what makes a good figure?

creating effective explanations of complex concepts

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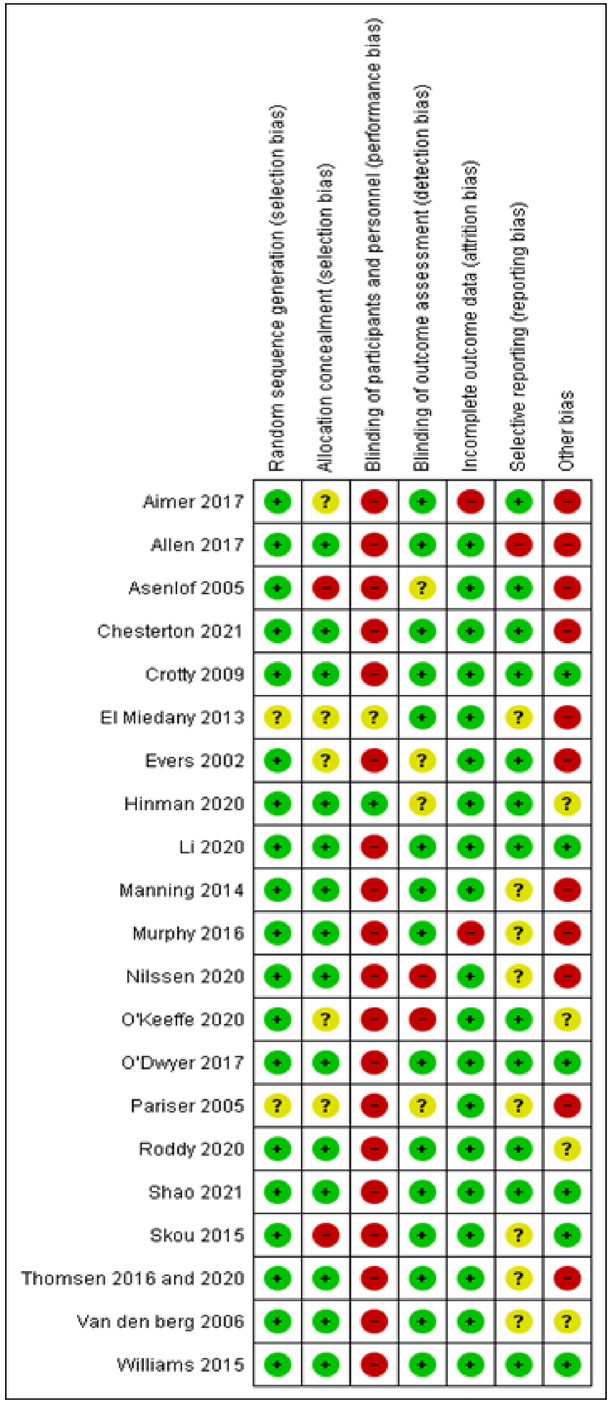
Luxembourg Centre for Systems Biomedicine (LCSB) · Belvaux, Luxembourg · 17 June 2024

putting it together — tables

include aggregate statistics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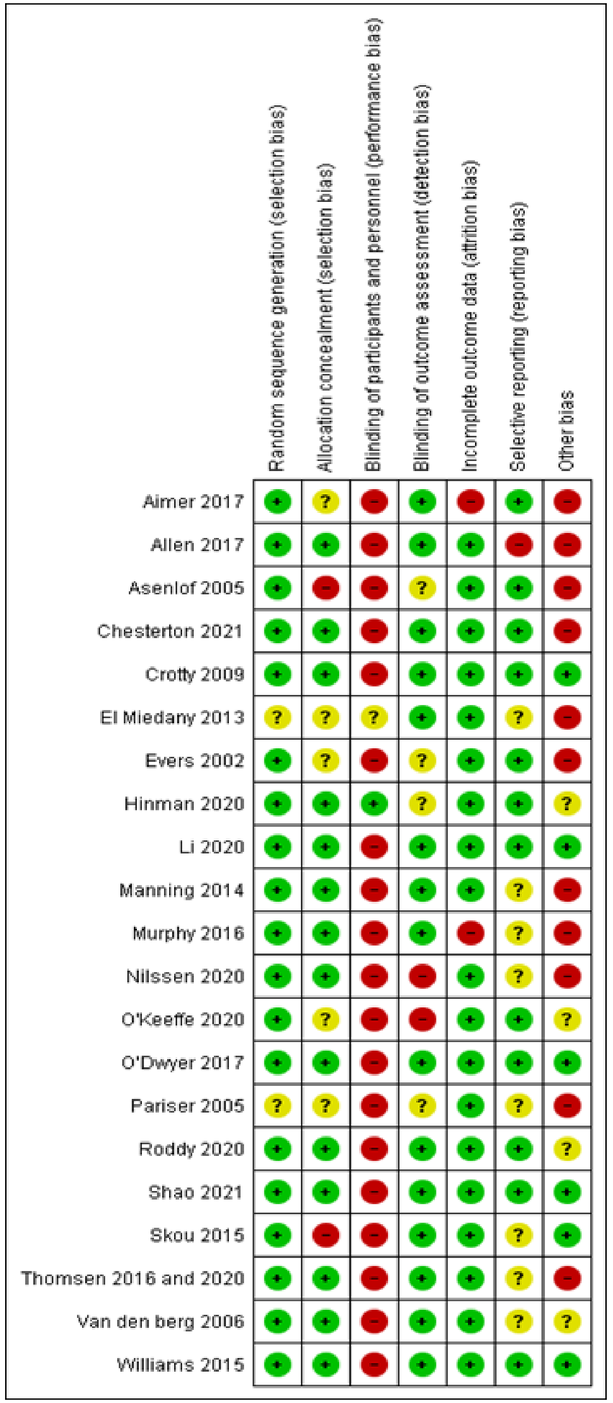
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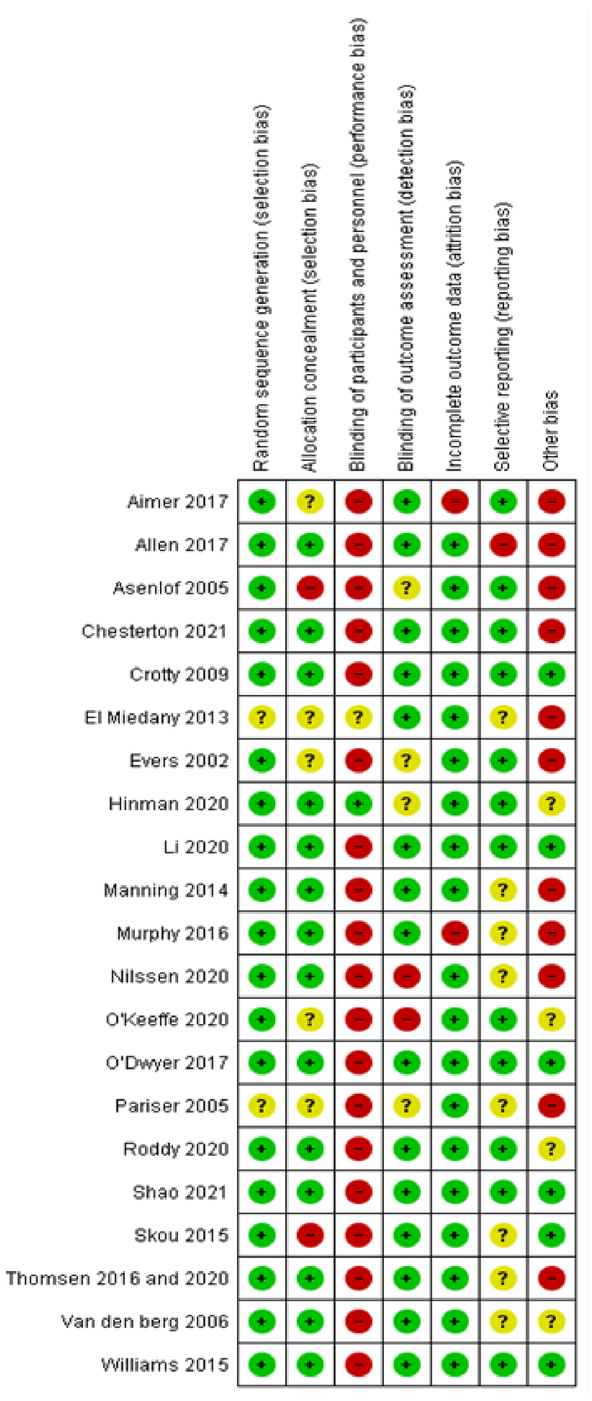
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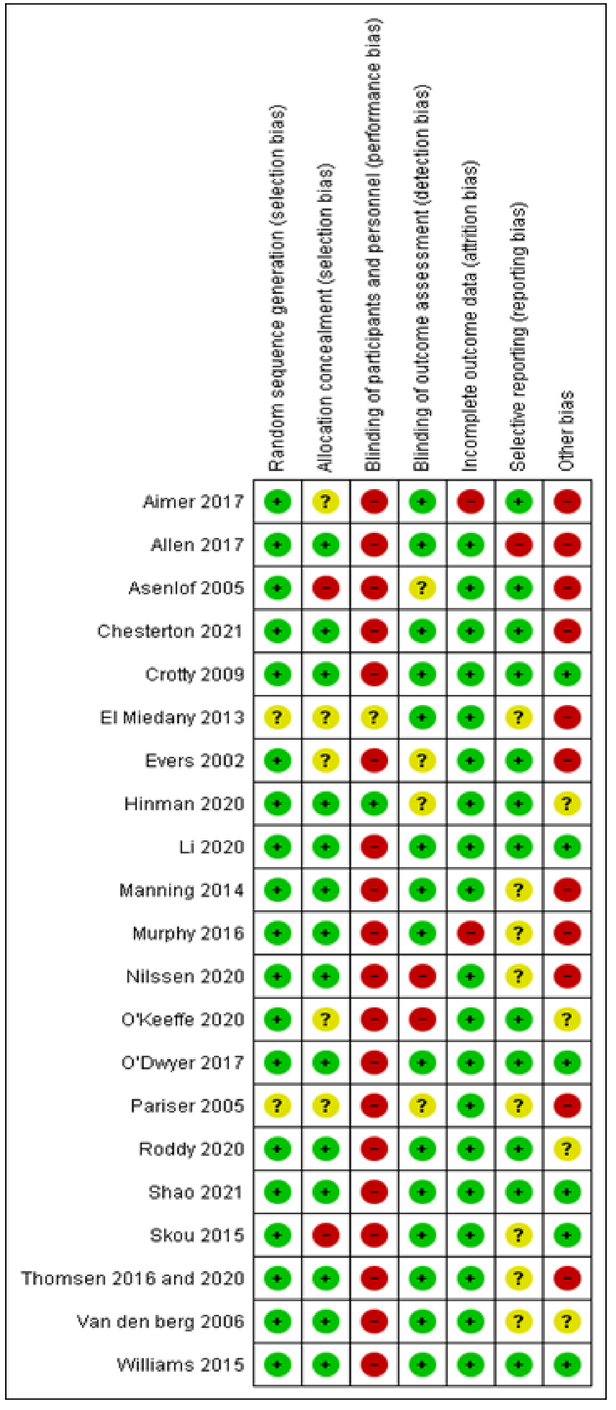
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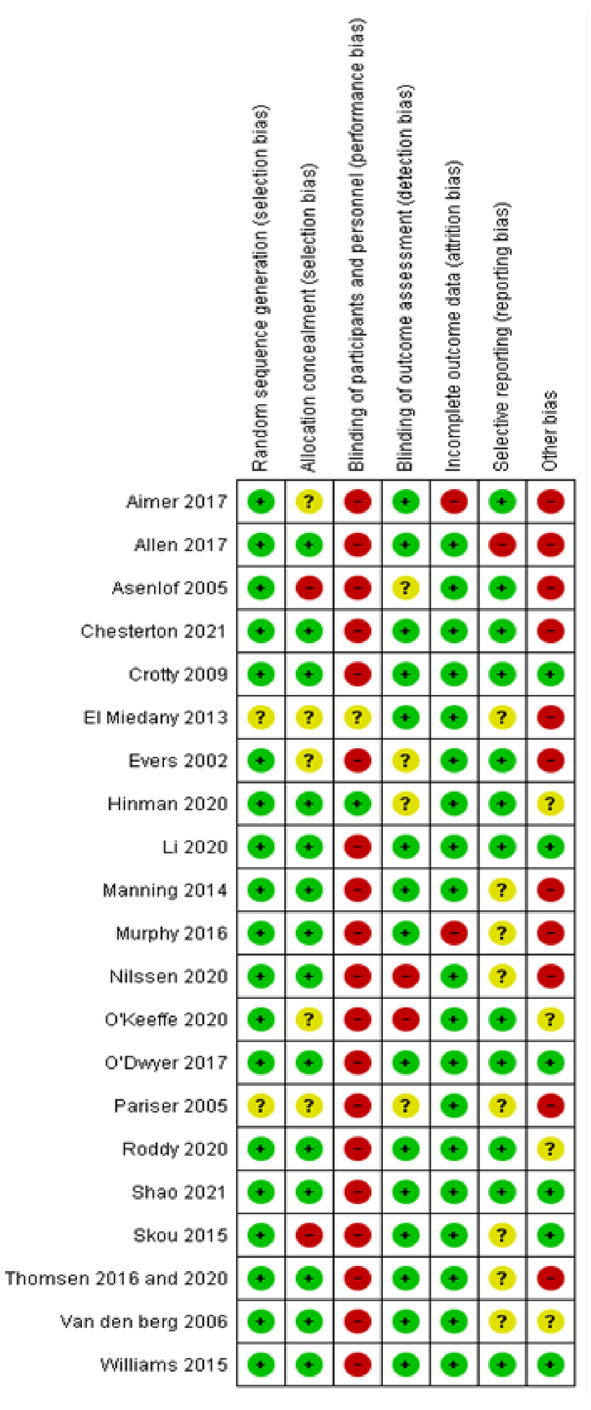
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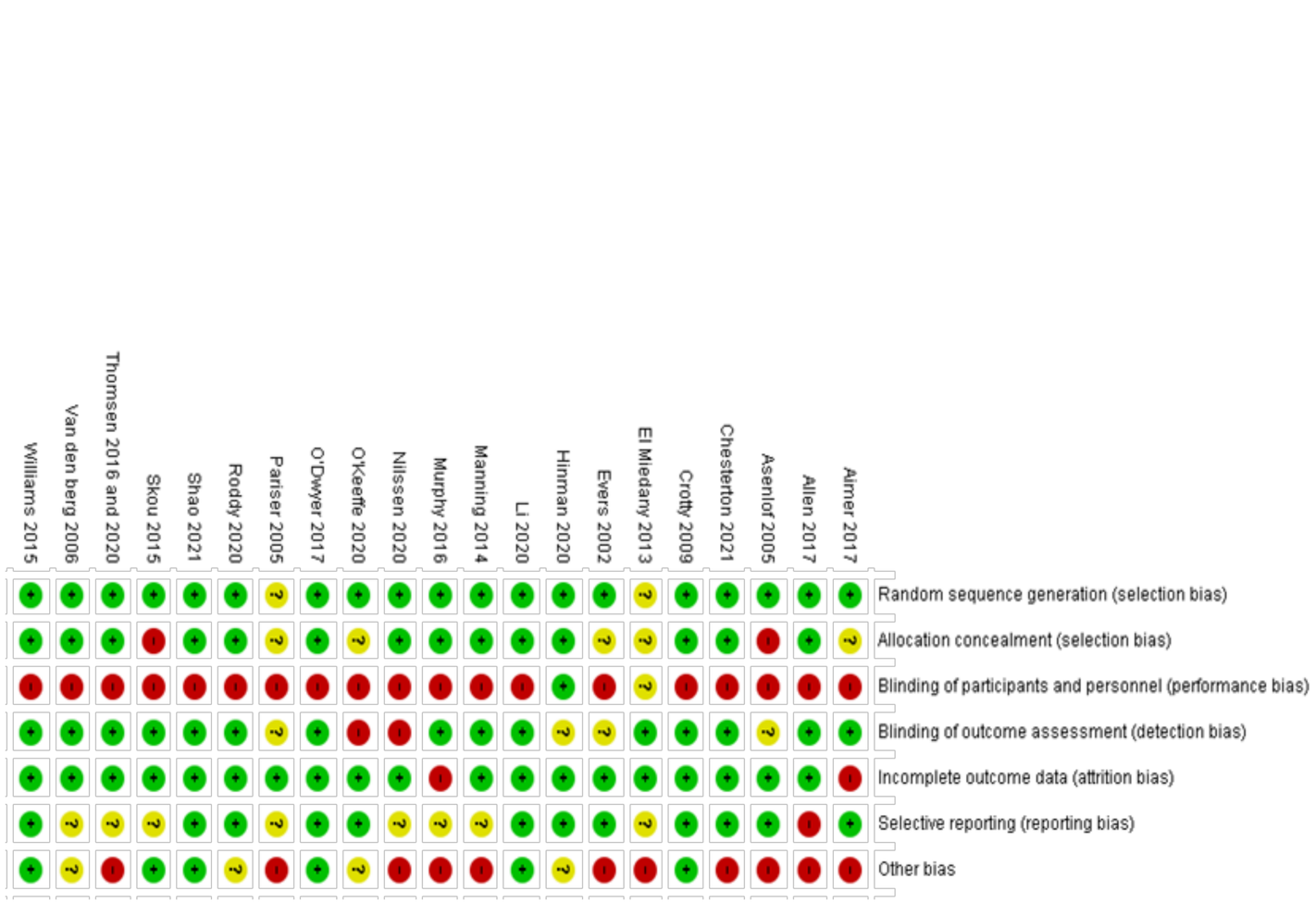
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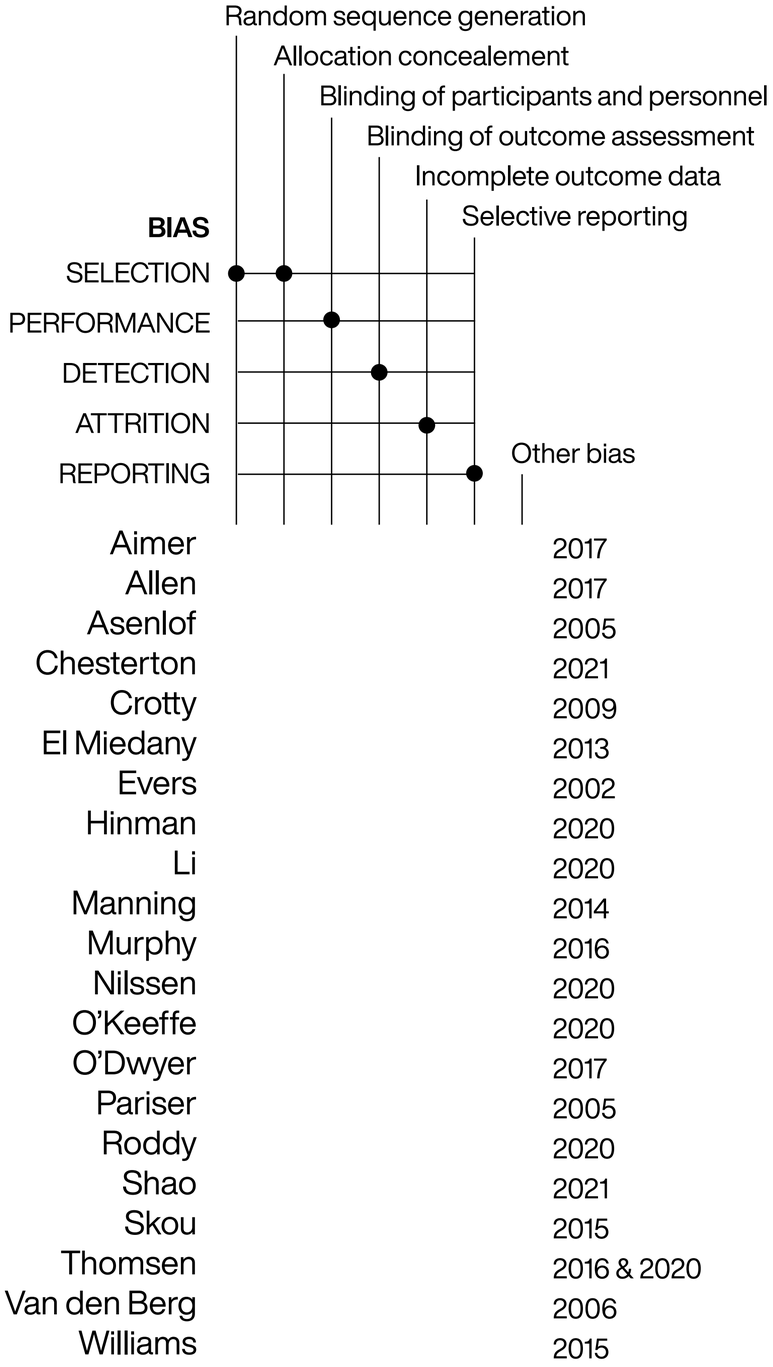
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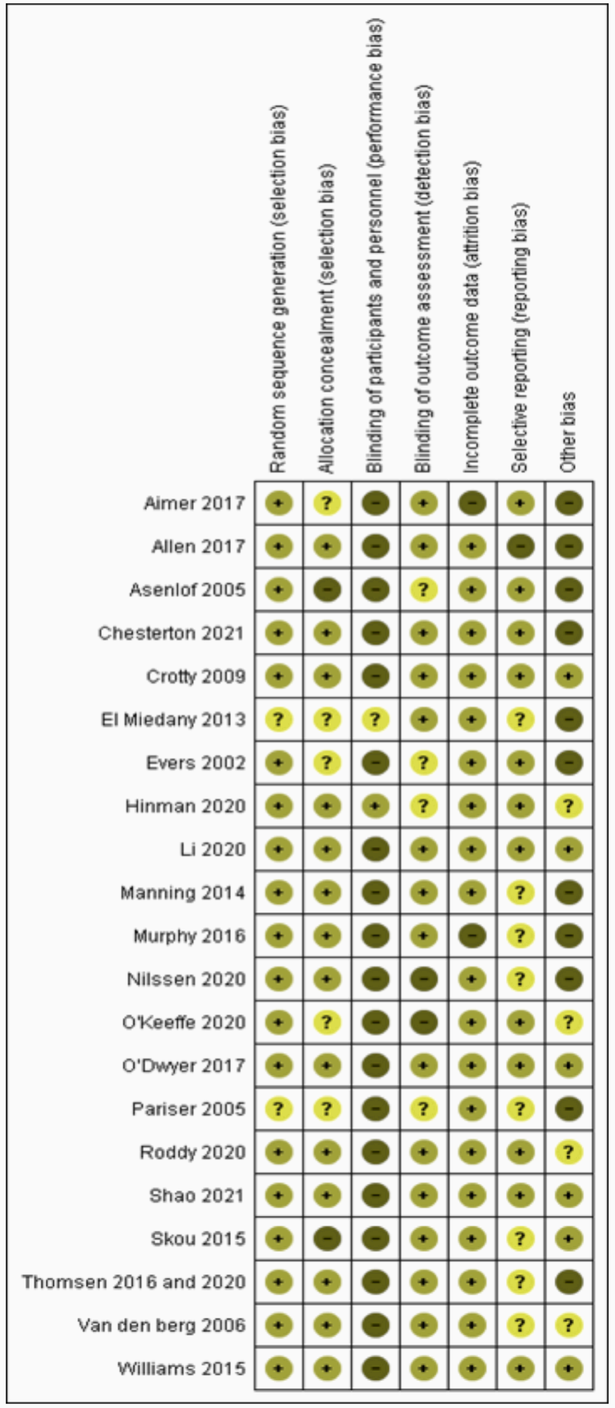
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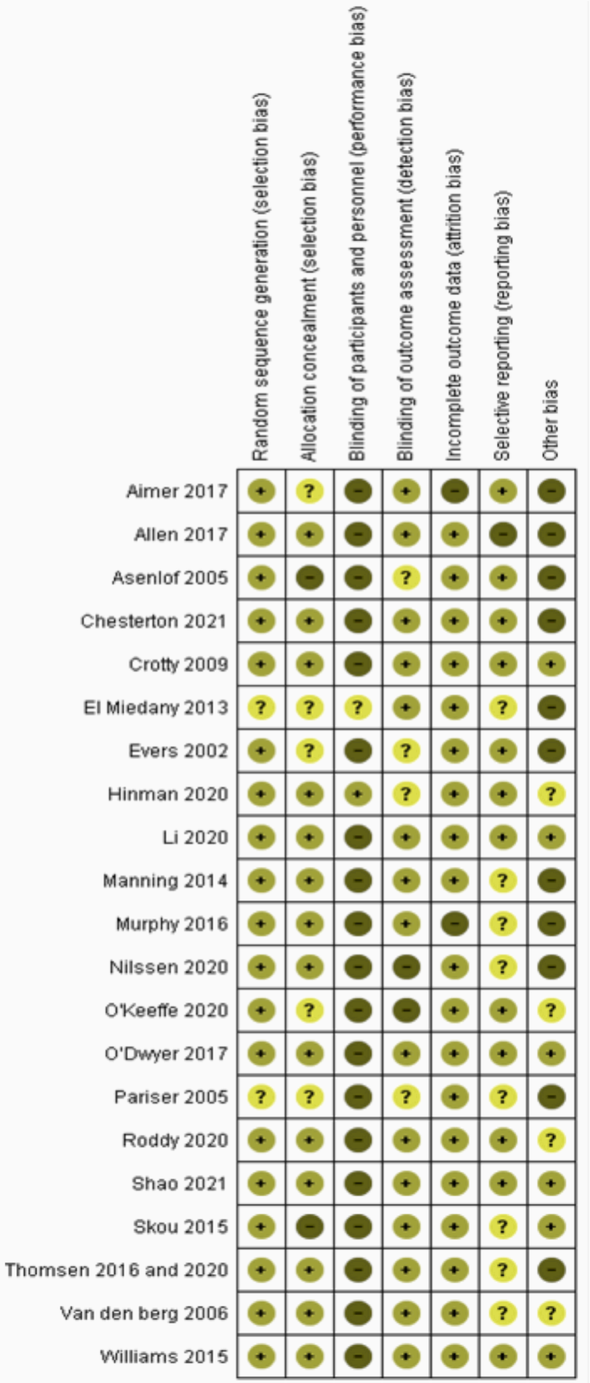
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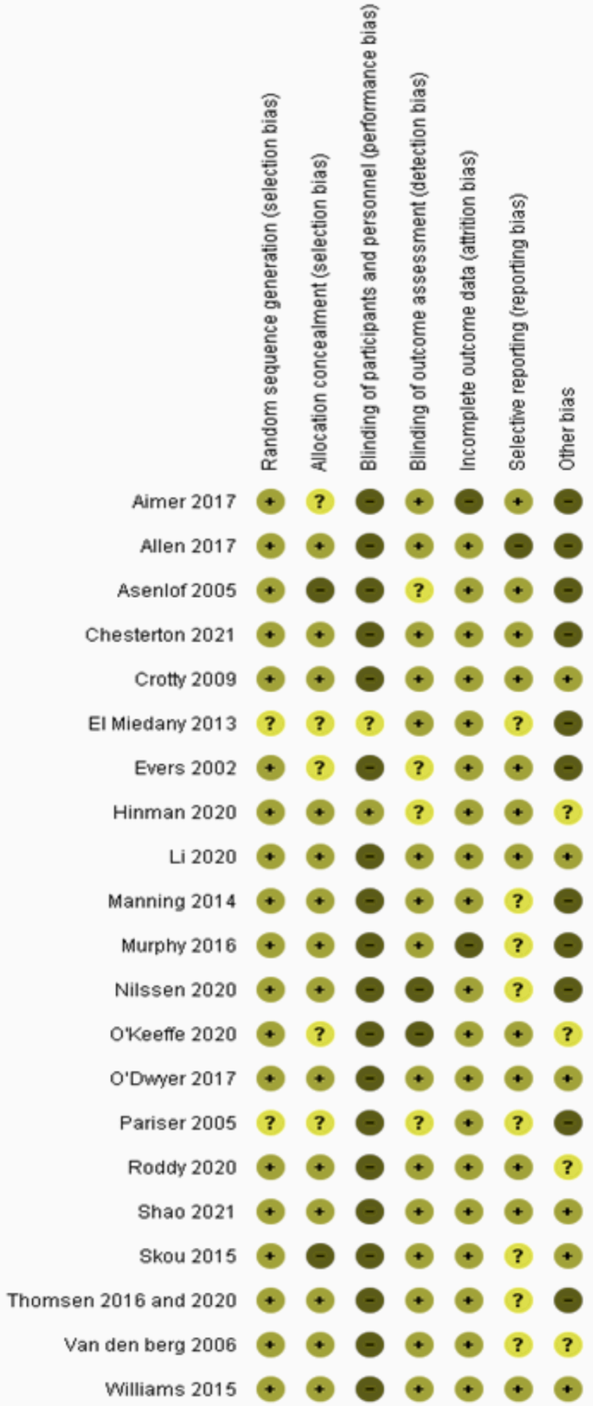
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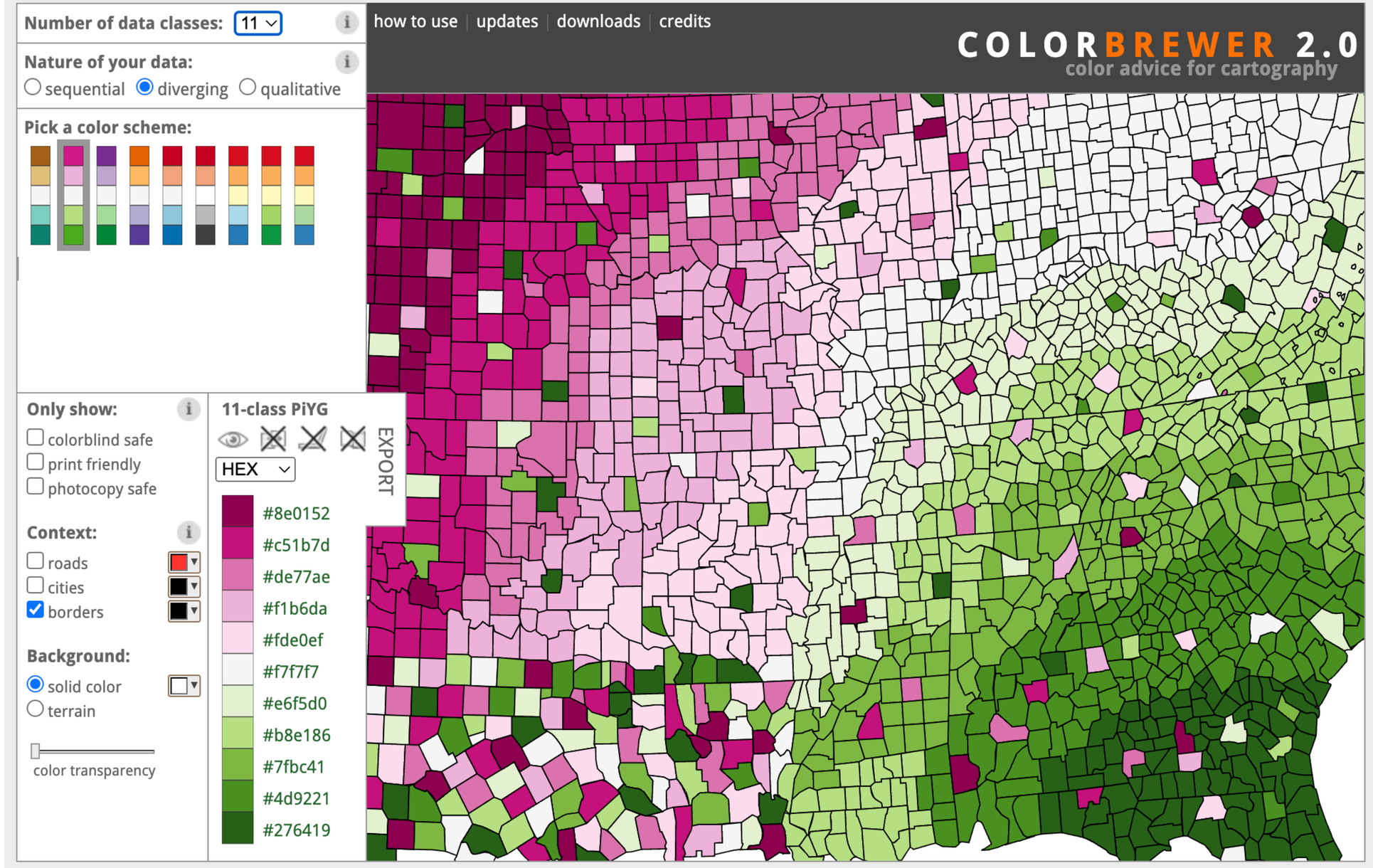

