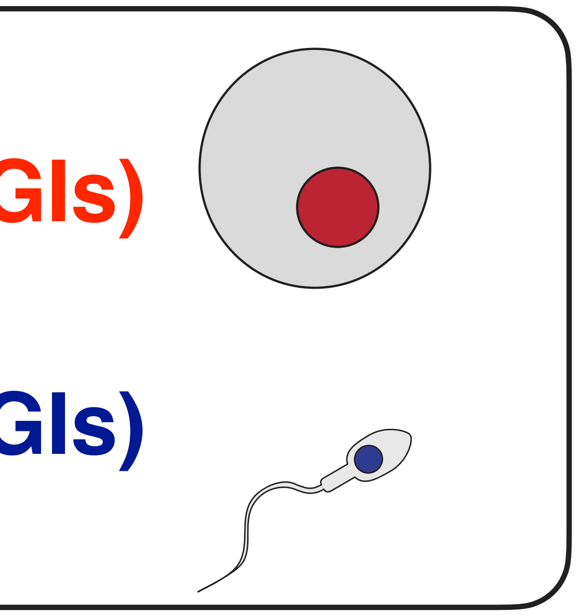
Maternal expression
Paternal expression

http://www.har.mrc.ac.uk/research/genomic_imprinting

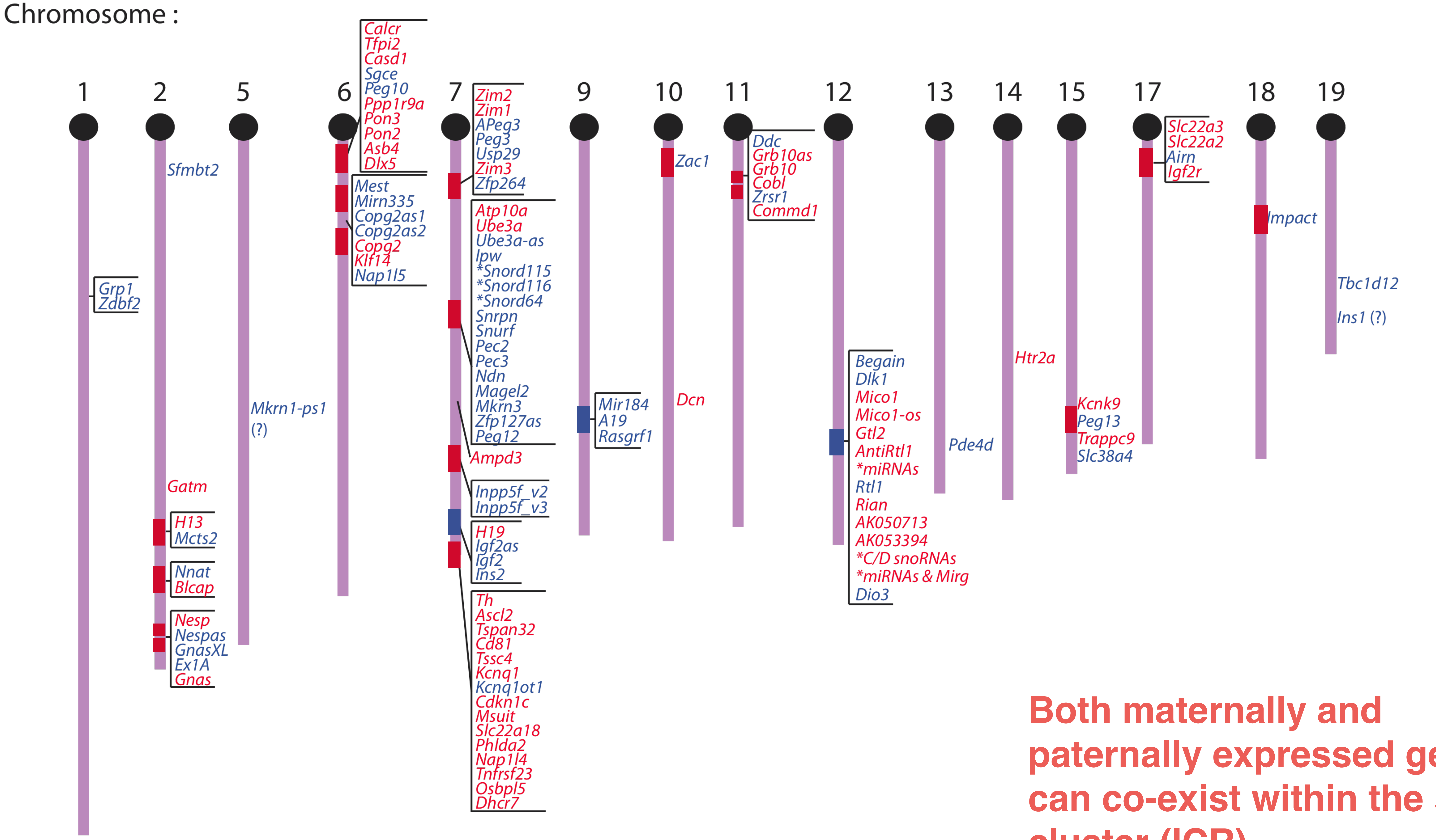
20 known ICRs



Dominant role of the oocyte in imprinting control!

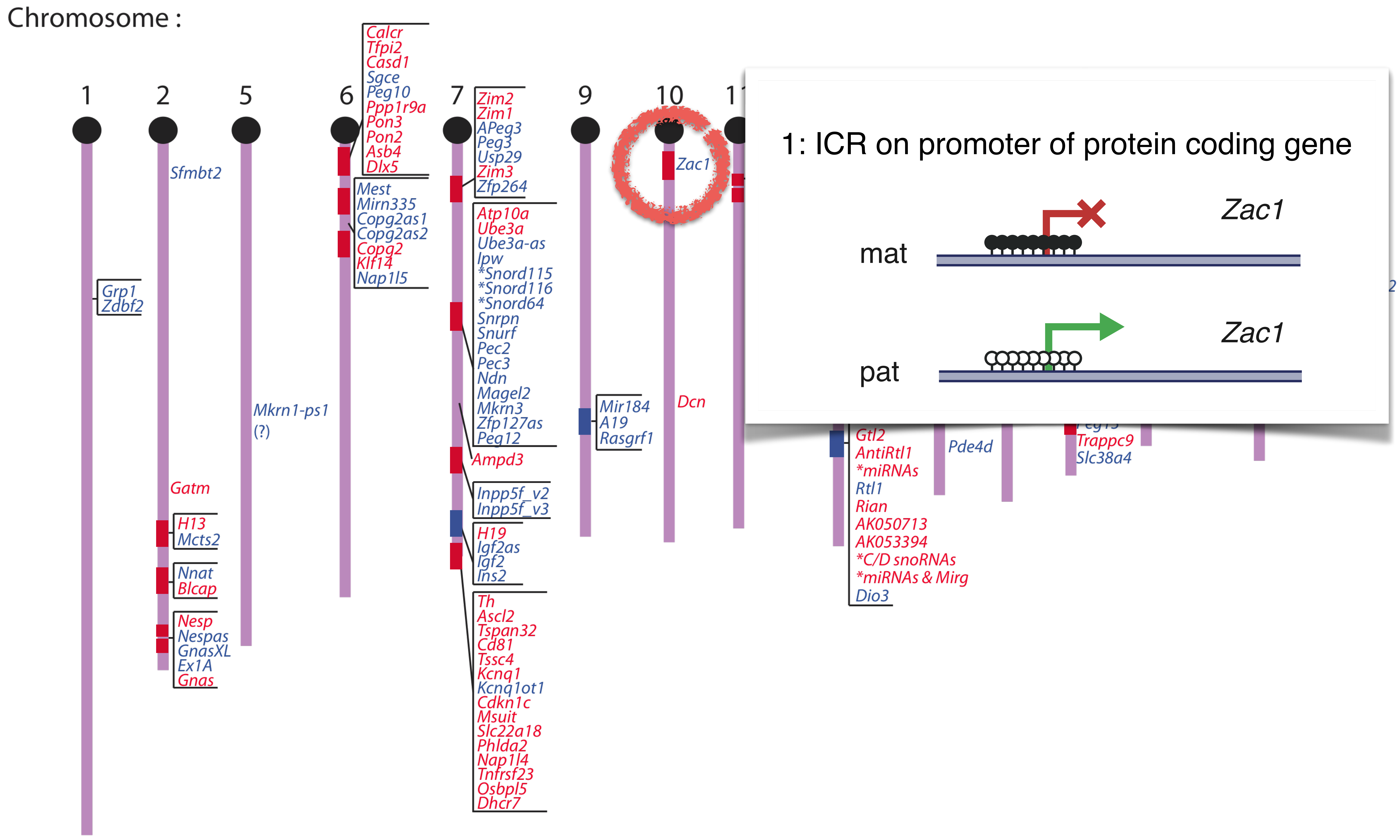


20 known ICRs

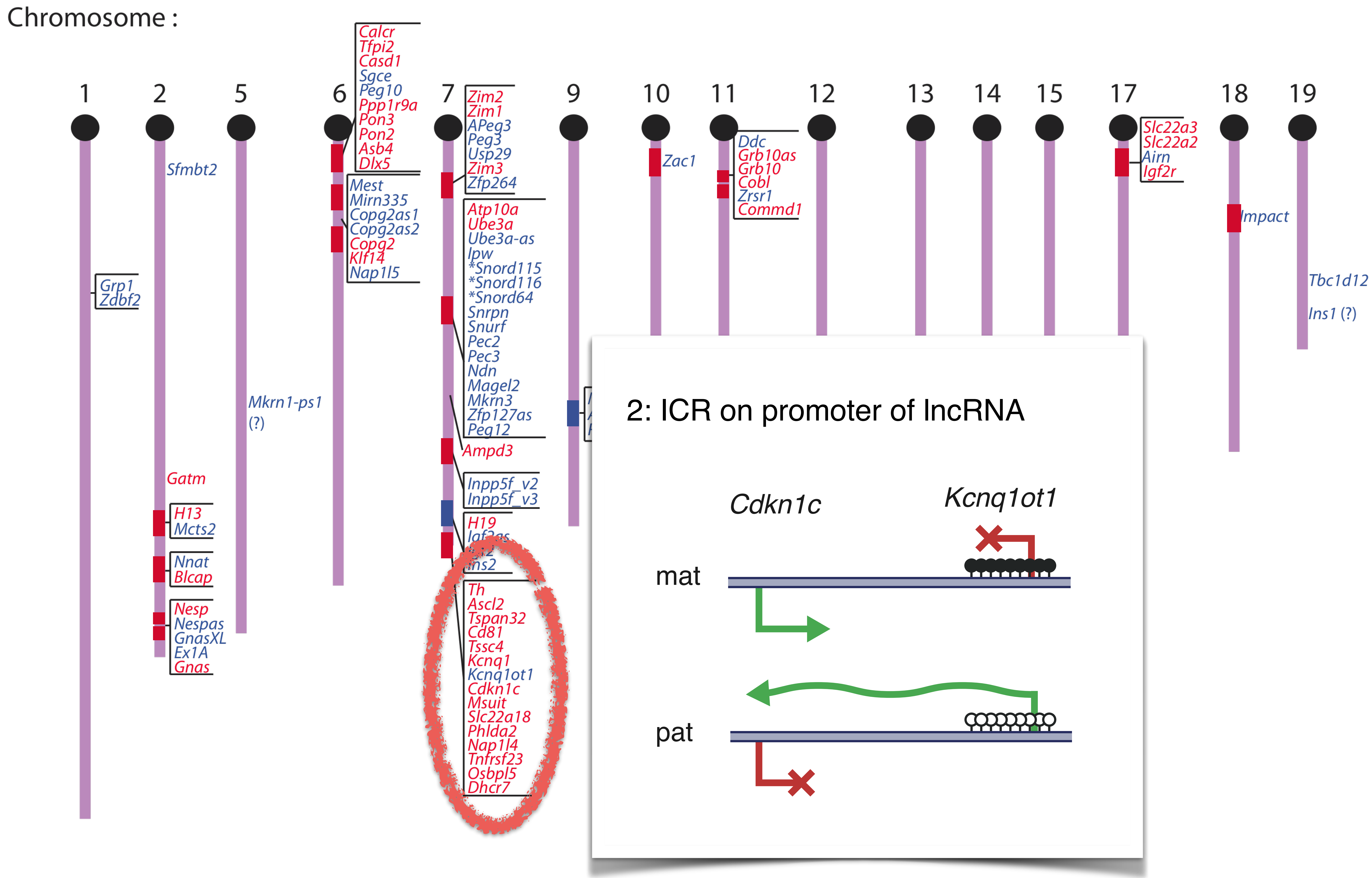


Both maternally and paternally expressed genes can co-exist within the same cluster (ICR)