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Source code and feedback

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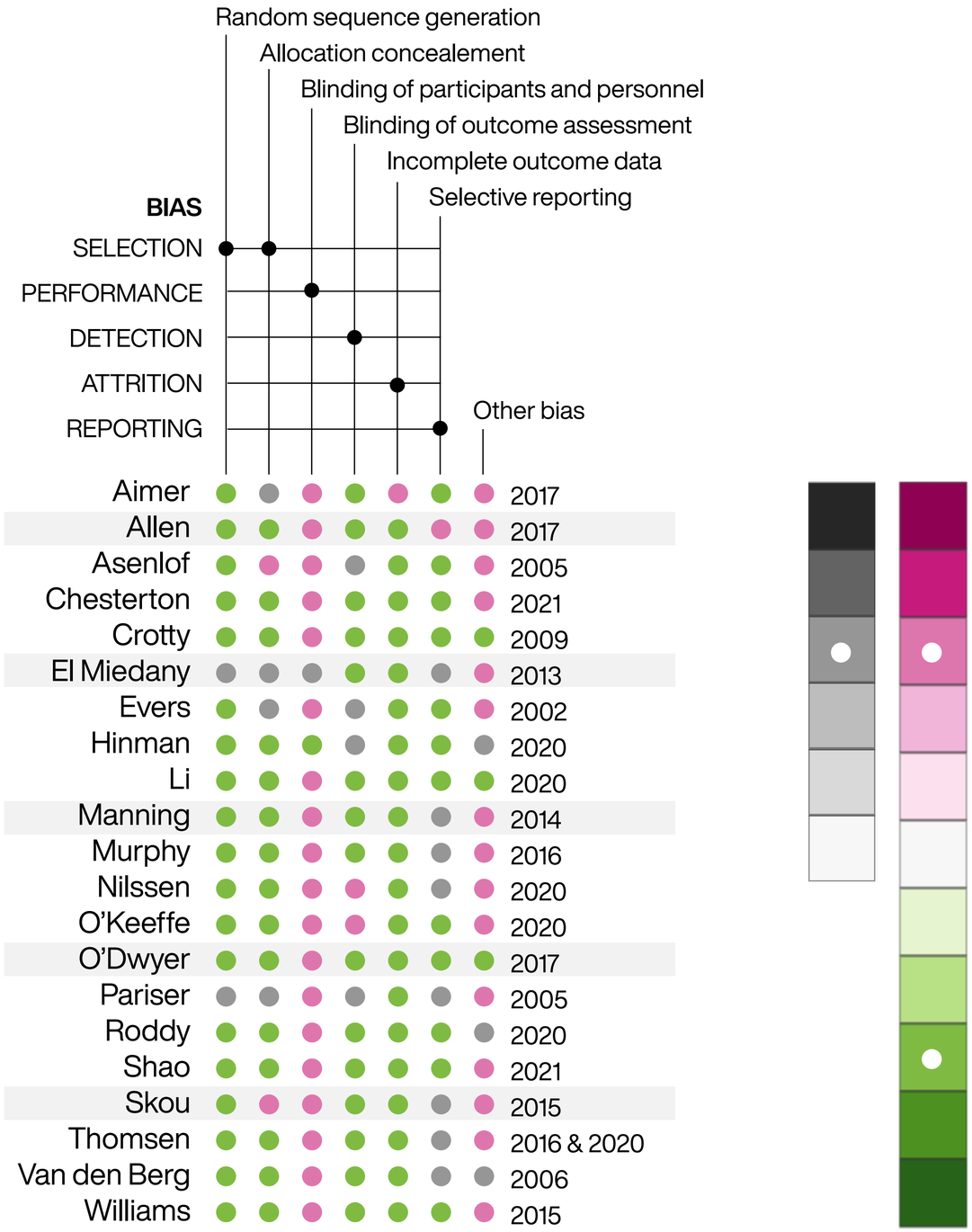
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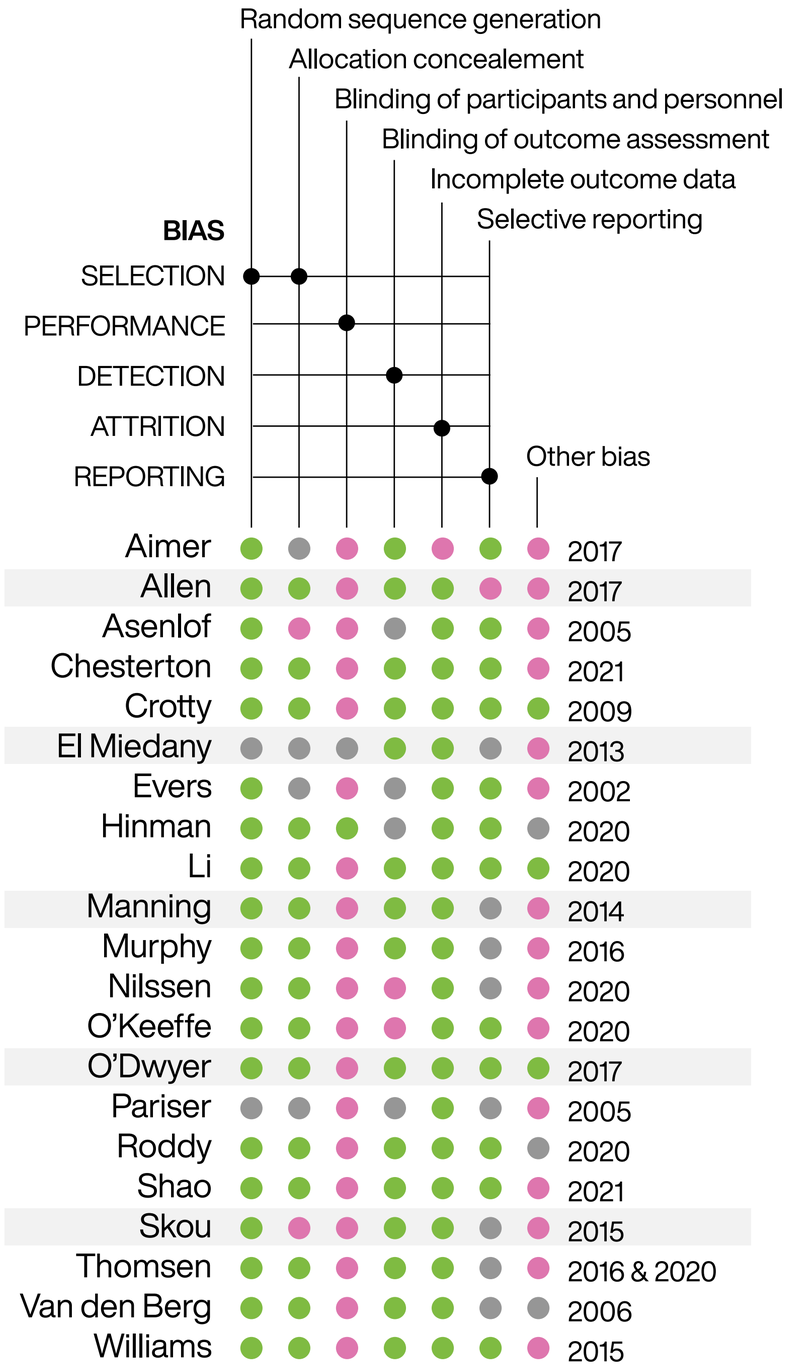
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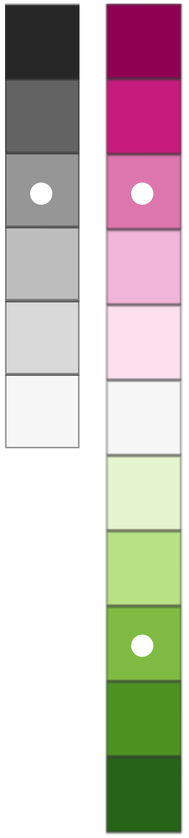

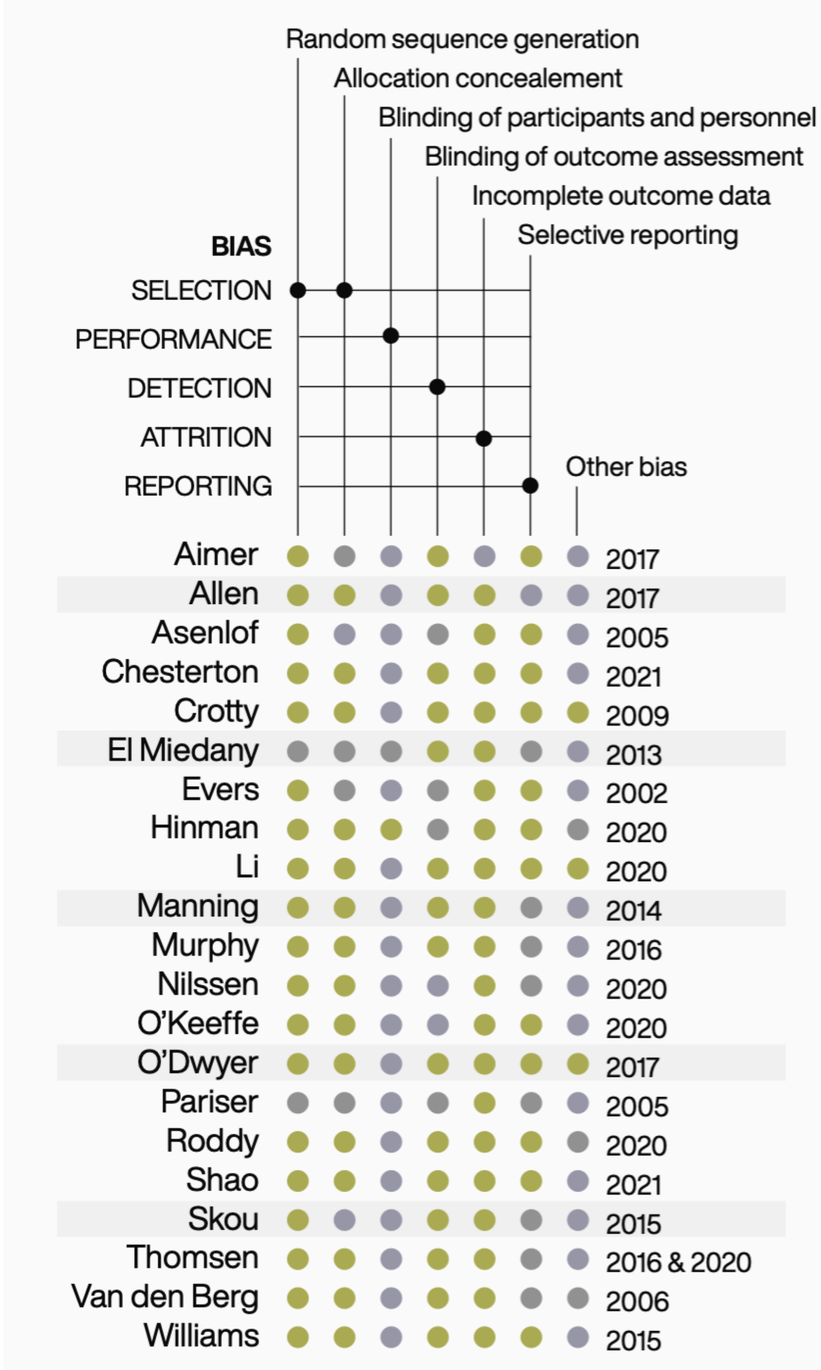
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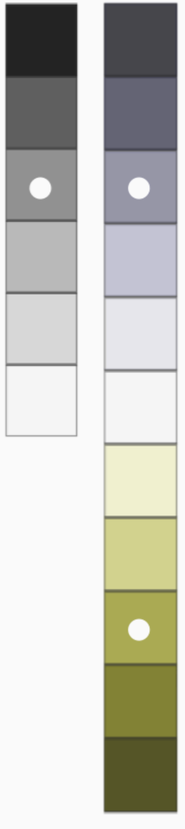

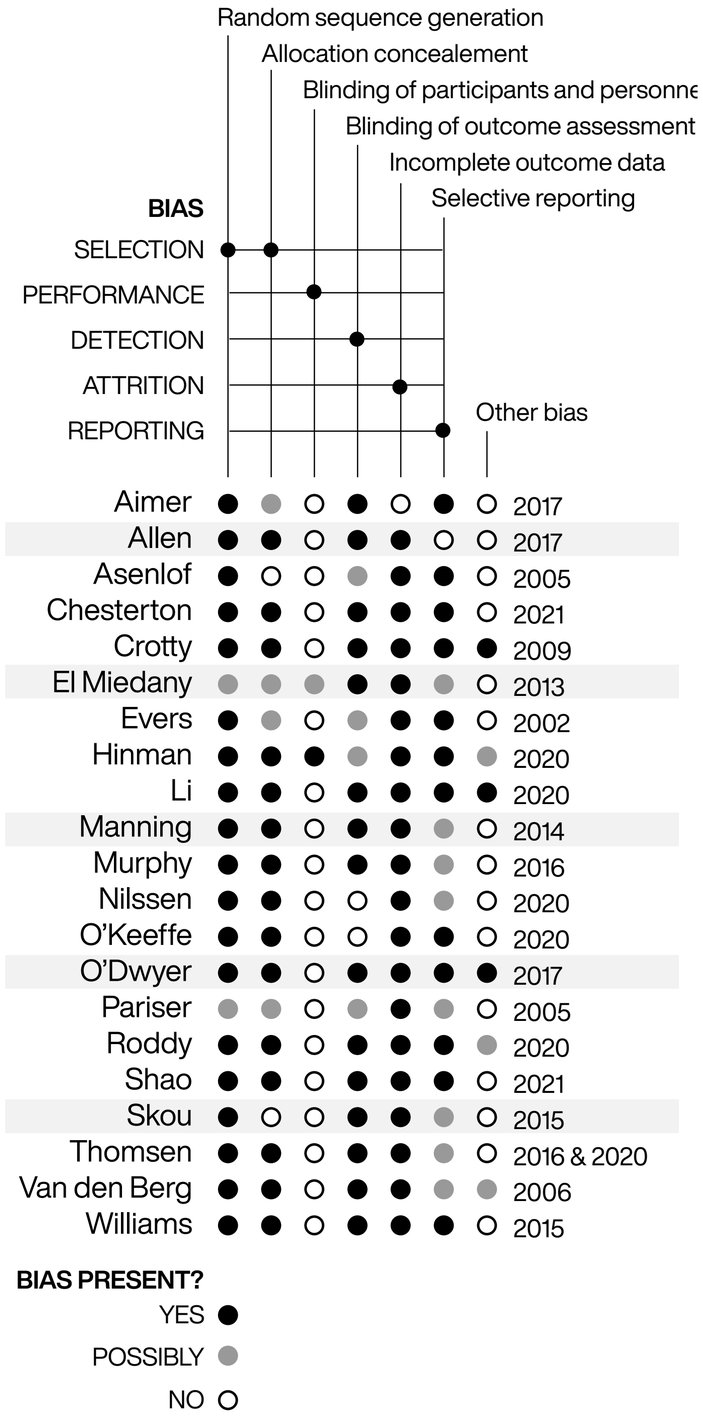
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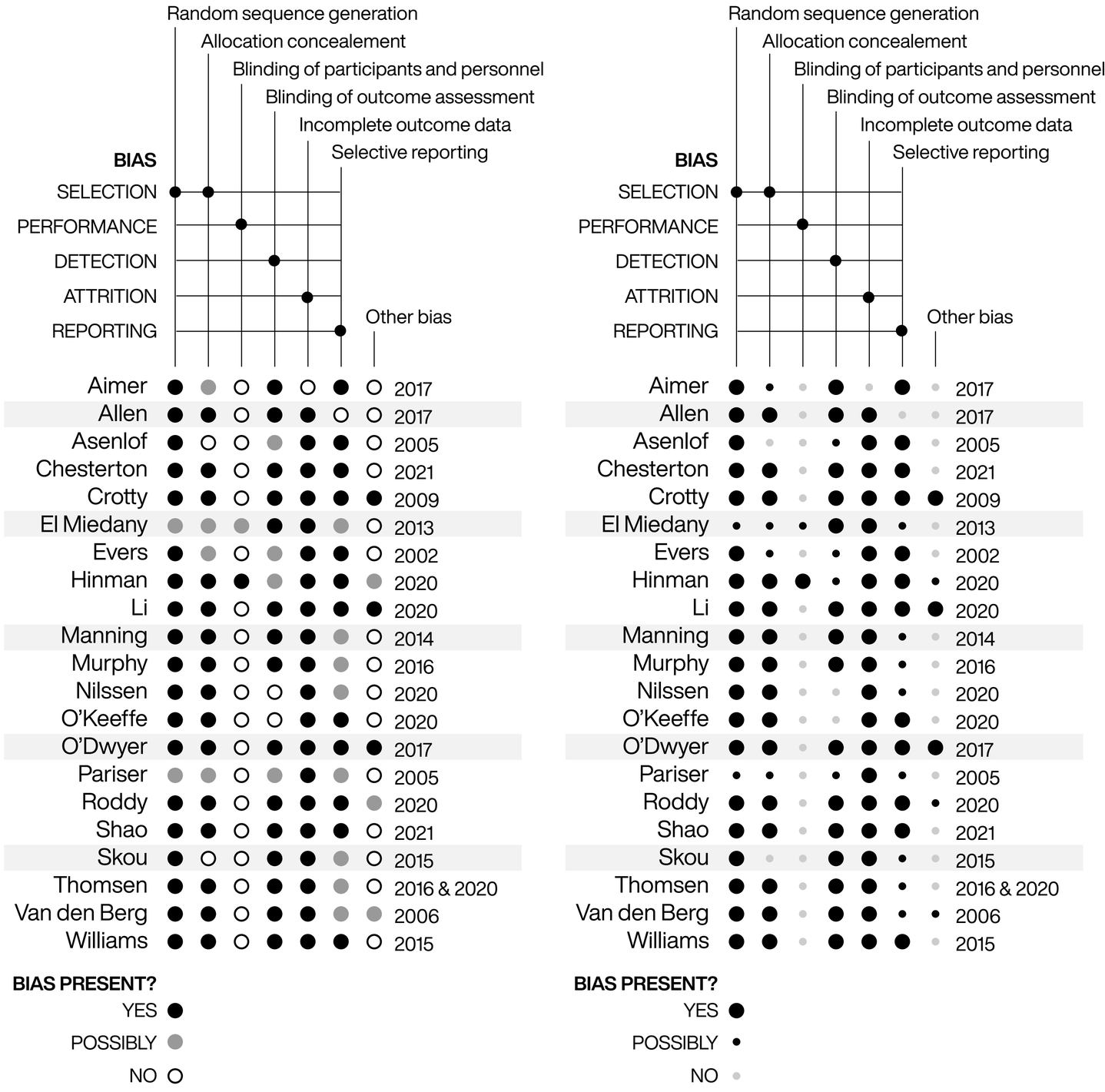
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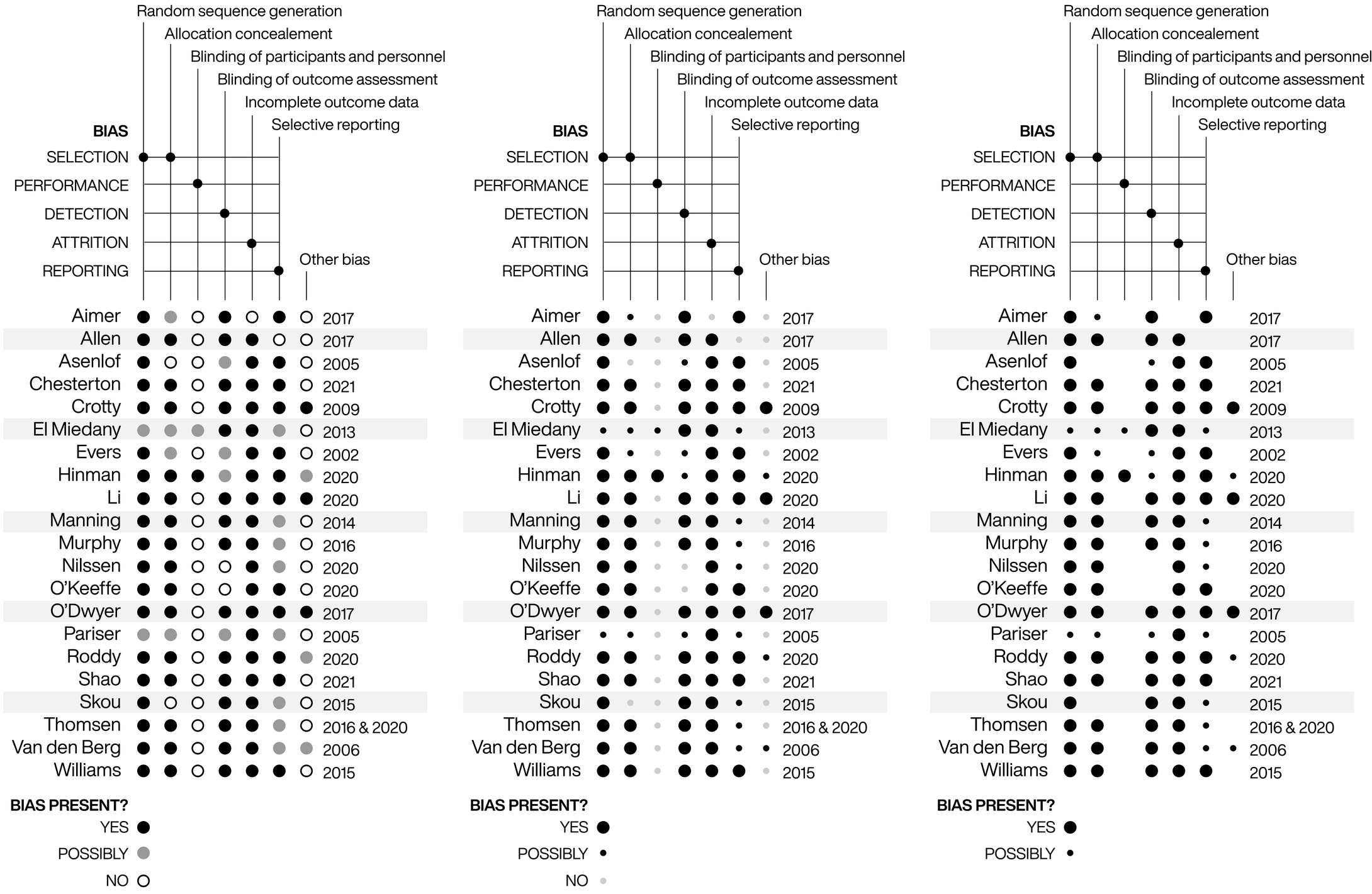
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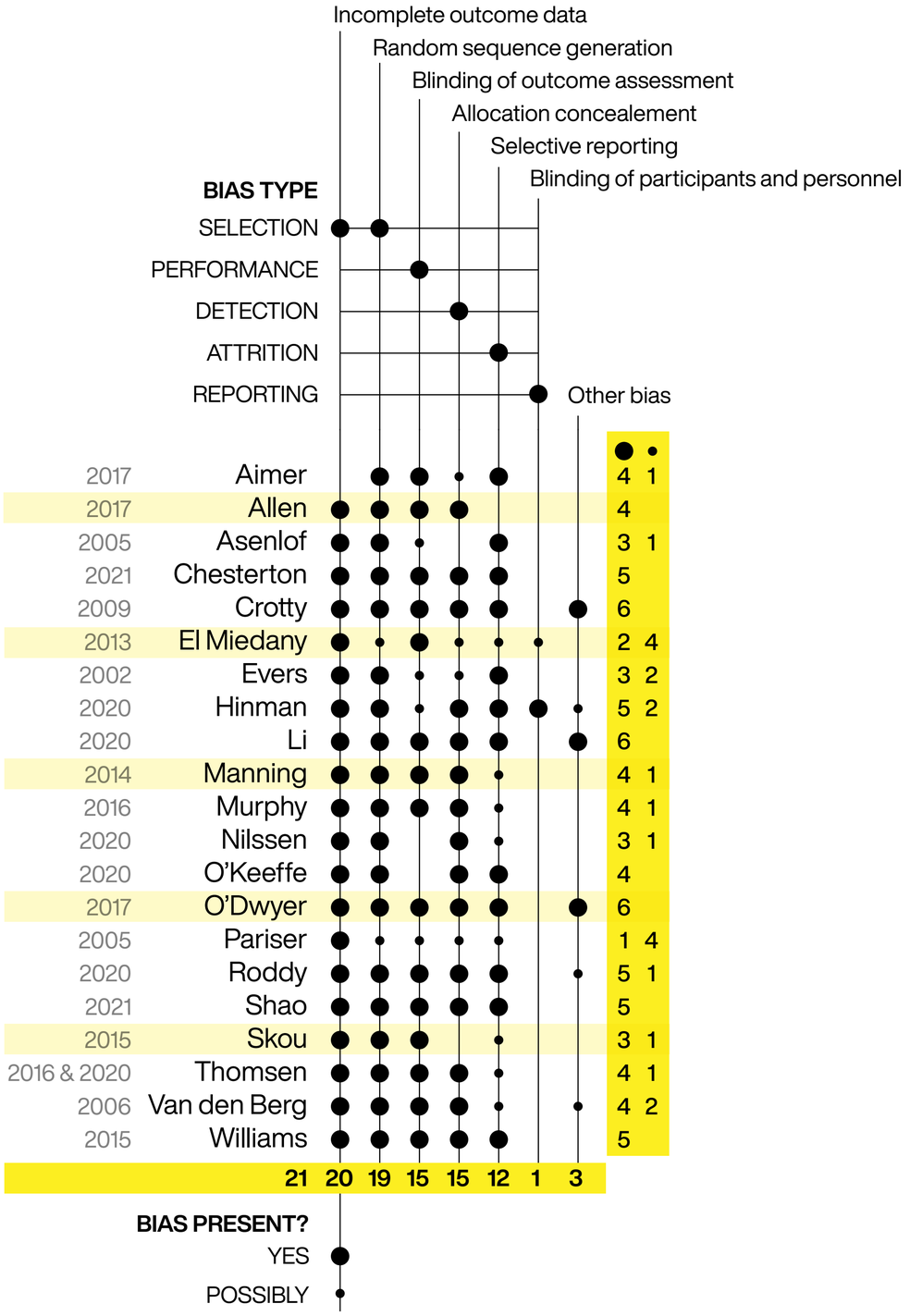
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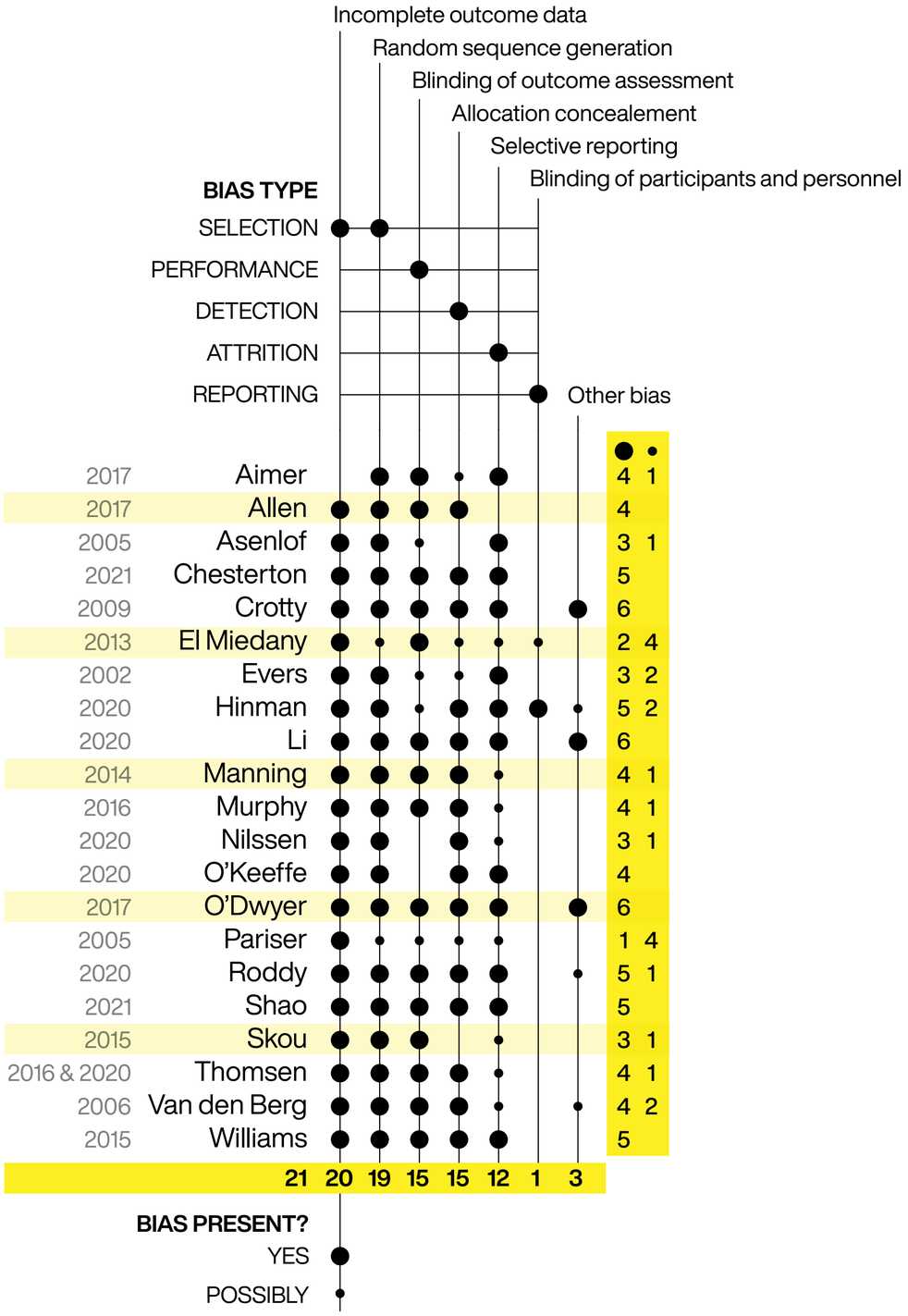
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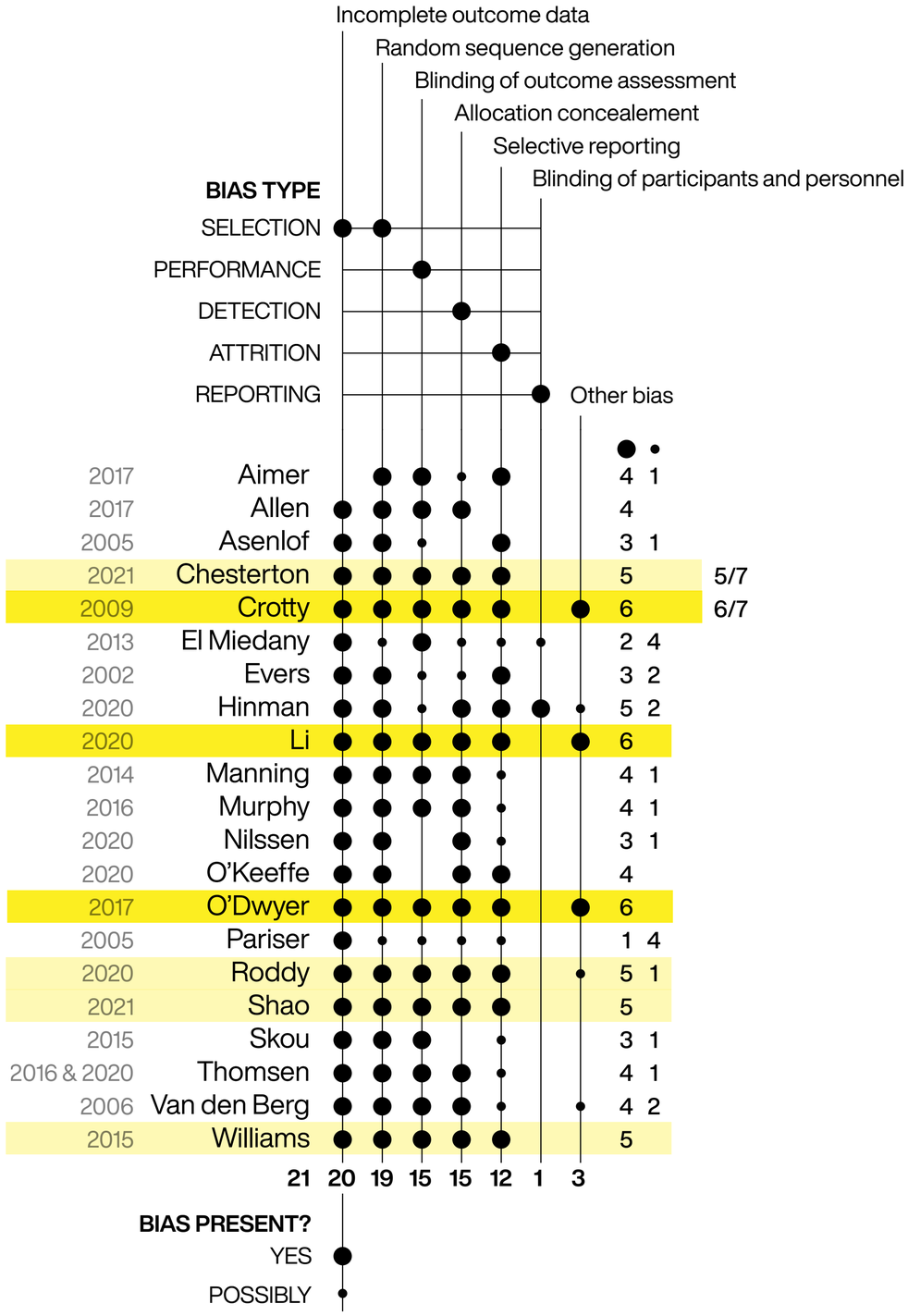
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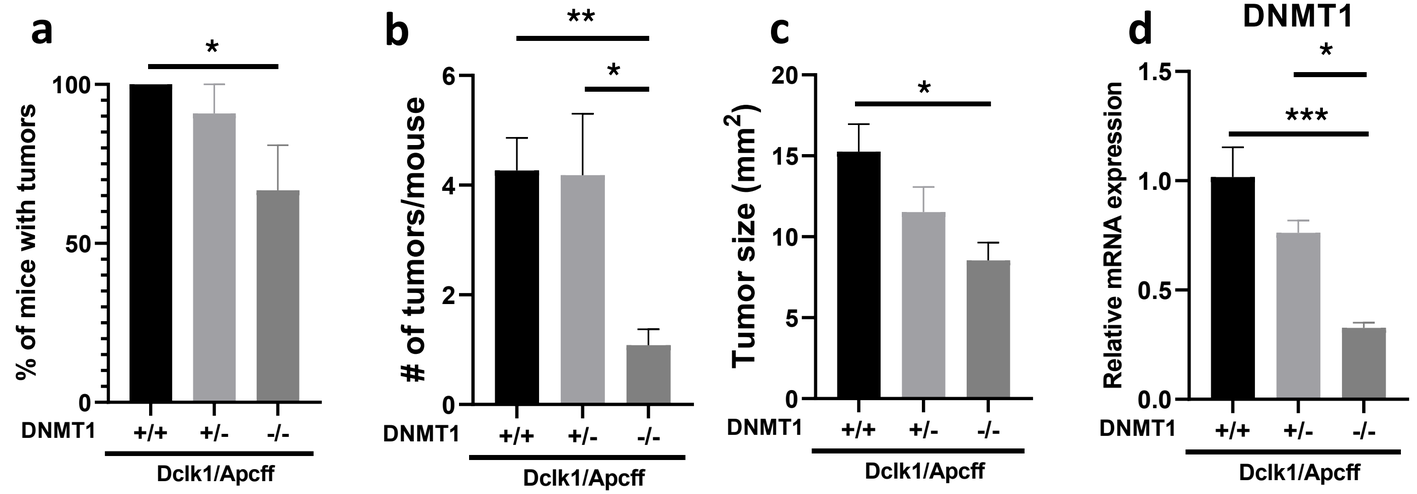
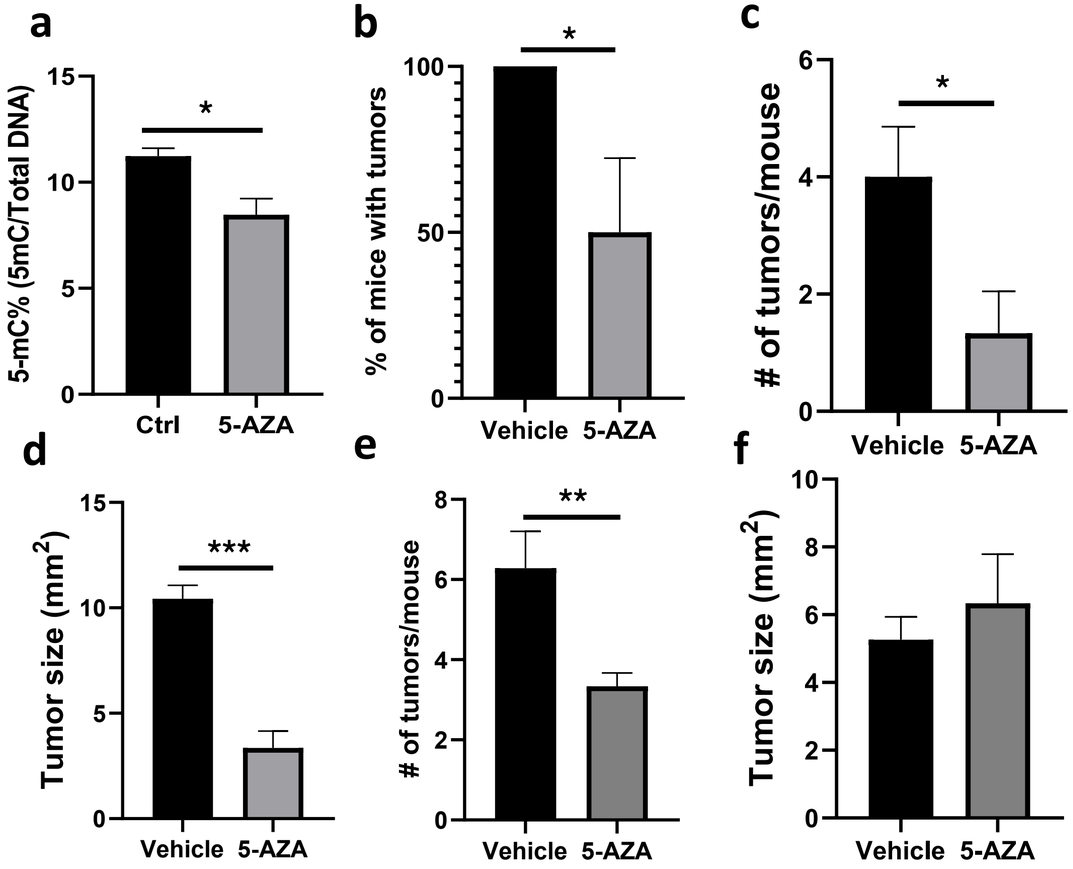
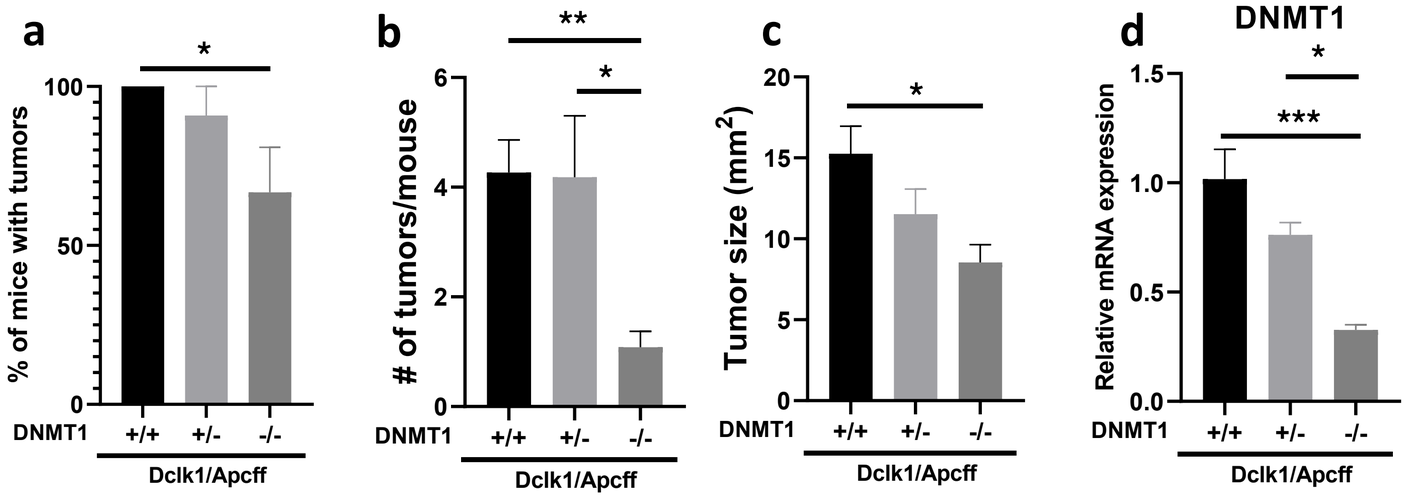
putting it together — plots

multiple panels become one (or few)