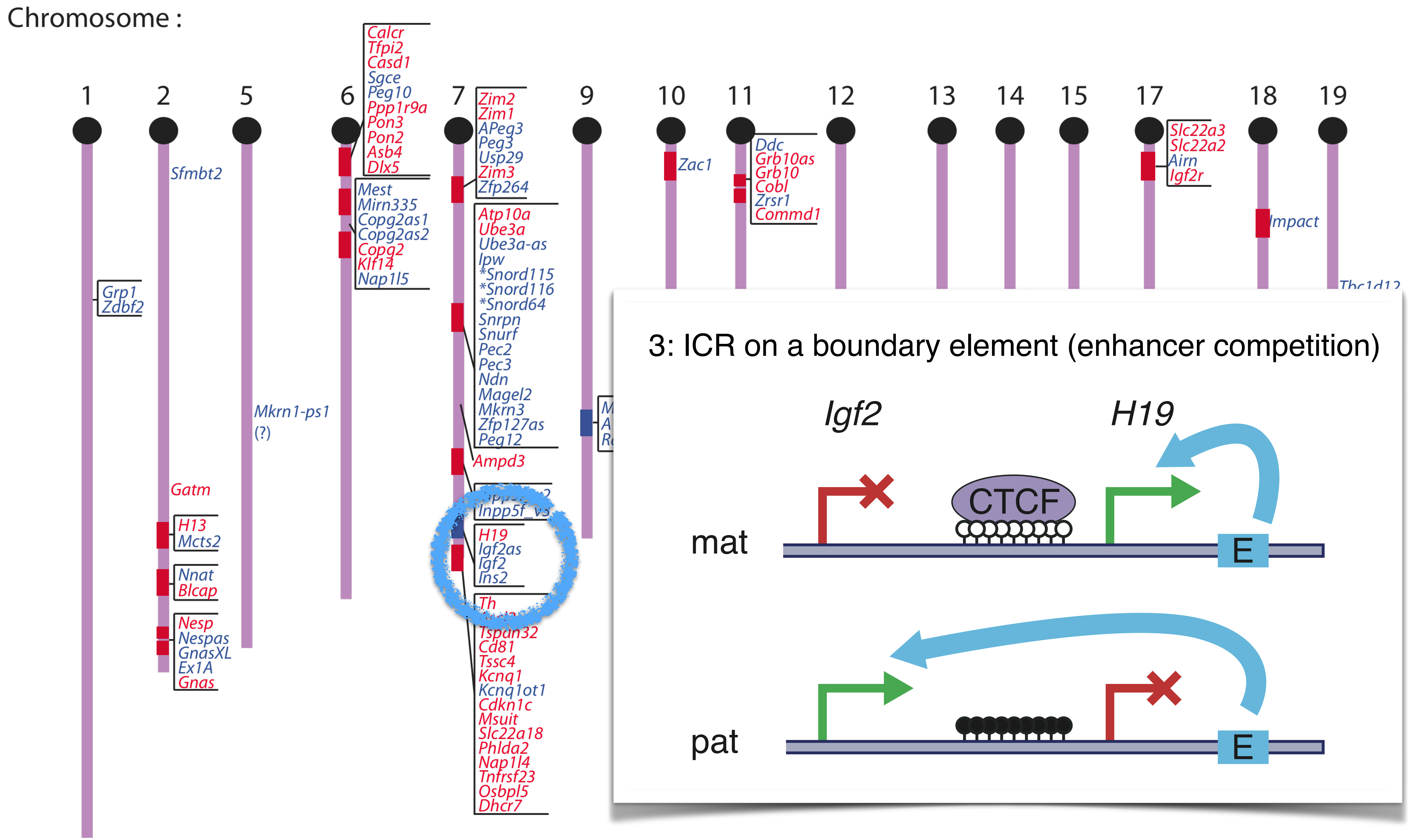
20 known ICRs



20 known ICRs



20 known ICRs



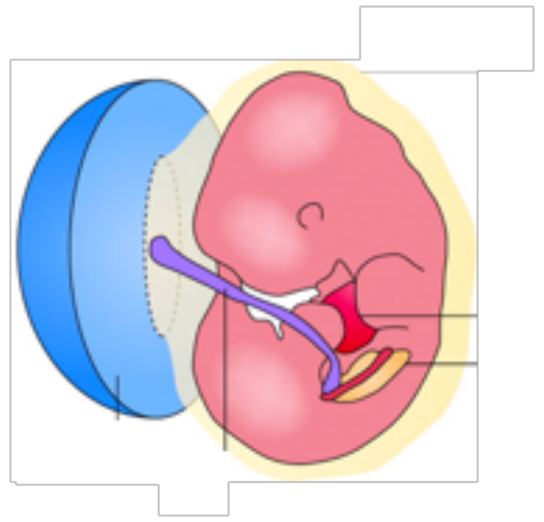
20 known ICRs

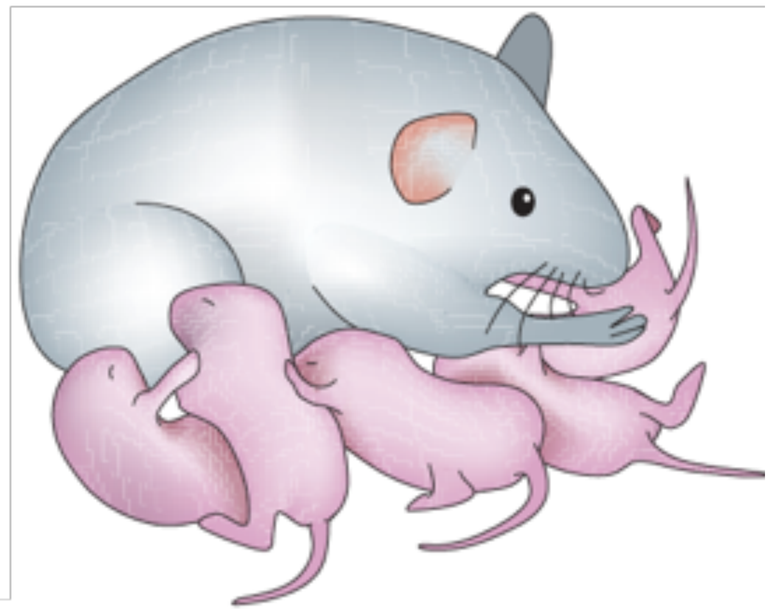
But they're important!

Mouse Phenotypes

- ✦ Growth & developmental defects

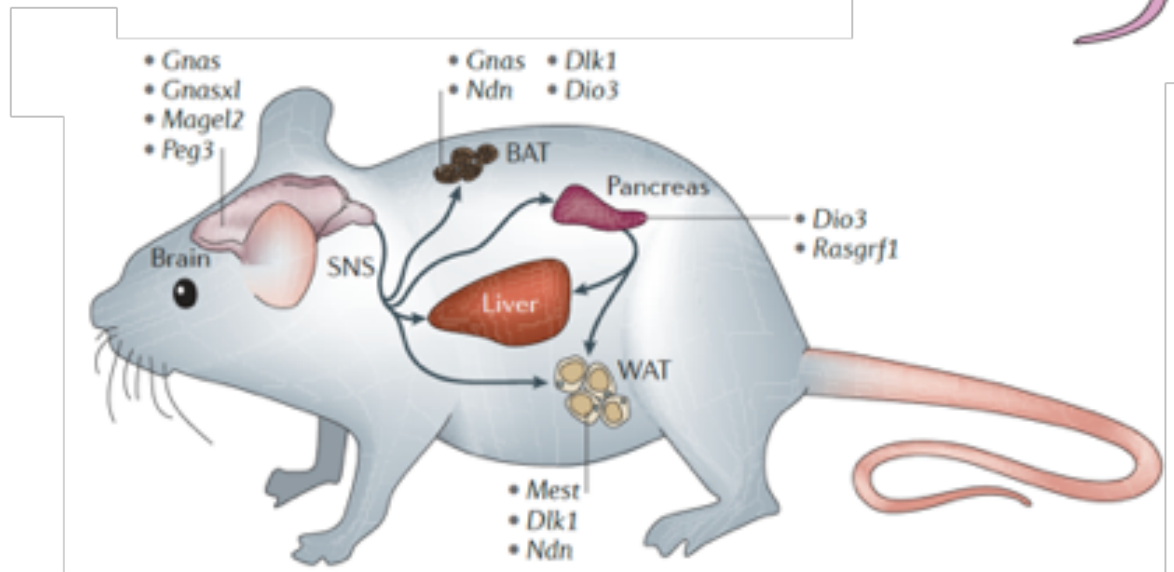
- Placental phenotype: embryonic lethality, growth restriction, alteration placental function
(*Peg10, Igf2, Cdkn1c, Phlda2, Slc22a3, Mash2…*)
- Foetal growth
(*Igf2, Igf2r, Dlk1, Grb10, Dio3, Peg3, Peg1, Zac1…*)
- Neonatal/ Post-natal growth
(*Igf2, Igf2r, H19, Rasgrf1, Grb10…*)





- ✦ Behaviour and neurological defects

- Neurogenesis
(*Dlk1*)
- Maternal care
(*Peg3, Nest*)
- Sleep
(*Gnas, Ube3a*)
- Memory
(*Gnas, Rasgrf1, Ube3a*)
- Social behaviour
(*Grb10, Nest*)
- Suckling (newborn)
(*Dlk1, Magel2, Peg3, GnasXL*)



Human Disease

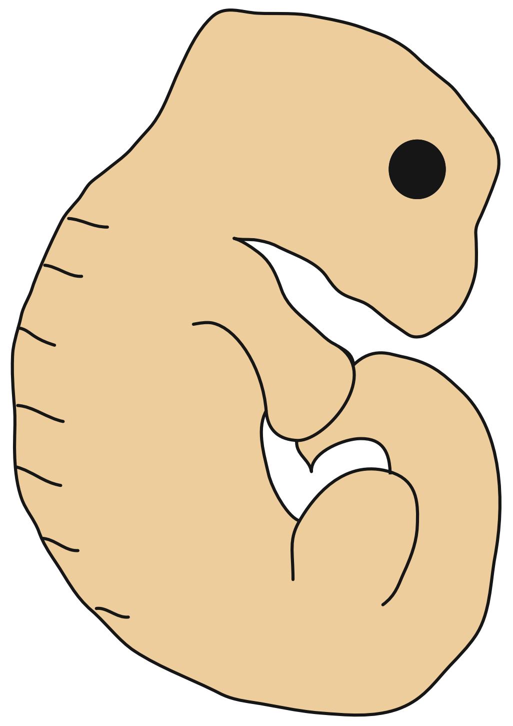
Syndrome	Clinical features	Etiology	Mouse chromosome
Angelman syndrome (AS)	Mental retardation, speech impairment, ataxia, seizure, microcephaly	15q11.2-q13 deletion (70%) PatUPD15 (7%), *UBE3A* mutation (11%), methylation defects (3%), epimutation	7C
Prader–Willi syndrome (PWS)	Neonatal hypotonia, childhood obesity, cognitive impairment, behavioral characteristics, hypogonadism	De novo paternal deletion in 15q11-q13 (70%), MatUPD15 (29%), imprinting defects (1%)	7C
Beckwith–Wiedemann syndrome (BWS)	Pre/postnatal overgrowth, neonatal hypoglycemia, exompholos, macroglossia, hemihypertrophy, increased embryonal tumors	Epimutation of *IGF2/H19* DMR1, epimutation of *KCNQ1/CDKN1C* DMR2 both on 11p15, hypomethylation of DMR2 (50%), hypermethylation DMR1 (2%–7%), PatUPD11, *CDKN1C* mutation	7F5
Silver–Russell syndrome (SRS)	Intrauterine/postnatal growth retardation, variable features (inc. 5th finger clinodactyl, learning disabilities)	Paternal DMR1 hypomethylation at 11p15 (>50%), MatUPD7 (5%) Matdup11p15, unknown (30%)	7F5
Maternal UPD14 (and UPD14 mat-like) syndrome	Low birth weight, short stature, characteristic facies, premature puberty, hypotonia	MatUPD14, paternal microdeletions at 14q32.2, hypomethylated DMRs at *DLK1/GTL2*	12F1
Paternal UPD14 (and UPD14 pat-like) syndrome	Bell-shaped thoracic cage, mental retardation, placentomegaly, polyhydramnios	PatUPD 14, maternal microdeletions at 14q32.2, hypermethylation at DMRs at *DLK1/GTL2*	12F1
Pseudo-hypoparathyroidism 1b	Resistance to parathyroid hormone, hypocalcaemia, hyperphophatemia	Microdeletion upstream of *GNAS* at 20q, maternal hypomethylation, PatUPD20	2H4
Transient neonatal diabetes mellitus	Growth retardation, hyperglycemia with low/undetectable insulin resolved by 6 months old, 40% Type2 diabetes later in life	Paternal UPD6, paternal duplication 6q22-q23, maternal hypomethylation at *ZAC1/PLAGL1* DMR	10A2

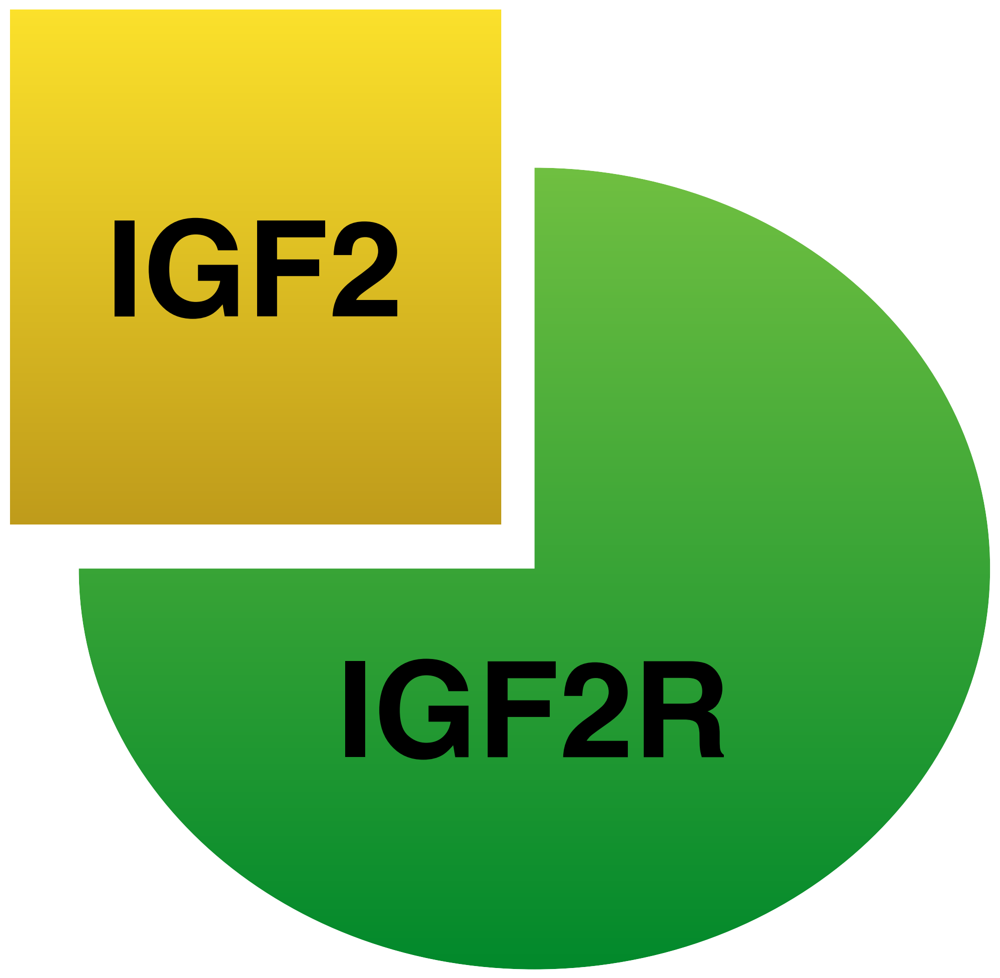
Bartolomei and Ferguson-Smith, *CSH Perspectives*, 2011

One *theory* for imprinting: Genetic Conflict

Dad wants one thing from his offspring (e.g. bigger)
Mom wants another (e.g., smaller)