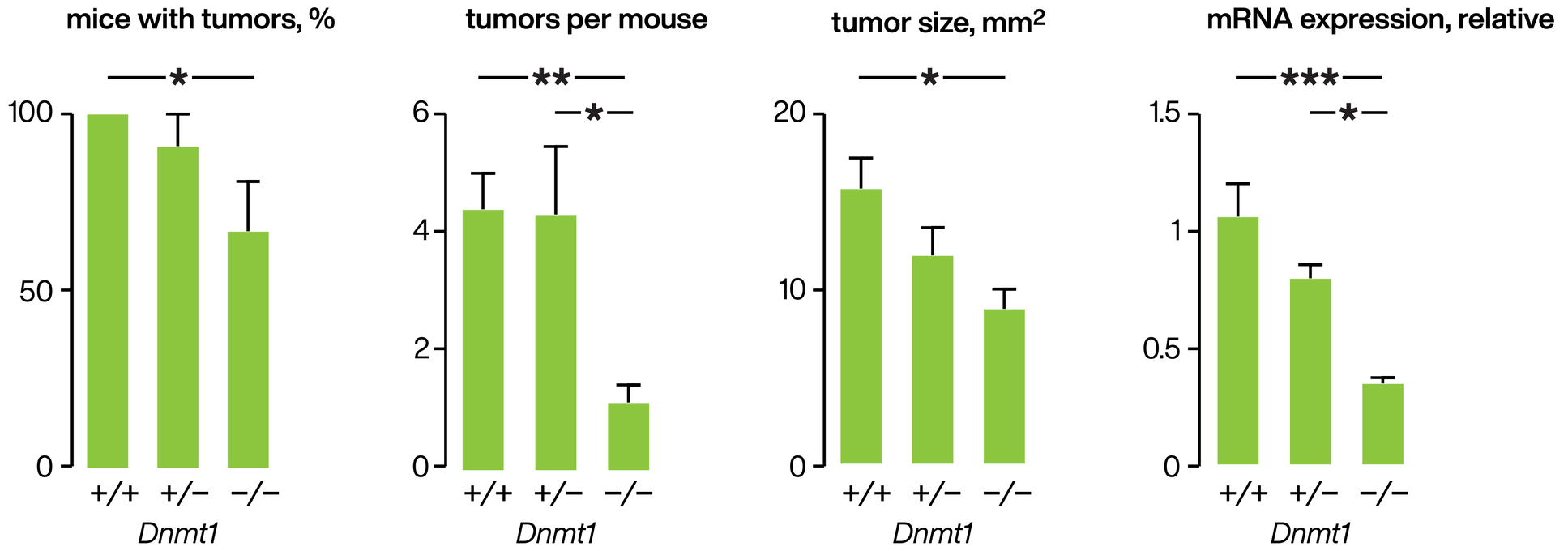

a
b
c
d
DNMT1
% of mice with tumors
# of tumors/mouse
Tumor size (mm2)
Relative mRNA expression
DNMT1
+/+
+/-
-/-
Dclk1/Apcff


a
b
c
d
e
f
5-mC% (5mC/Total DNA)
Ctrl
5-AZA
% of mice with tumors
Vehicle
# of tumors/mouse
Tumor size (mm2)



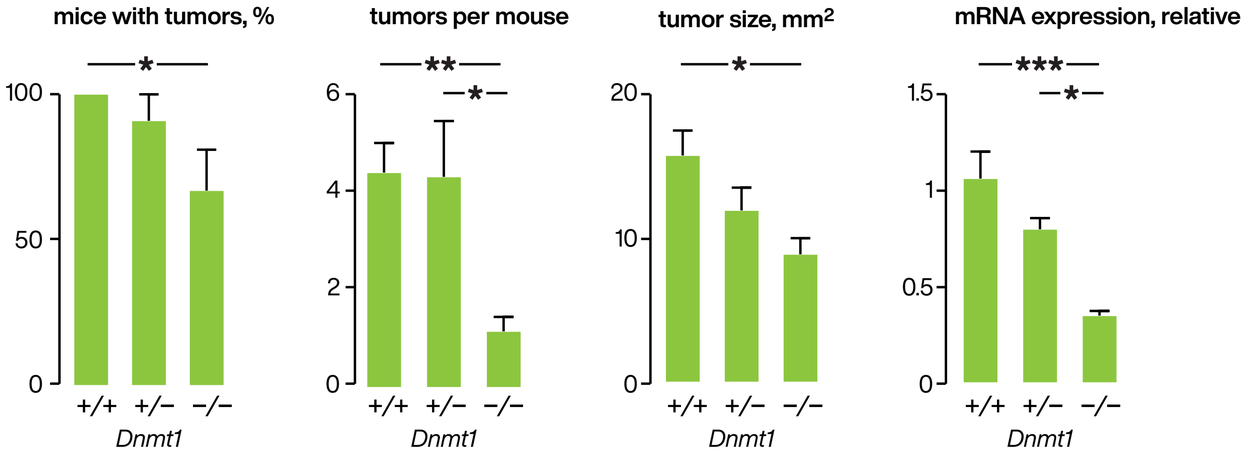
Loss of DNMT1 inhibits colitis-associated cancer

Dclk[1] *Apc*[f/f] *Dnmt1*[*/*]



Loss of DNMT1 inhibits colitis-associated cancer

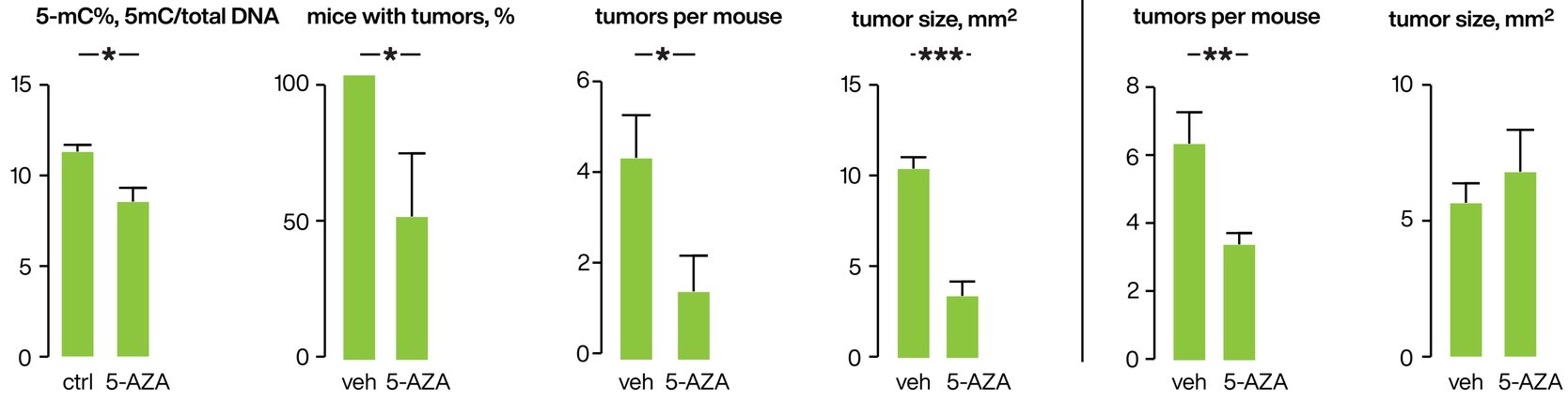
Dclk^{1} *Apc*$^{f/f}$ *Dnmt1*$^{*/*}$



(a) Percentage of mice with tumors, (b) tumor number, and (c) size of Dclk1CreERT2;Apcfl/fl;Dnmt1fl/fl mice compared to Dnmt1fl/+ and Dnmt1+/+ mice. (d) Relative mRNA expression of DNMT1 of tumors from Dnmt1+/+ Dnmt1fl/+ and Dnmt1fl/fl mice. The percentage of mice with tumors was significantly reduced with DNMT1 deletion as well as tumor number and size compared to Dnmt1fl/+ and Dnmt1+/+ mice. Data are presented as mean±SEM. (*p<0.05, **p<0.01, ***p<0.001)

5-AZA inhibits colitis-associated cancer

Dclk^{1} *Apc*$^{f/f}$ *CreERT*^{2}



(a) Percentage 5-methylcytosine in colonic epithelial cells from WT mice treated acutely with vehicle vs 5-AZA, (b) percentage of mice with tumors, (c) tumor number, and (d) tumors size of Dclk1CreERT2;Apcfl/fl.mice treated with vehicle (left) or 5-AZA (right). (e) Tumor number and (f) size in AOM/DSS mice treated with vehicle (left) or 5-AZA (right). Mice treated with 5-AZA show a significant reduction in colonic tumor number and size. Data are presented as mean±SEM. (*p<0.05, **p<0.01, ***p<0.001)

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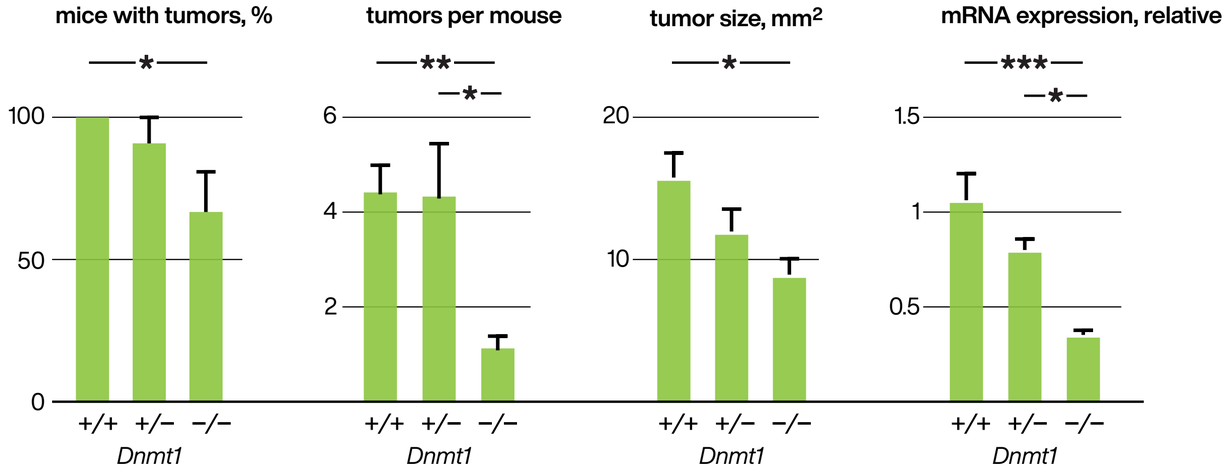

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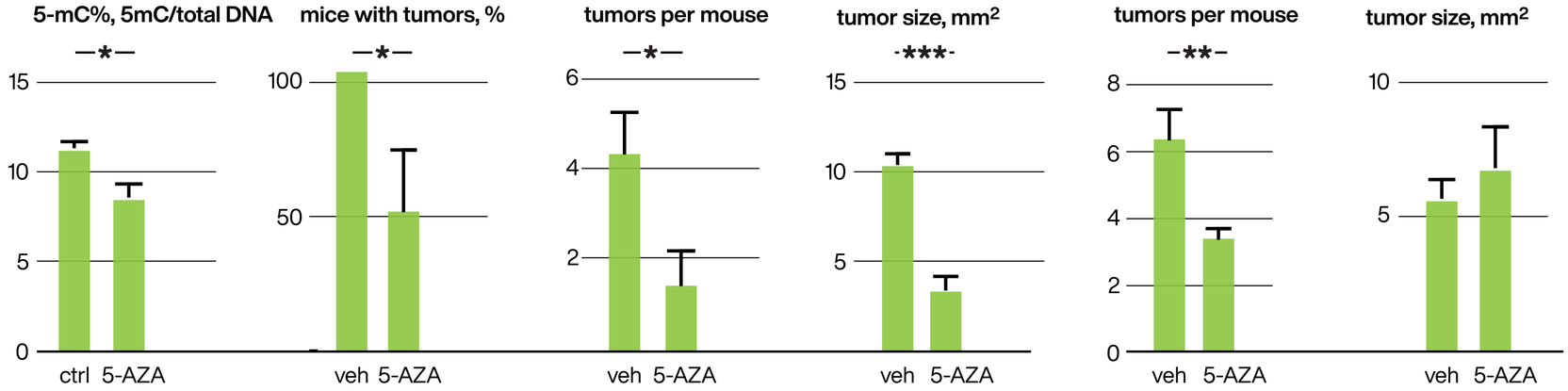
5-AZA inhibits colitis-associated cancer

Dclk^{1} *Apc*$^{f/f}$ *CreERT2*

AOM/DSS



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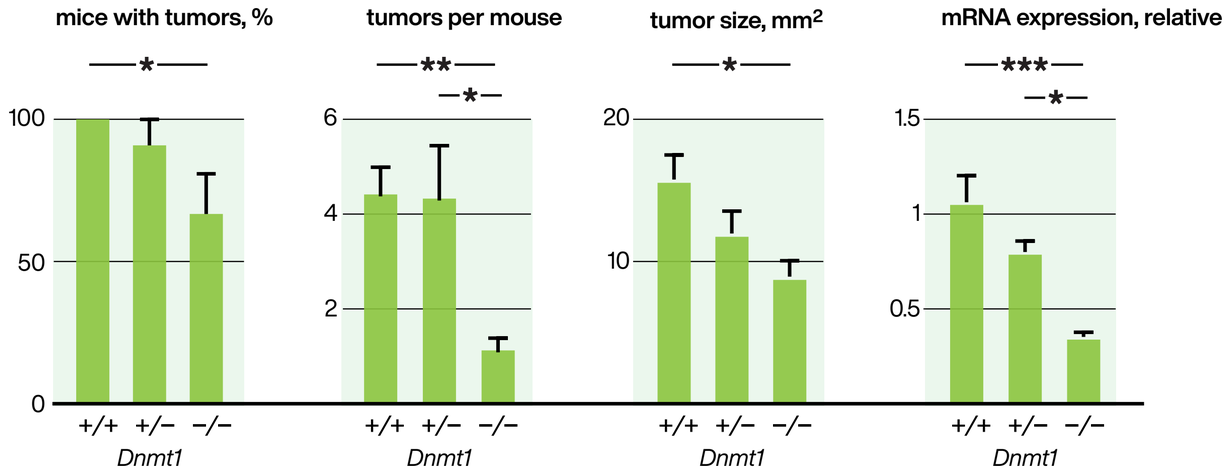

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Dclk^{1} *Apc*$^{f/f}$ *CreERT*^{2}

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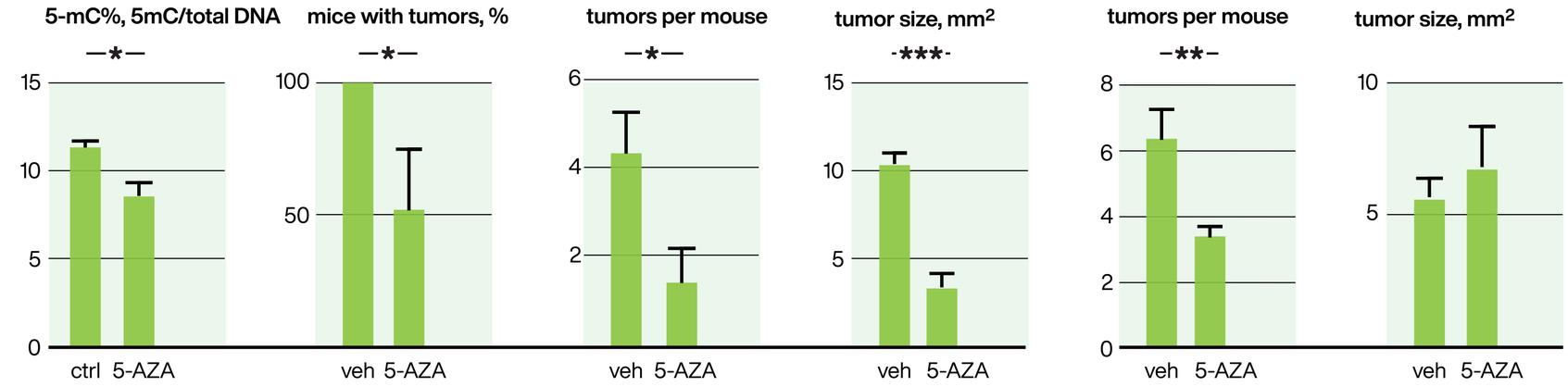

(a) Percentage 5-methylcytosine in colonic epithelial cells from WT mice treated acutely with vehicle vs 5-AZA, (b) percentage of mice with tumors, (c) tumor number, and (d) tumors size of Dclk1CreERT2;Apcfl/fl.mice treated with vehicle (left) or 5-AZA (right). (e) Tumor number and (f) size in AOM/DSS mice treated with vehicle (left) or 5-AZA (right). Mice treated with 5-AZA show a significant reduction in colonic tumor number and size. Data are presented as mean±SEM. (*p<0.05, **p<0.01, ***p<0.001)

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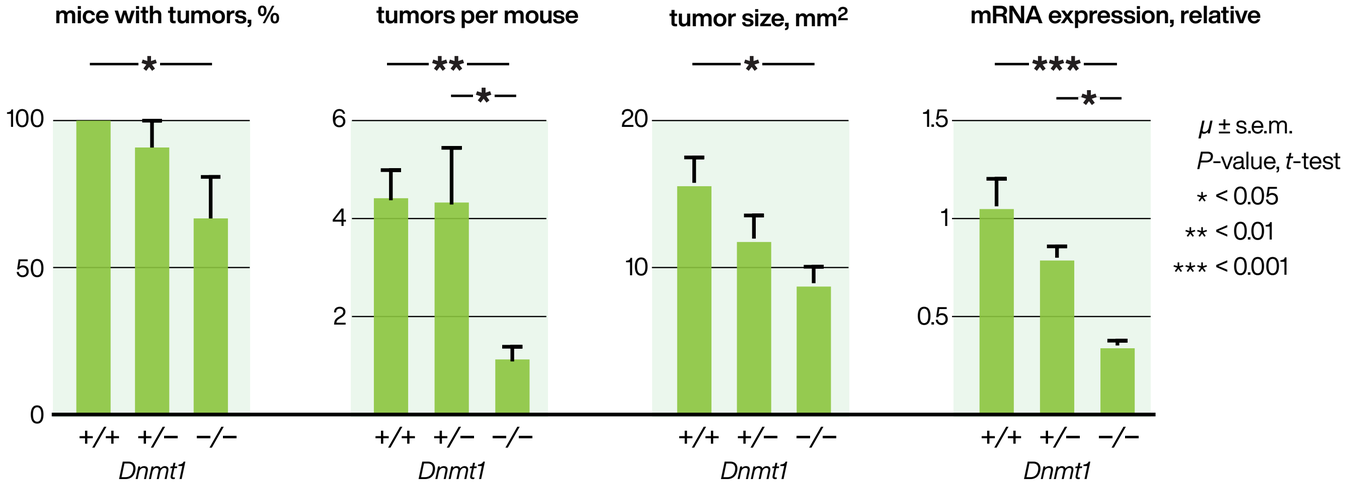

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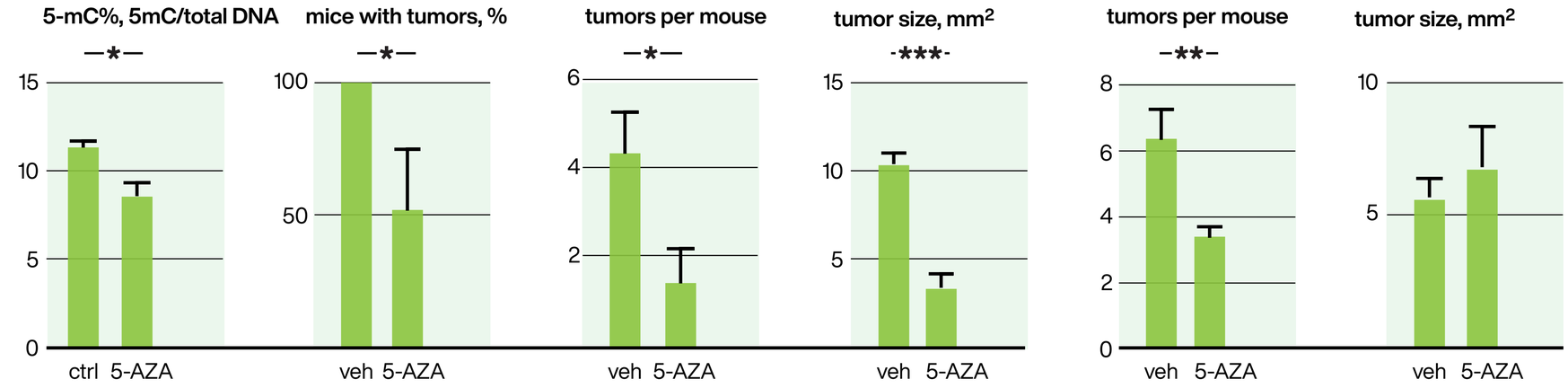

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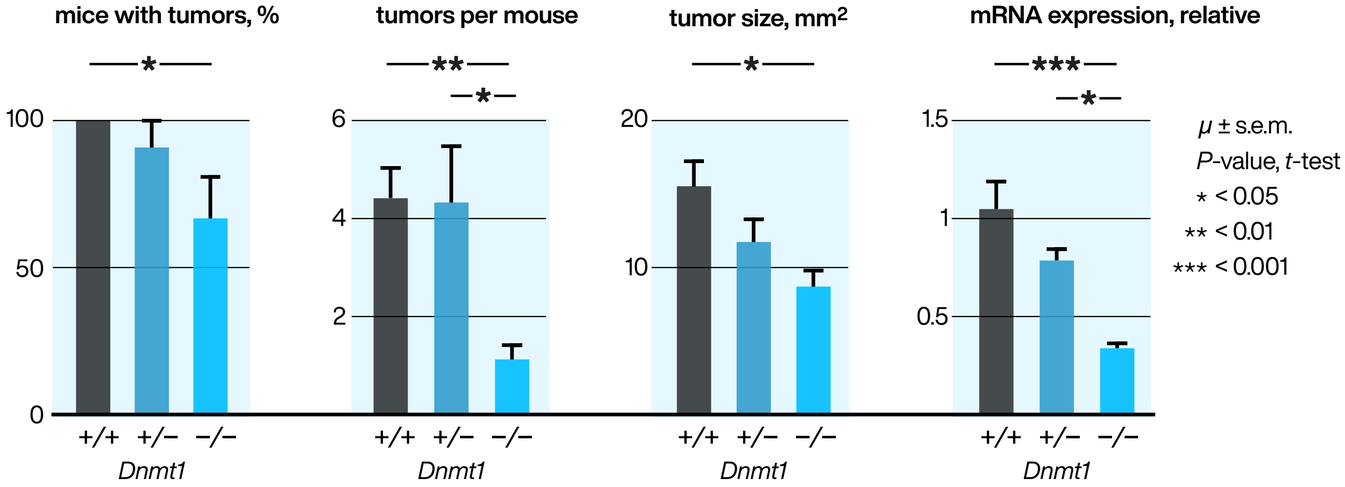

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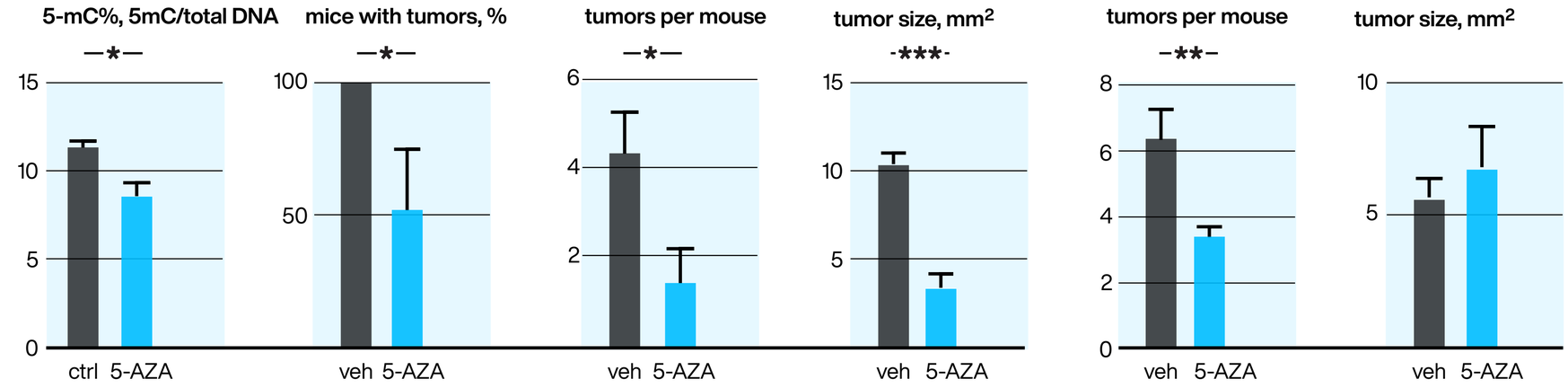

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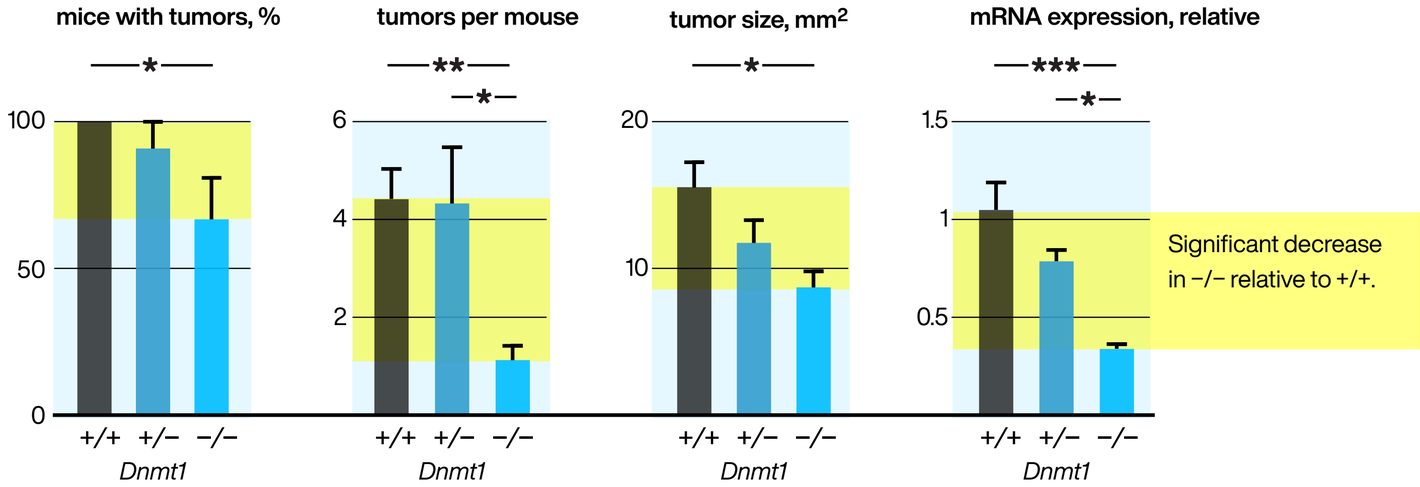

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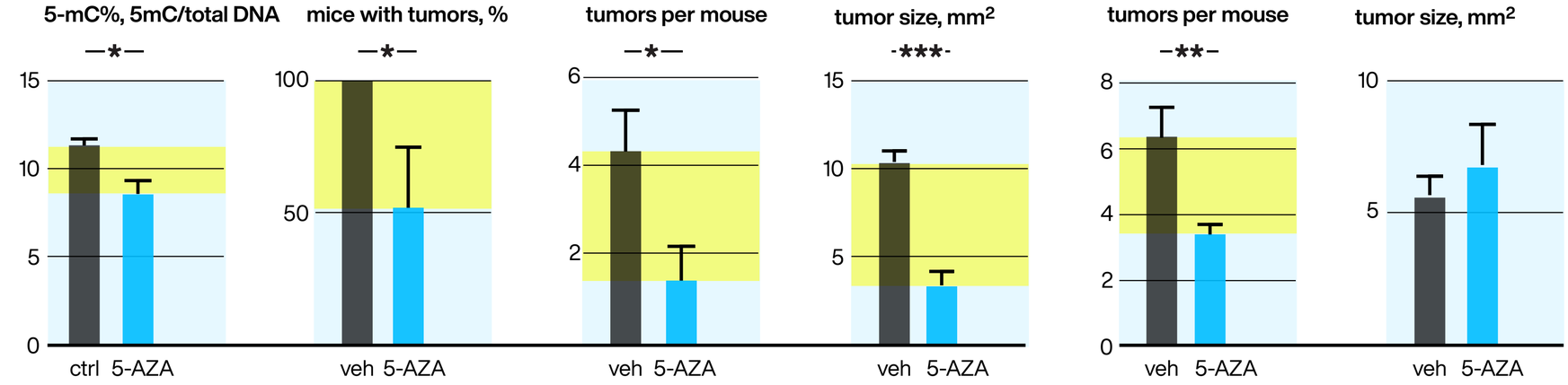

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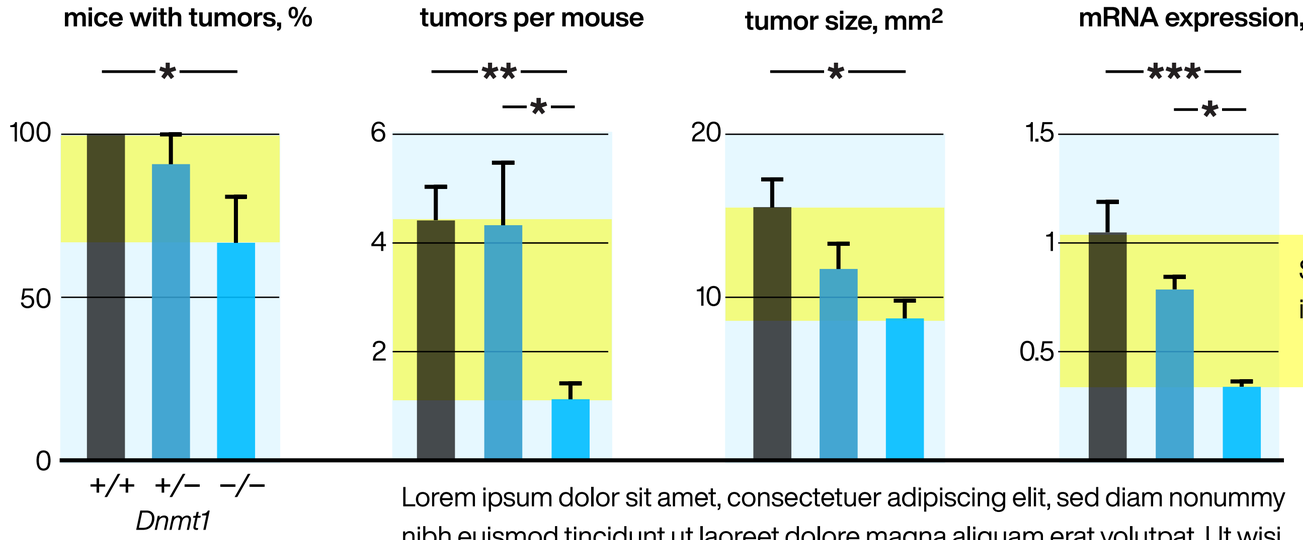

Significant decrease in −/− relative to +/+.

$\mu \pm$ s.e.m.
P-value, *t*-test
* < 0.05
** < 0.01
*** < 0.001

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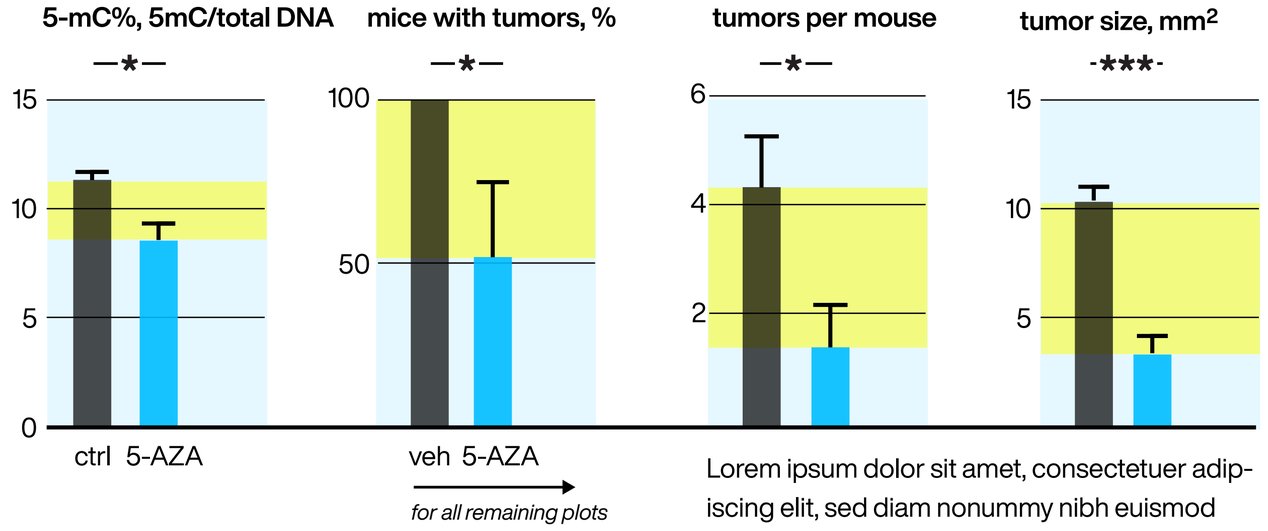

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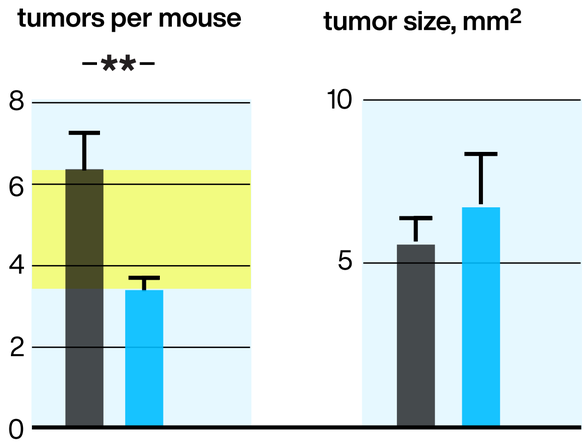
AOM/DSS



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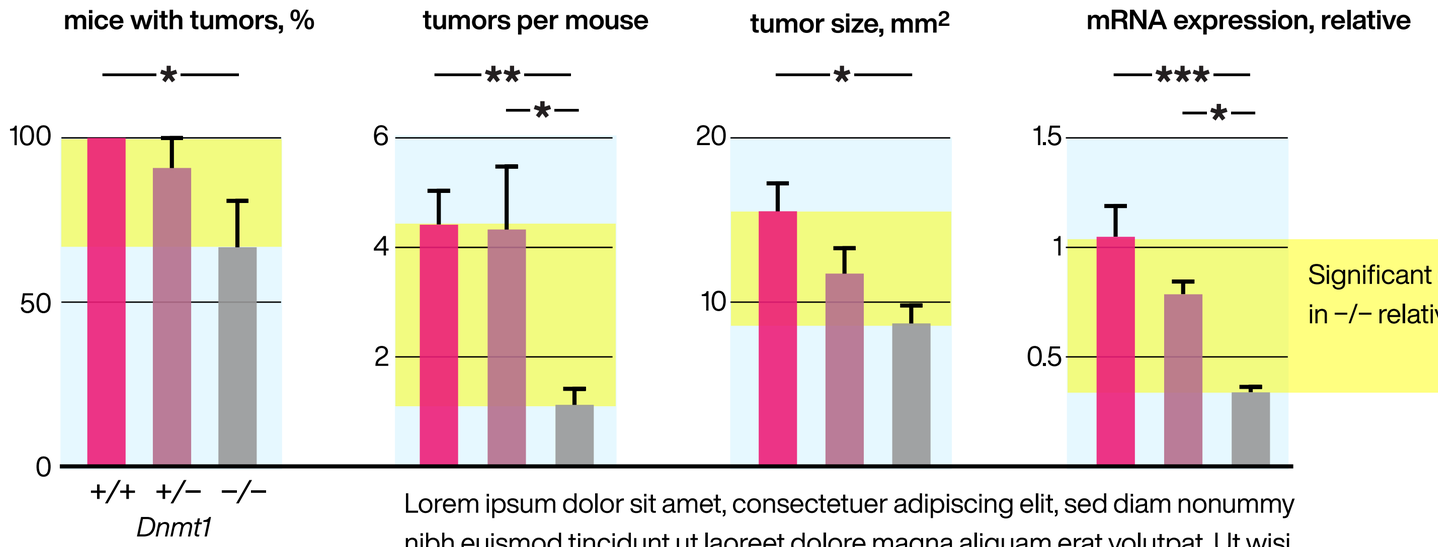
Dclk[1] *Apc*[f/f] *Dnmt1*[*/*]



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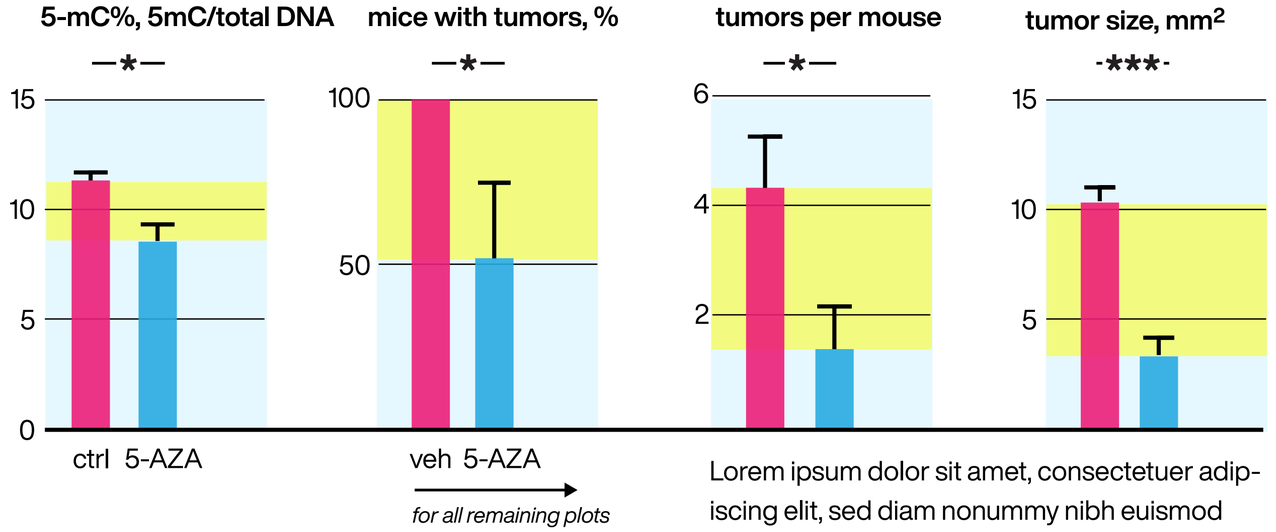
5-AZA inhibits colitis-associated cancer

Dclk[1] *Apc*[f/f] *CreERT*[2]



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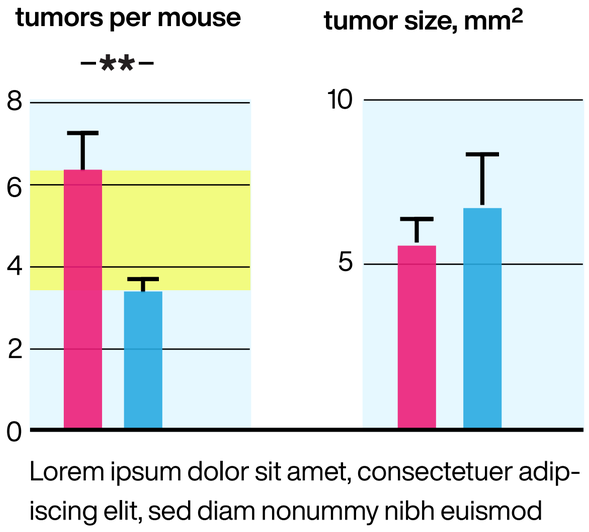
AOM/DSS



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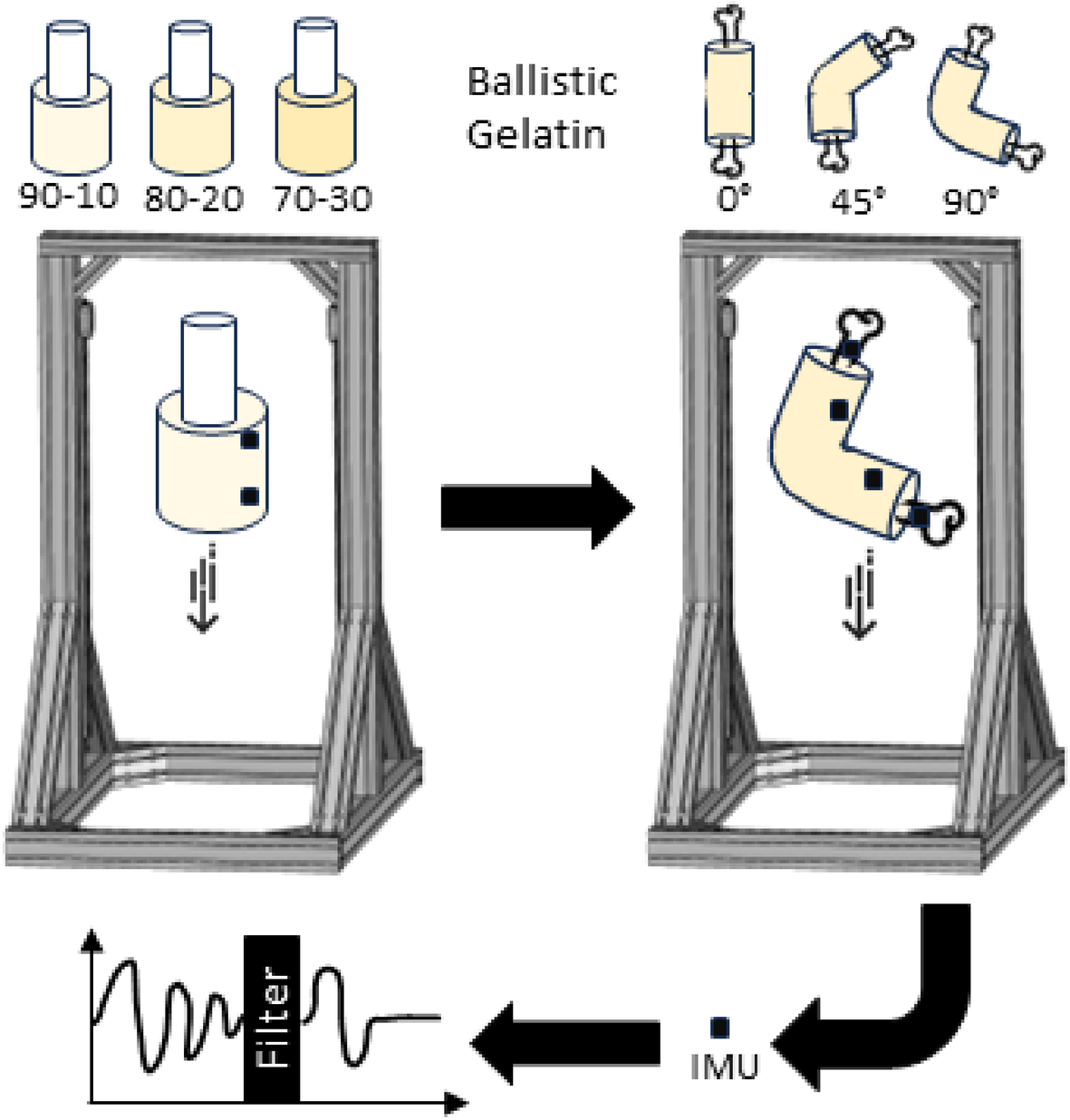
putting it together — graphical abstracts

problem & solution


90-10
80-20
70-30
Ballistic
Gelatin
0°
45°
90°
Filter
IMU

IMPROVED KALMAN FILTERS MITIGATE SOFT TISDUE ARTEFACTS IN MODELLING ACL INJURY

knee joint

ACL

causes of ACL injury

rapid deceleration

pivot move

error in measurement

sensors on soft tissue may experience different acceleration than knee joint

Kalman filters partially mitigate this via correction δ

ballistic gelatin models

optimizing the Kalman filter

variable gelatin stiffness

measure IMU deceleration

optimize Kalman filter

stiff cable

predicted optimized measured

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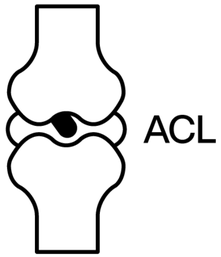
knee joint



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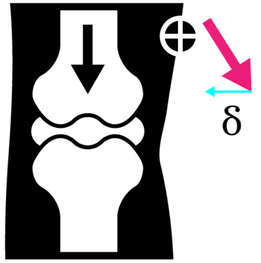

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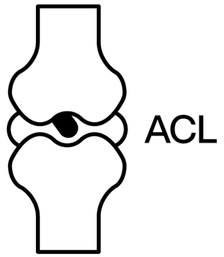

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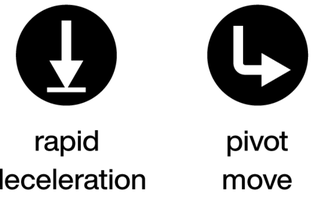

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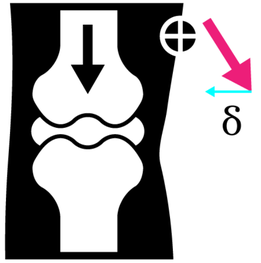

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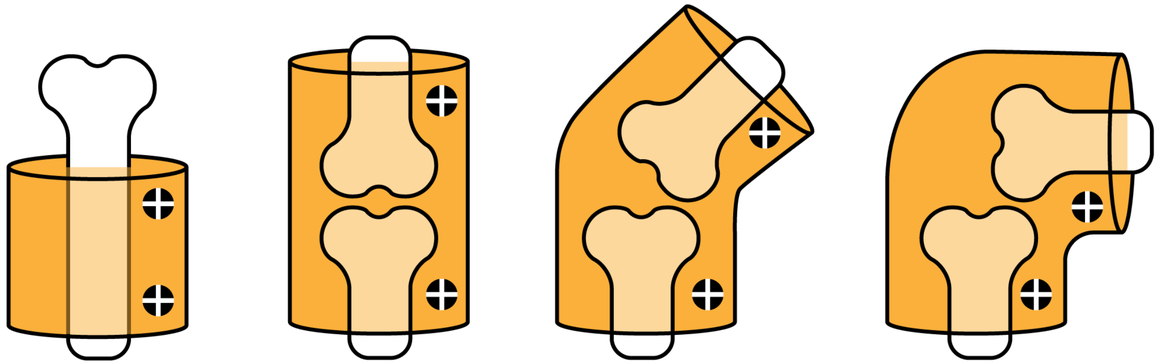

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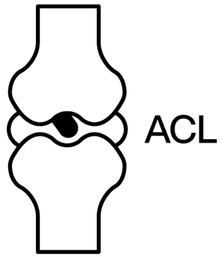

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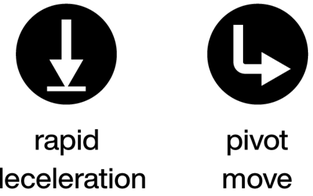

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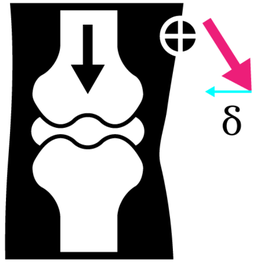

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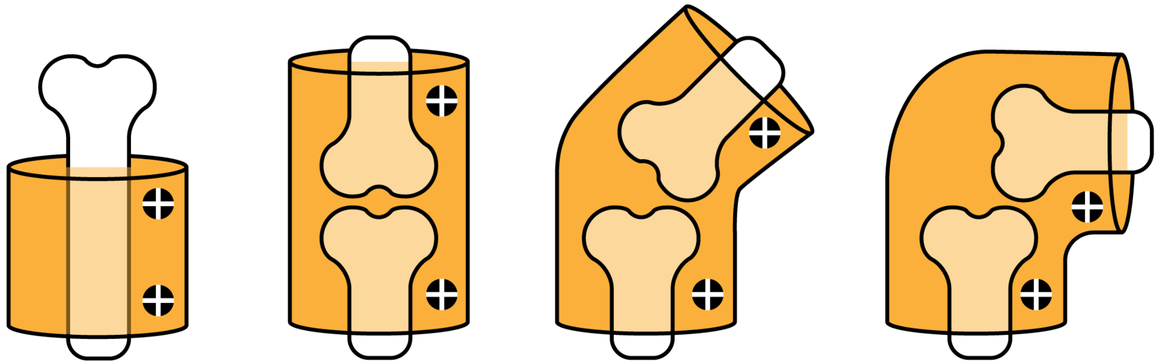

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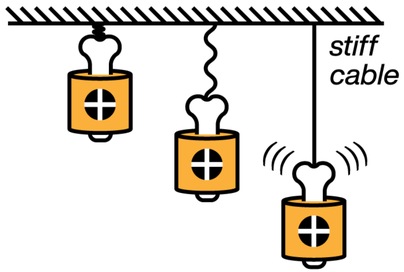
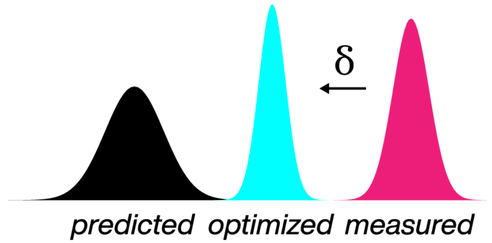
variable gelatin stiffness

measure IMU deceleration



optimize Kalman filter



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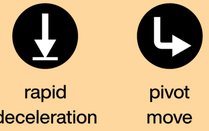

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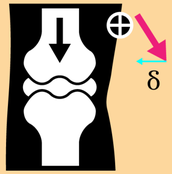

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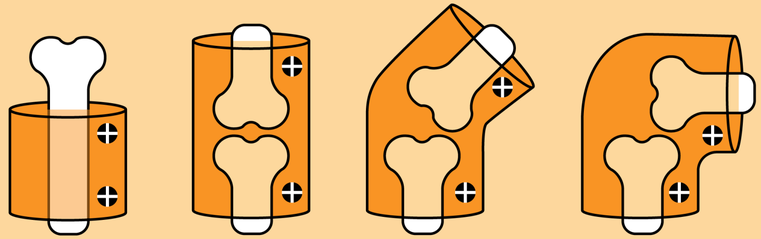

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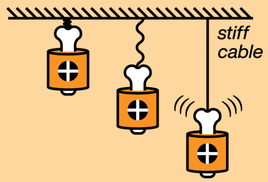
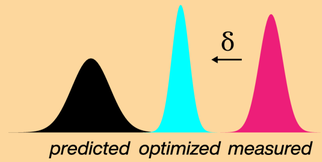
variable gelatin stiffness