Example: Insulin-like growth factor 2 (IGF2) and receptor





Alleles from mom

IGF2	*Off*
IGF2 Receptor	*On*

Alleles from dad

IGF2	*On*
IGF2 Receptor	*Off*

One *theory* for imprinting: Genetic Conflict

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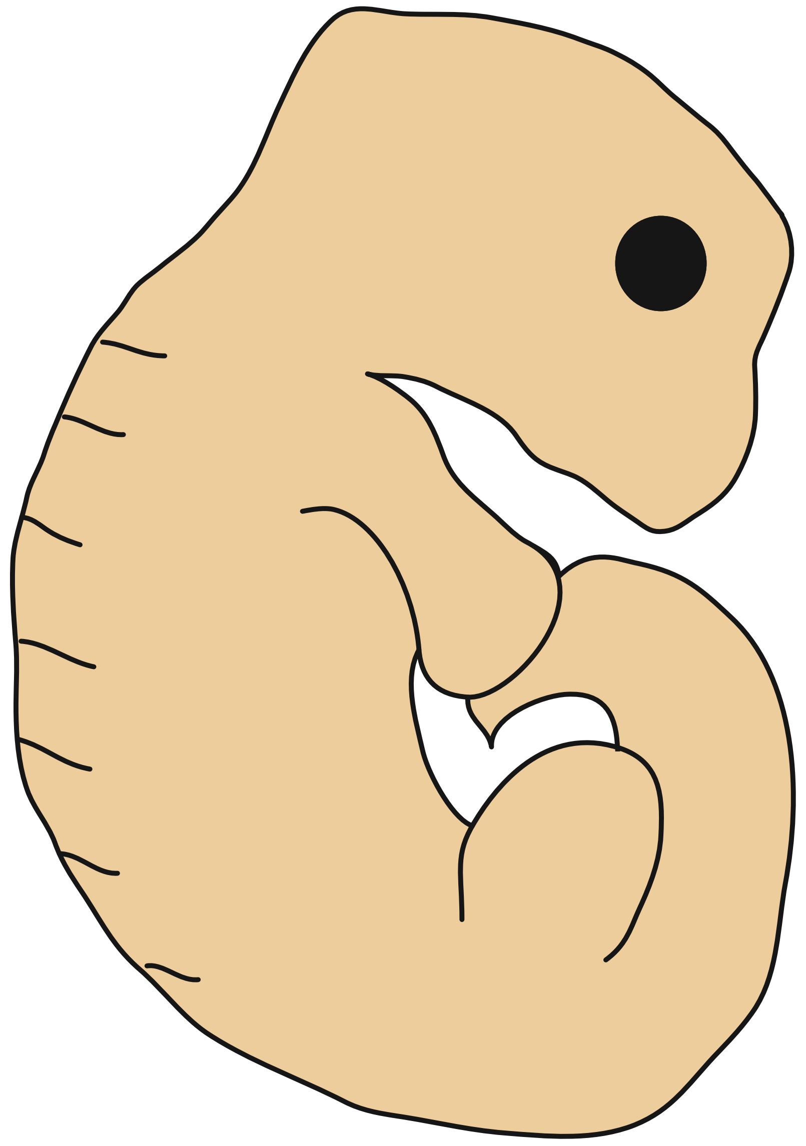
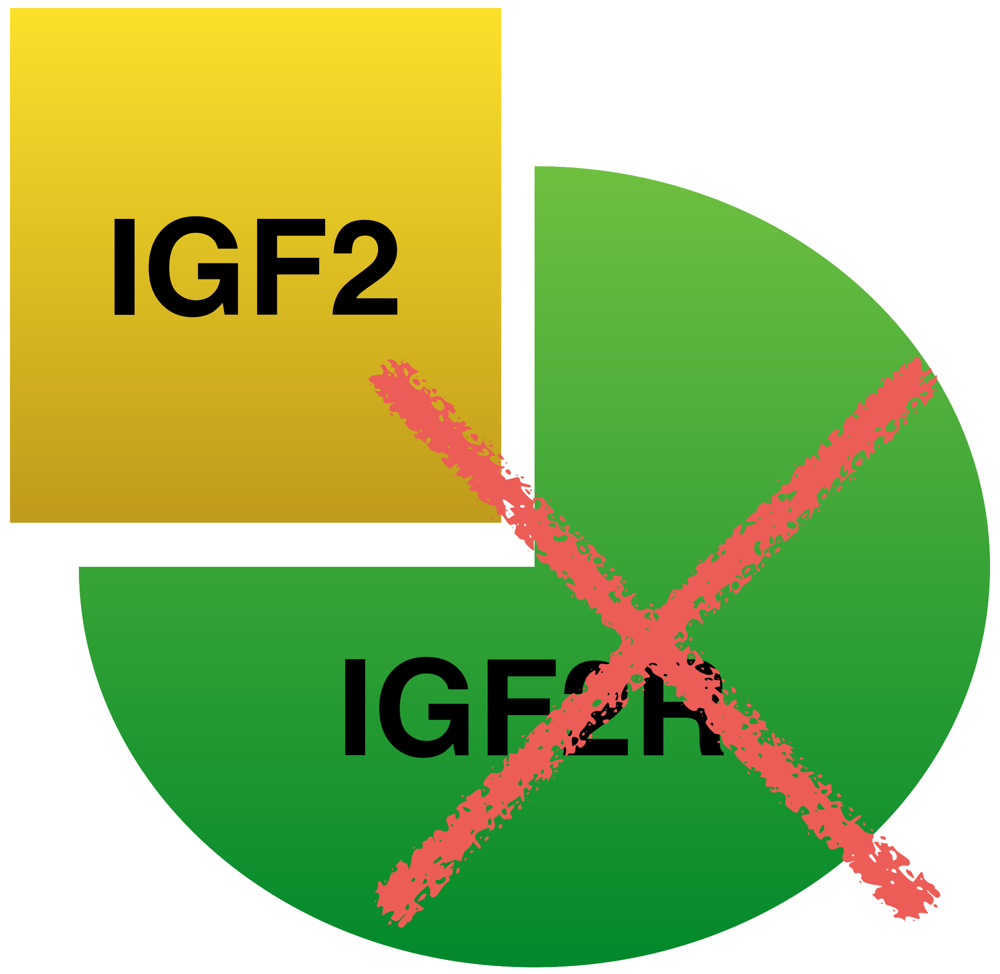
Example: Insulin-like growth factor 2 (IGF2) and receptor





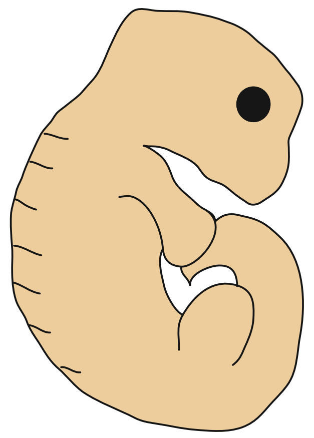
Alleles from mom
IGF2 *Off*
~~IGF2 Receptor *On*~~

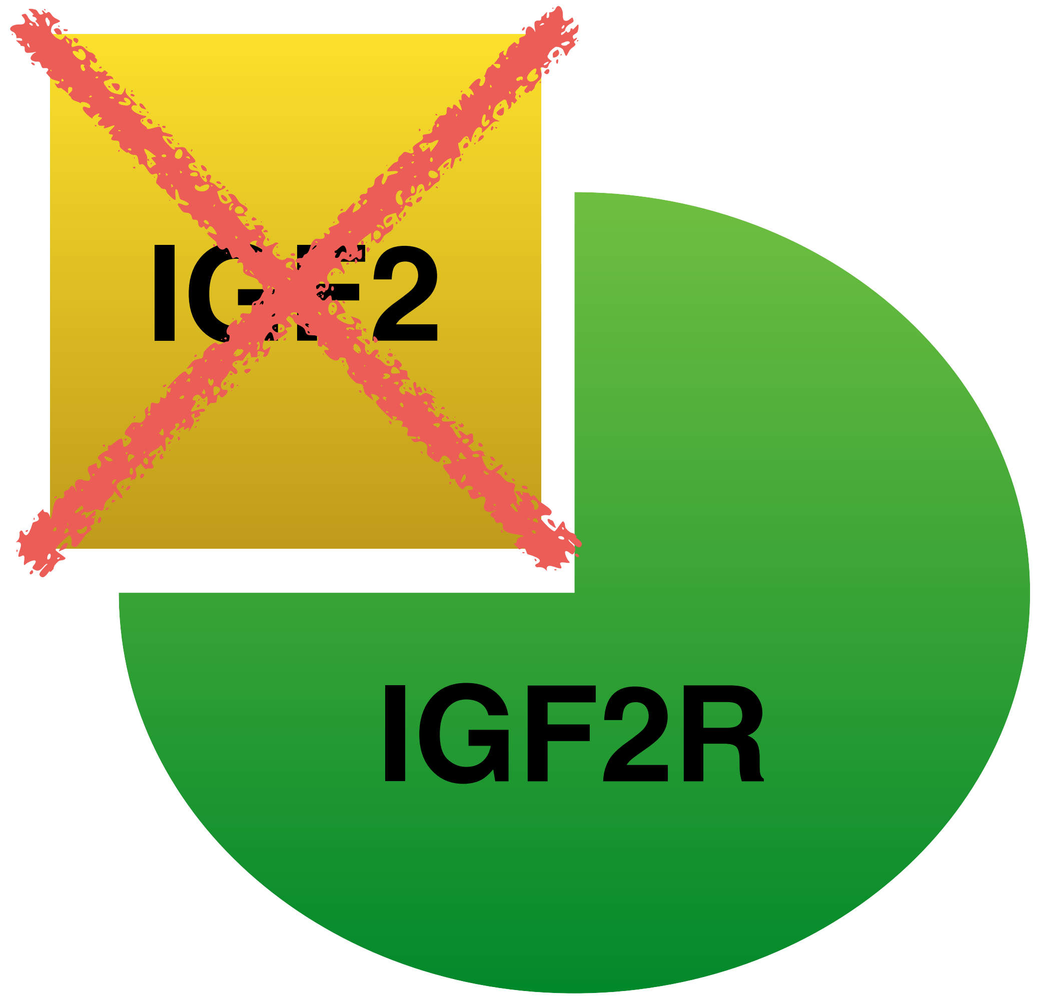
Alleles from dad
IGF2 *On*
IGF2 Receptor *Off*

One *theory* for imprinting: Genetic Conflict

Dad wants one thing from his offspring (e.g. bigger)
Mom wants another (e.g., smaller)

Example: Insulin-like growth factor 2 (IGF2) and receptor





Alleles from mom
IGF2 *Off*
IGF2 Receptor *On*

Alleles from dad
~~IGF2 *On*~~
IGF2 Receptor *Off*

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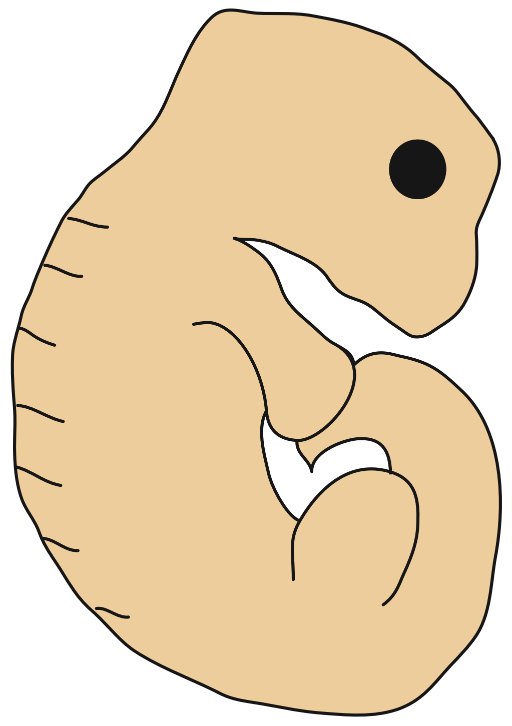
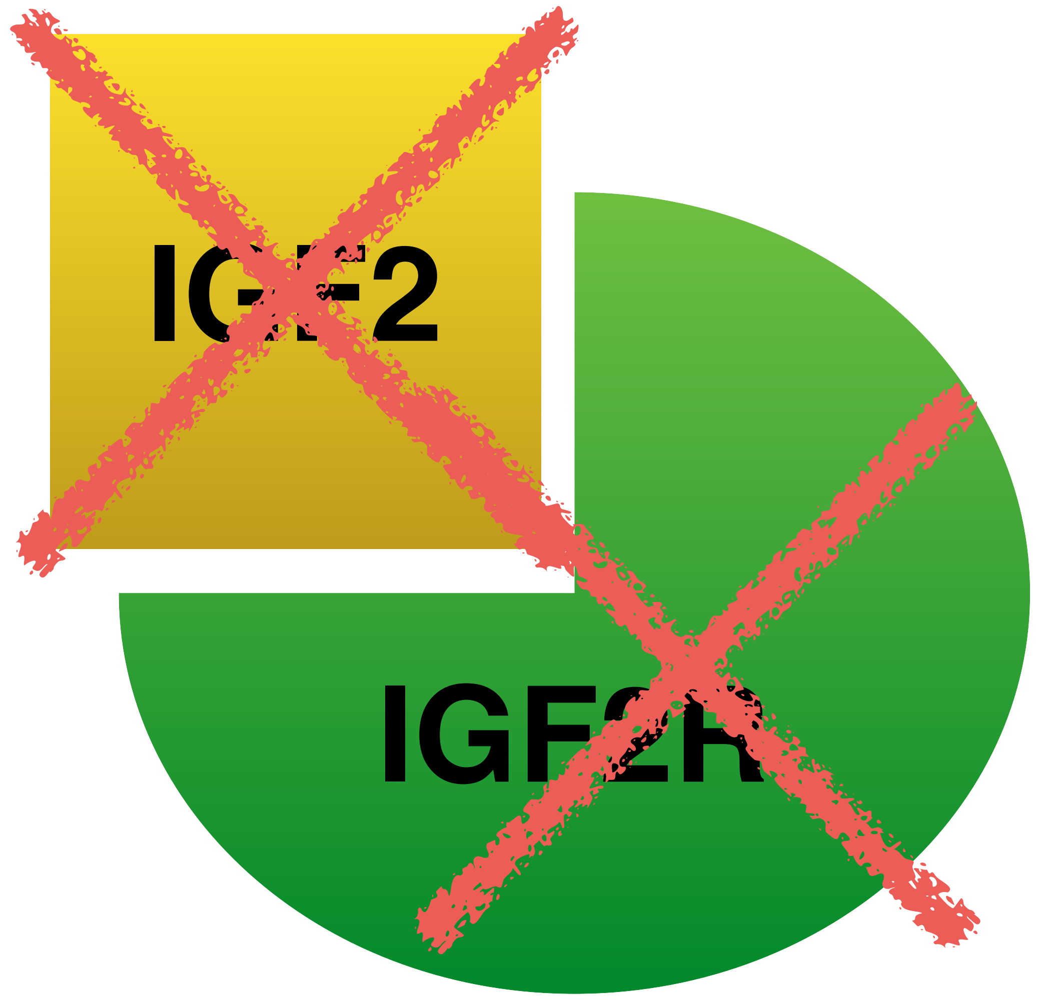


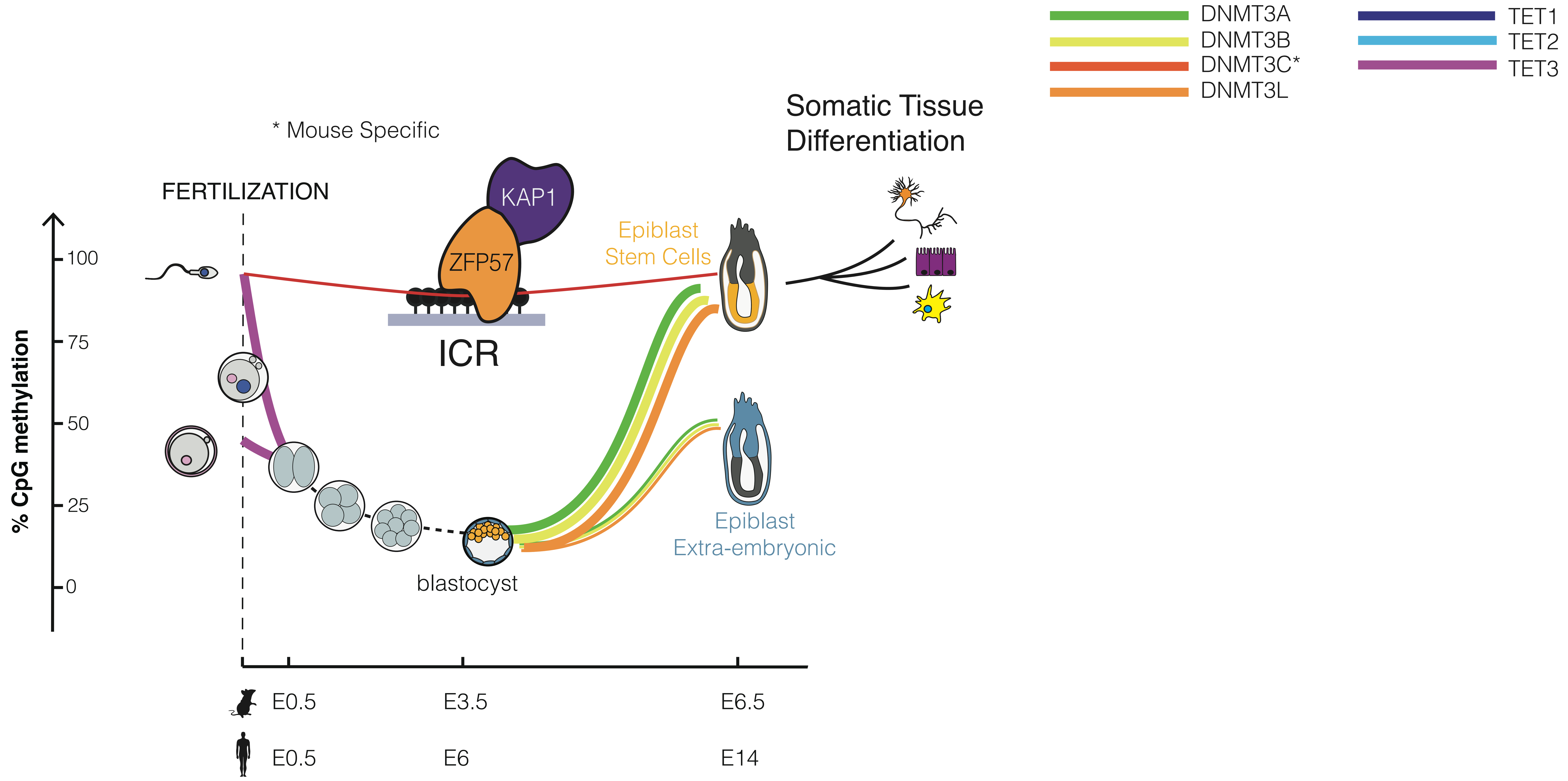


Alleles from mom
IGF2 *Off*
~~IGF2 Receptor *On*~~

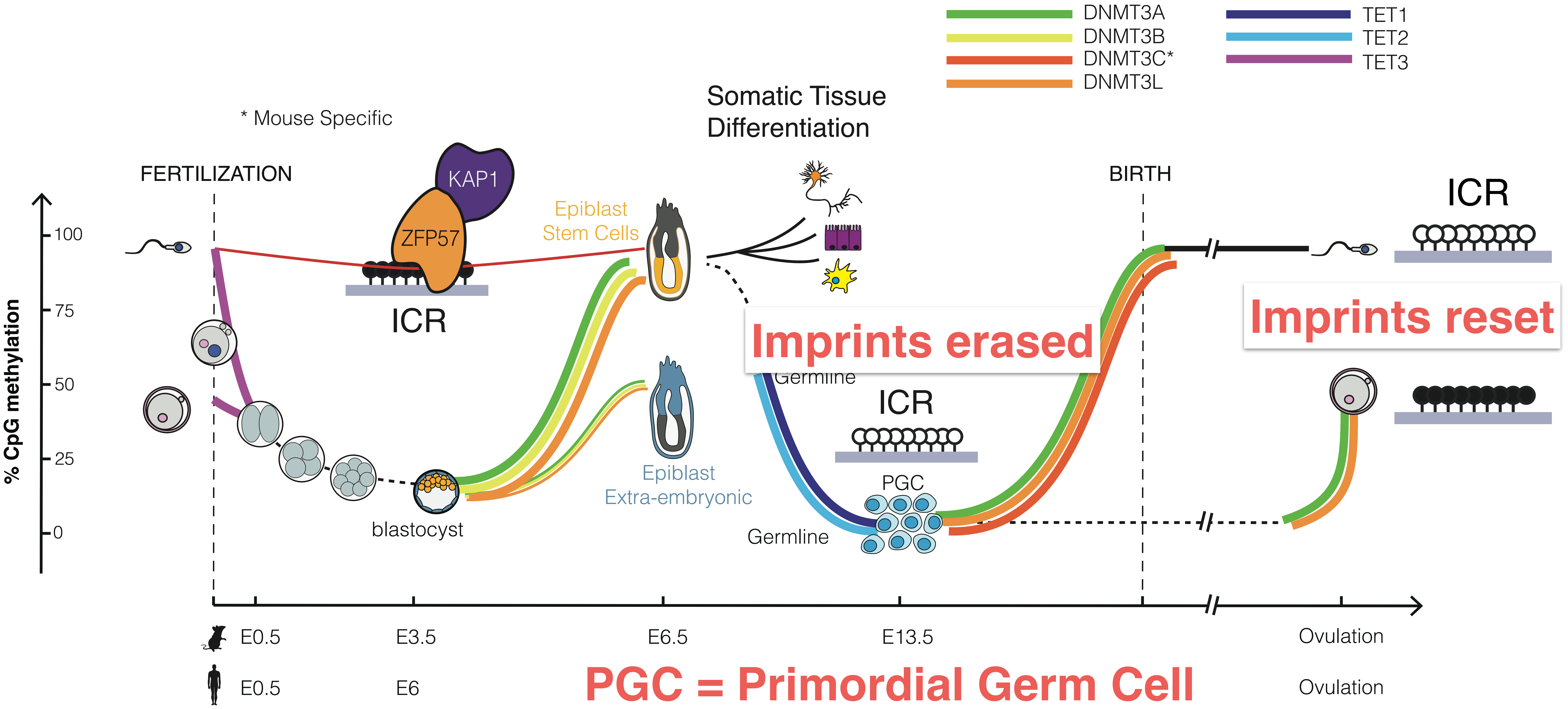
Alleles from dad
~~IGF2 *On*~~
IGF2 Receptor *Off*

Can imprints perisist “transgenerationally”?

NO!

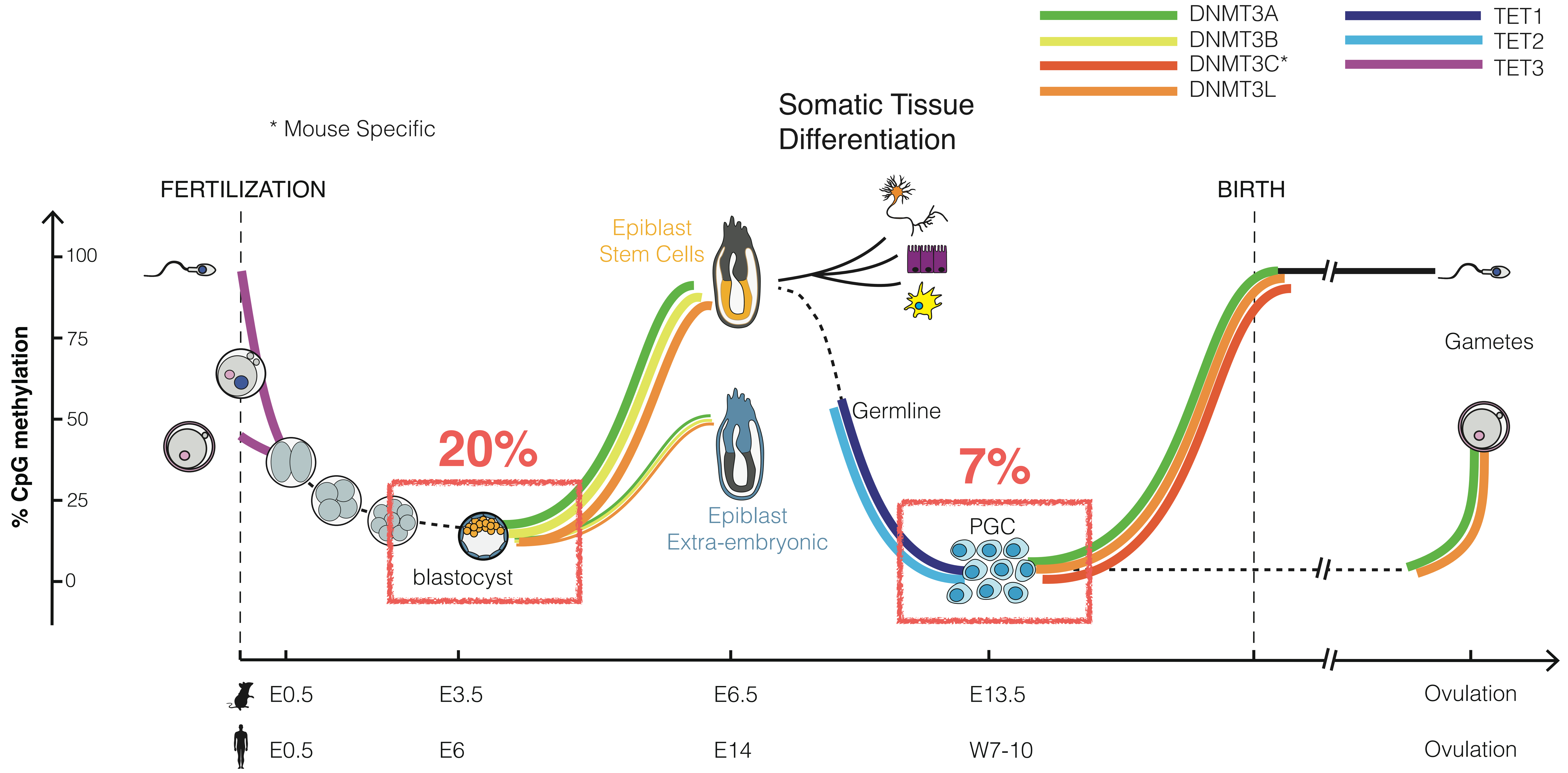


Second wave of embryonic reprogramming in germline

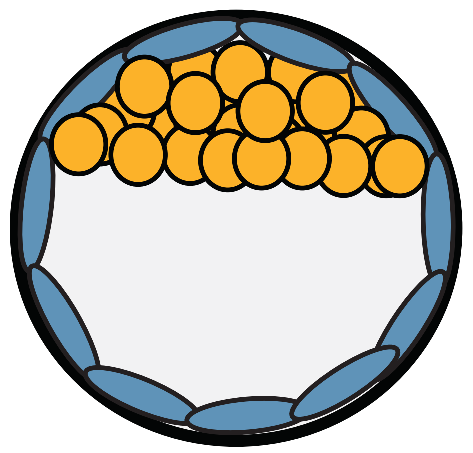


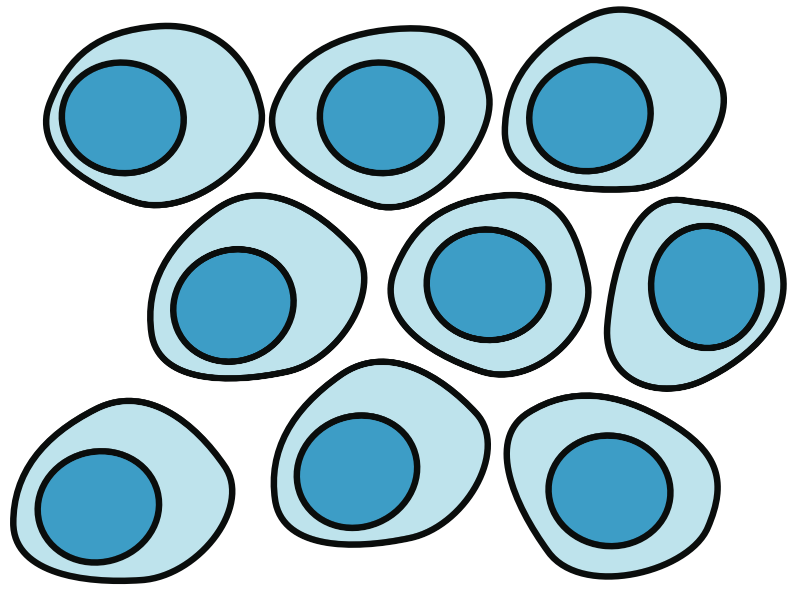
PGC = Primordial Germ Cell

Can *any* epigenetic signatures persist transgenerationally?

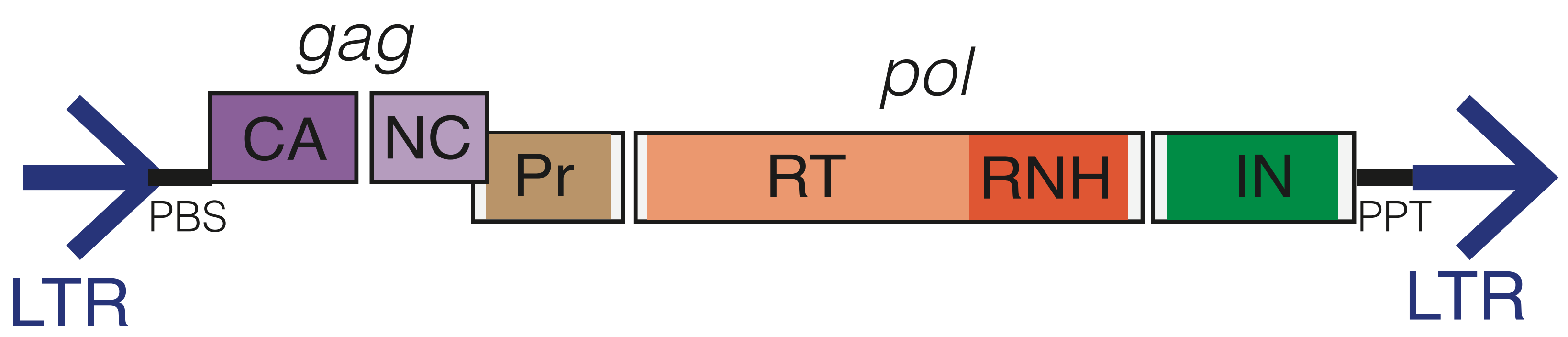


Residually methylated sequences are mainly retrotransposons



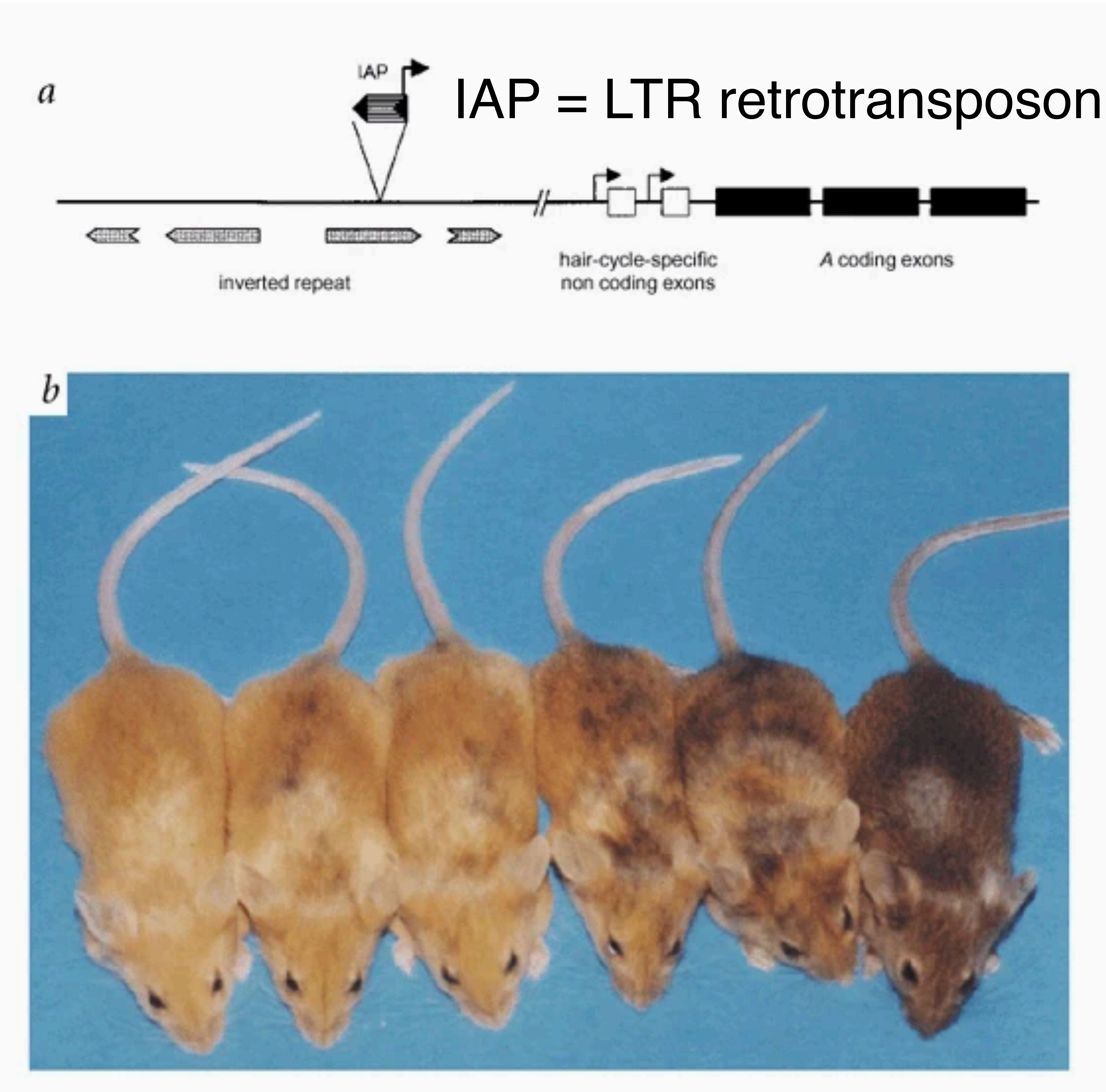


LTR Retrotranspson



Can these TEs be vectors of epigenetic inheritance?