measure IMU deceleration



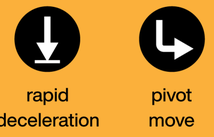
optimize Kalman filter



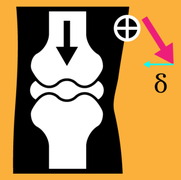
IMPROVED KALMAN FILTERS MITIGATE SOFT TISDUE ARTEFACTS IN MODELLING ACL INJURY

knee joint

causes of ACL injury



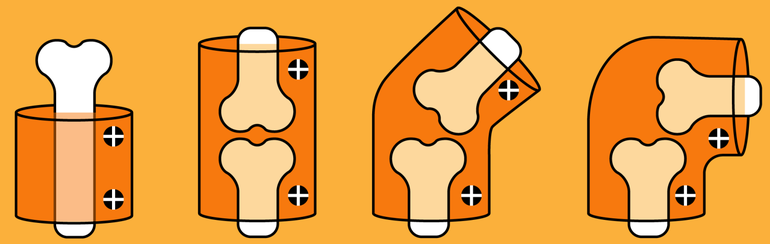
error in measurement



sensors on soft tissue may experience different acceleration than knee joint

Kalman filters partially mitigate this via correction δ

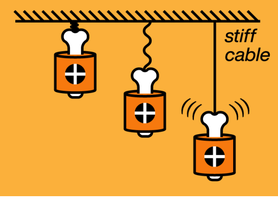
ballistic gelatin models



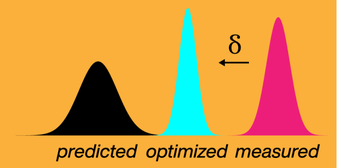
optimizing the Kalman filter

variable gelatin stiffness

measure IMU deceleration



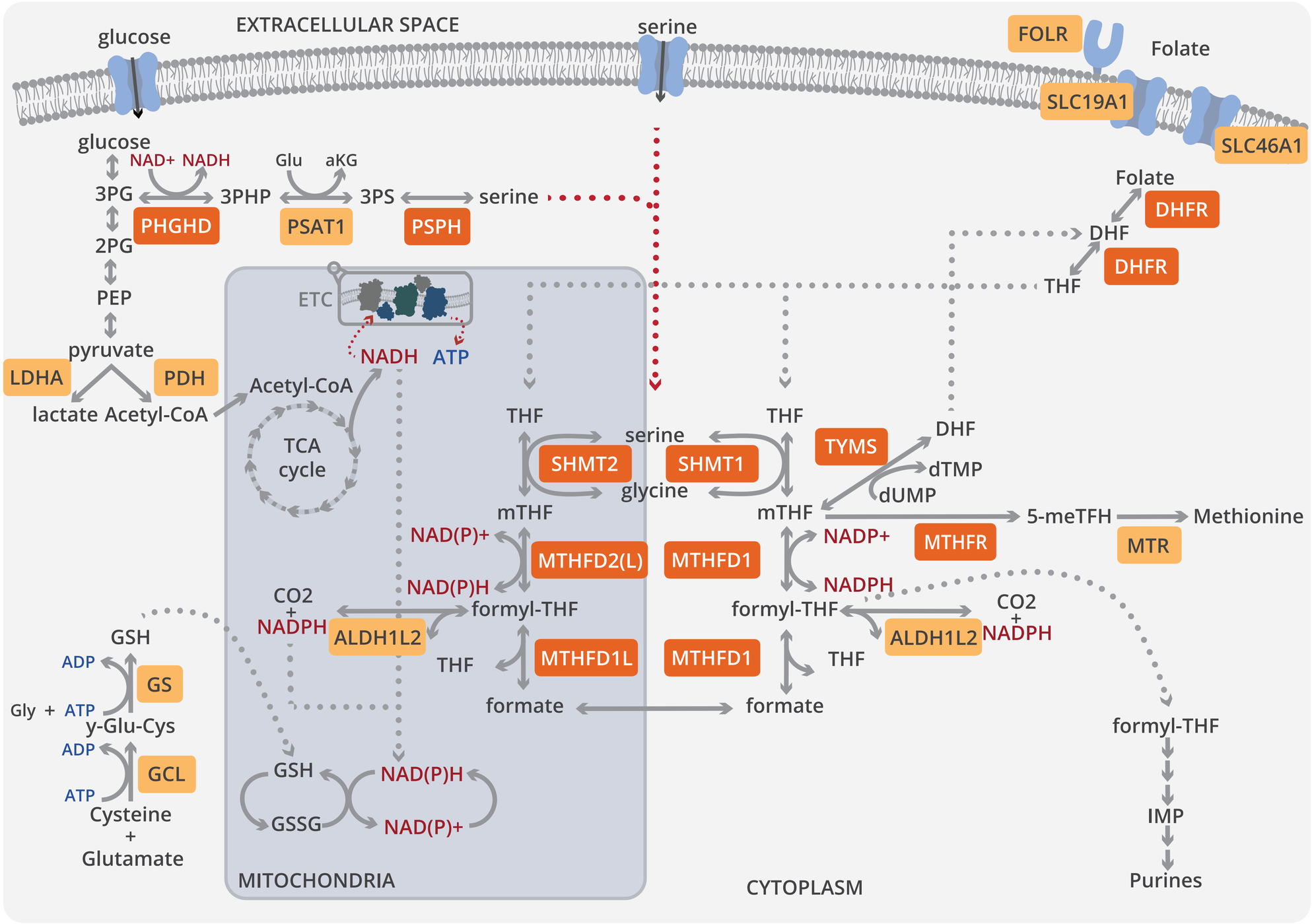
optimize Kalman filter



putting it together — pathways

arrows & compartments

this way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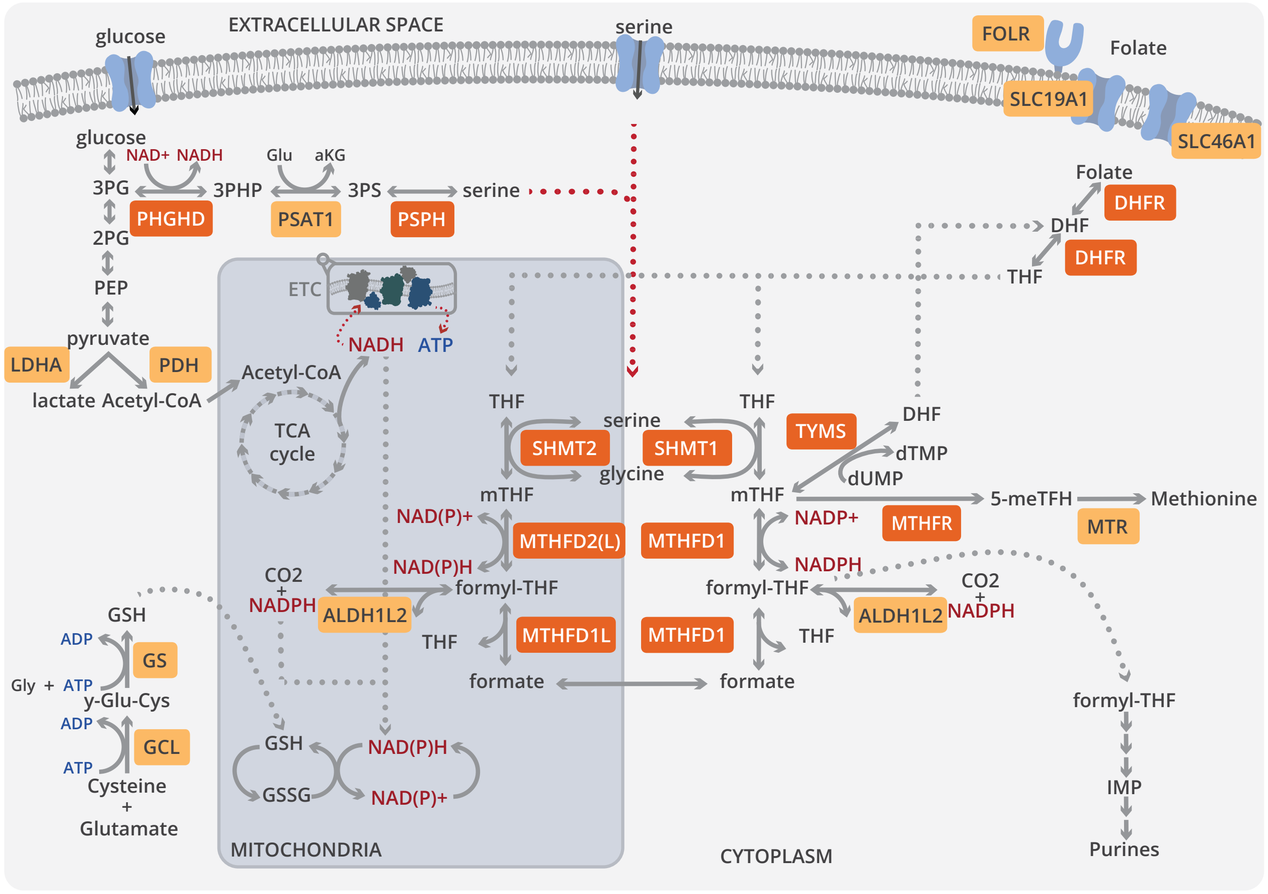
glucose
serine
FOLR
Folate
SLC19A1
SLC46A1
glucose
NAD+ NADH
Glu aKG
3PG
3PHP
3PS
serine
PHGHD
PSAT1
PSPH
2PG
PEP
pyruvate
LDHA
PDH
lactate
Acetyl-CoA
ETC
NADH
ATP
TCA cycle
Folate
DHFR
DHF
DHFR
THF
THF
serine
SHMT2
SHMT1
glycine
THF
TYMS
DHF
dTMP
dUMP
mTHF
mTHF
5-meTFH
Methionine
NAD(P)+
MTHFD2(L)
MTHFD1
NADP+
MTHFR
MTR
NAD(P)H
NADPH
CO2
+
NADPH
formyl-THF
formyl-THF
CO2
+
NADPH
ALDH1L2
ALDH1L2
GSH
ADP
GS
Gly + ATP
y-Glu-Cys
THF
MTHFD1L
MTHFD1
THF
formate
formate
formyl-THF
ADP
GCL
ATP
Cysteine
+
Glutamate
GSH
NAD(P)H
GSSG
NAD(P)+
IMP
Purines
MITOCHONDRIA
CYTOPLASM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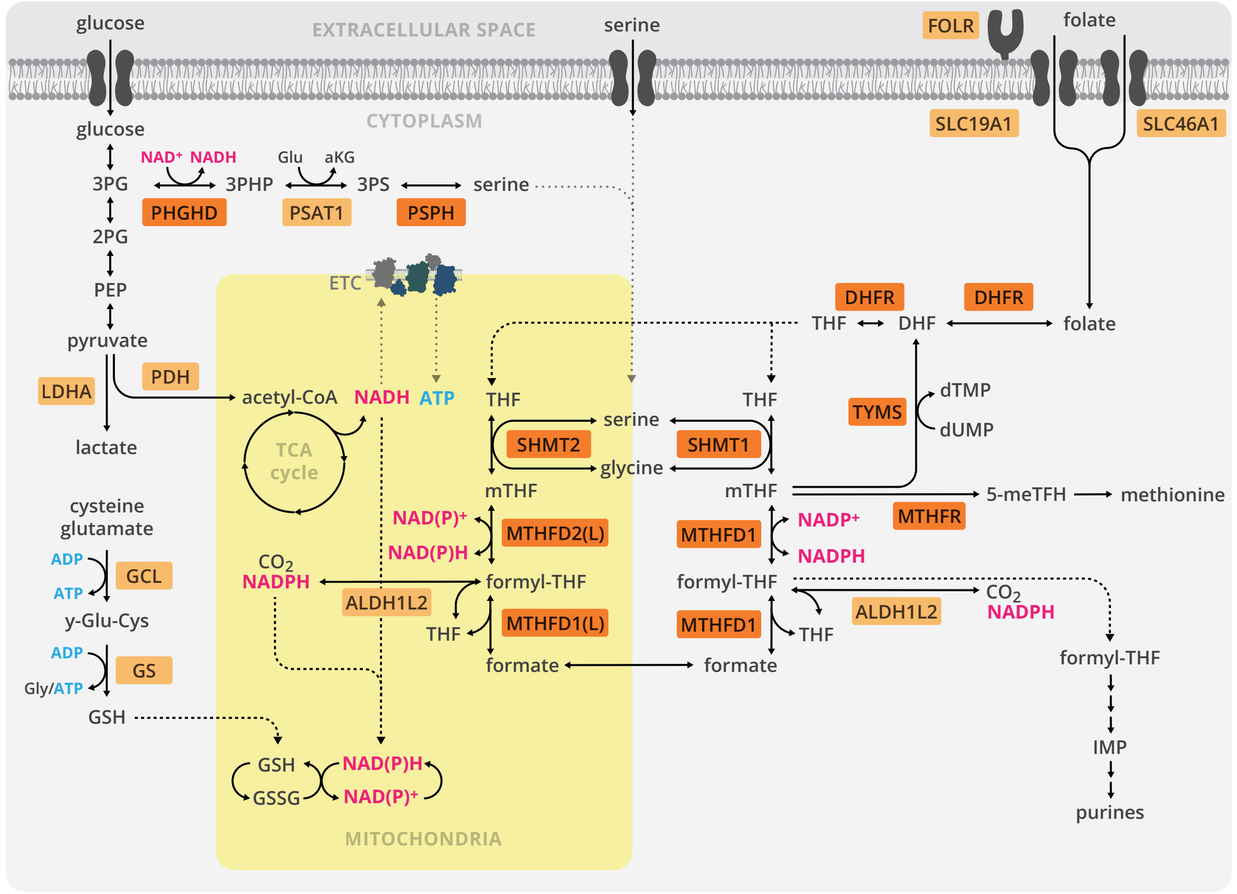
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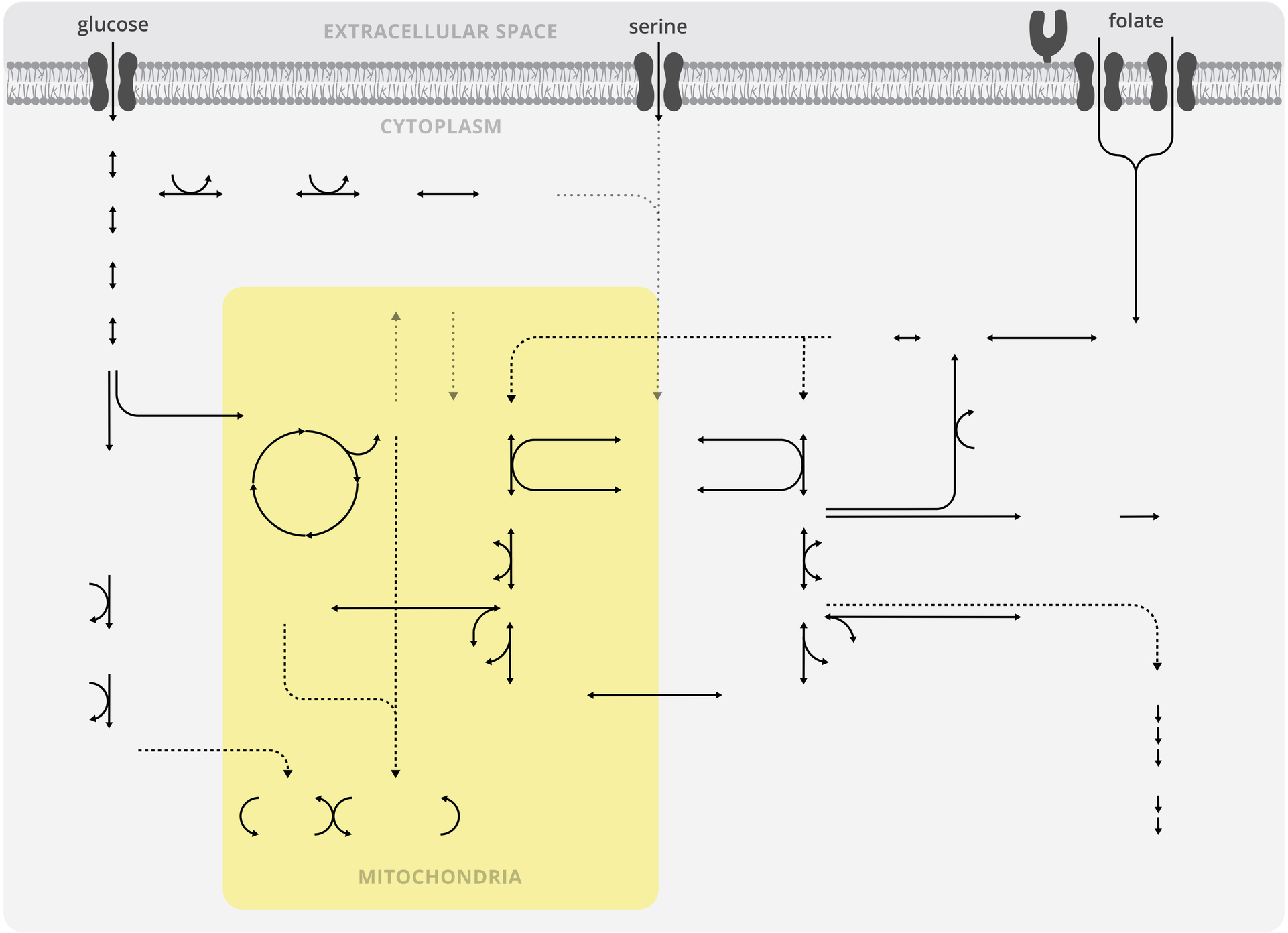
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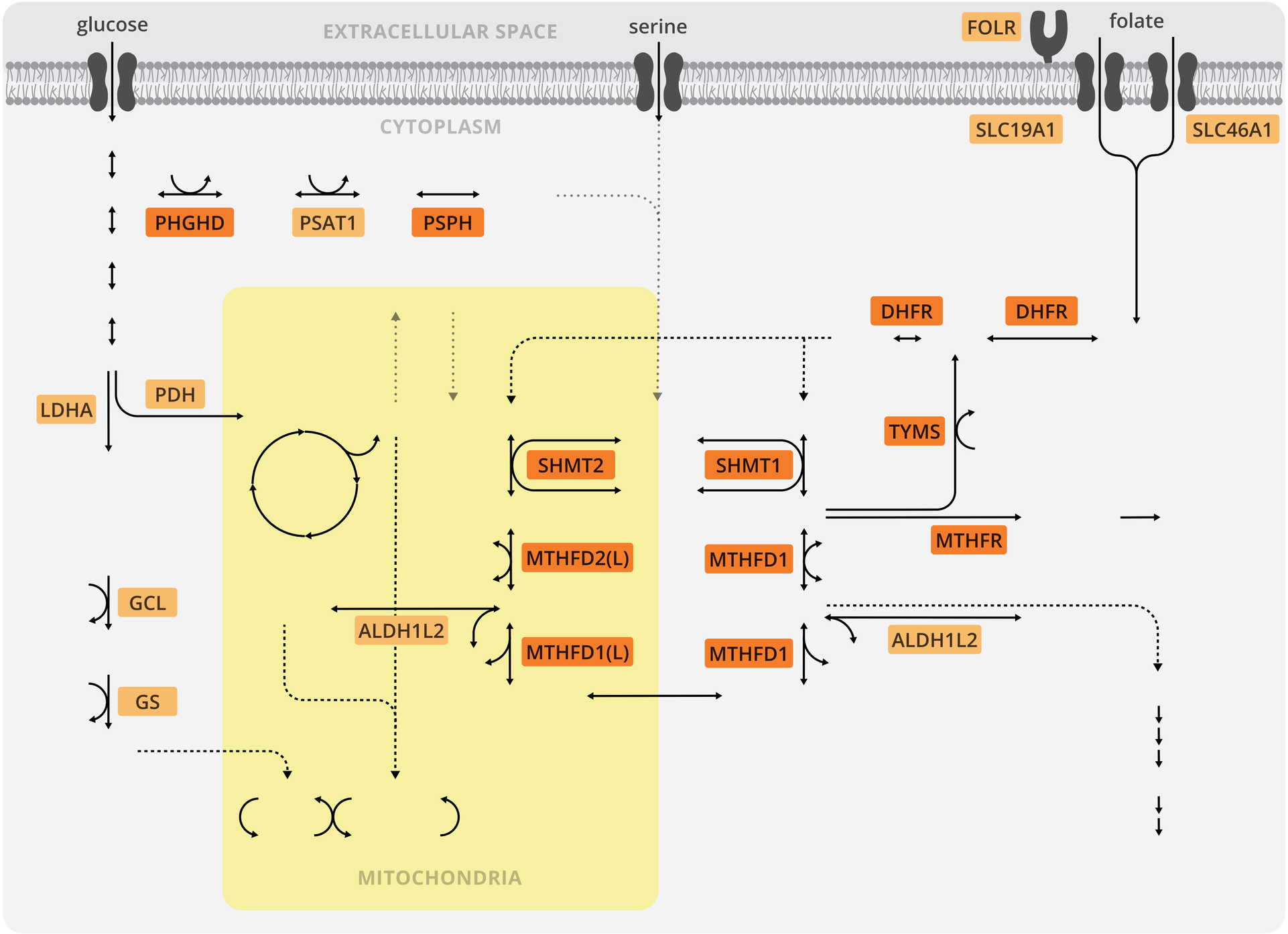
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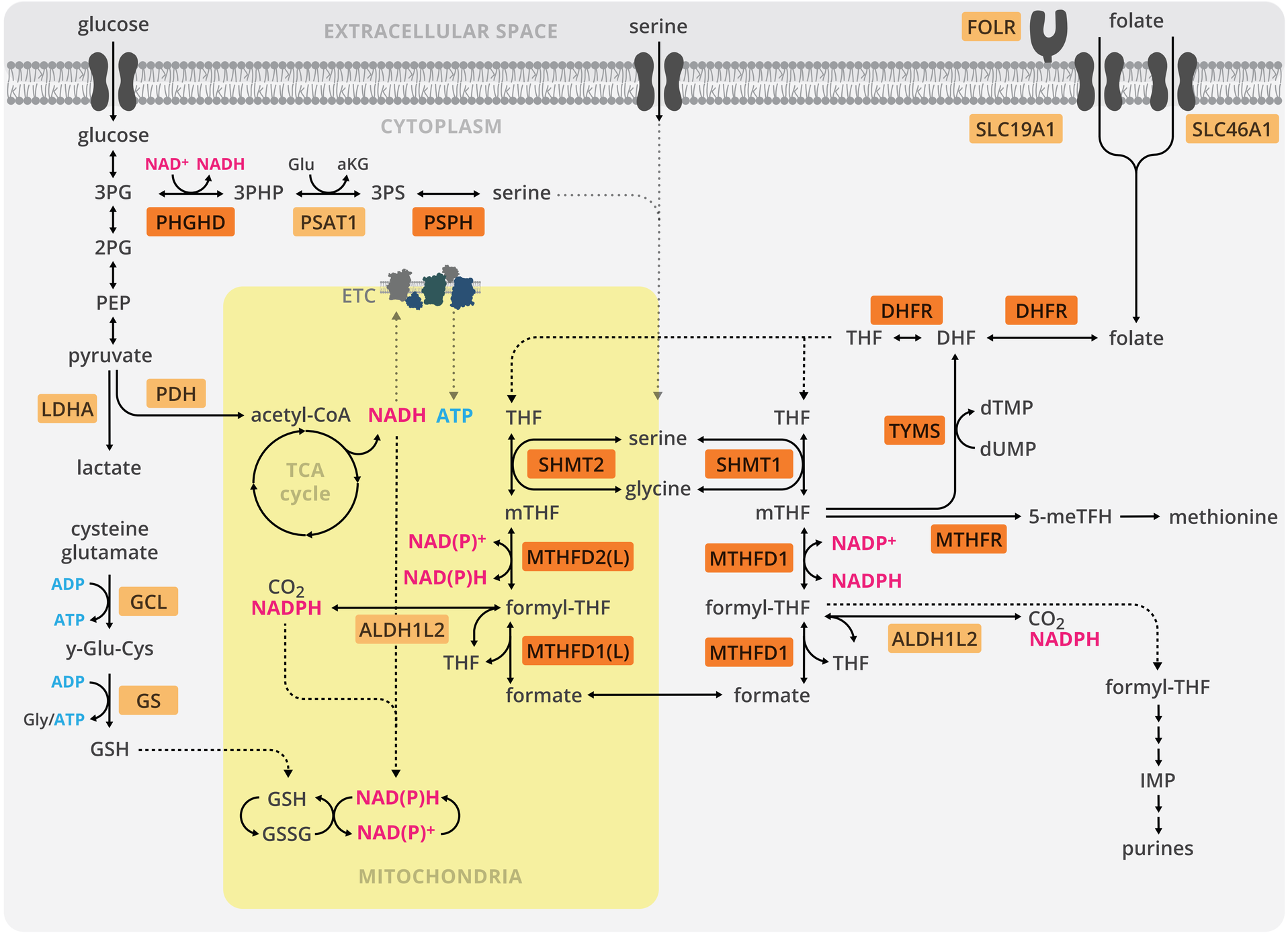
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ALDH1L2
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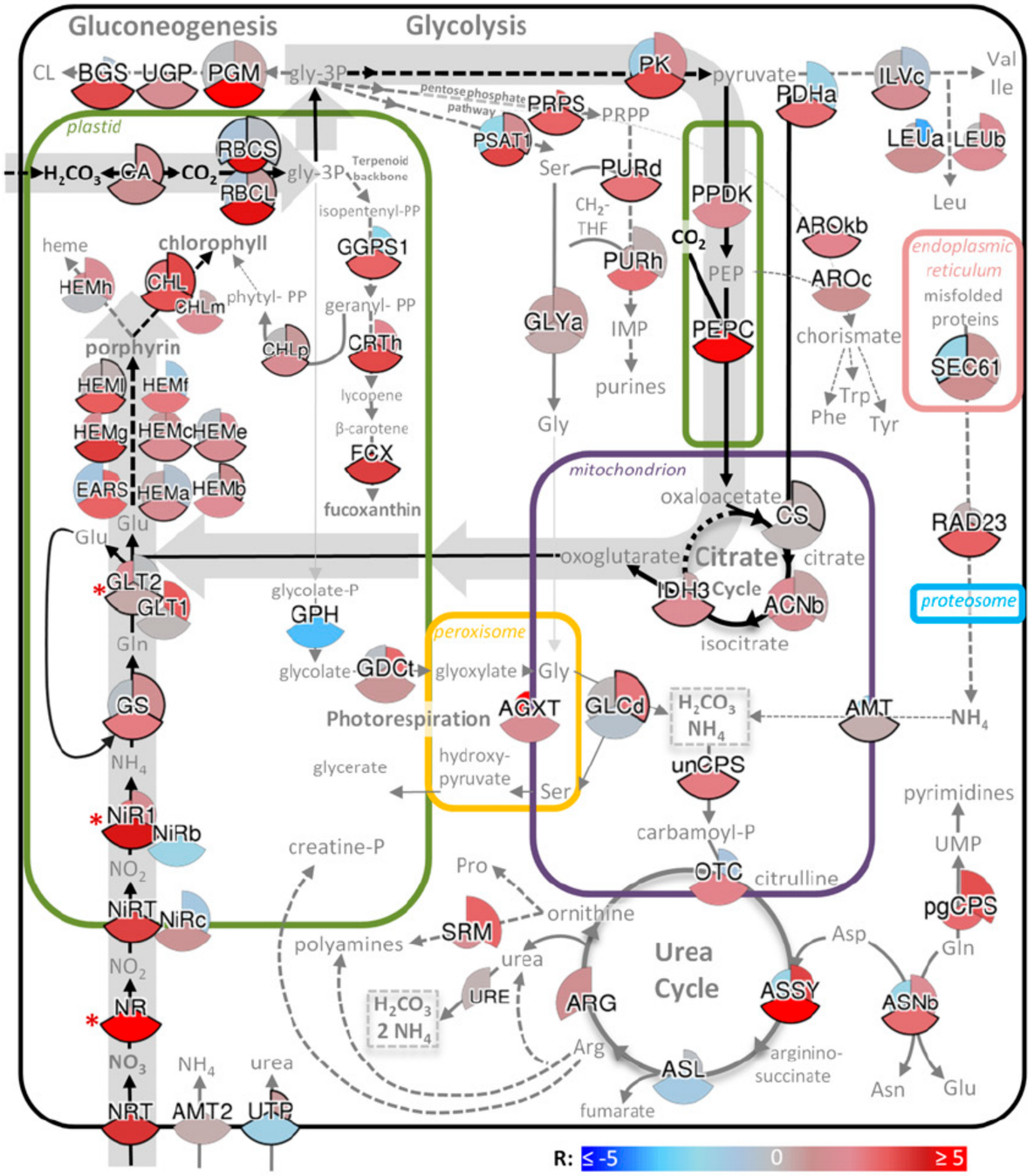
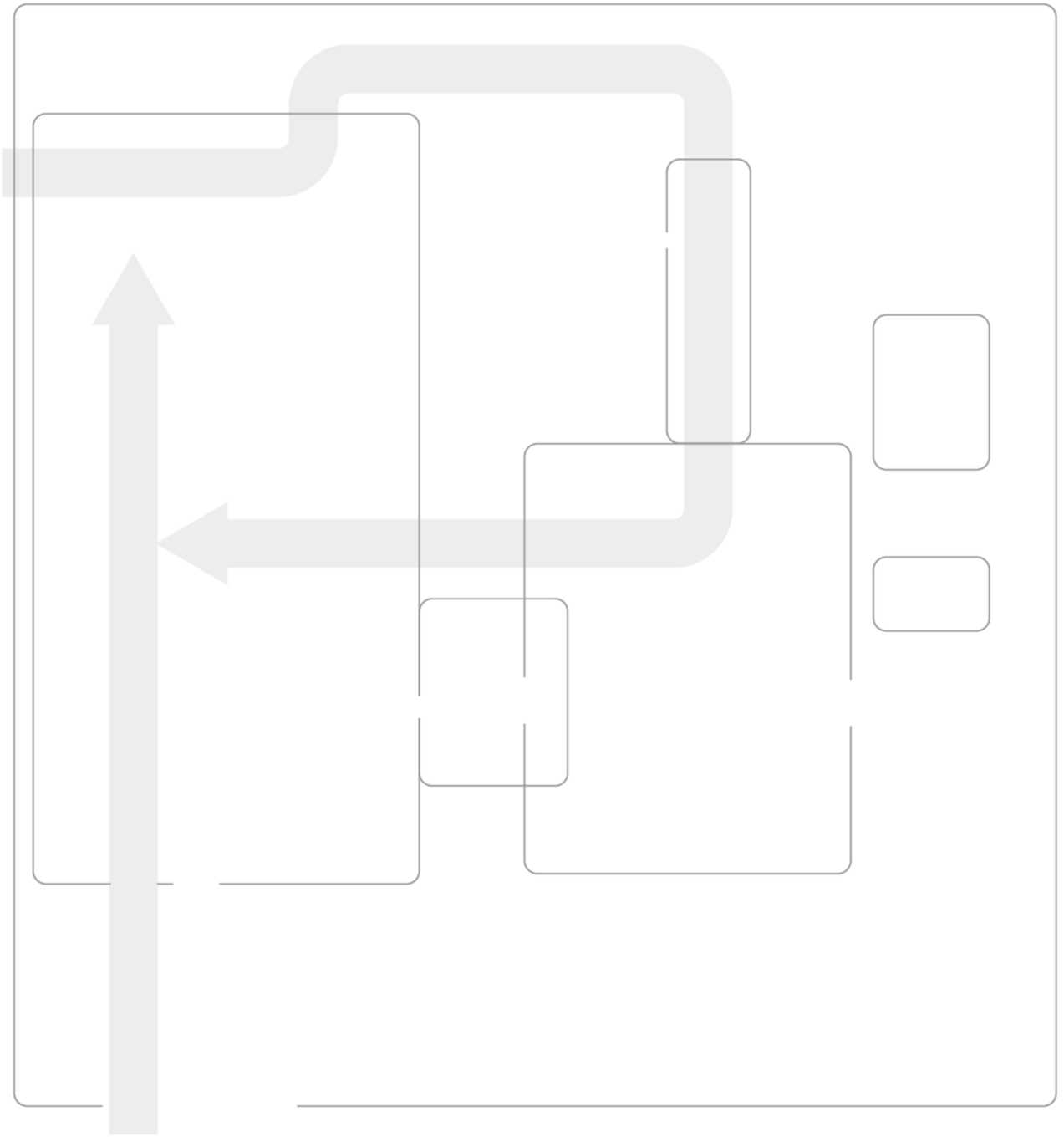
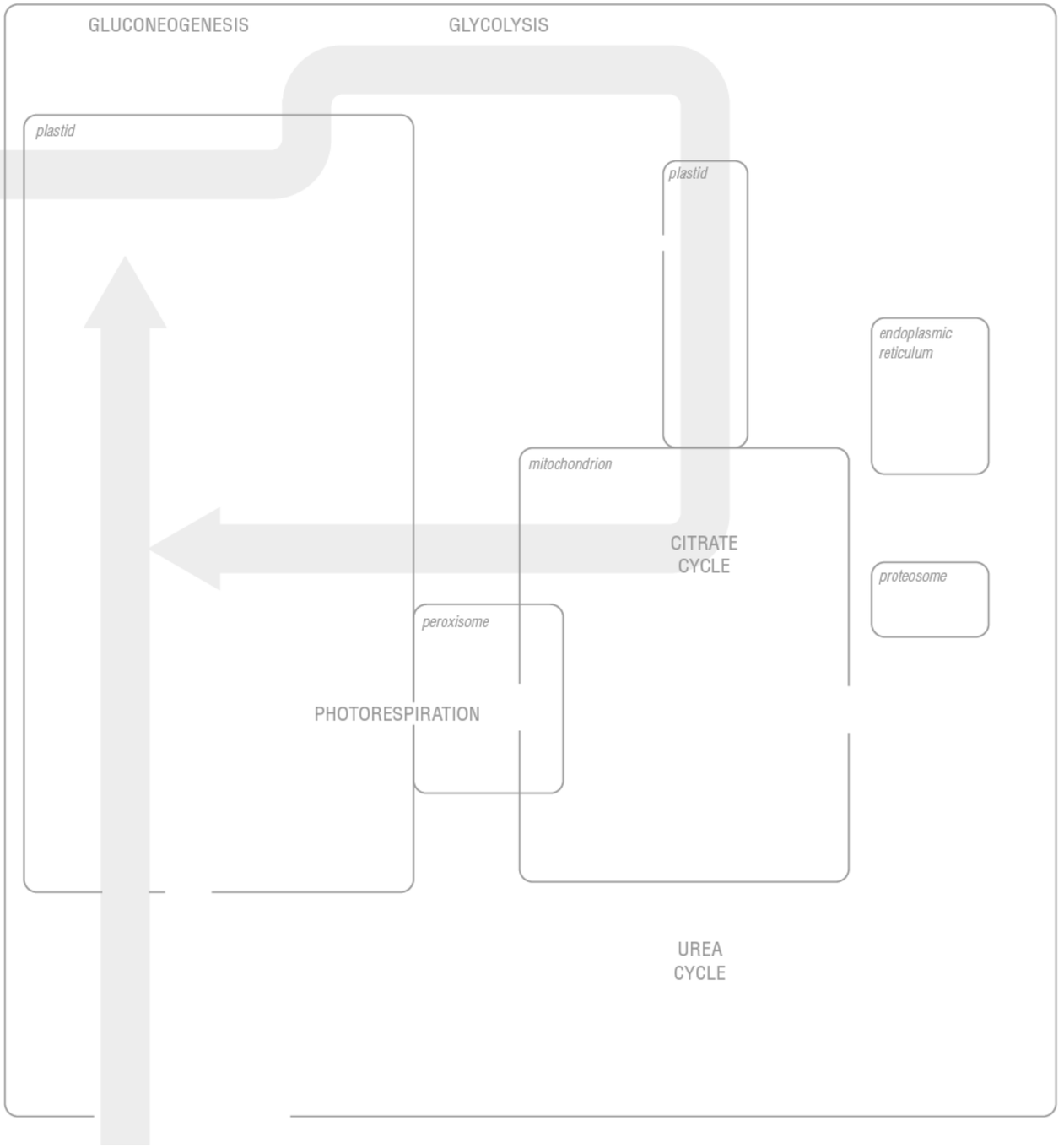


ACNb	aconitate hydratase 2
AGXT	serine--pyruvate aminotransferase
AMT, 2	**ammonium transporter**
ARG	arginase/agmatinase/formimionoglutamate hydrolase
AROc	chorismate synthase
AROkb	3-dehydroquinate synthase
ASL	argininosuccinate lyase
ASNb	asparagine synthase (glutamine-hydrolysing)
ASSY	**argininosuccinate synthase**
BGS	**callose synthase**
CA	**carbonic anhydrase**
CHL	**magnesium chelatase**
CHLm	magnesium-protoporphyrin O-methyltransferase
CHLp	geranylgeranyl reductase
CRTh	carotenoid isomerase
CS	citrate synthase
EARS	glutamyl-tRNA synthetase
FCX	**Lhcr7; fucoxanthin chl a/c light-harvesting protein**
GDTC	glycine dehydrogenase subunit 1
GGPS1	geranylgeranyl pyrophosphate synthetase
GLCD	glycolate oxidase
GLT1, 2	**glutamate synthase (NADPH/NADH, ferredoxin)**
GLYa	glycine hydroxymethyltransferase
GPH	2-phosphoglycolate phosphatase
GS	**glutamine synthetase**
HEMa	glutamyl-tRNA reductase
HEMb	porphobilinogen synthase
HEMc	hydroxymethylbilane synthase
HEMe	uroporphyrinogen decarboxylase
HEMf	coproporphyrinogen III oxidase
HEMg	protoporphyrinogen oxidase
HEMh	ferrochelatase
HEMl	glutamate-1-semialdehyde 2,1-aminomutase
IDH3	isocitrate dehydrogenase (NAD+)
ILVc	ketol-acid reductoisomerase
LEUa,b	2,3 isopropylmalate synthase, dehydrogenase
NiRb, 1	**nitrite reductase (NADH/NADPH, ferredoxin)**
NiRT, c	**nitrite transporters**
NR	**nitrate reductase**
NRT	**nitrate transporter**
OTC	**ornithine carbamoyltransferase**
PDHa	pyruvate dehydrogenase E1 component subunit alpha
PEPC	**phosphoenolpyruvate carboxylase**
pgCPS	carbamoyl-phosphate synthase / aspartate carbamoyltransferase
PGM	**phosphoglucomutase**
PK	pyruvate kinase
PPDK	pyruvate,orthophosphate dikinase
PRPS	ribose-phosphate pyrophosphokinase
PSAT1	phosphoserine aminotransferase
PURd	phosphoribosylamine--glycine ligase / phosphoribosylglycinamide
PURh	phosphoribosylaminoimidazolecarboxamide formyltransferase
RAD23	UV excision repair protein
RBCS, L	**ribulose-bisphosphate carboxylase (small , large) chain**
SEC61	transport protein subunit alpha
SRM	spermidine synthase
UGP	**precursor of phosphorylase udp-glucose diphosphorylase**
unCPS	**carbamoyl-phosphate synthase mitochindrial precursor**
URE	urease
UTP	**urea transporter**

Marchetti, A., et al. Proceedings of the National Academy of Sciences of the United States of America, 2012. 109(6): p. E317-E325.

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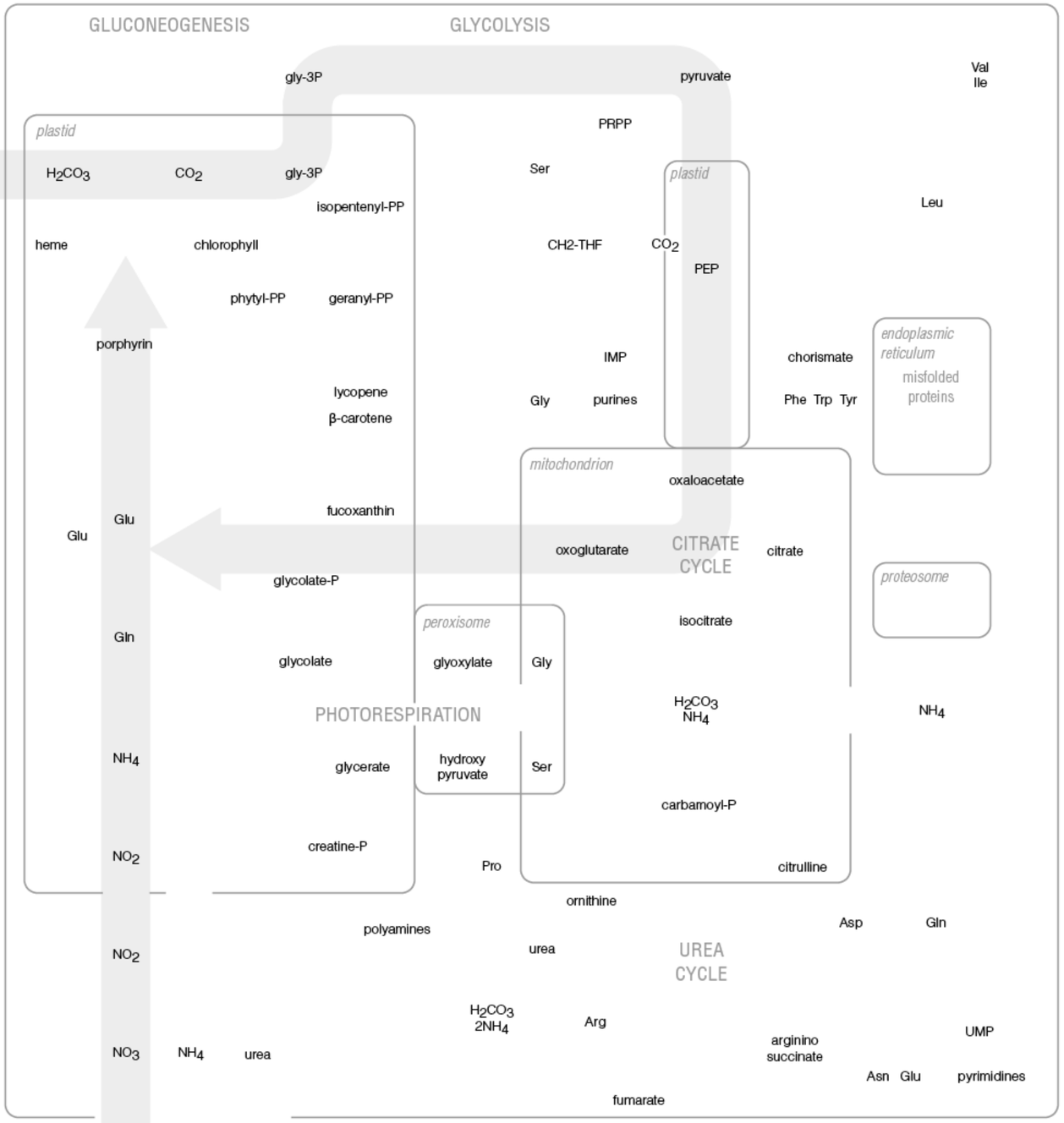
plastid
endoplasmic reticulum
mitochondrion
CITRATE CYCLE
proteosome
peroxisome
PHOTORESPIRATION
UREA CYCLE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CO_2
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Leu
heme
chlorophyll
CH2-THF
CO_2
PEP
phytyl-PP
geranyl-PP
endoplasmic reticulum
misfolded proteins
porphyrin
IMP
chorismate
lycopene
β-carotene
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purines
Phe Trp Tyr
mitochondrion
oxaloacetate
fucoxanthin
Glu
Glu
oxoglutarate
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citrate
proteosome
glycolate-P
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isocitrate
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glyoxylate
Gly
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PHOTORESPIRATION
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hydroxy pyruvate
Ser
carbamoyl-P
NO_2
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Pro
citrulline
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Gln
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urea
UREA CYCLE
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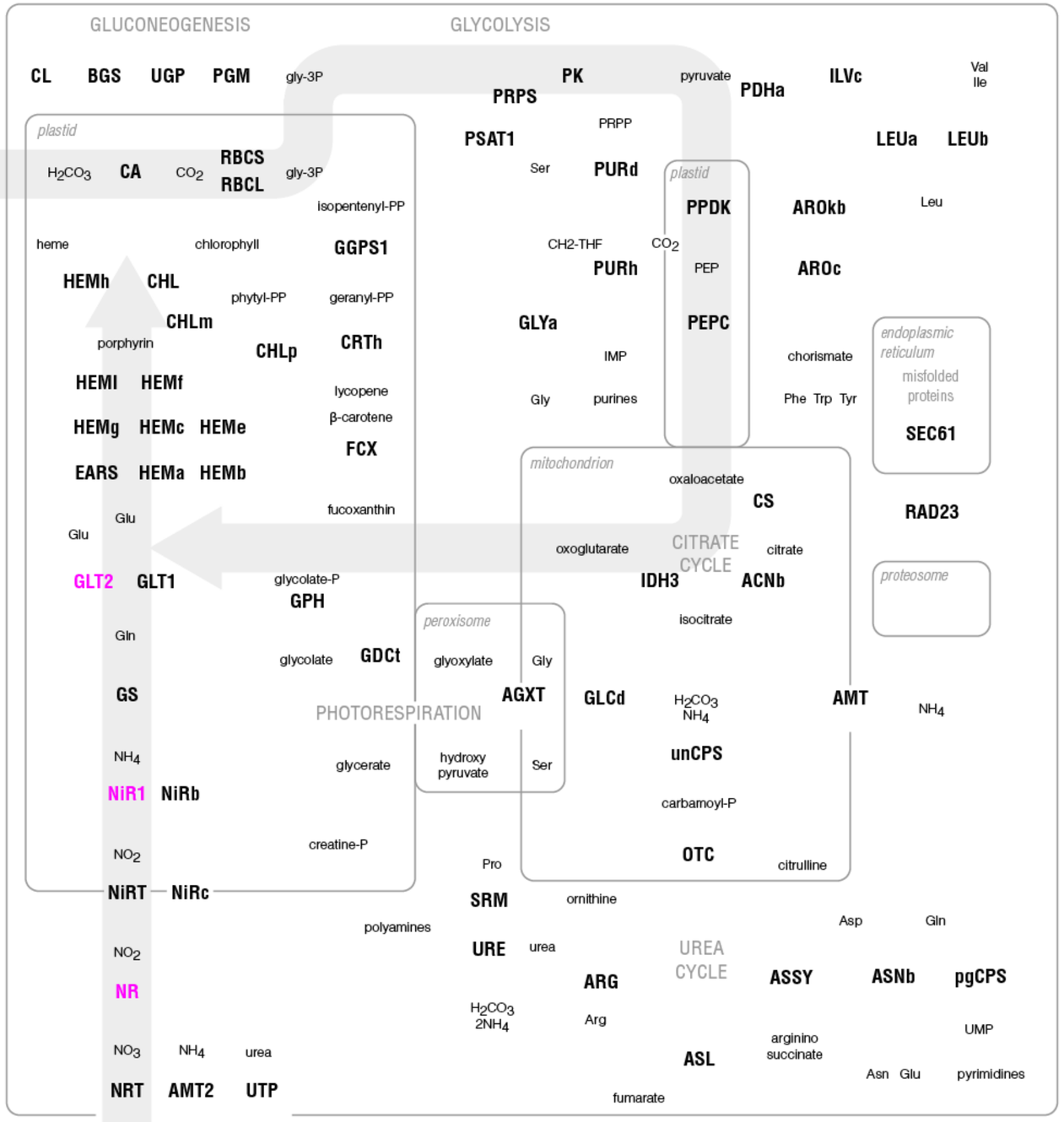
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CO_2
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RBCL
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PPDK
AROkb
Leu
isopentenyl-PP
heme
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GDCt
glyoxylate
Gly
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NiRb
carbamoyl-P
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NO_2
OTC
citrulline
Pro
NiRT
NiRc
SRM
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NO_2
URE
urea
UREA
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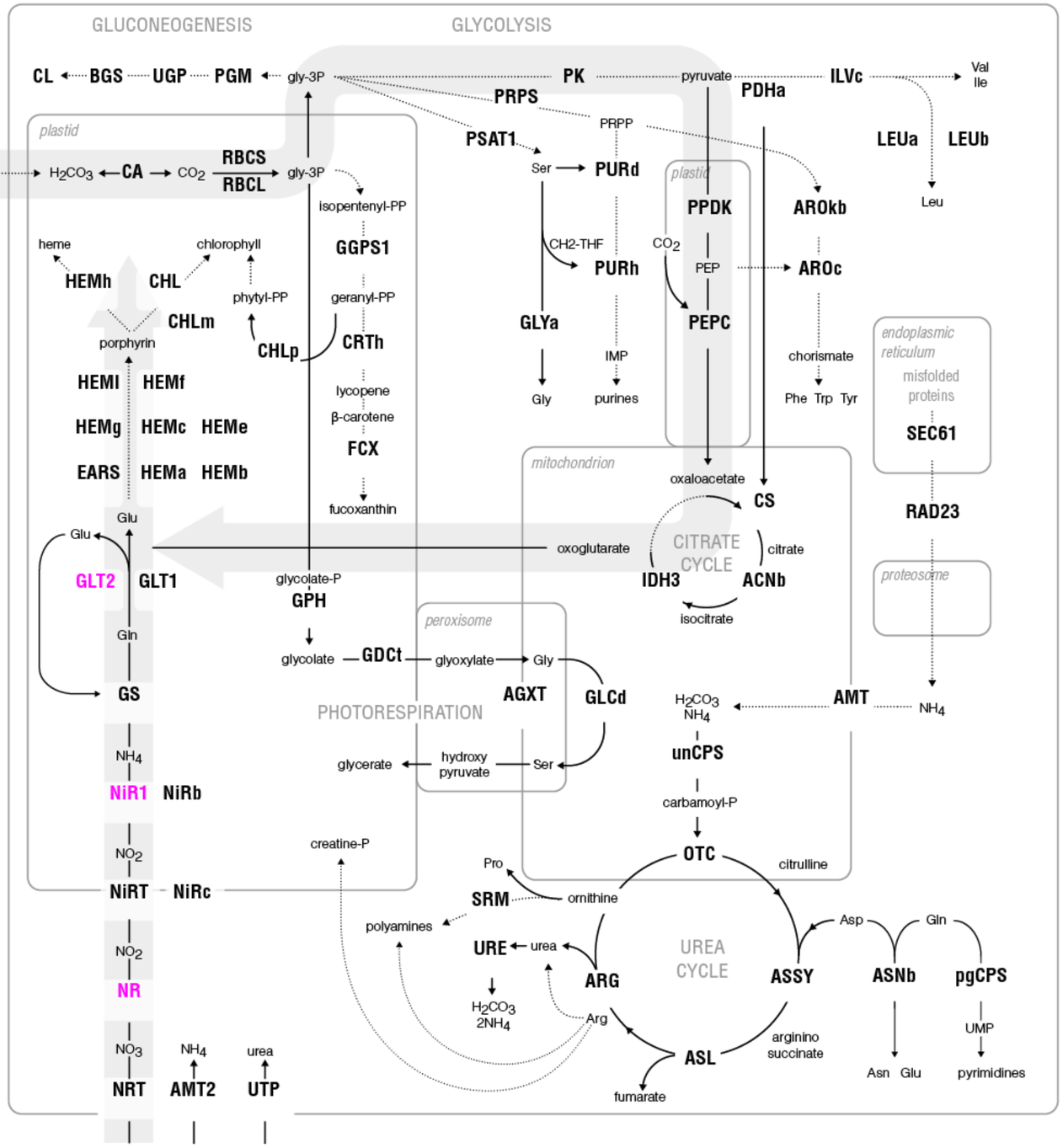
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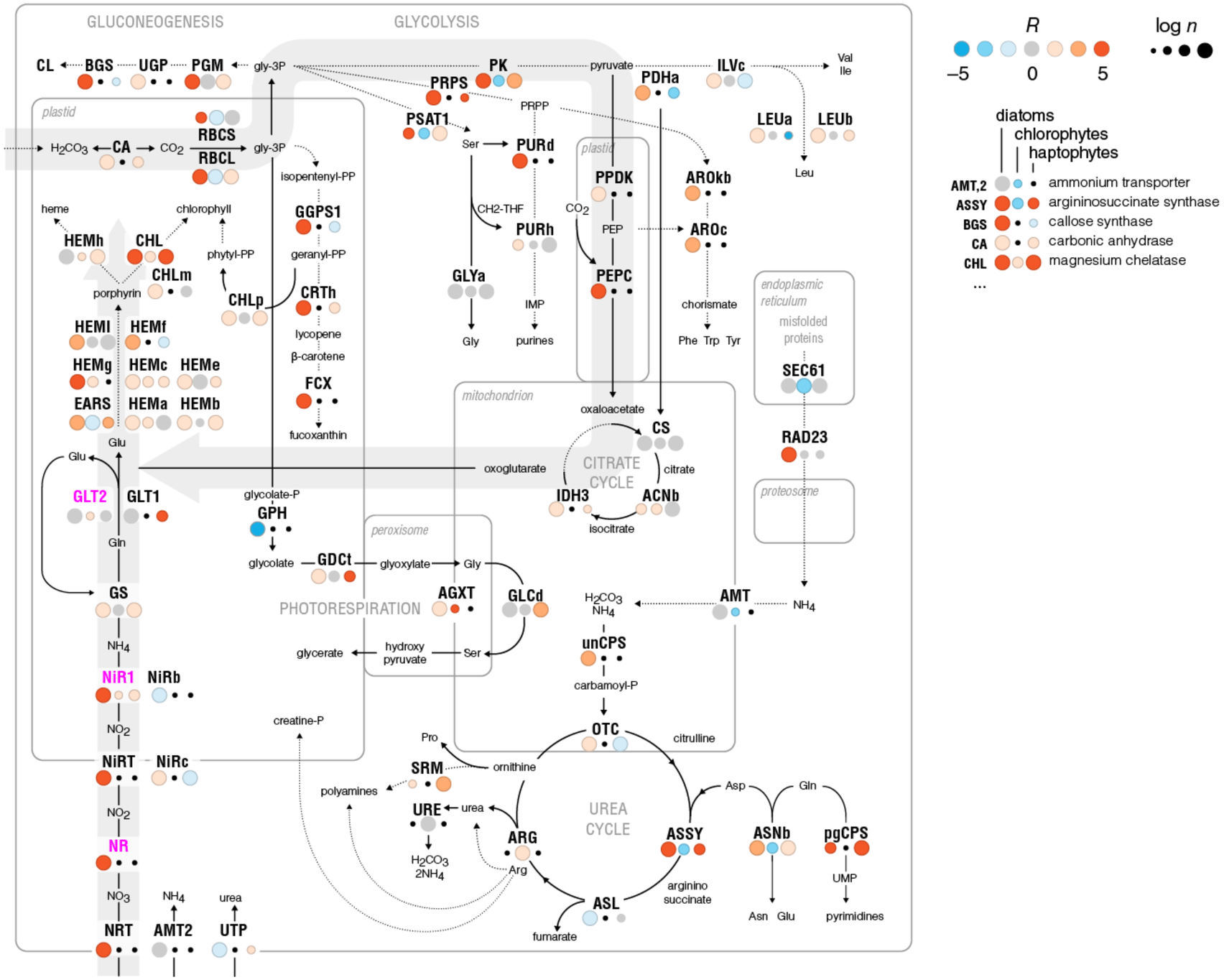
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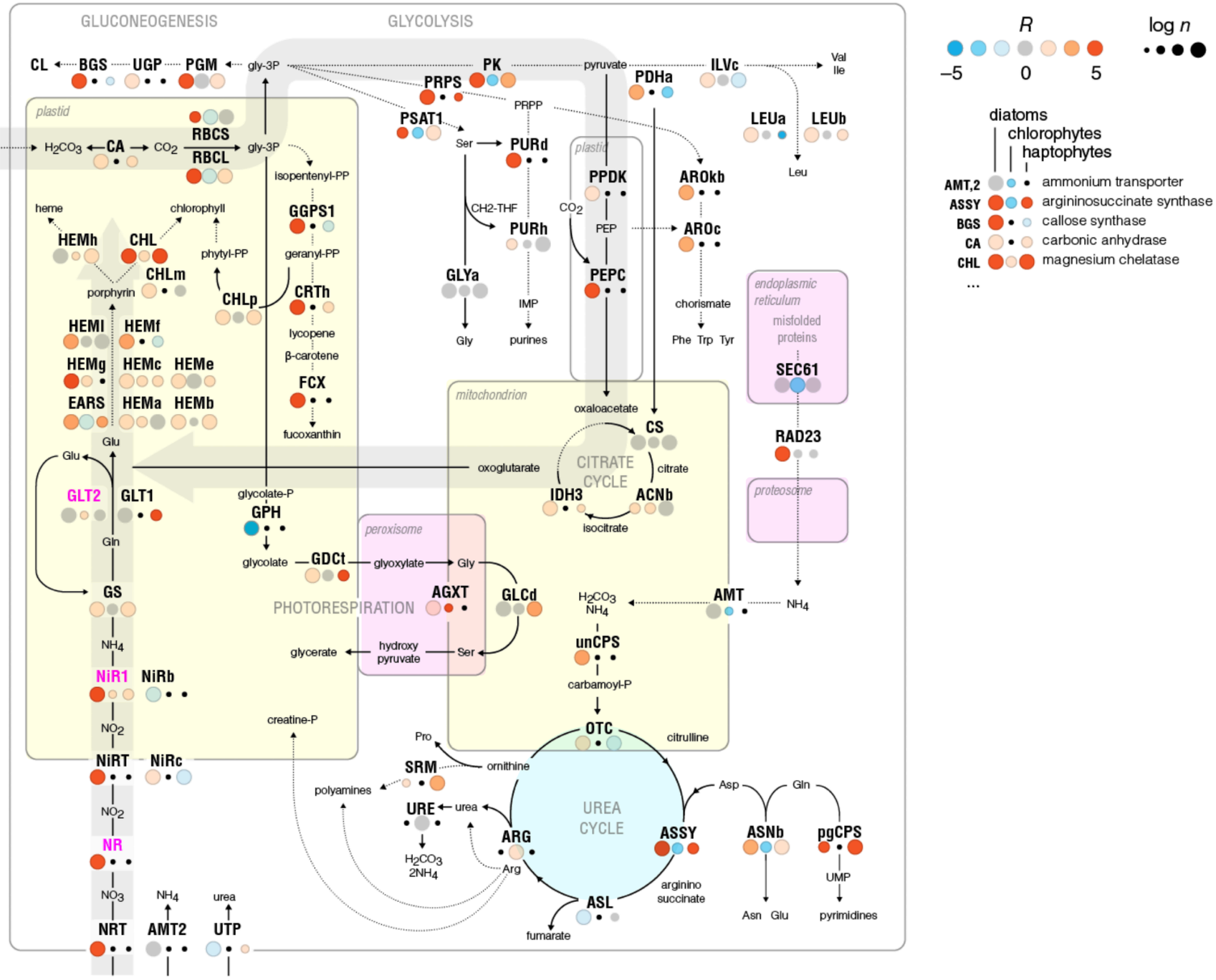
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citrate
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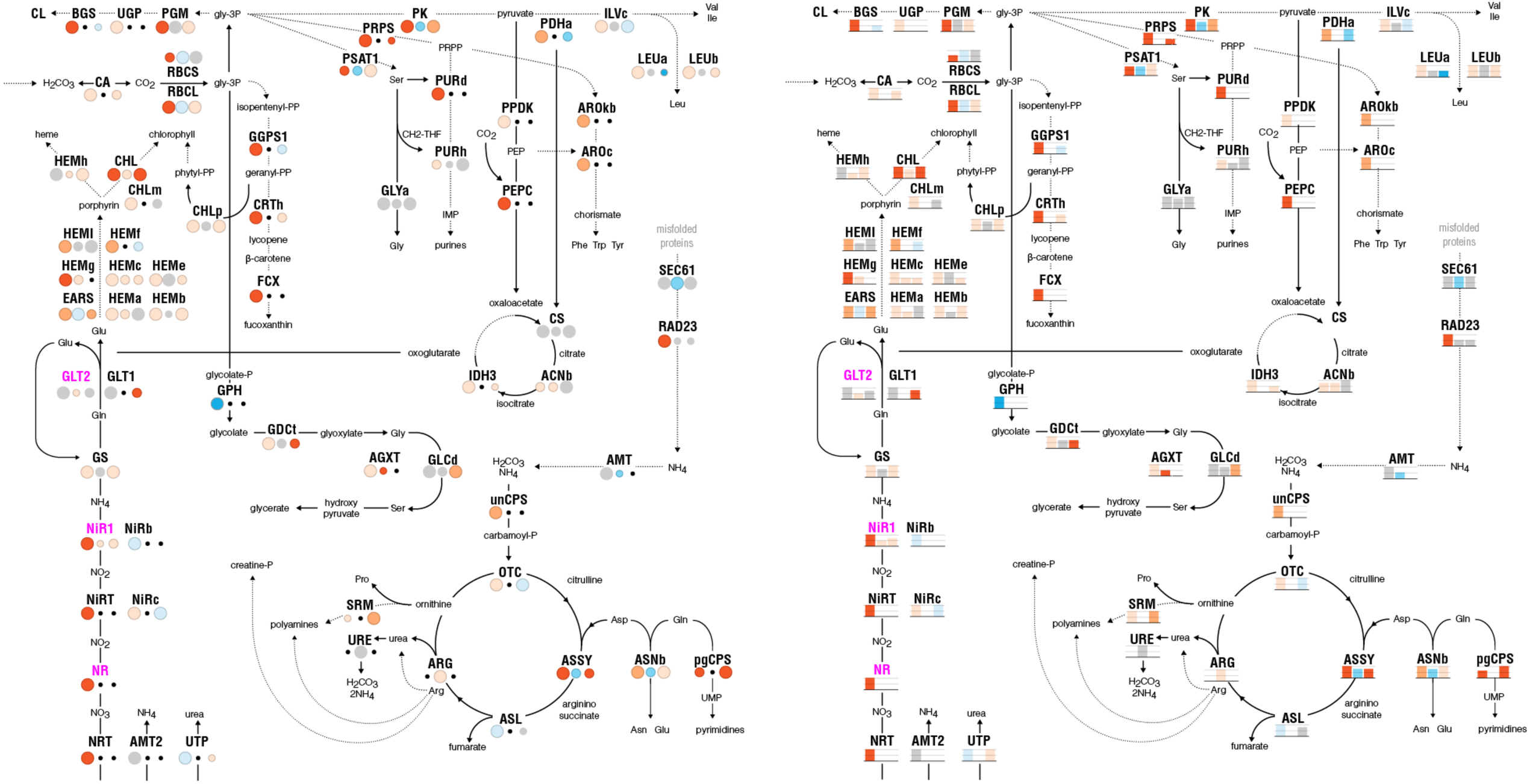
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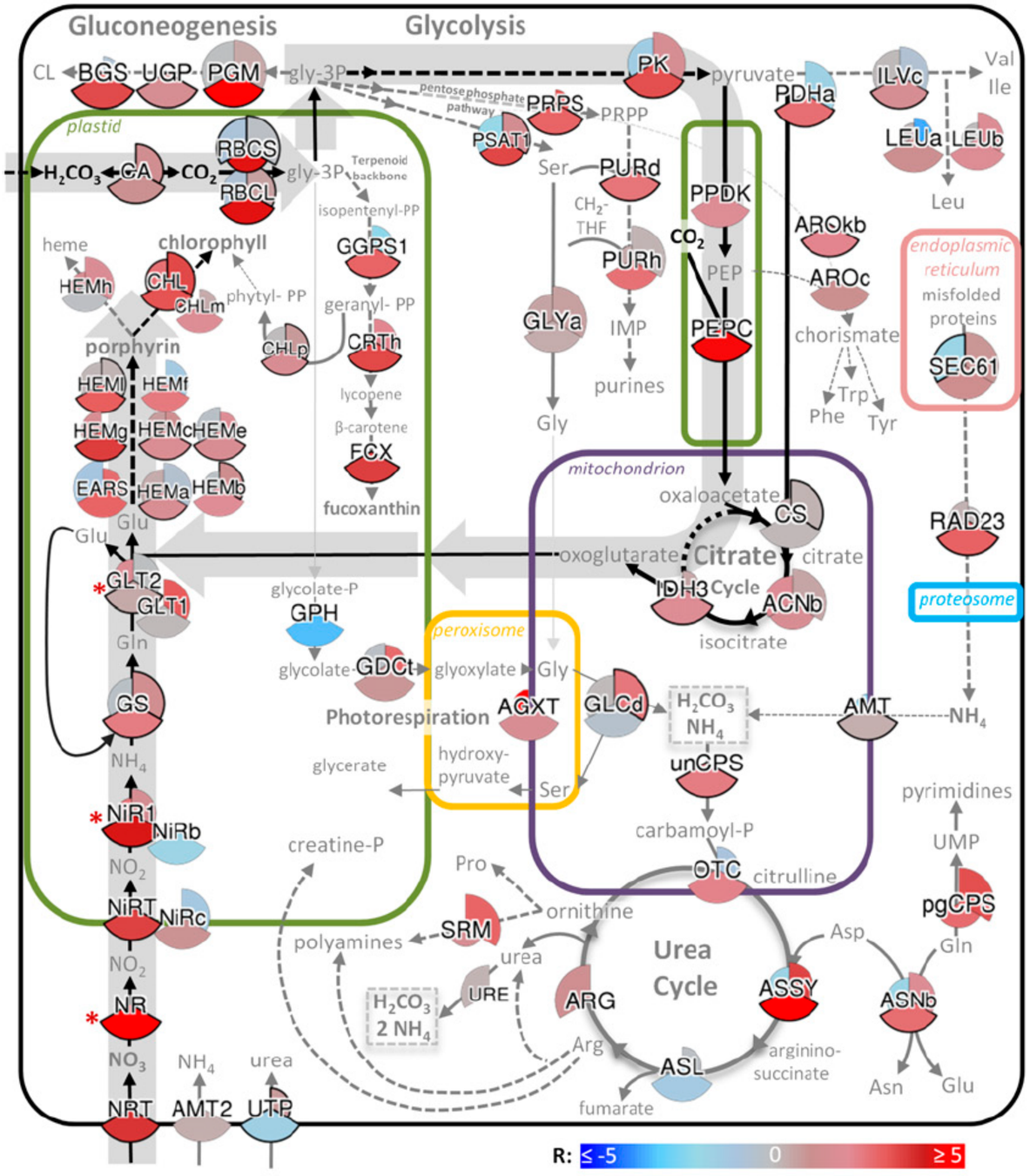
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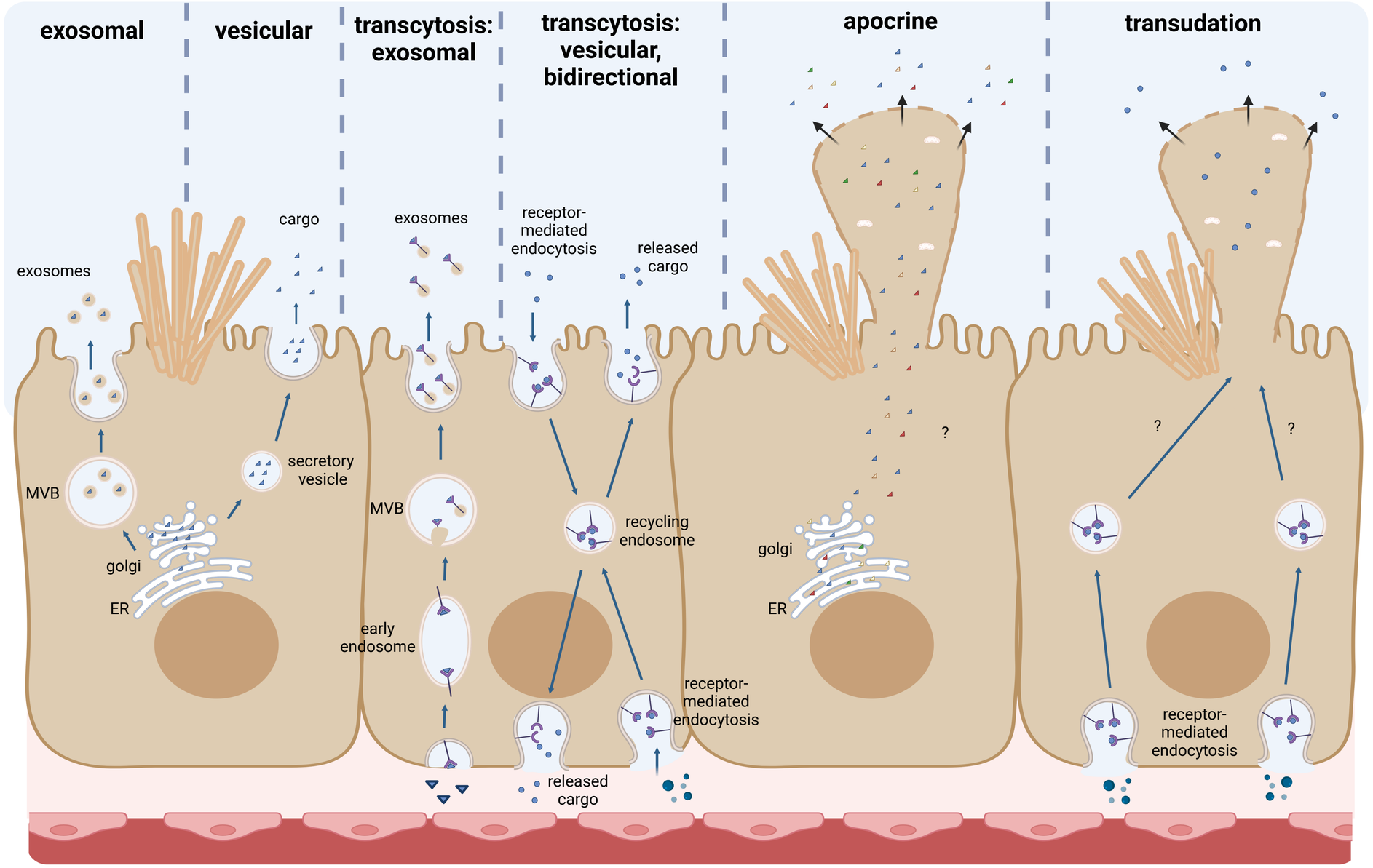
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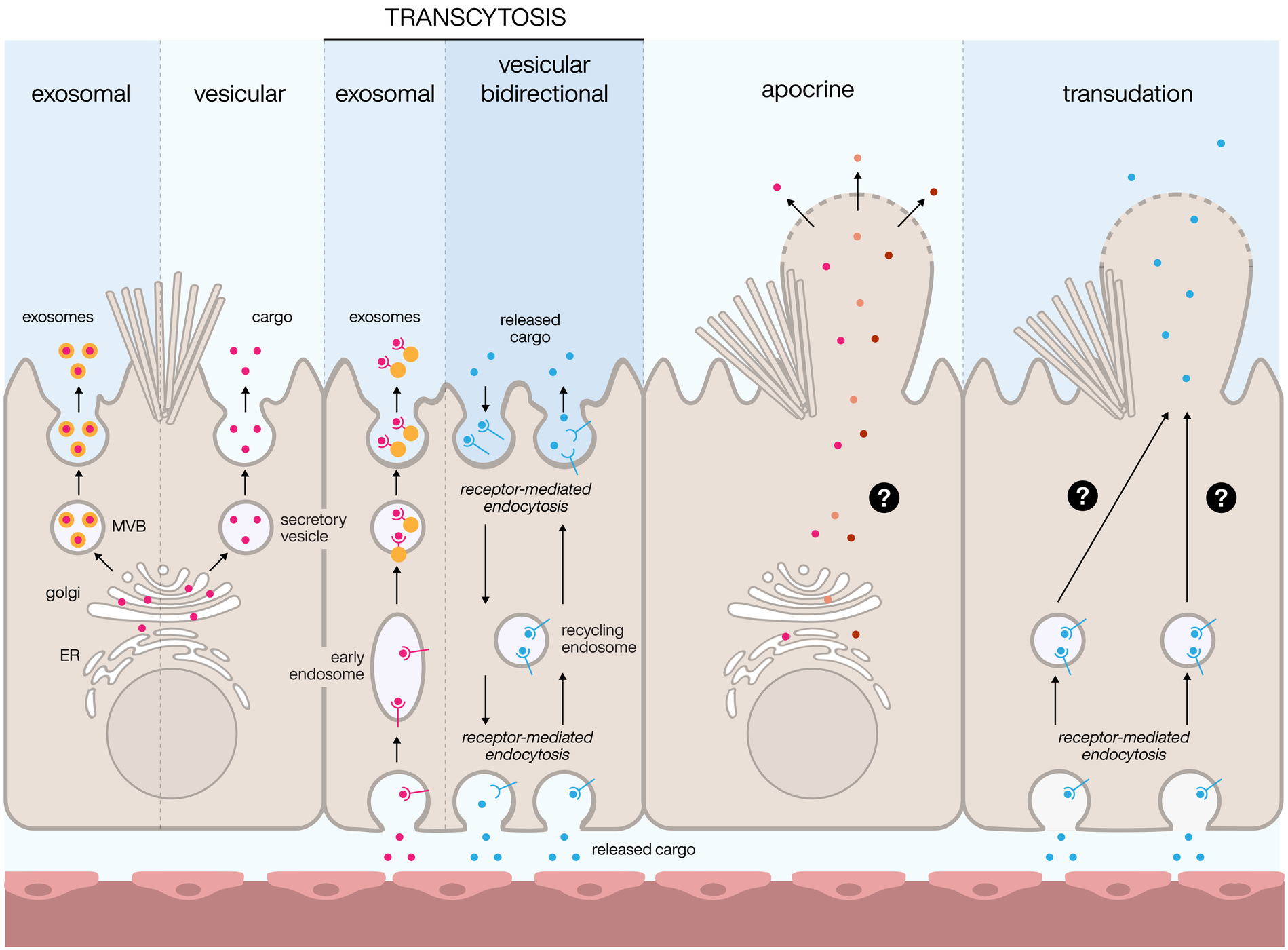
receptor-mediated endocytosis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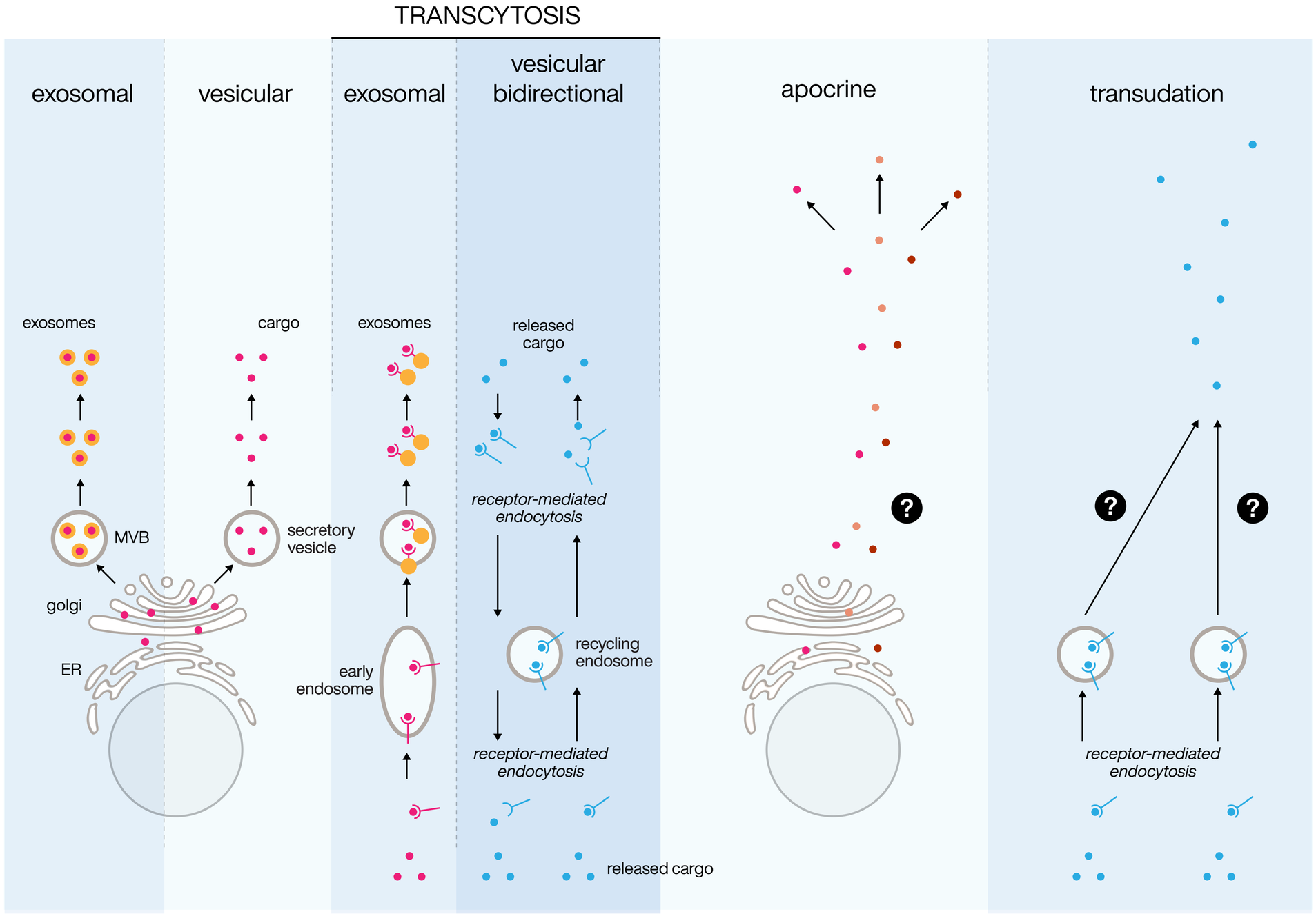
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receptor-mediated
endocytosis

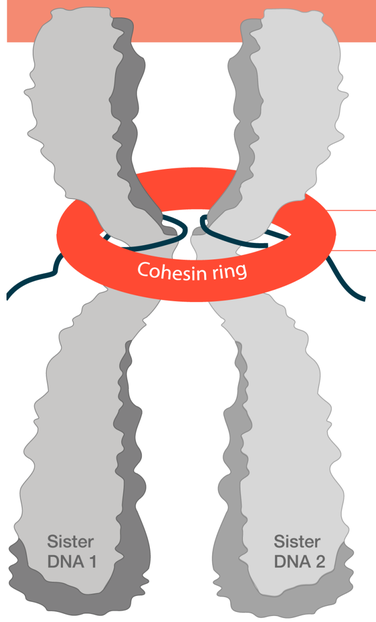
putting it together — posters

you can’t explain everything all at once

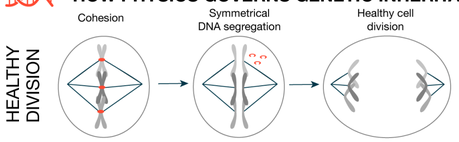
One ring to rule them all: how cohesin holds together X-shaped chromosomes

Martina Richeldi[1,2,3], Frank Uhlmann[2], Maxim Molodtsov[1,3]

[1]Biophysics and Mechanobiology and [2]Chromosome Segregation Laboratories at The Francis Crick Institute | London, UK
[3]Department of Astrophysics, University College London | London, UK

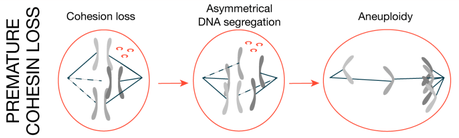


HOW PHYSICS GOVERNS GENETIC INHERITANCE



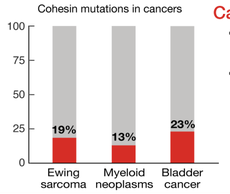
When cells divide, the DNA needs to be duplicated and segregated symmetrically.

The ring-shaped protein **Cohesin** is responsibe for keeping chromosomes together.



If symmetrical segregation fails, genetic diseases and cancers can arise.

COHESIN IN CANCER AND DISEASE



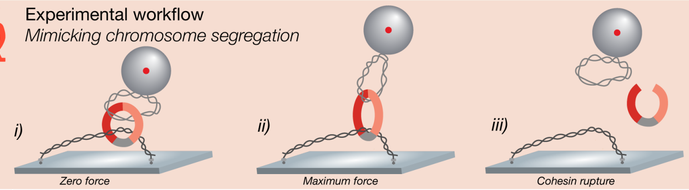
Cancer:
- mutated in 30% of pan-cancer cases
- found in 32 different cancer types

Cohesinopathies:
- 1 in 10,000 live births
- severe developmental and physical abnormalities

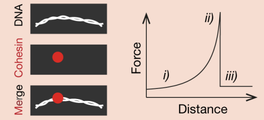
Understanding how cohesin works will help treat genetic diseases

HOW DOES COHESIN KEEP TOGETHER CHROMOSOMES BALANCING EXTERNAL FORCES?

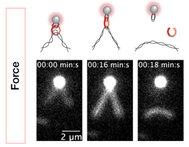
Experimental workflow
Mimicking chromosome segregation

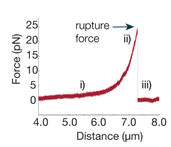


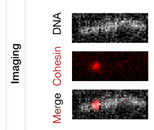
Output
Merging imaging and biophysics

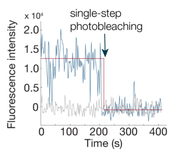


COHESIN RESISTS FORCES UP TO 20 PN, WHEN IT DISENGAGES AT ITS WEAKEST INTERFACE

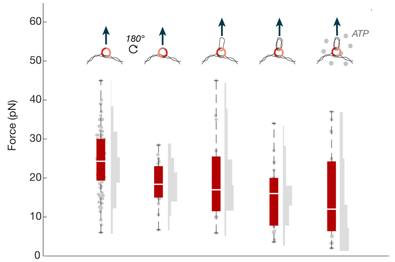








- Maximum force is ~20 pN: cohesin has detached from DNA
- Significant force! *e.g.* protein kinesin generates ~5 pN force
- Real-time imaging: A single-step drop in fluorescence represents one labelled molecule.
- Ensured that one cohesin only was being tested at any time.



Cohesin always broke at the least stable interface: a ring that always breaks at its weakest point.

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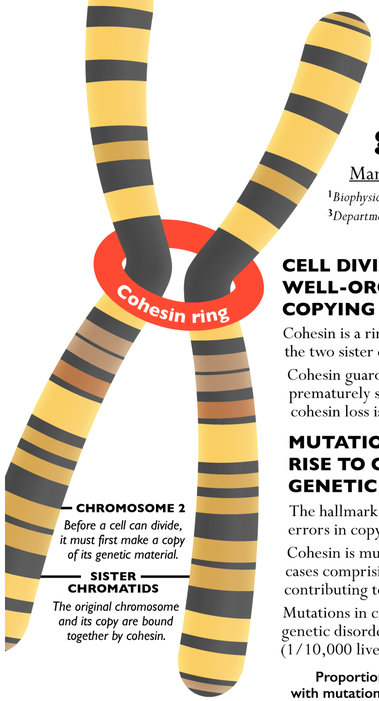
The Francis Crick Institute
Boehringer Ingelheim Fonds Stiftung für medizinische Grundlagenforschung

Cohesin's weakest interface is mutated in disease: knowing how this interface is important allows us to target the dysfunctional mutations



ONE RING TO RULE THEM ALL
Cohesin: the tiny and strong guardian of our genetic inheritance

Martina Richeldi[1,2,3], Frank Uhlmann[2], Maxim Molodtsov[1,3]

[1]Biophysics and Mechanobiology and [2]Chromosome Segregation Laboratories at The Francis Crick Institute, London, UK
[3]Department of Astrophysics, University College London, London, UK martina.richeldi@crick.ac.uk @MartinaRicheld1

CELL DIVISION REQUIRES WELL-ORGANISED DNA COPYING AND SEGREGATION

Cohesin is a ring-shaped protein that keeps the two sister chromatids together.

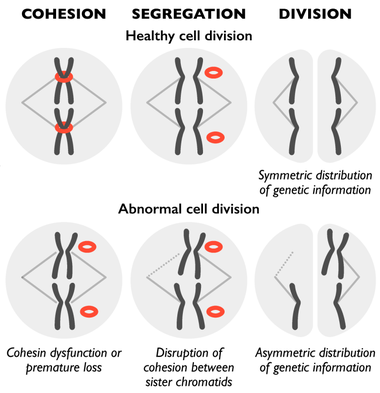
Cohesin guards against these becoming prematurely separated — the consequence of cohesin loss is an imbalance of genetic material.

MUTATIONS IN COHESIN GIVE RISE TO CANCER AND OTHER GENETIC DISEASES

The hallmark of cancer is genomic instability: errors in copying and distributing DNA.

Cohesin is mutated in 30% of all cancer cases comprising 32 different cancer types, contributing to genomic instability.

Mutations in cohesin also directly cause severe genetic disorders called cohesinopathies (1/10,000 live births).



SEEING AND MOVING TINY THINGS — TESTING THE LIMITS AND WEAKNESSES OF COHESIN

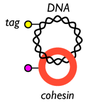
How do we move molecules?

To understand the role of cohesin in cell division and chromosome segregation we use a biophysical technique called *optical tweezers*.

Through a glass bead, which we can attach to specific molecules, the laser of these tiny tweezers can hold and apply forces to DNAs that are encircled by cohesin. Like this,where the bead goes, the molecule goes with it.

How do we see molecules?

Fluorescent tags can be attached to molecules, like DNA and cohesin. When illuminated by a laser, these tagged molecules light up, allowing us to monitor their movement and behaviour.



The intersection of imaging & biophysics

For the first time, combining visualisation and optical tweezers, we see and move single molecules of cohesin. This allows us to measure cohesin's strength and identify its weaknesses.

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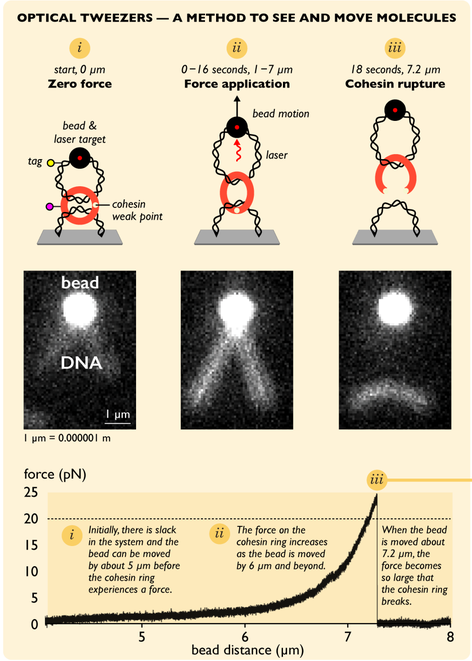
martina.richeldi@crick.ac.uk

OPTICAL TWEEZERS — A METHOD TO SEE AND MOVE MOLECULES



Targeting the weak link

The cohesin ring always broke when the force applied to it reached ~20 pN. We observed this regardless of the direction of pulling and what kind of DNA was used.

Importantly, the cohesin ring broke always at the same position in its structure: this weak point is precisely the part of the protein that is mutated in disease.

Watch the cohesin ring break in real time.

THE WAY FORWARD

Understanding how mutations affect the biophysics of cohesin will eventually help us treat cancer and cohesinopathies.

Cohesin is incredibly strong! It can resist a pulling force of ~20 piconewtons (20 trillionth of a newton). This is 4 times as much as generated by kinesin, a protein that moves molecules around.

A Newton is a measure of force. The Earth pulls on a 100 gram mass with a force of about 1 Newton.

Boehringer Ingelheim Fonds
Stiftung für medizinische Grundlagenforschung

One ring to rule them all: how cohesin holds together X-shaped chromosomes

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HOW PHYSICS GOVERNS GENETIC INHERITANCE

Cohesion

Symmetrical DNA segregation

Healthy cell division

When cells divide, the DNA needs to be duplicated and segregated symmetrically.

The ring-shaped protein **Cohesin** is responsibe for keeping chromosomes together.

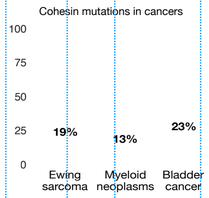
Cohesion loss

Asymmetrical DNA segregation

Aneuploidy

If symmetrical segregation fails, genetic diseases and cancers can arise.