Residually methylated sequences are mainly retrotransposons



Morgan et al, *Nat Gen* 1999

- Agouti viable yellow (A^{vy})
- Expression of IAP causes ectopic *A* expression and yellow fur
- IAP methylation and silencing silencing results in wild-type fur
- Maternal methylation status is heritable

Residually methylated sequences are mainly retrotransposons

Article

Cell 2018

Identification, Characterization, and Heritability of Murine Metastable Epialleles: Implications for Non-genetic Inheritance

Authors

Anastasiya Kazachenka,
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"Only in rare instances do they act as promoters controlling adjacent gene expression…Variably methylated [TEs] are **reprogrammed** after fertilization and **re-established** as variable loci in the next generation….challenging the generalizability of non-genetic inheritance at these regions"

Summary Part III

- Parents can exert epigenetic influence to progeny via imprinting mechanism (intergenerationl epigenetic inheritance)

- Imprints are erased and reset in germline

- Residual methylation during reprogramming is mainly at TEs

- No strong evidence that DNA methylation-based regulation can be transmitted transgenerationally

Questions We Will Address Today

I. What is epigenetic reprogamming?

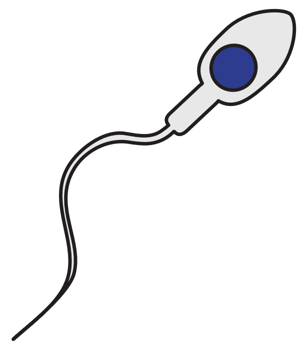
II. How does epigenetic reprogramming occur?

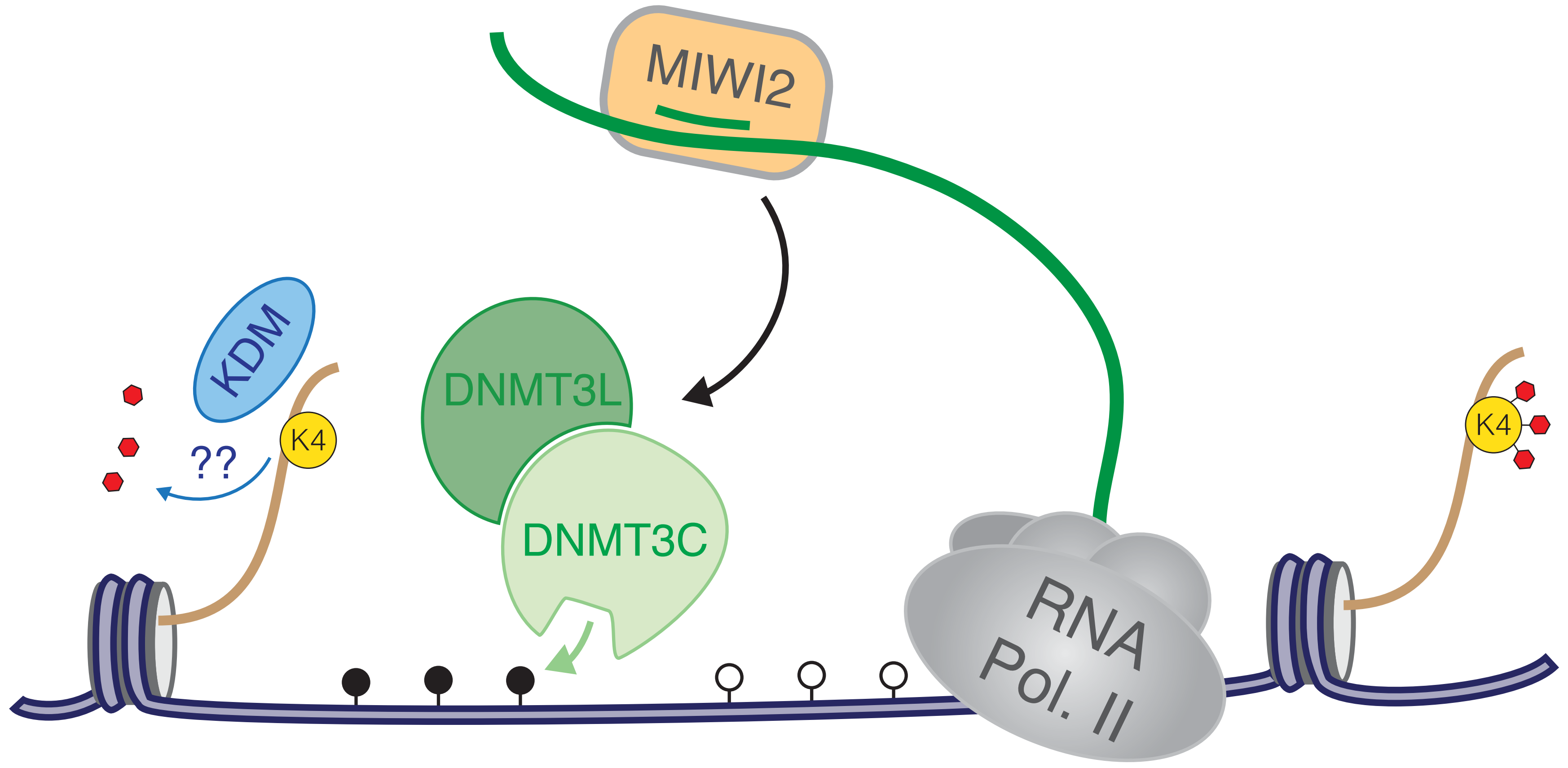
III. Can any regions of the genome escape reprogramming?

IV. **Why** does epigenetic reprogramming occur????

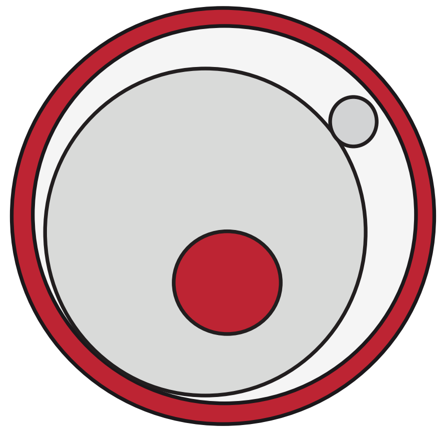
Male and Female Methylomes Highly Divergent

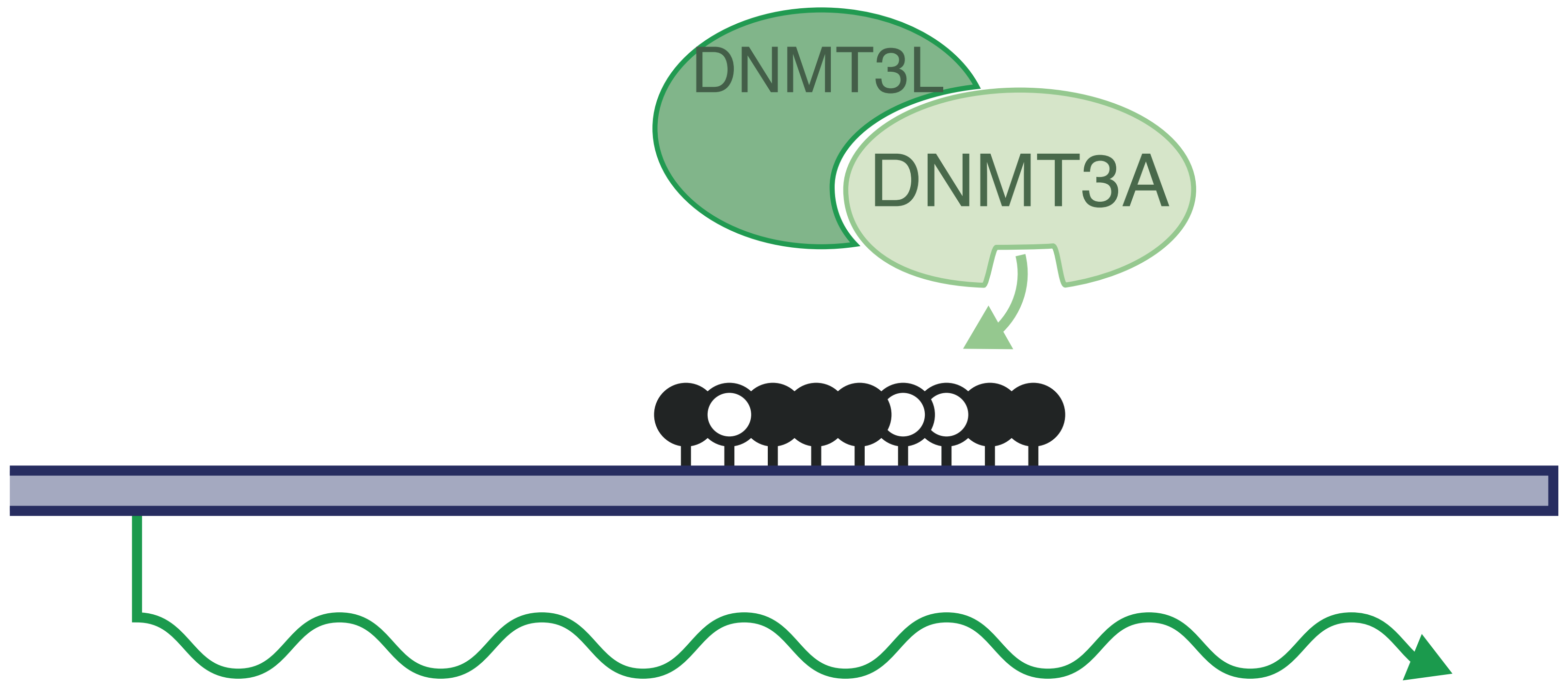
Mechanistically





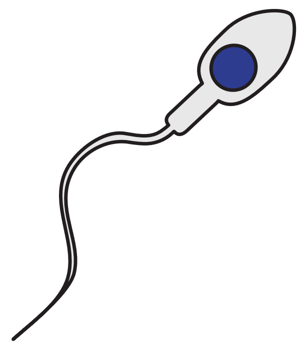
Small-RNA directed DNA methylation of TEs (mice)

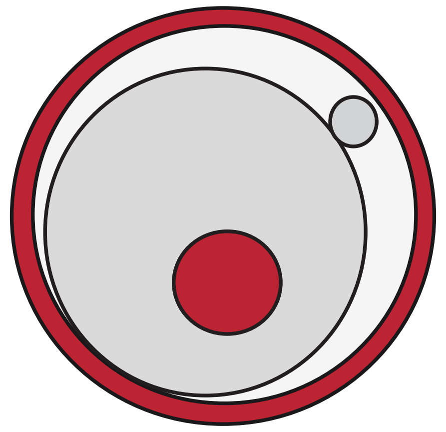




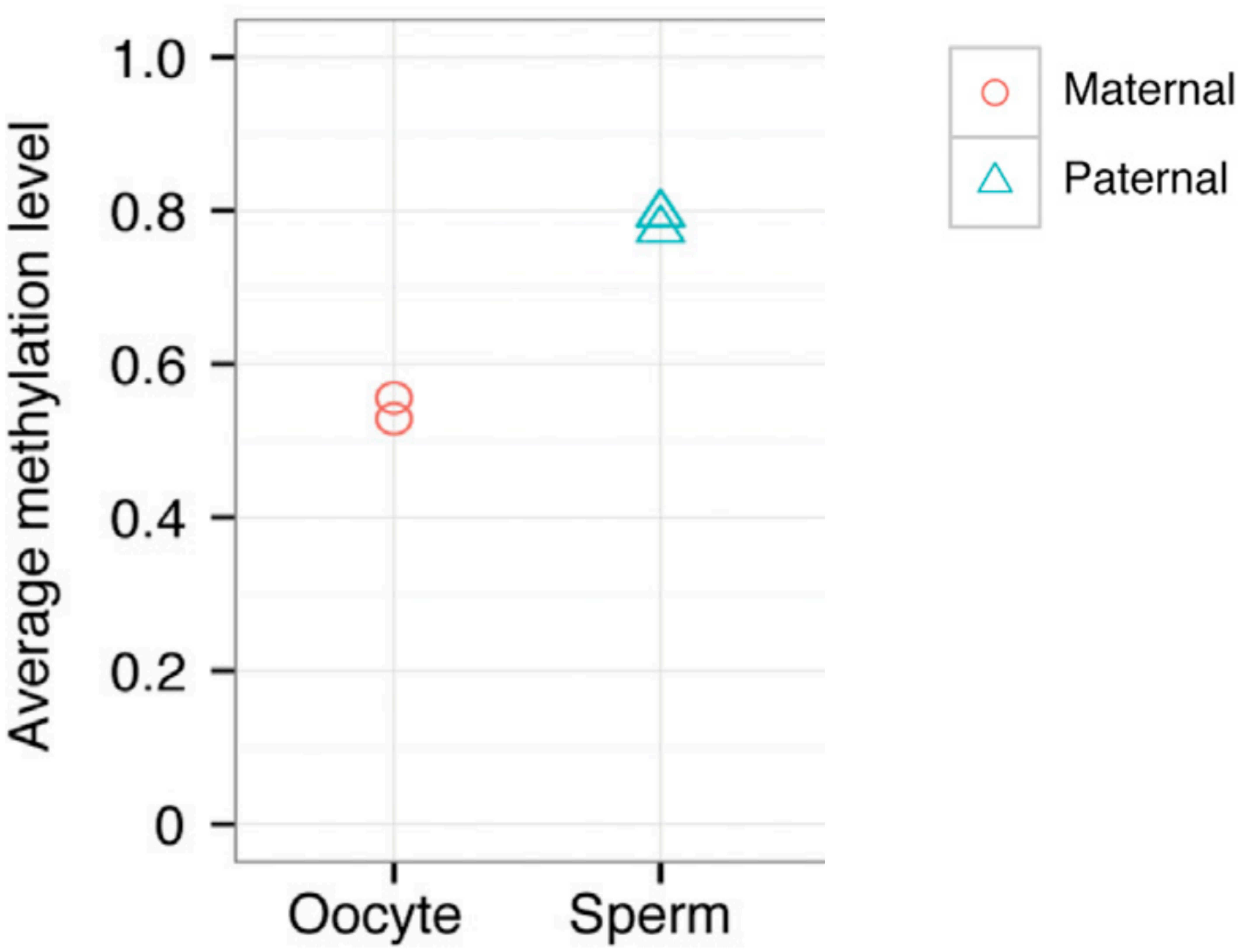
Transcription dependent DNA methylation in gene bodies

Male and Female Methylomes Highly Divergent

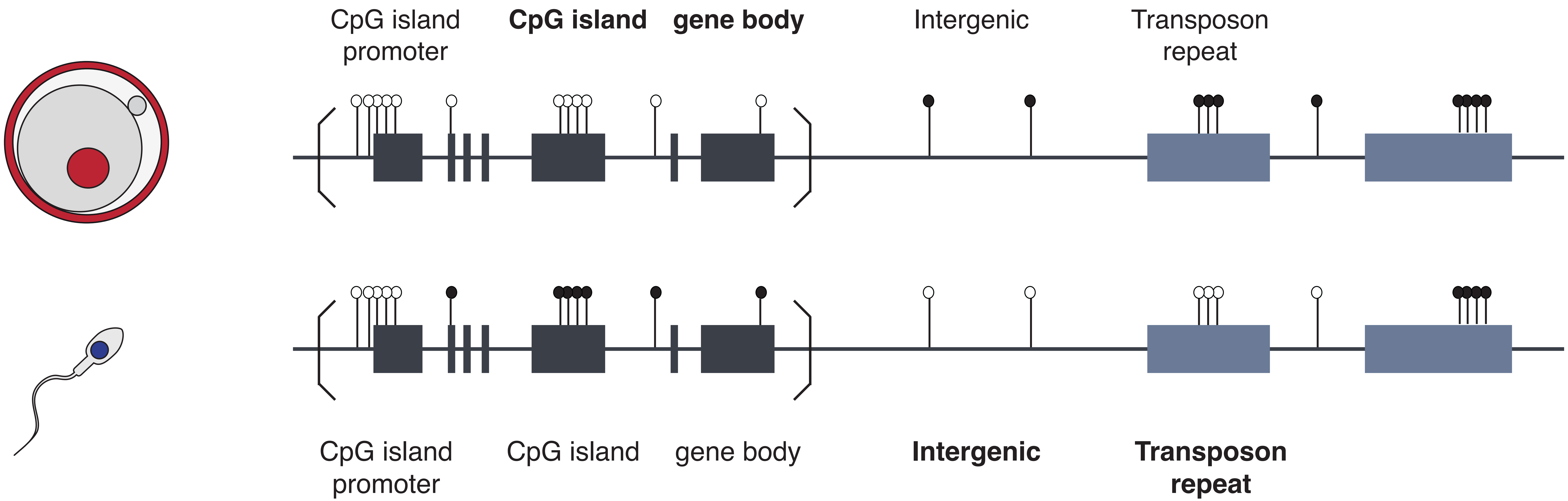




Global Levels

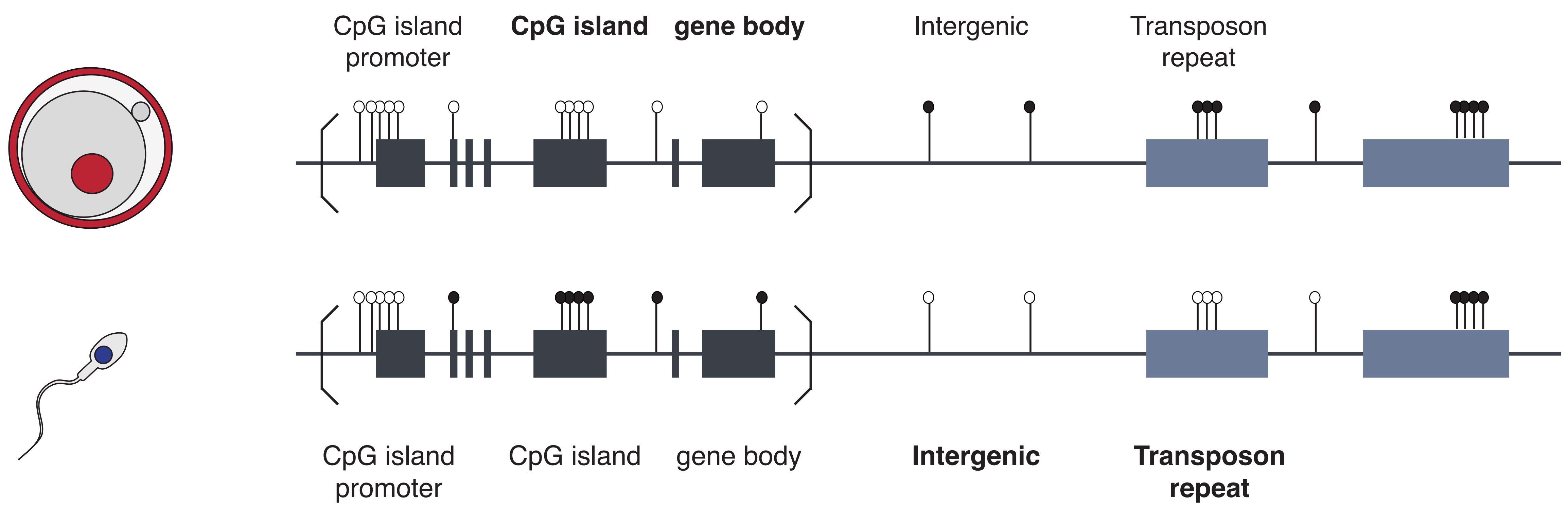


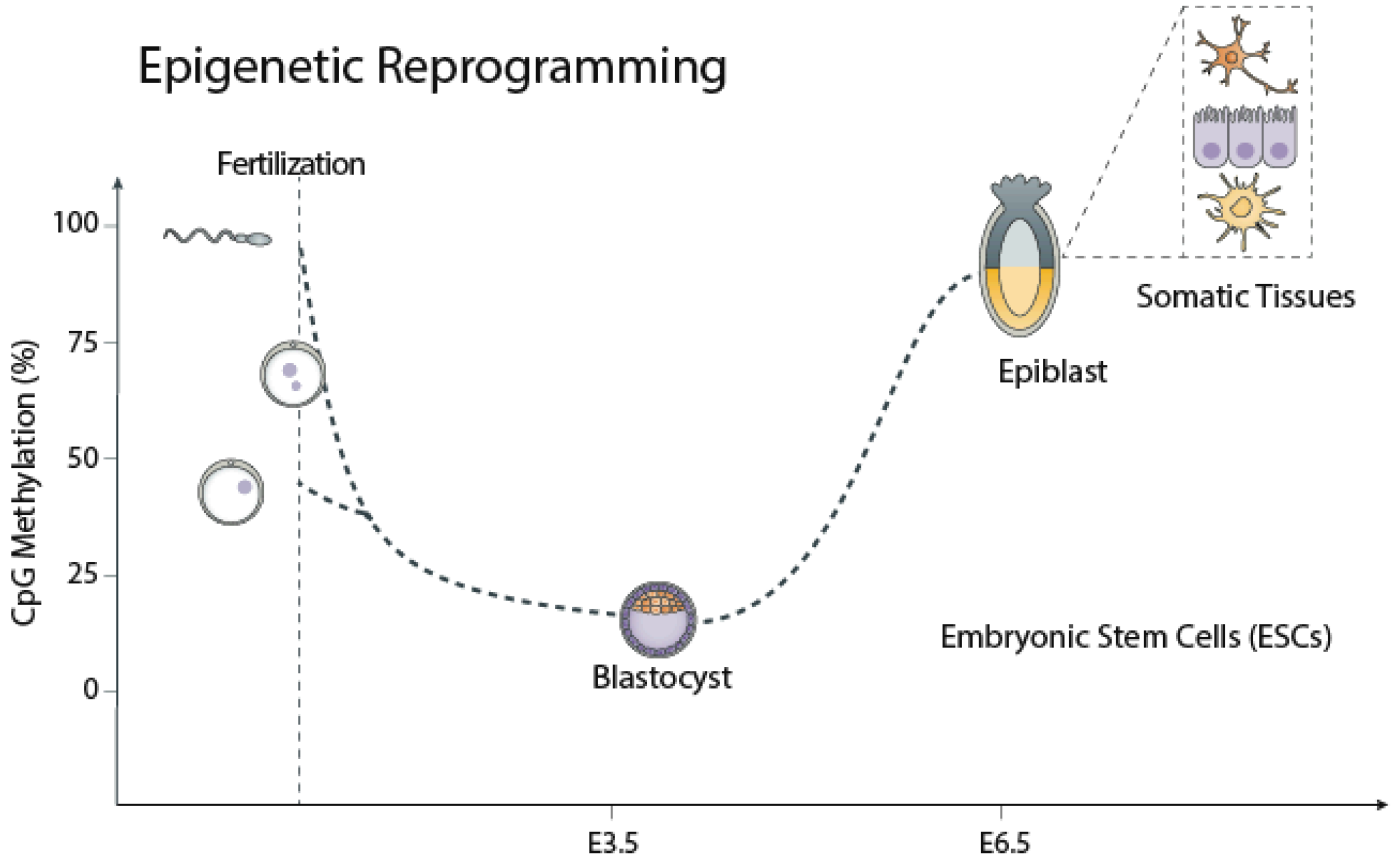
DNA methylation patterns: Fertilization



● Methylated CpG
○ Unmethylated CpG

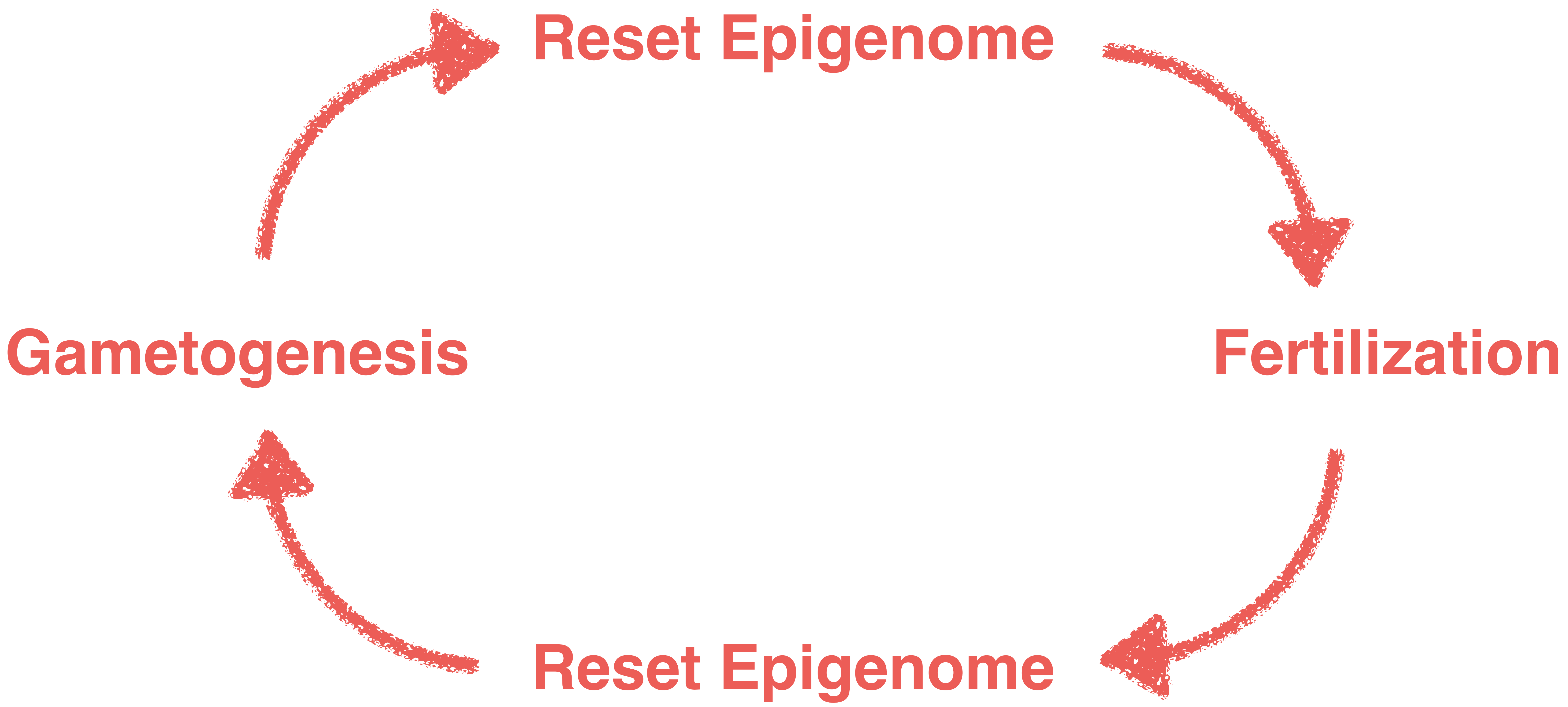
DNA methylation patterns: Fertilization



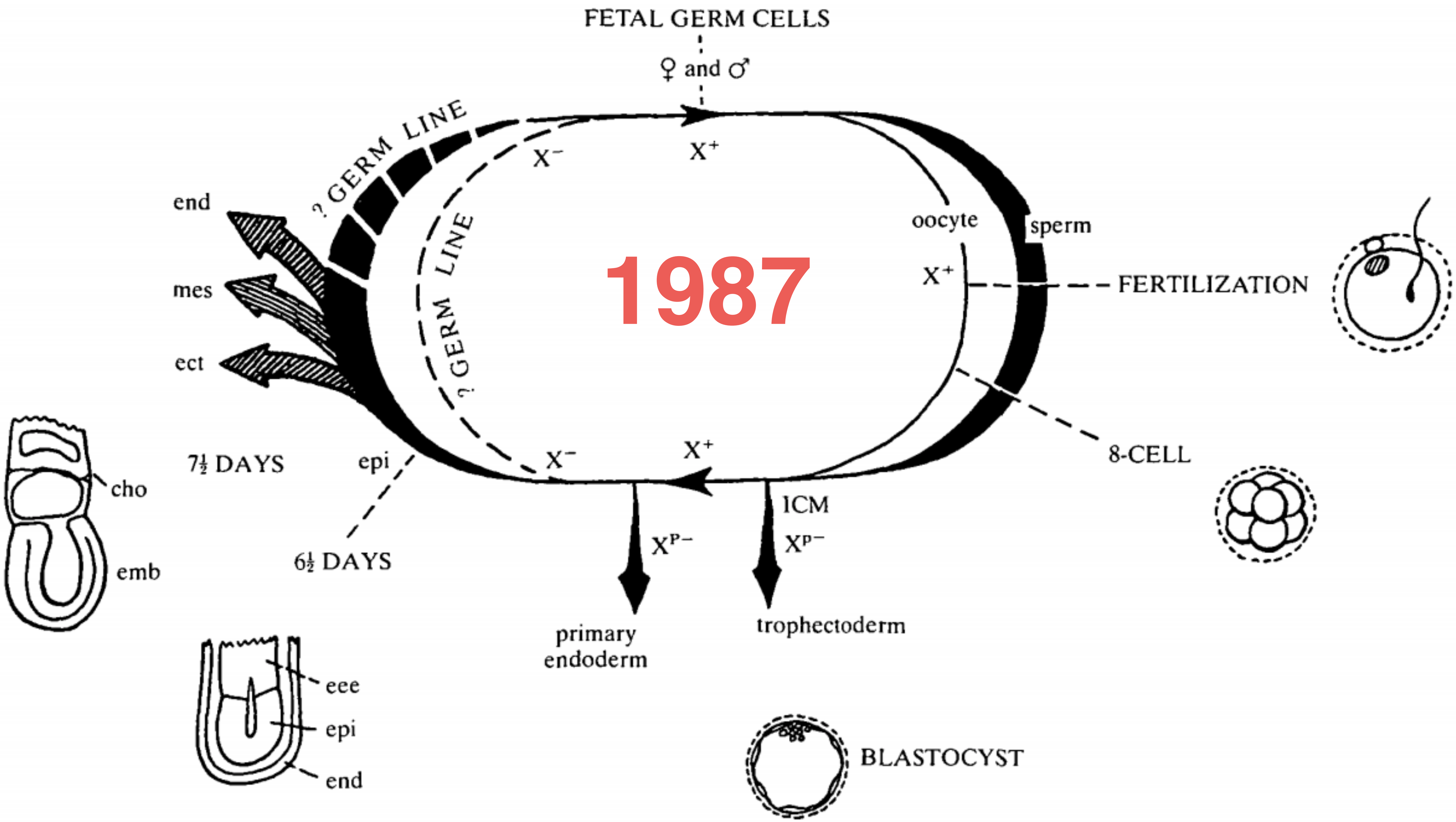


Embryo *must* level methylation landscape to reduce gene dosage discrepancies

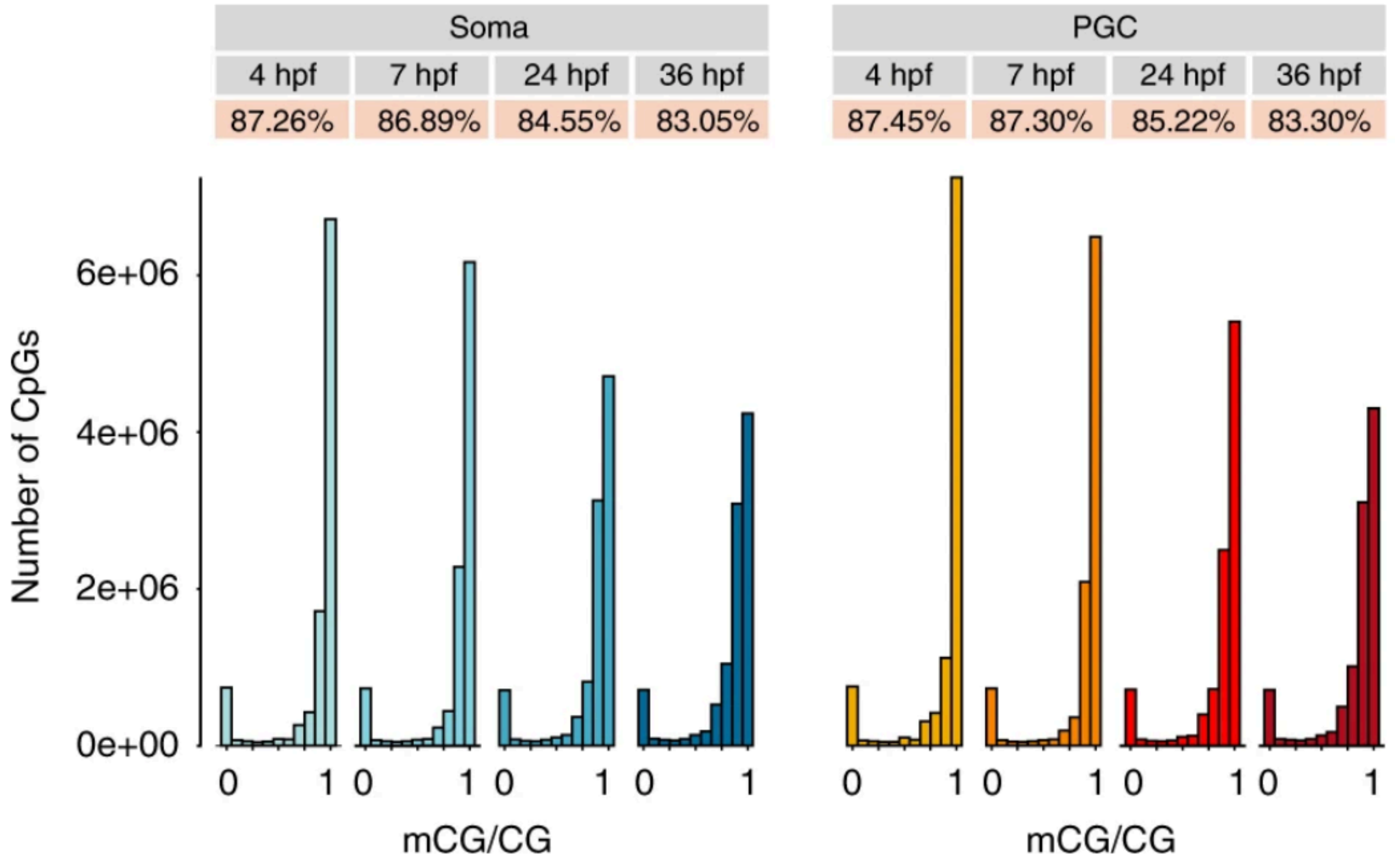
Perpetual Cycle



Perpetual Cycle



Epigenetic Reprogramming: Peculiar Phenomenon

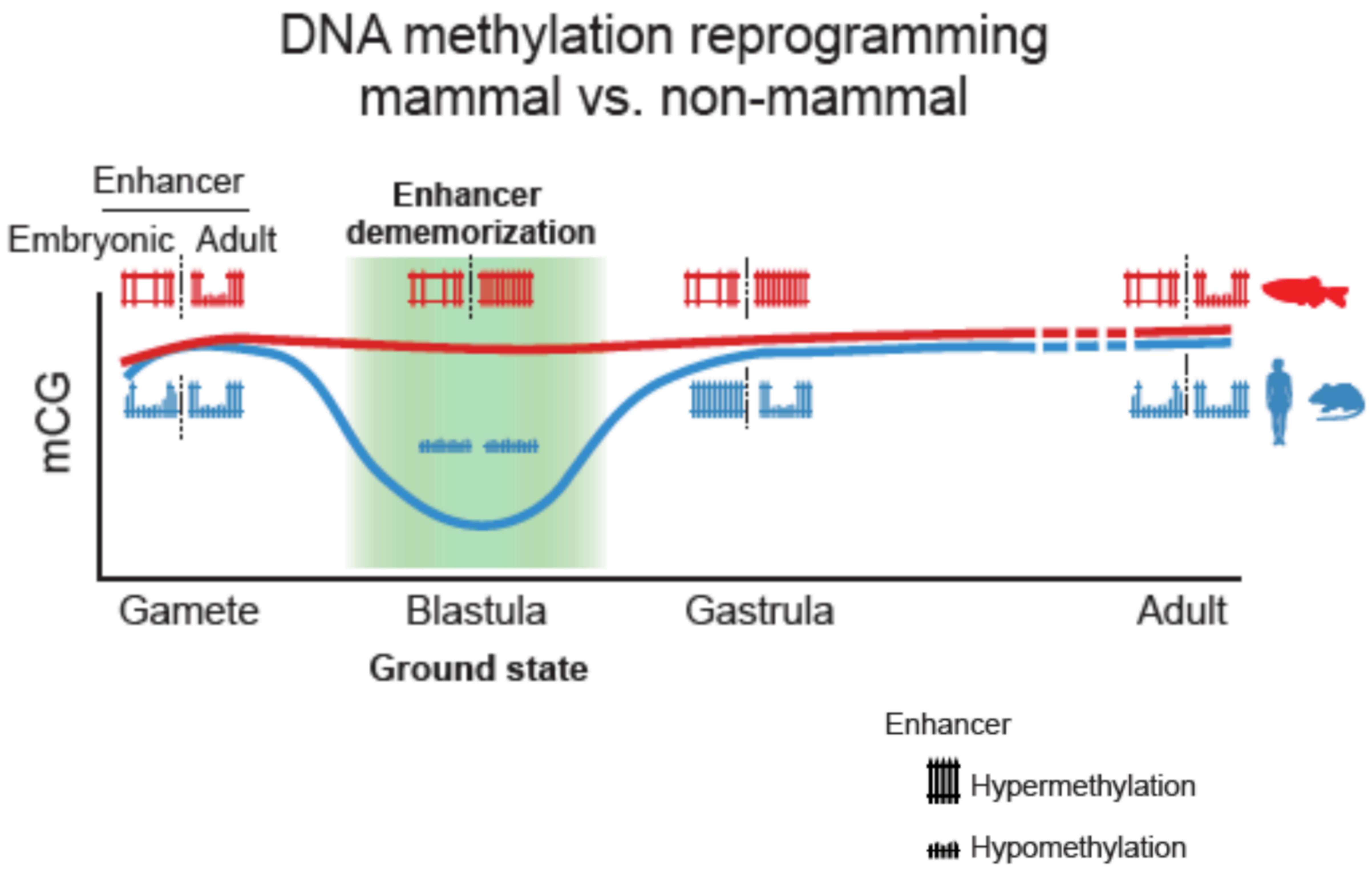


Fish don't *globally* reprogram DNA methylome