COHESIN IN CANCER AND DISEASE



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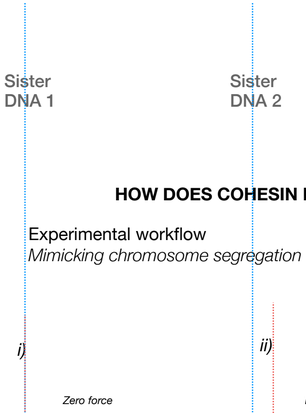

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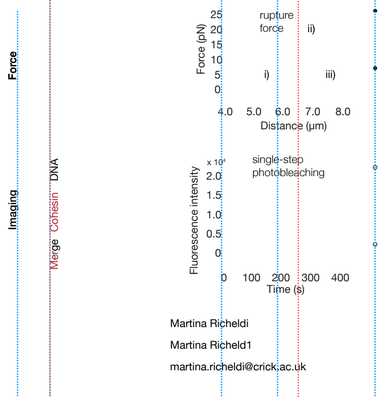

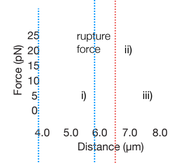

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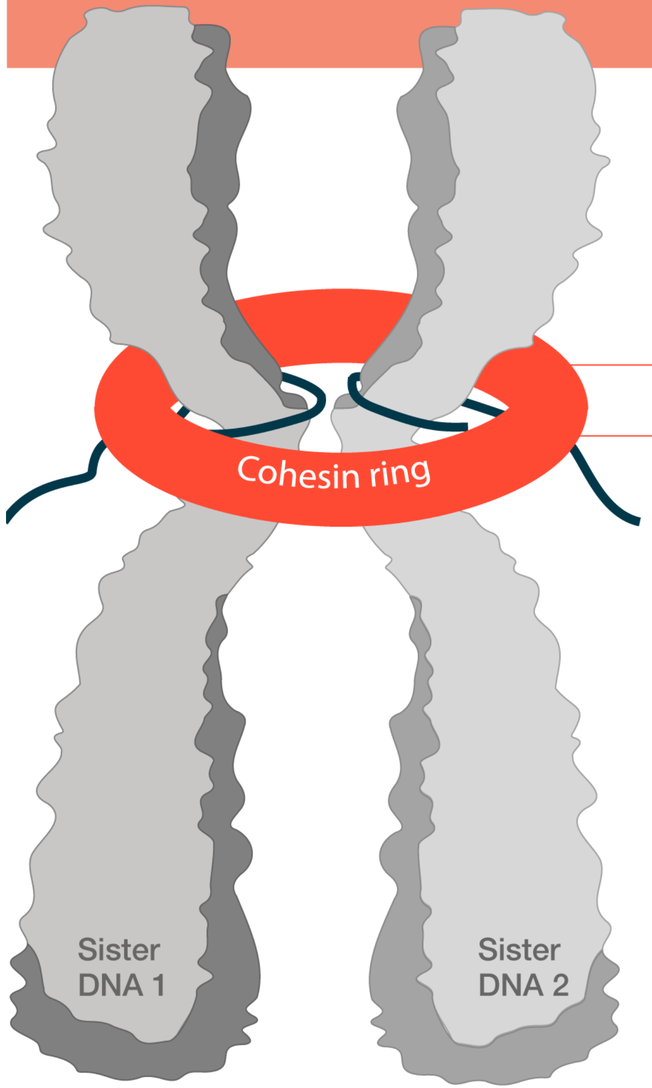
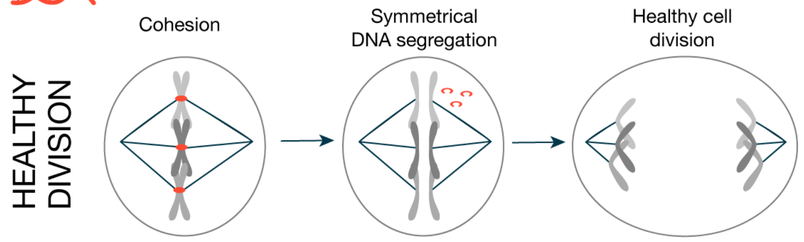


HOW PHYSICS GOVERNS GENETIC INHERITANCE



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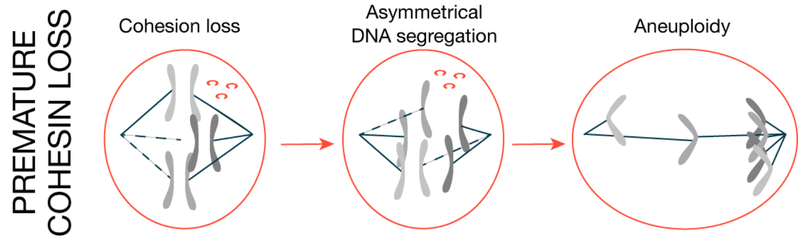
The ring-shaped protein **Cohesin** is responsibe for keeping chromosomes together.



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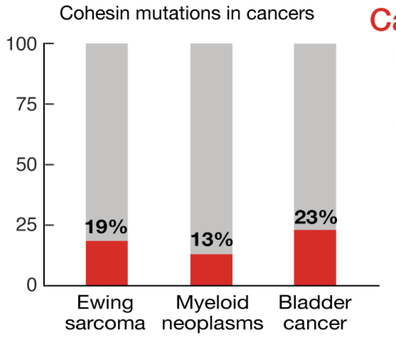
COHESIN IN CANCER AND DISEASE



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Cohesinopathies:
- 1 in 10,000 live births
- severe developmental and physical abnormalities

Understanding how cohesin works will help treat genetic diseases

One ring to rule them all: how cohesin holds together X-shaped chromosomes

Martina Richeldi[1,2,3], Frank Uhlmann[2], Maxim Molodtsov[1,3]

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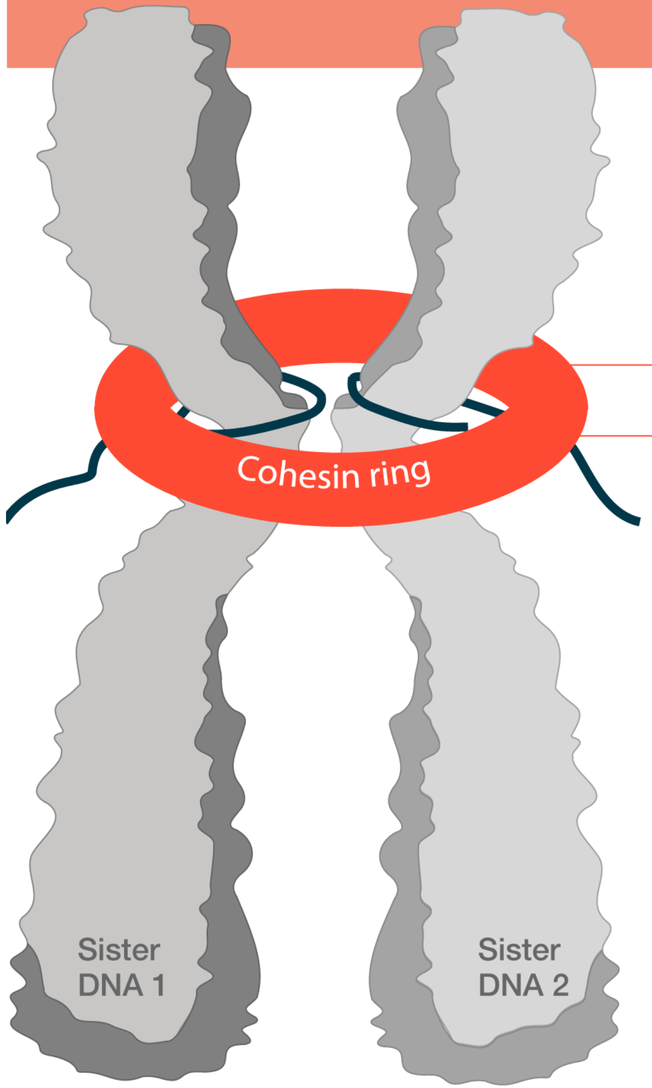

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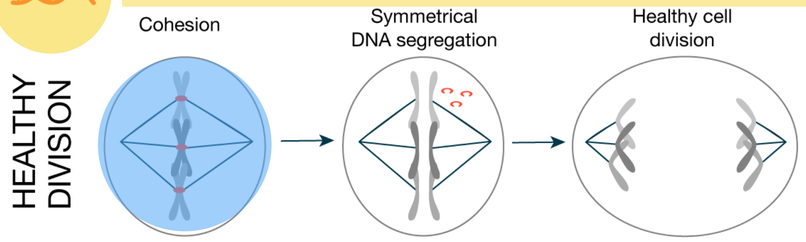

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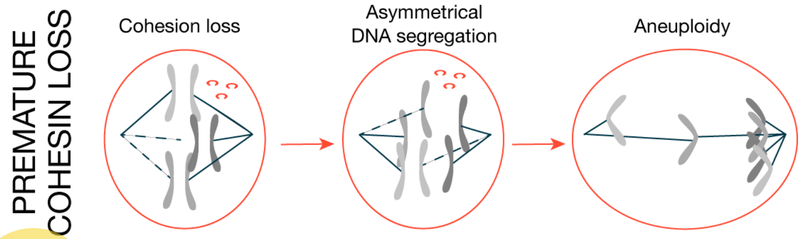

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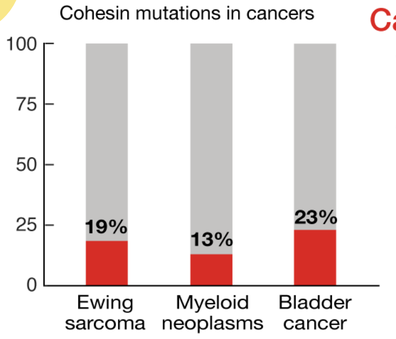

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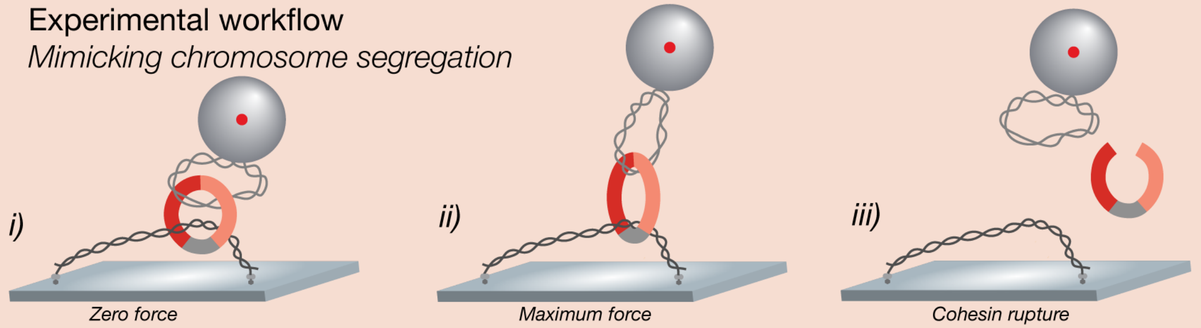
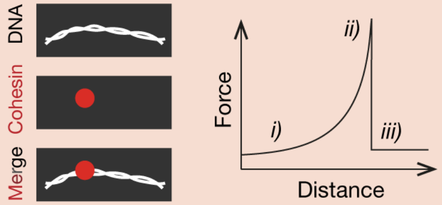
Experimental workflow
Mimicking chromosome segregation



Output
Merging imaging and biophysics



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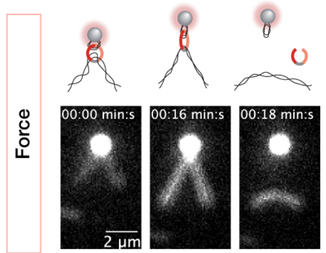

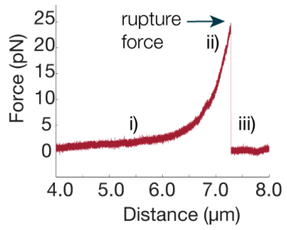

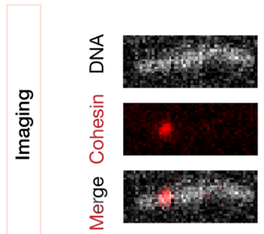

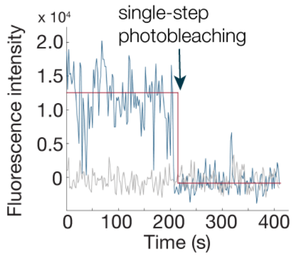

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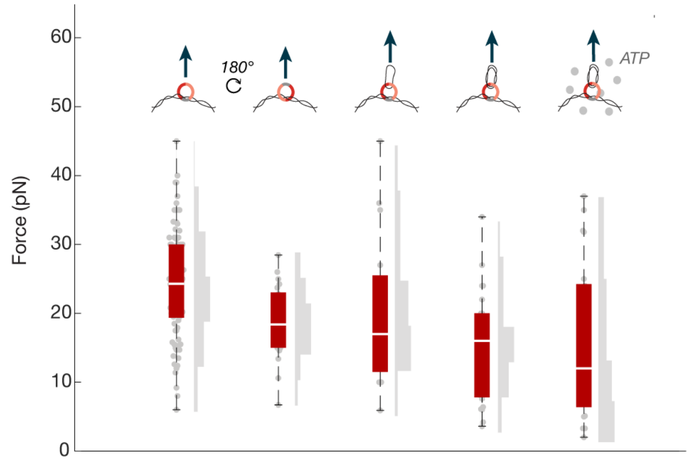

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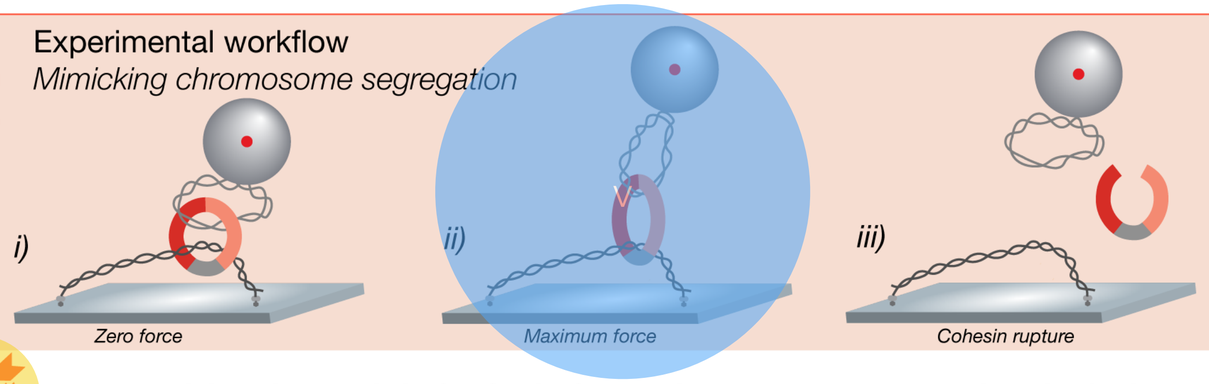

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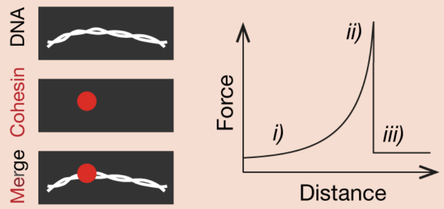

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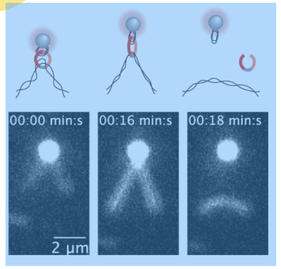
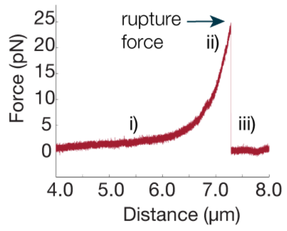
Force





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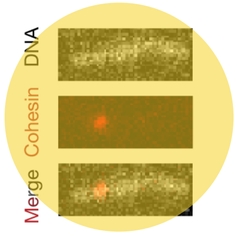

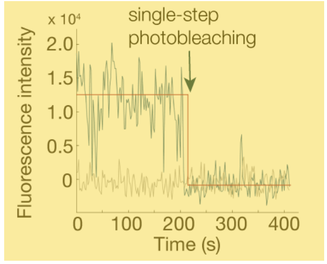

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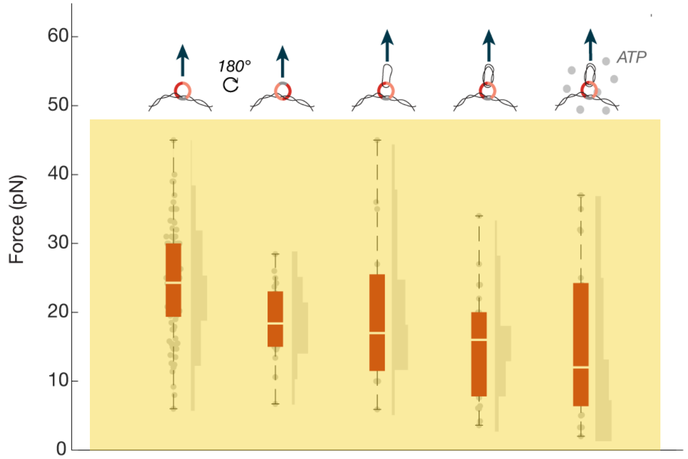

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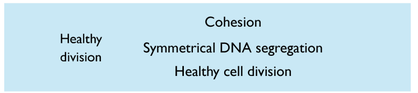

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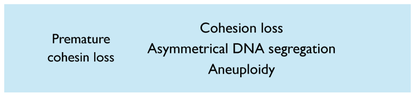

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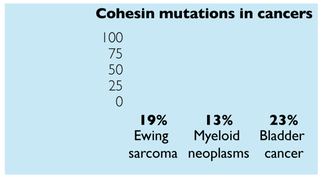

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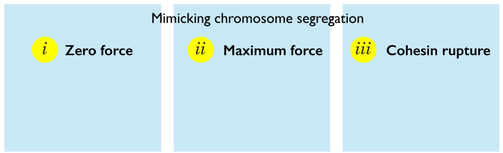

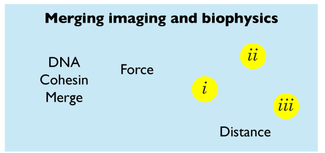

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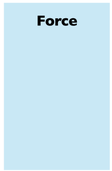

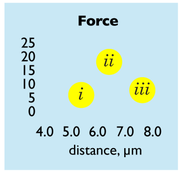

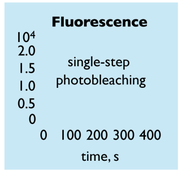

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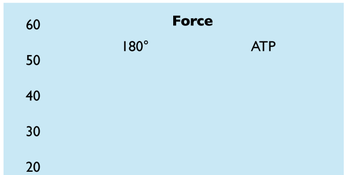

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Premature cohesin loss	Cohesion loss Asymmetrical DNA segregation Aneuploidy

Sister DNA 1

Sister DNA 2

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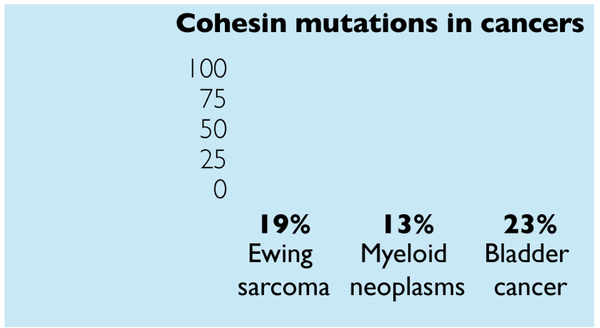

HOW DOES COHESIN KEEP TOGETHER CHROMOSOMES BALANCING EXTERNAL FORCES?

Mimicking chromosome segregation

i Zero force

ii Maximum force

iii Cohesin rupture

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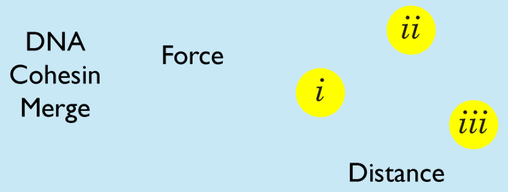

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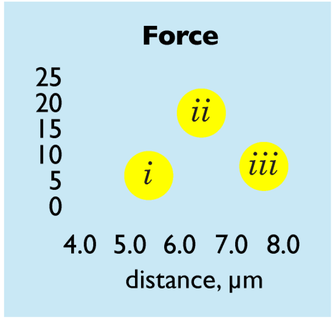
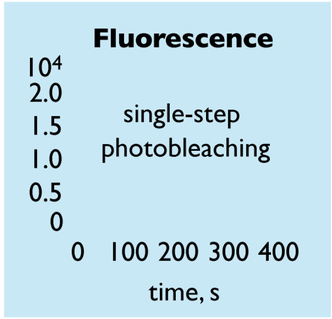


Force

DNA
Cohesin
Merge



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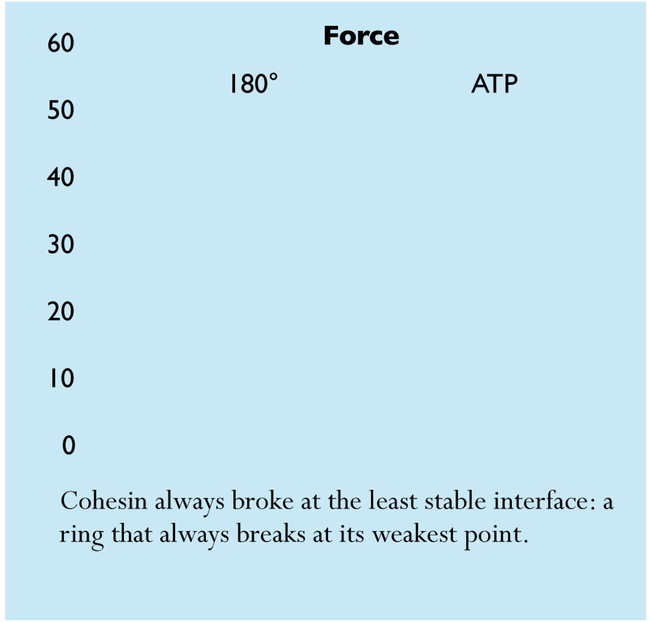

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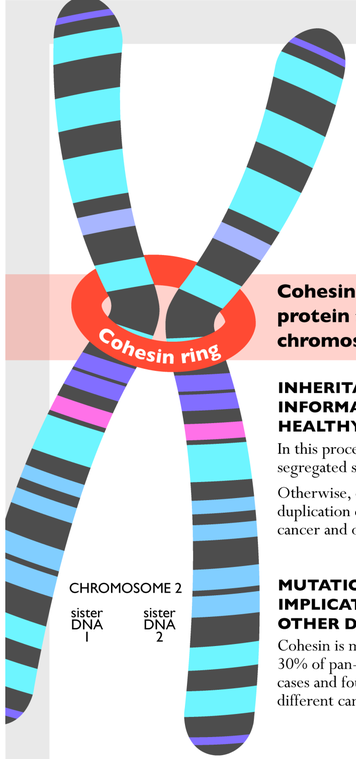

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In this process, DNA must be segregated symmetrically.

Otherwise, errors in DNA duplication can occurr, leading to cancer and other genetic diseases.

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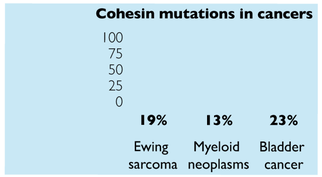

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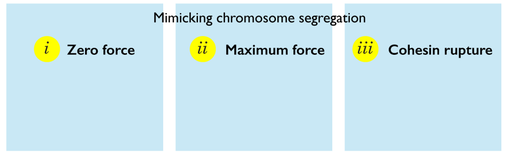

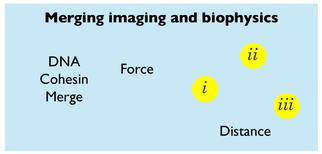

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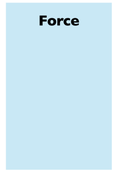

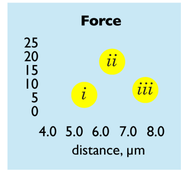

Maximum force is ~20 pN: cohesin has detached from DNA. Significant force!
e.g. protein kinesin generates ~5 pN force

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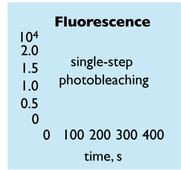
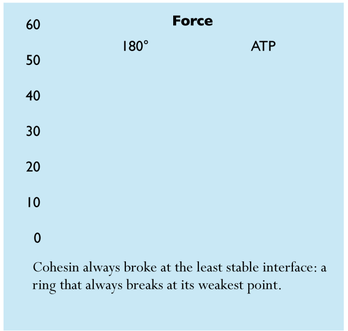
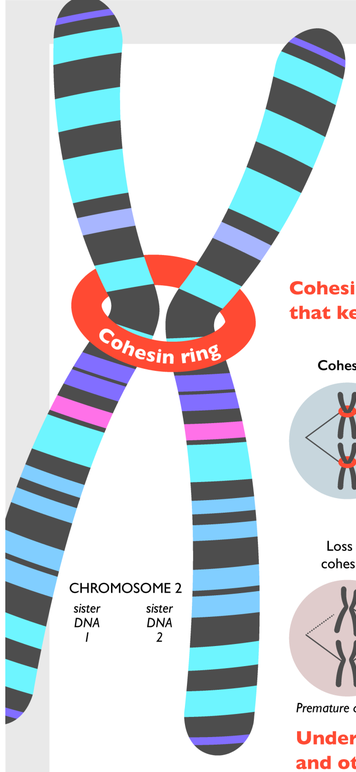




Cohesin always broke at the least stable interface: a ring that always breaks at its weakest point.

COHESIN'S WEAKEST INTERFACE IS MUTATED IN DISEASE: KNOWING HOW THIS INTERFACE IS IMPORTANT ALLOWS US TO TARGET THE DYSFUNCTIONAL MUTATIONS

0
Mbp
93.9
Mbp
Mbp
400 bphs
550 bphs
850 bphs
Human chromosome 2



ONE RING TO RULE THEM ALL
What holds a chromosome together and why it matters

Martina Richeldi[1,2,3], Frank Uhlmann[2], Maxim Molodtsov[1,3]

[1]Biophysics and Mechanobiology and [2]Chromosome Segregation Laboratories at The Francis Crick Institute, London, UK
[3]Department of Astrophysics, University College London, London, UK

martina.richeldi@crick.ac.uk @MartinaRicheld1

Cohesin is a ring-shaped protein that keeps a chromosome together.

HEALTHY DIVISION

Cohesion | Segregation | Cell division

ABNORMAL DIVISION

Loss of cohesion | Asymmetrical segregation | Chromosome imbalance

Premature cohesin loss

INHERITANCE OF GENETIC INFORMATION RELIES ON HEALTHY CELL DIVISION

In this process, DNA must be segregated symmetrically. Otherwise, errors in DNA duplication can occurr, leading to cancer and other genetic diseases.

MUTATIONS IN COHESIN ARE IMPLICATED IN CANCER AND OTHER DISEASES

Cohesin is mutated in 30% of pan-cancer cases and found in 32 different cancer types. Mutations in cohesin account for severe developmental and physical abnormalities in 1 out of 10,000 live births.

Proportion of cancers with mutations in cohesin

Ewing sarcoma **19%**
Myeloid neoplasms **13%**
Bladder cancer **23%**

Understanding how cohesin works will help treat cancer and other genetic diseases.

HOW DOES COHESIN KEEP TOGETHER CHROMOSOMES BALANCING EXTERNAL FORCES?

How do we see molecules?

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Imaging & Biophysics

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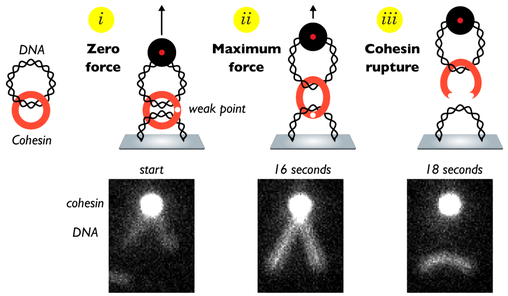


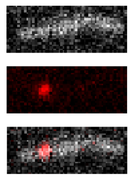
Targeting the weak link

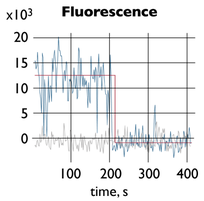
Cohesin always breaks at its weakest point and it is precisely this region in the protein that is mutated in disease.

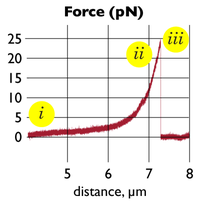
By understanding how mutations affect the biophysics of cohesin rupture will help pinpoint targeted treatment.

Imaging in real-time

Real-time imaging: A single-step drop in fluorescence represents one labelled molecule. Ensured that one cohesin only was being tested at any time.







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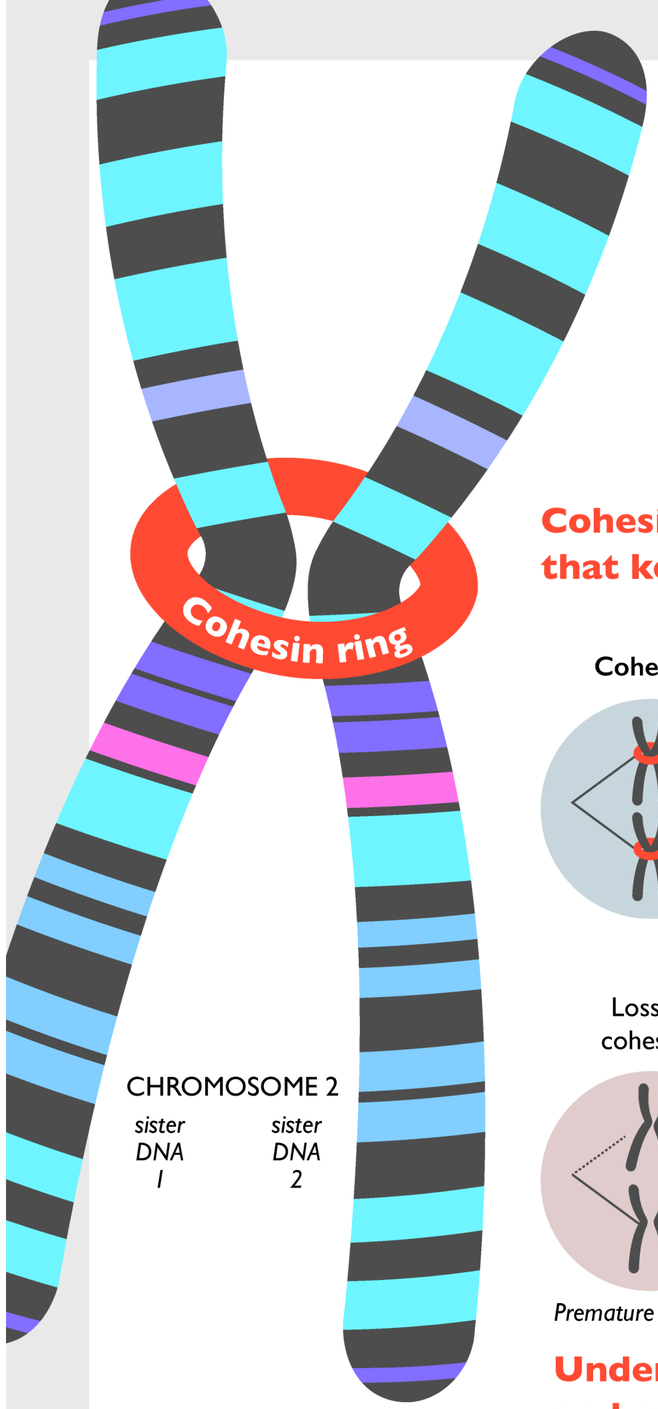

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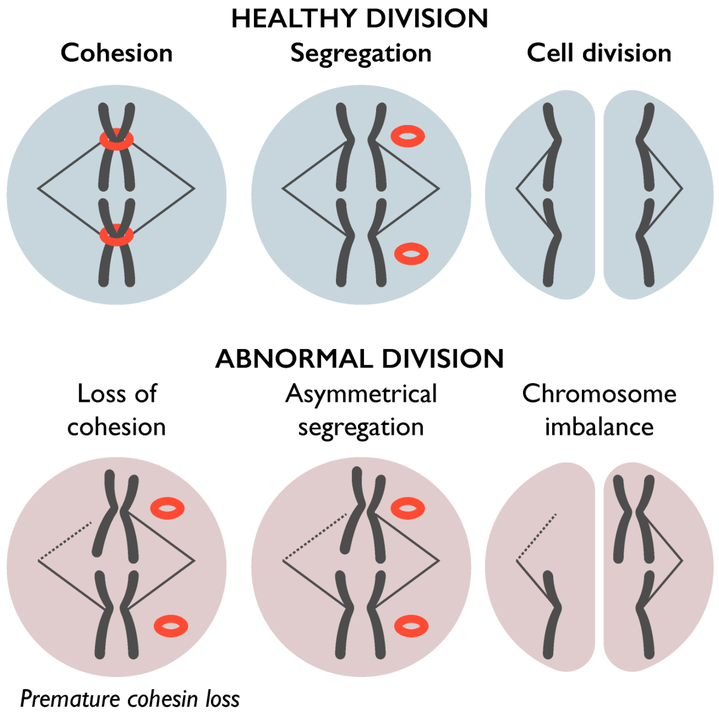

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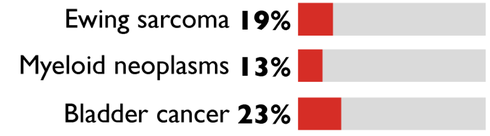

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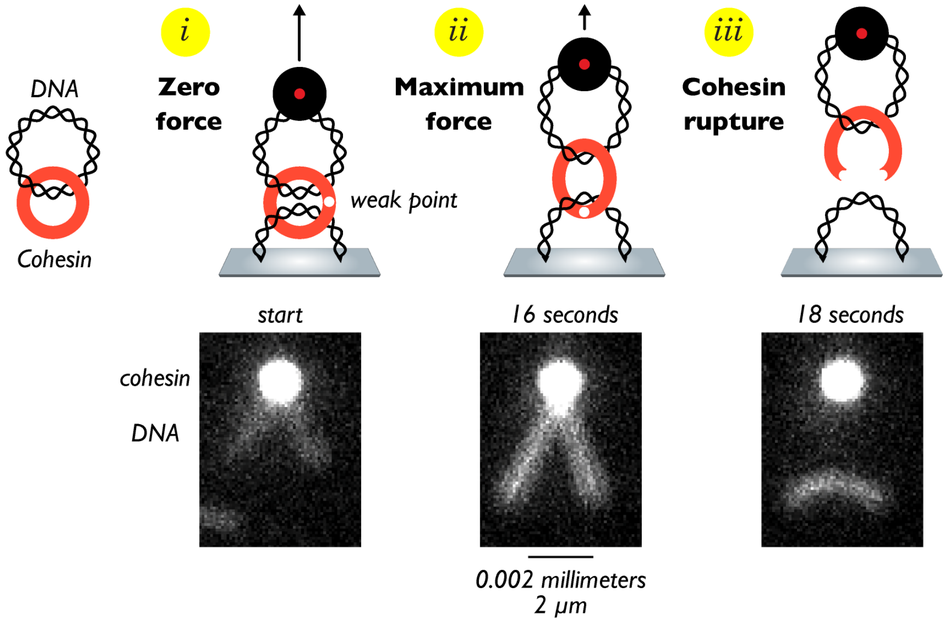

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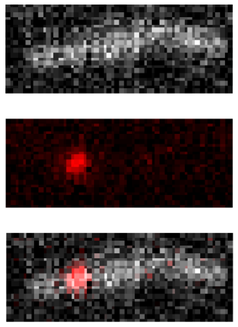

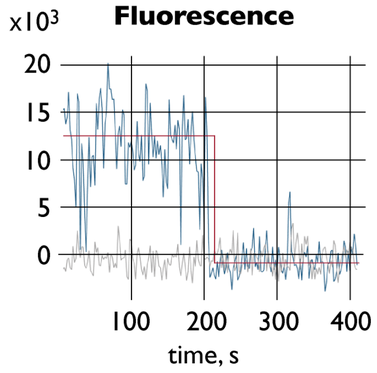

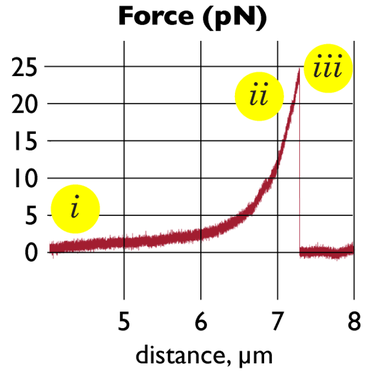

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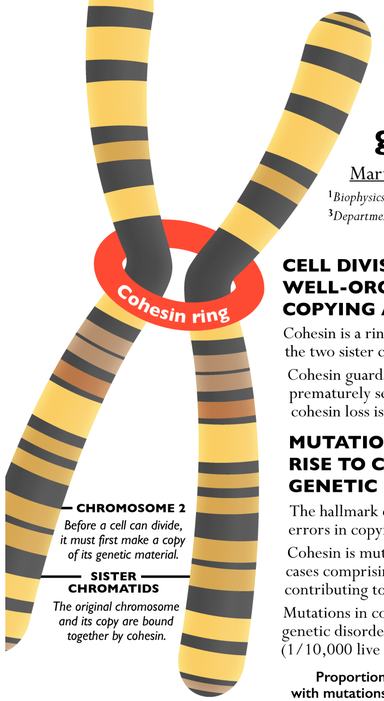


CELL DIVISION REQUIRES WELL-ORGANISED DNA COPYING AND SEGREGATION

Cohesin is a ring-shaped protein that keeps the two sister chromatids together.