Neither do reptiles or birds

Skvortsova et al., *Nat Comms* 2019

Epigenetic Reprogramming: Peculiar Phenomenon



Wu et al., *Science Advances* 2021

Fish don't *globally* reprogram DNA methylome

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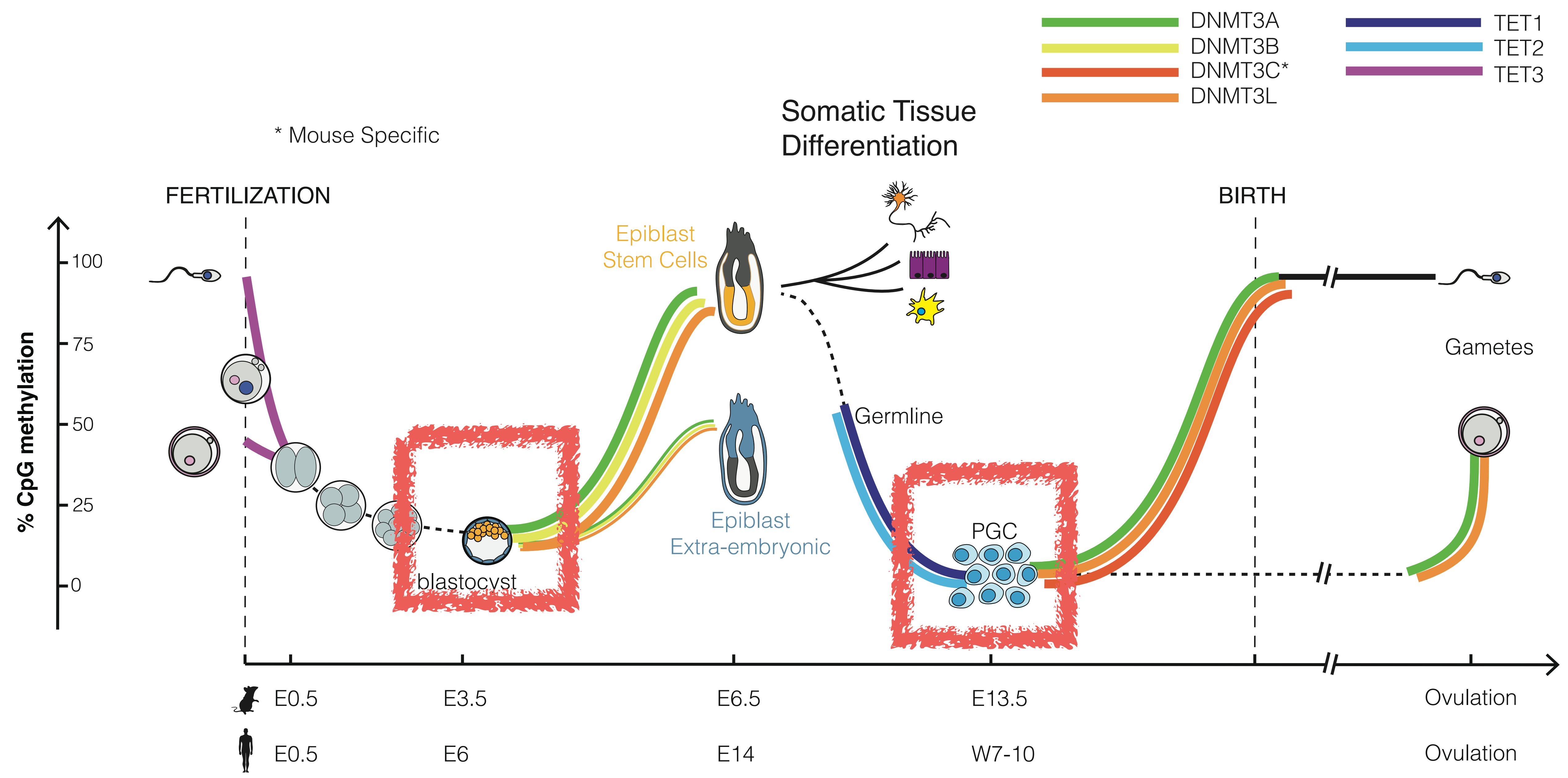
Proper embryogenesis occurs with enhancer reprogramming

Mammals are weird!!

Epigenetic Reprogramming: Peculiar Phenomenon



Fish don't reprogram DNA methylome

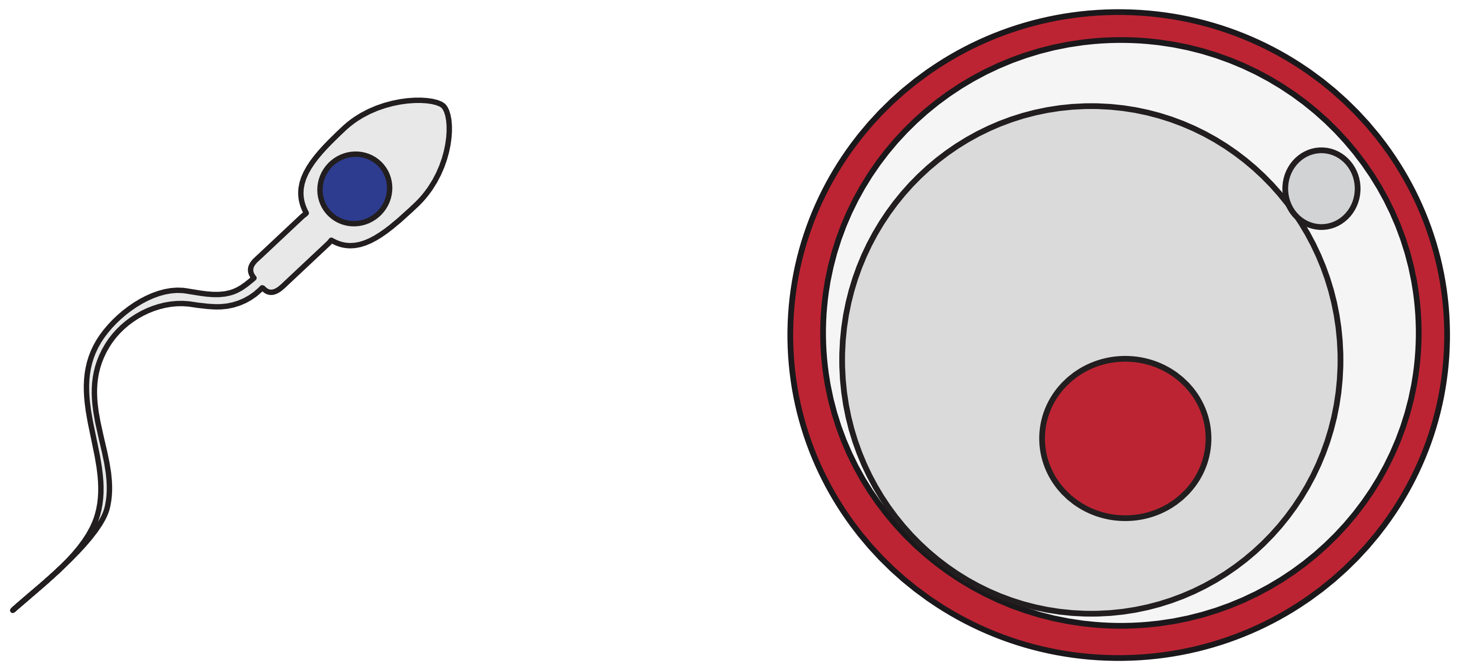
Neither do reptiles or birds

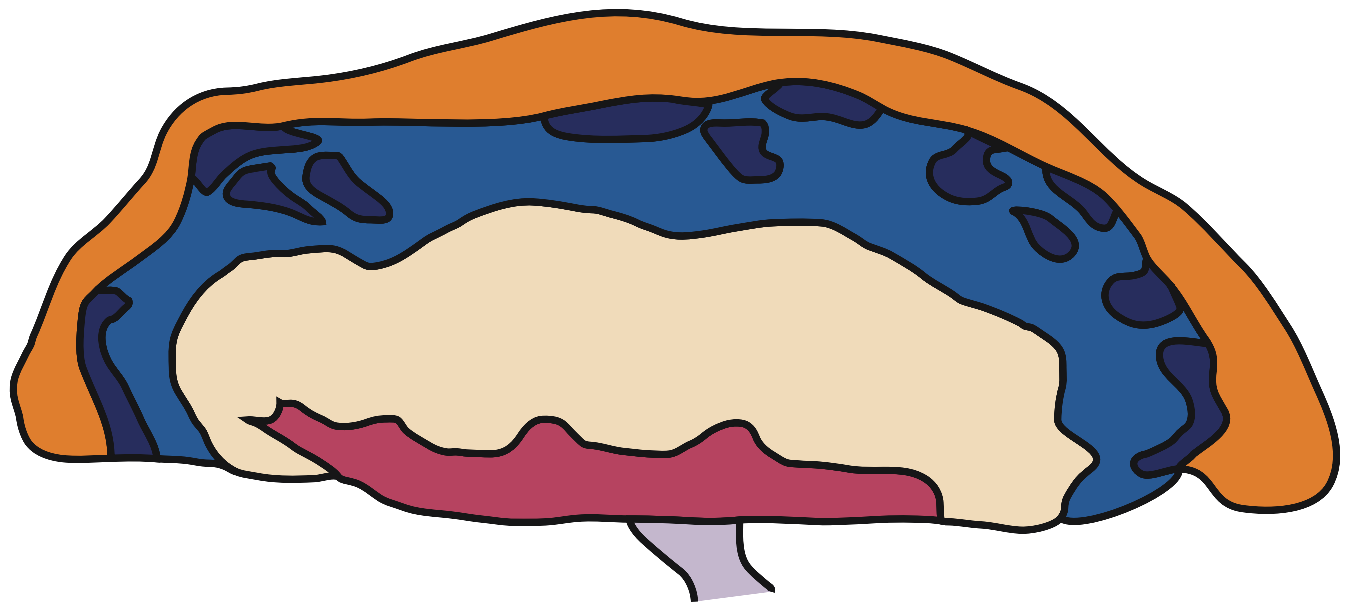
Proper embryogenesis occurs with enhancer reprogramming

Mammals are weird!!

Mammals reprogram during the most vulnerable periods of development!

Better question:
Why are gamete epigenomes so divergent?

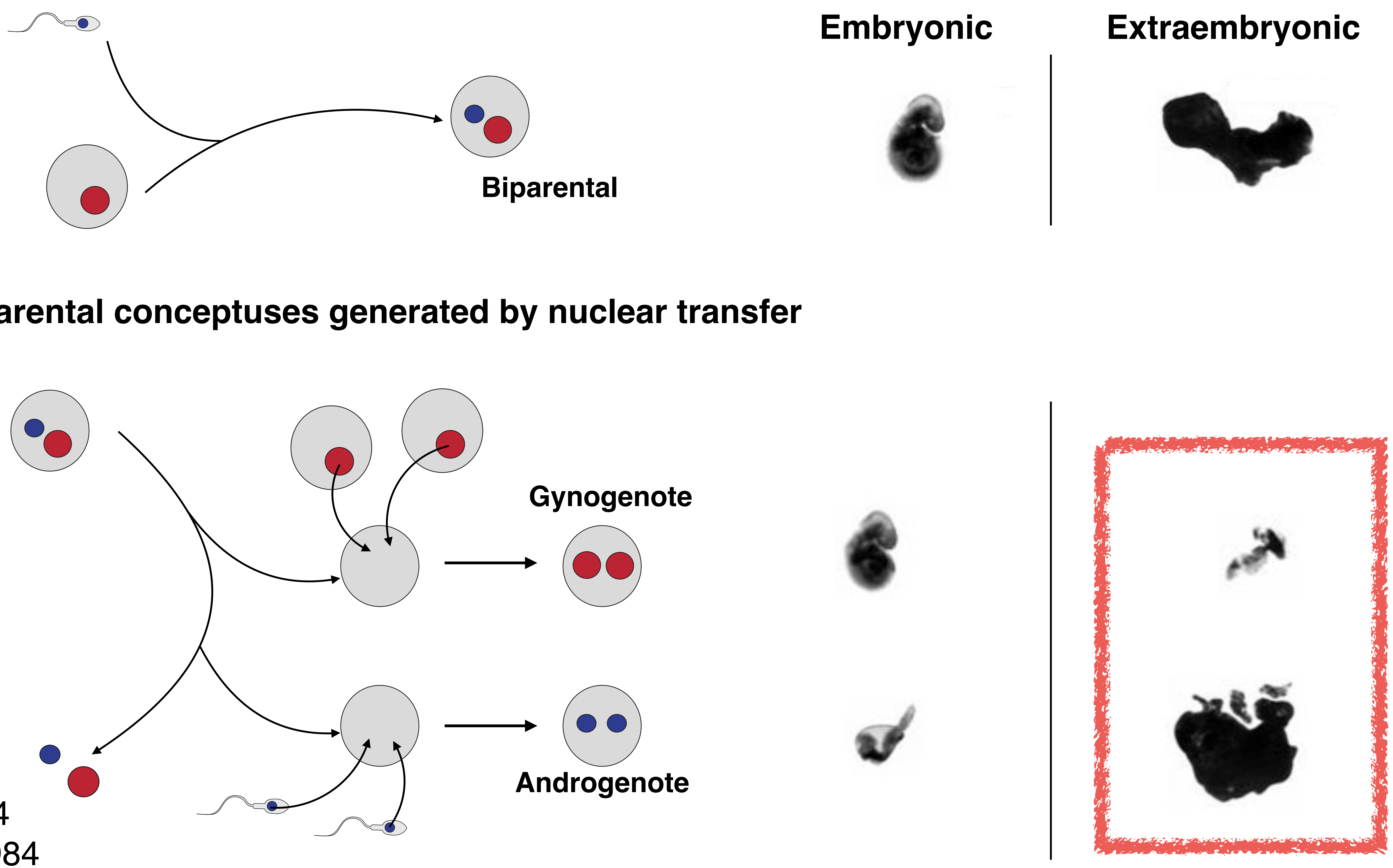




The answer might be the placenta

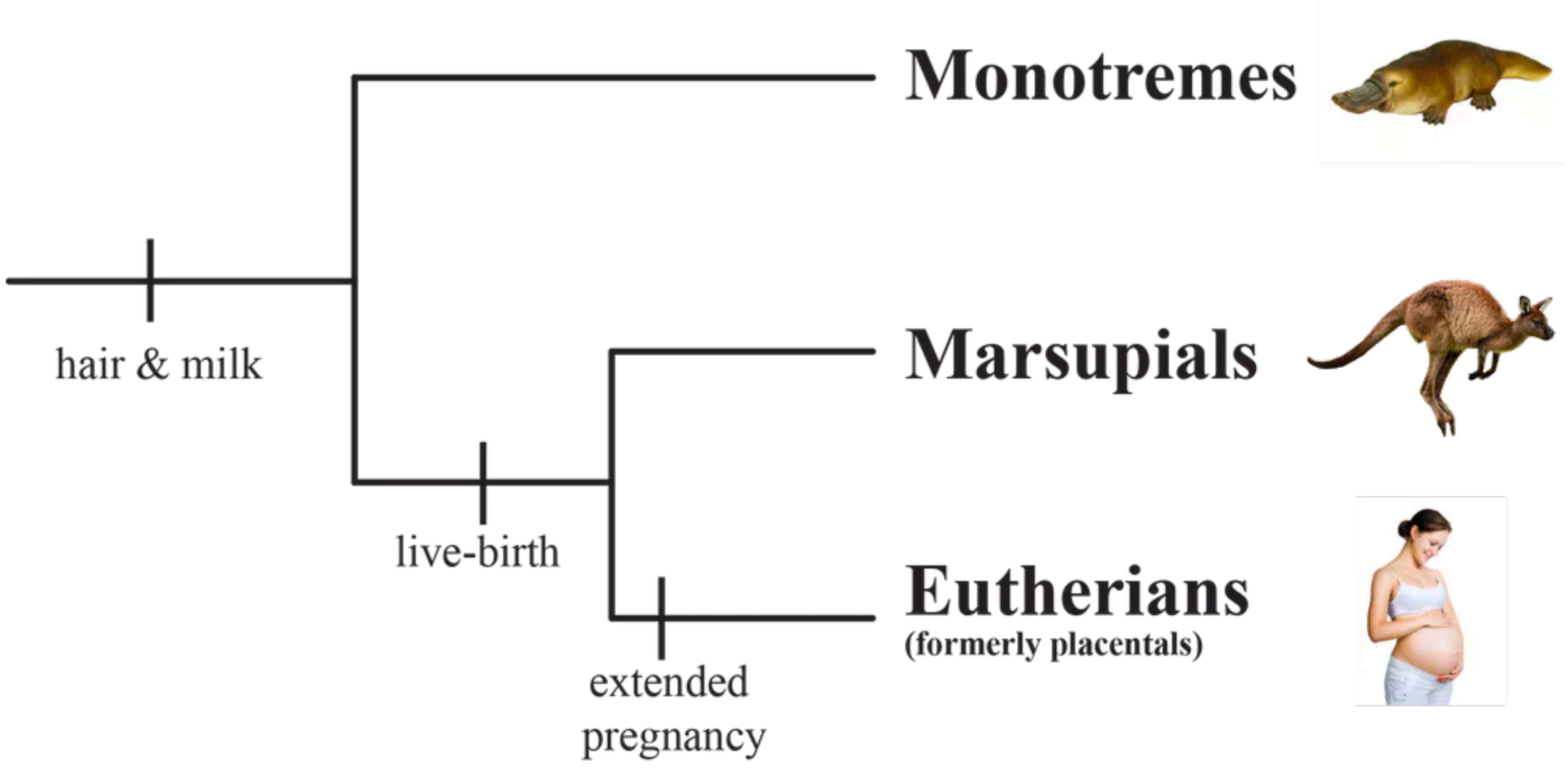
Placenta regulates fetal growth (interface between mother and fetus)

Uniparental conceptuses generated by nuclear transfer



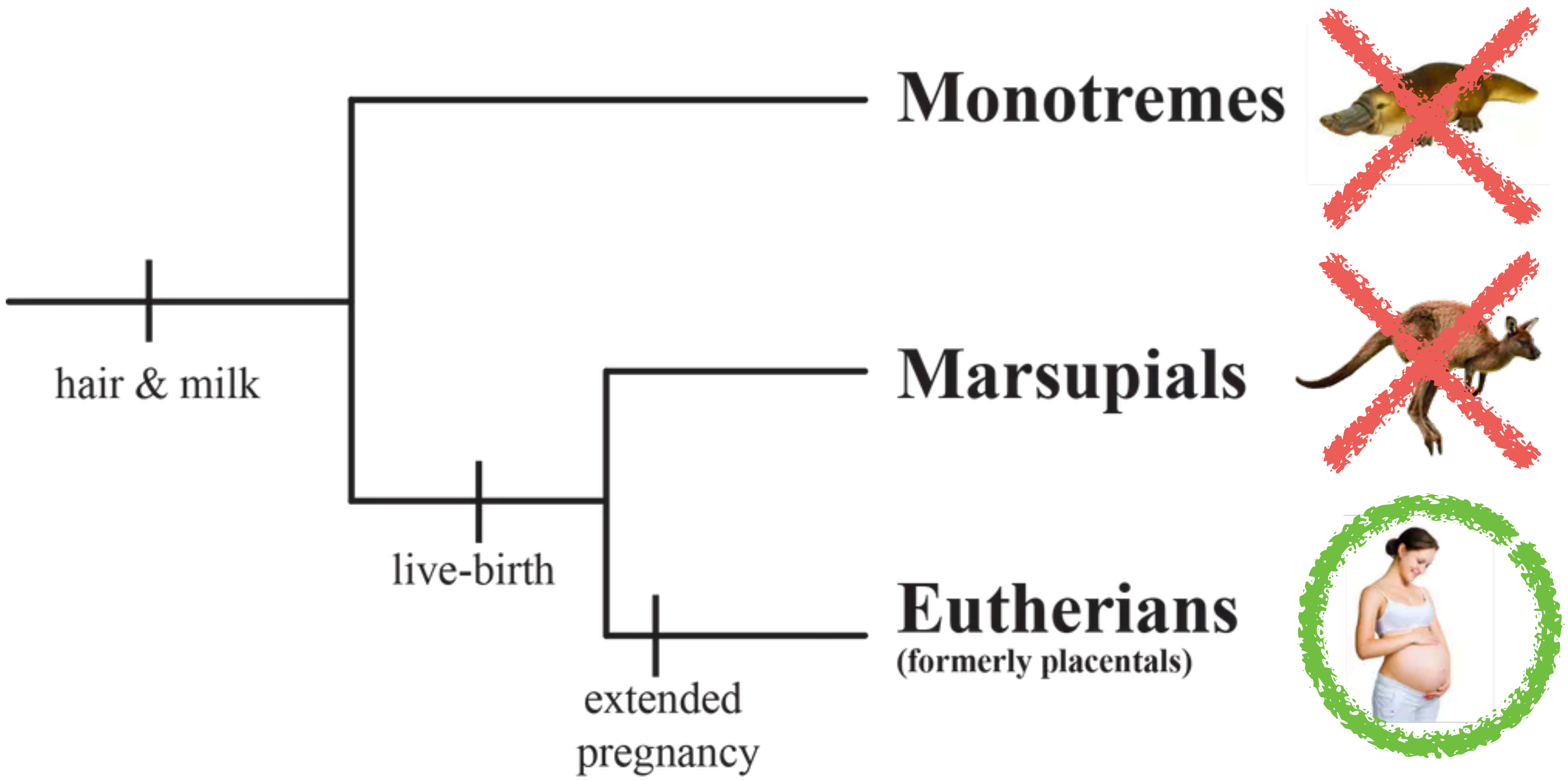
Barton *et al.*, 1984
McGrath *et al.*, 1984

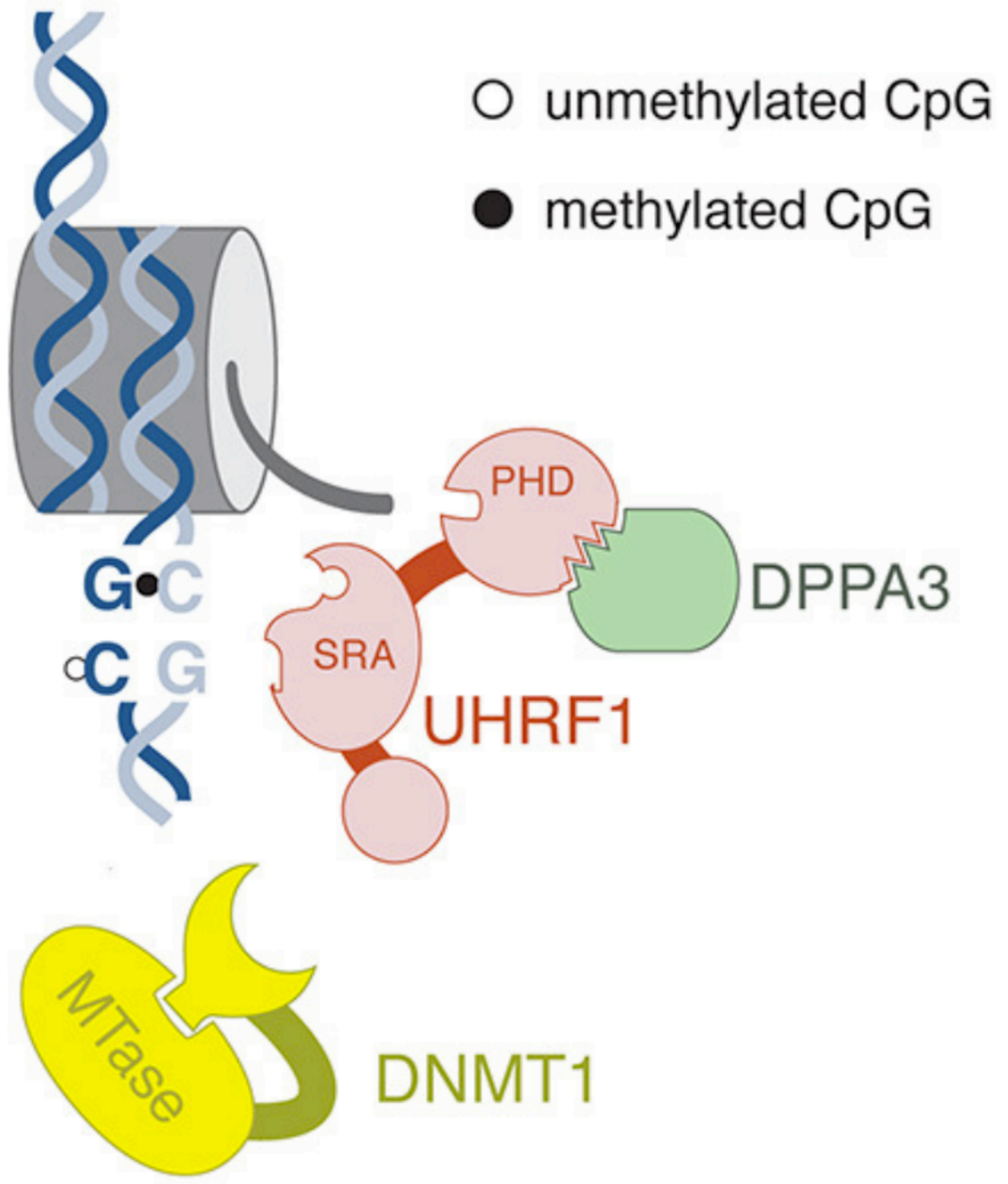
Egg-laying mammals don't exhibit imprinting



Do they exhibit epigenetic reprogramming??

Egg-laying mammals don't exhibit imprinting





DPPA3 only found in placental mammals. Is this the key?

Summary Part IV

- Reprogramming resolves asymmetrically methylated gametes
- Epigenetic reprogramming (DNA methylation) appears to be unique to mammals
- May have evolved because of parental conflict, and allocation of resources to fetus

Thank You for Your Attention!

"Chromatin Development in Mammalian Development"

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