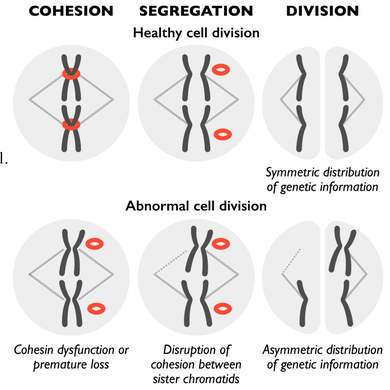
Cohesin guards against these becoming prematurely separated — the consequence of cohesin loss is an imbalance of genetic material.

MUTATIONS IN COHESIN GIVE RISE TO CANCER AND OTHER GENETIC DISEASES

The hallmark of cancer is genomic instability: errors in copying and distributing DNA.

Cohesin is mutated in 30% of all cancer cases comprising 32 different cancer types, contributing to genomic instability.

Mutations in cohesin also directly cause severe genetic disorders called cohesinopathies (1/10,000 live births).



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23% Bladder cancer **19%** Ewing sarcoma **13%** Myeloid neoplasms

SEEING AND MOVING TINY THINGS — TESTING THE LIMITS AND WEAKNESSES OF COHESIN

How do we move molecules?

To understand the role of cohesin in cell division and chromosome segregation we use a biophysical technique called *optical tweezers*.

Through a glass bead, which we can attach to specific molecules, the laser of these tiny tweezers can hold and apply forces to DNAs that are encircled by cohesin. Like this, where the bead goes, the molecule goes with it.

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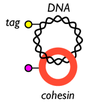
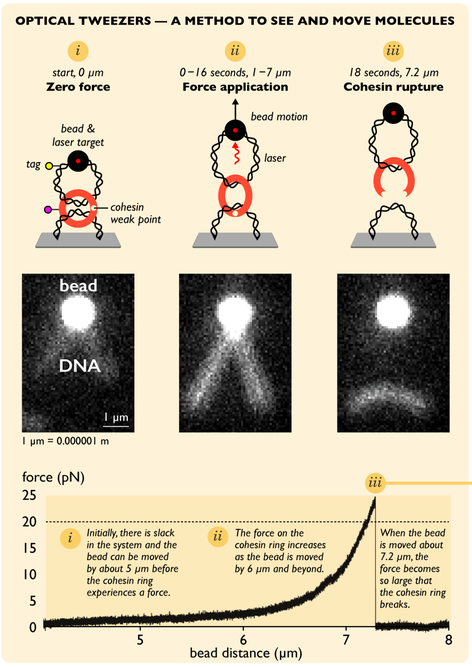
Fluorescent tags can be attached to molecules, like DNA and cohesin. When illuminated by a laser, these tagged molecules light up, allowing us to monitor their movement and behaviour.



The intersection of imaging & biophysics

For the first time, combining visualisation and optical tweezers, we see and move single molecules of cohesin. This allows us to measure cohesin's strength and identify its weaknesses.



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Importantly, the cohesin ring broke always at the same position in its structure: this weak point is precisely the part of the protein that is mutated in disease.

Watch the cohesin ring break in real time.

THE WAY FORWARD

Understanding how mutations affect the biophysics of cohesin will eventually help us treat cancer and cohesinopathies.

Cohesin is incredibly strong! It can resist a pulling force of ~20 piconewtons (20 trillionth of a newton). This is 4 times as much as generated by kinesin, a protein that moves molecules around.

A Newton is a measure of force. The Earth pulls on a 100 gram mass with a force of about 1 Newton.

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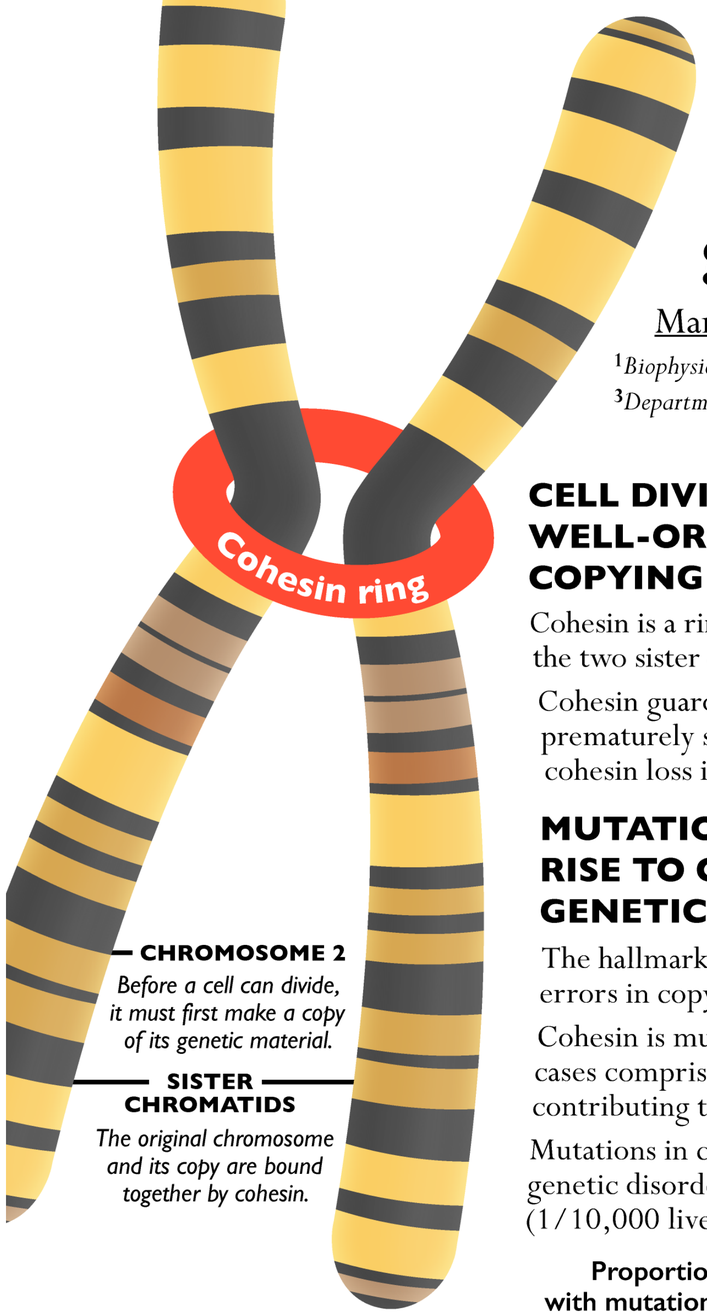

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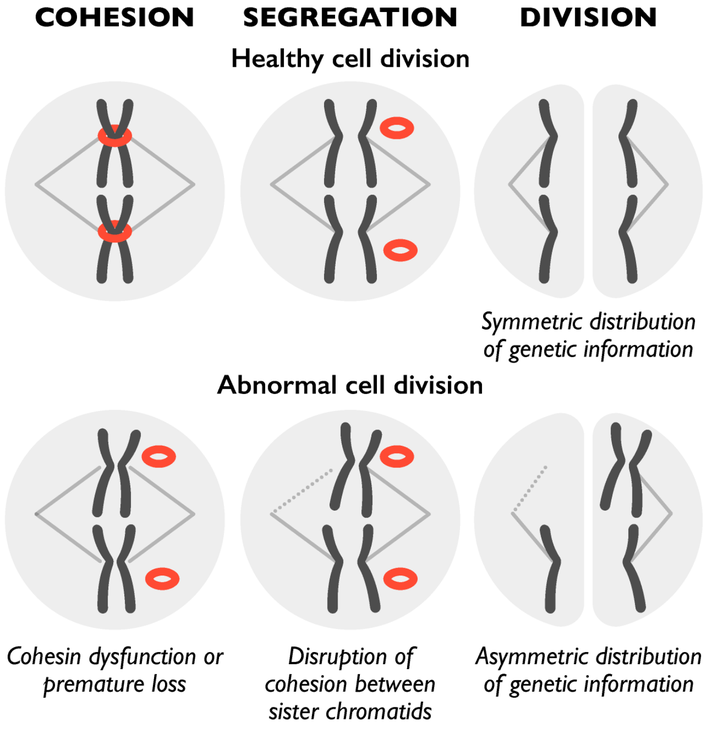

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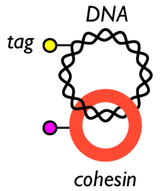

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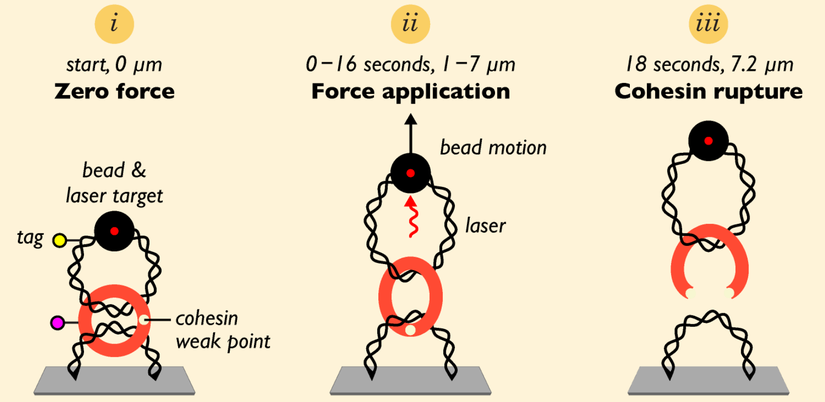
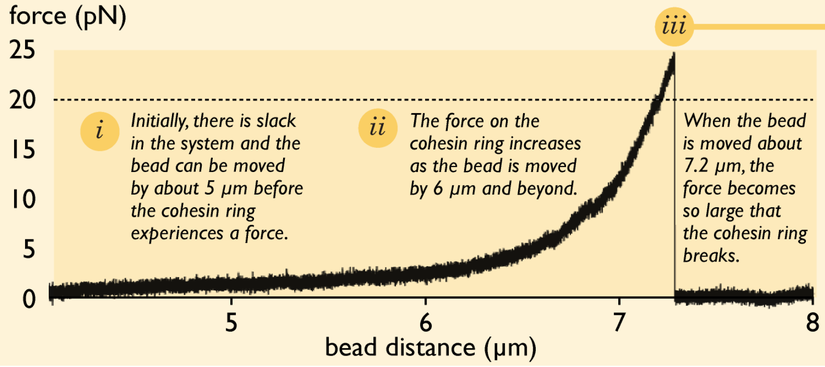


1 µm = 0.000001 m



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your submissions

thank you

information graphics

engage without distracting

Evaluation satisfaction survey pdp 2023

Survey period: mid-October – mid-November 2023

422

satisfaction surveys were sent out
(excluded: demented, deceased)

response rate:
N = 206

49 %

87.9 % ◄ ► 12.1 %

Quantitative results

Strongly disagree	Somewhat disagree	Neutral	Somewhat agree	Strongly agree
1	2	3	4	5

91% would recommend the pdp to others

291 comments from **138 participants**

„Ich habe viel dazu gelernt und auch wo meine schwächeren Punkte sind und ich möchte daran arbeiten!“

“Just taking part helped me a lot.”

„Ich freue mich immer, wenn ein neuer Termin ansteht!“

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„Le fait de savoir qu'il y a quelqu'un qui se soucie de mes préoccupations. “

pdp voices

„Wichtig ist für mich, dass man nach jedem pdp Programm sich nicht verschlechtert hat. Das ist mein Ziel.“

„J'éspère pouvoir encore participer longtemps.“

„Freundliche, effizient arbeitende Menschen die in einem sehr gut organisierten Umfeld nützliche Leistungen erbringen, nicht nur für mich und über die Forschung für andere.“

„D'abord: le fait qu'il existe. Ensuite, le fait qu'il continue à exister, c'est un projet sérieux qui s'inscrit dans la durée et qui pourra m'être utile à moi personnellement. “

ideas for improvement

- Gruppentherapie im Norden.
- Bei der Terminvergabe danach in der Zitha. Da hat es lange gedauert, bis Platz für mich da war.
- Avoir une application d'entraînement en français.
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ideas for improvement

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- Arrêt de bus devant le PDP.
- Avoir le résultat des tests par rapport au(x) dernier(s) réalisés avec un graphisme simple […]

PDF 2023

Evaluation satisfaction survey

mid-October – mid-November 2023

ENGAGEMENT

206 49%

422

88% 12% respondents

satisfaction surveys mailed out
excluded: demented, deceased

FEEDBACK

strongly disagree 1 · somewhat disagree 2 · neutral 3 · somewhat agree 4 · strongly agree 4.5 5

team
professionalism
comfortable feeling
dedicated time
waiting time on site
organisation & communication of appointments

91%

would recommend PDP to others

291 COMMENTS / 138 PARTICIPANTS

IDEAS FOR IMPROVEMENT

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422

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(excluded: demented, deceased)

response rate:
N = 206

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87.9 % ◄ ► 12.1 %

Quantitative results

Strongly disagree	Somewhat disagree	Neutral	Somewhat agree	Strongly agree
1	2	3	4	5

91% would recommend the pdp to others

professionalism
team
comfortable feeling
> 4.5/5
waiting time on site
dedicated time
organisation and communication of appointments

291 comments from **138 participants**

„Ich habe viel dazu gelernt und auch wo meine schwächeren Punkte sind und ich möchte daran arbeiten!“

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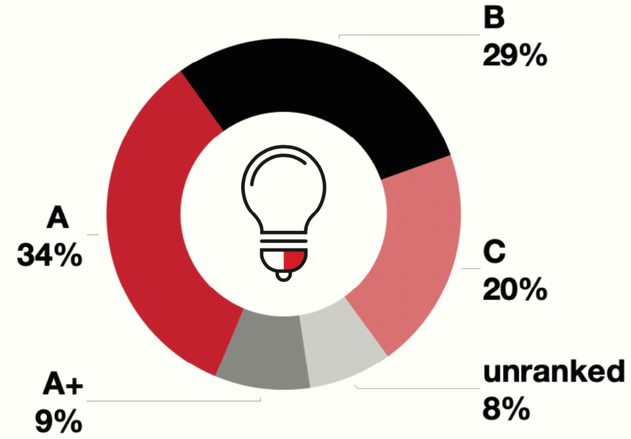
5-YEAR Research Portfolio

Carleton University | Sprott School of Business



399 peer-reviewed articles

233 conference proceedings

276 presentations

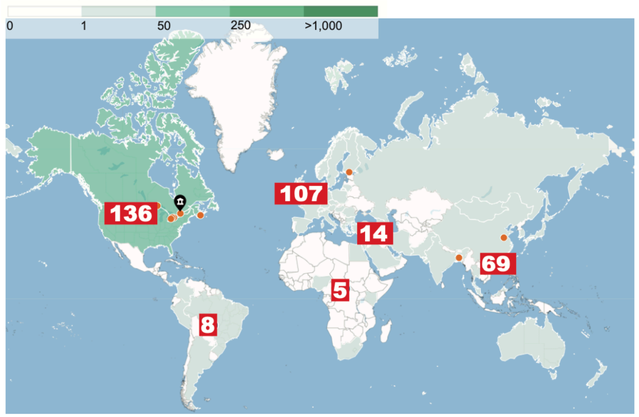
47 chapters

5 books

246 editorships

INTERNATIONAL ENGAGEMENT

Co-authored publications per country/region:



Collaborating Institutions
Top Institutions worldwide by co-authored publications

CMYK	PANTONE	RGB	HEX
0 / 100 / 100 / 0	185	233 / 28 / 36	E91C24

CMYK	RGB	HEX
0 / 0 / 0 / 100	0 / 0 / 0	000000

TINTS

90%	80%	70%	60%	50%	40%	30%	20%	10%	5%

CMYK	RGB	HEX
0 / 0 / 0 / 0	255 / 255 / 255	FFFFFF

Photography → Guiding principles

Engaging original photography should be used where possible. To accurately tell our story and reflect the Carleton brand, there are four guiding principles to consider.

SELECTED IMAGES SHOULD BE:

Bold

Carleton is an exciting place doing exciting things. Photography should be confident and distinctive, not quiet and generic.

Active

There's a lot going on in every part of the university. Our subjects should be captured either taking action or taking a brief break from what they're doing to interact with our audience.

Authentic

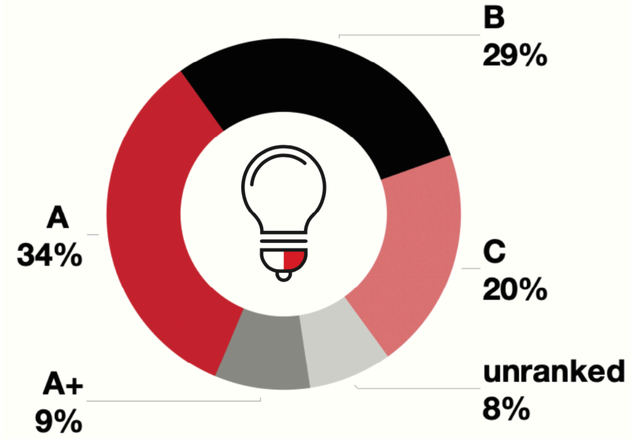
Photography must reflect the actual story of Carleton. We should capture real students, faculty, and staff in environments that reflect Carleton. We have a unique story to tell, so must emphasize this through the imagery we use.

Inclusive

The Carleton community is diverse in every way. Photography should proportionately represent diversity in all forms.

5-YEAR Research Portfolio

Carleton University | Sprott School of Business



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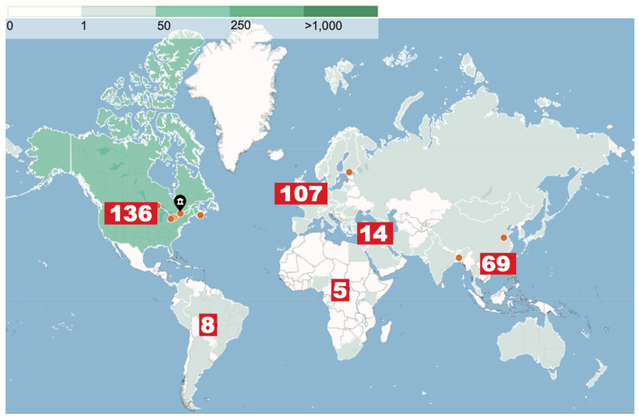

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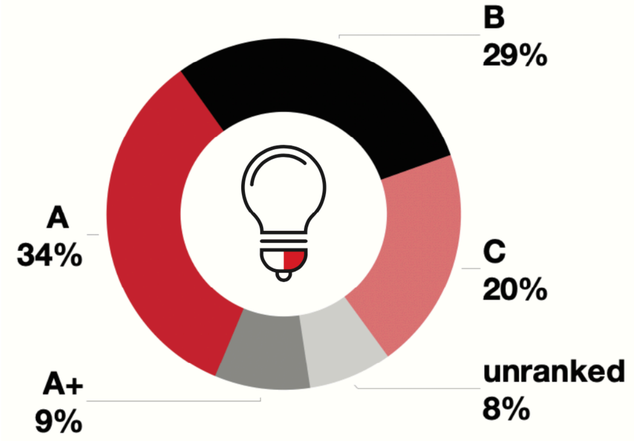

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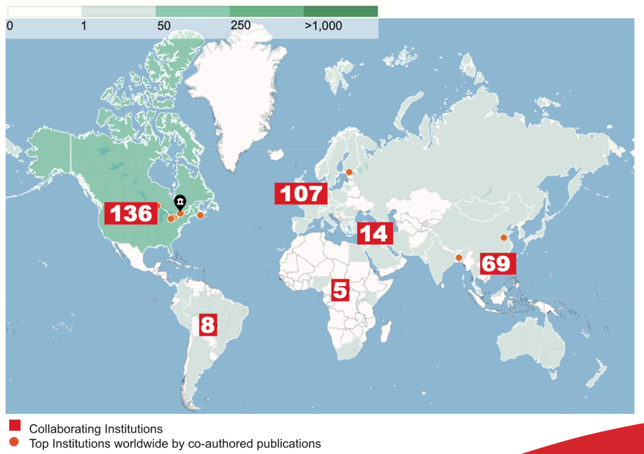

Carleton University | Sprott School of Business

2018–2023

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Research programs *XYZ rank*

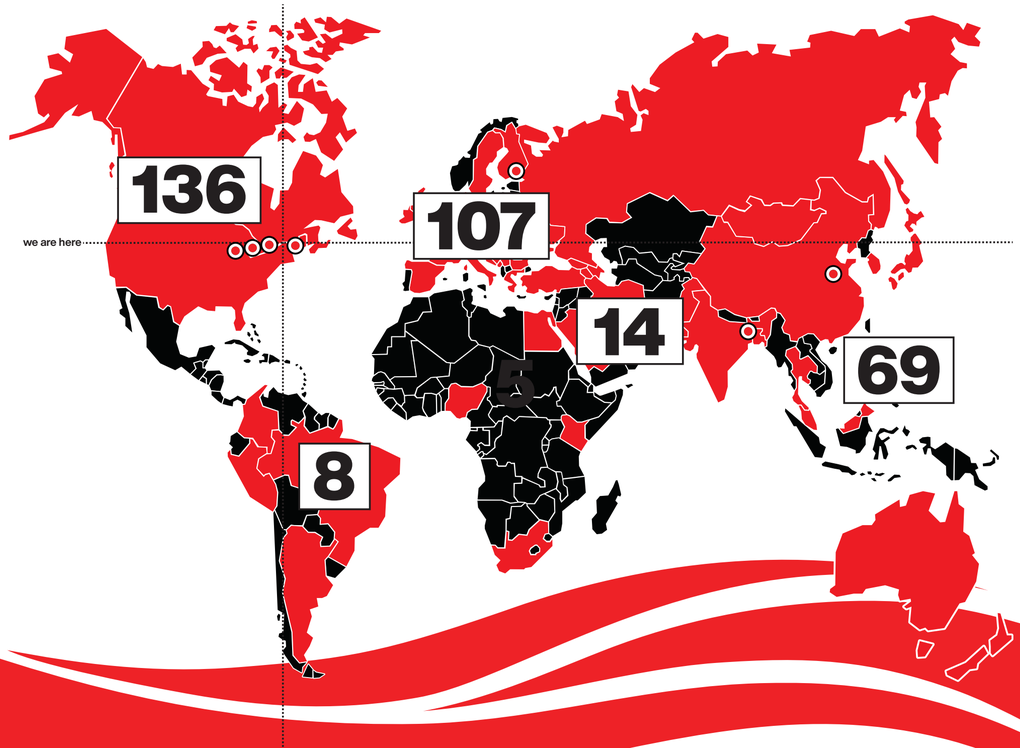
A+	A	B	C	not ranked
9%	34%	29%	20%	8%

International engagement

co-authored publications, by region

top 10, by publications

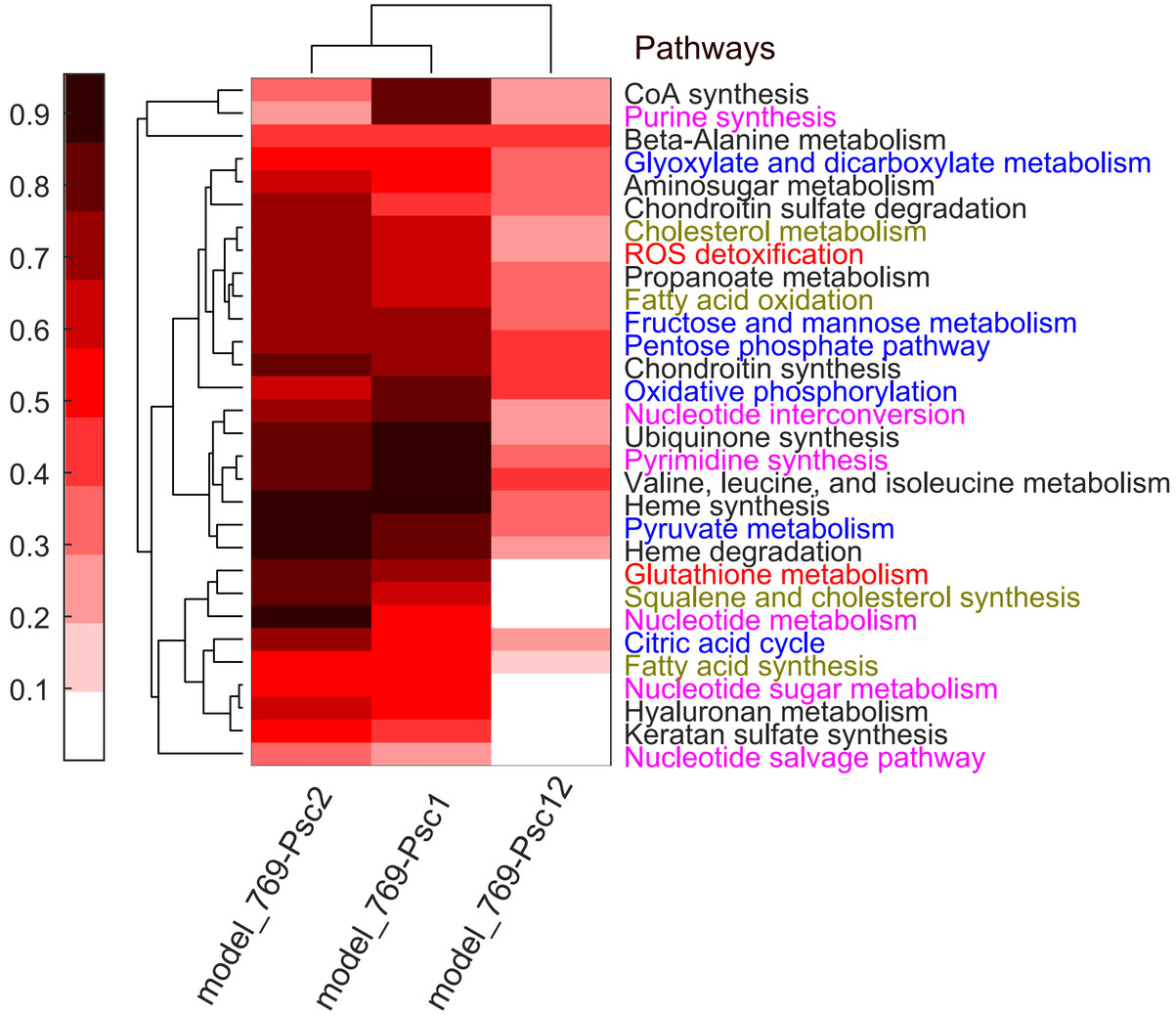
Organization 1, Organization 2, Organization 3, Organization 4, Organization 5
Organization 6, Organization 7, Organization 8, Organization 9, Organization 10



data plots

legends and shortcuts

Similarity based on Flux Variability Analysis RCC



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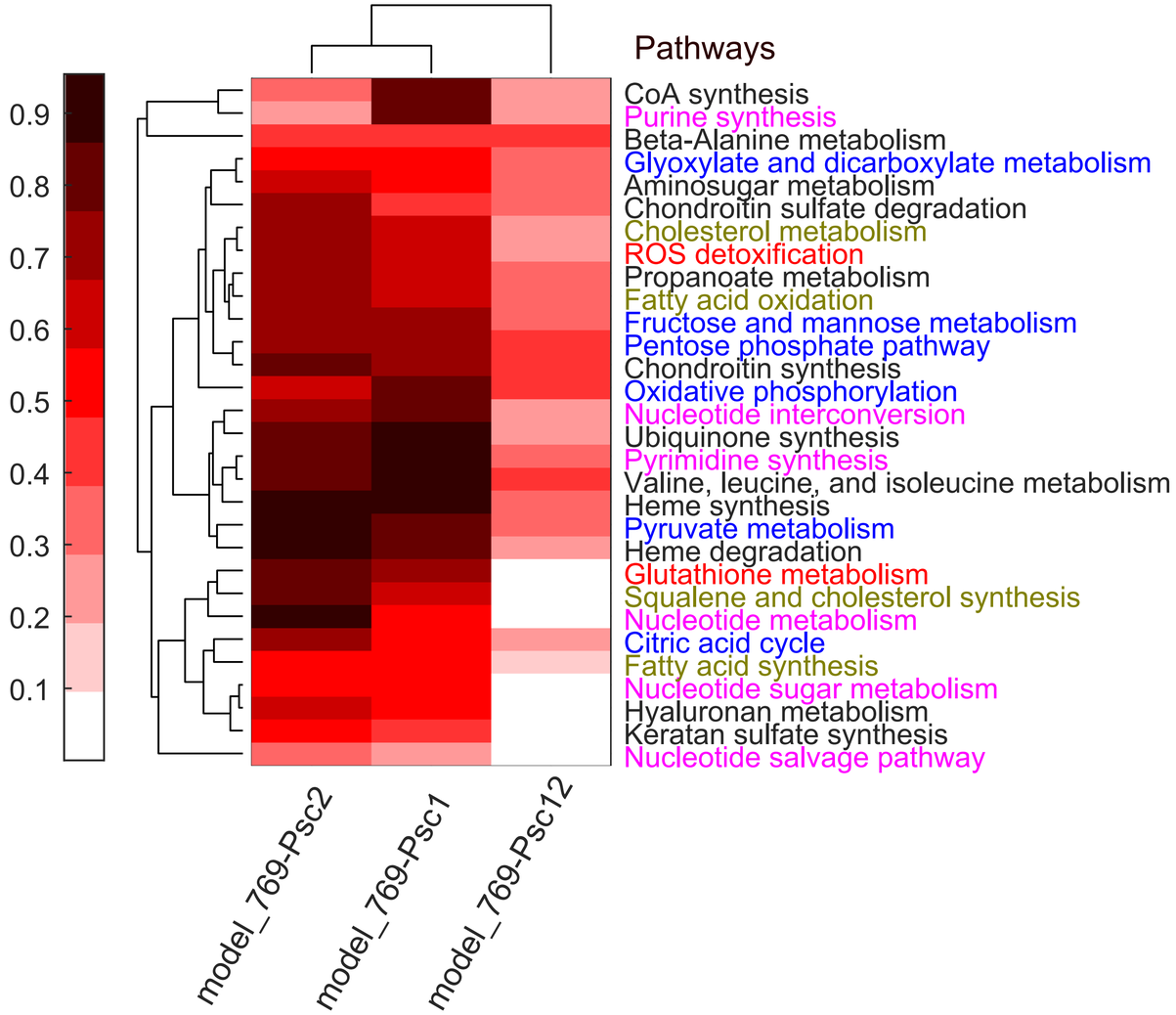

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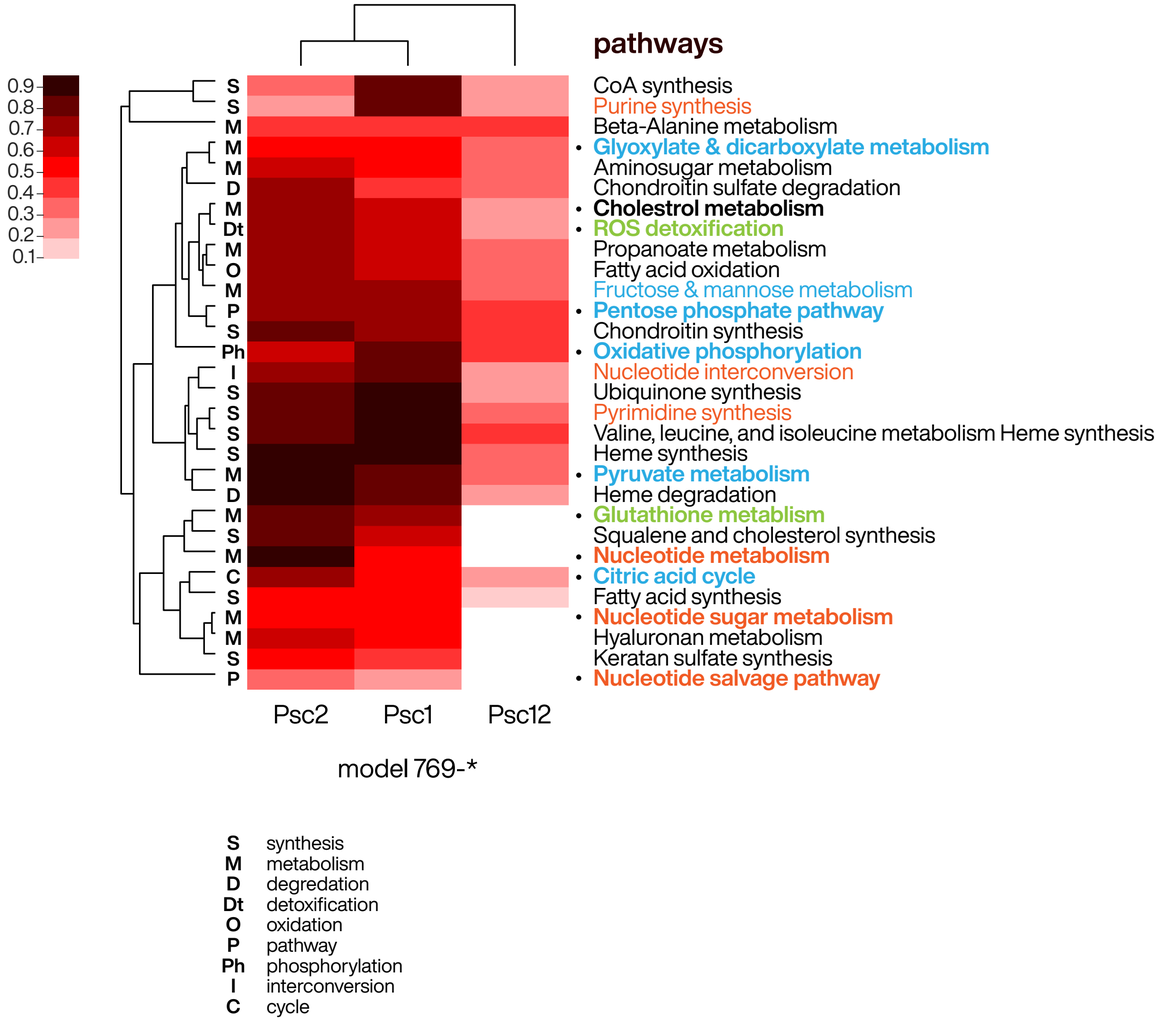
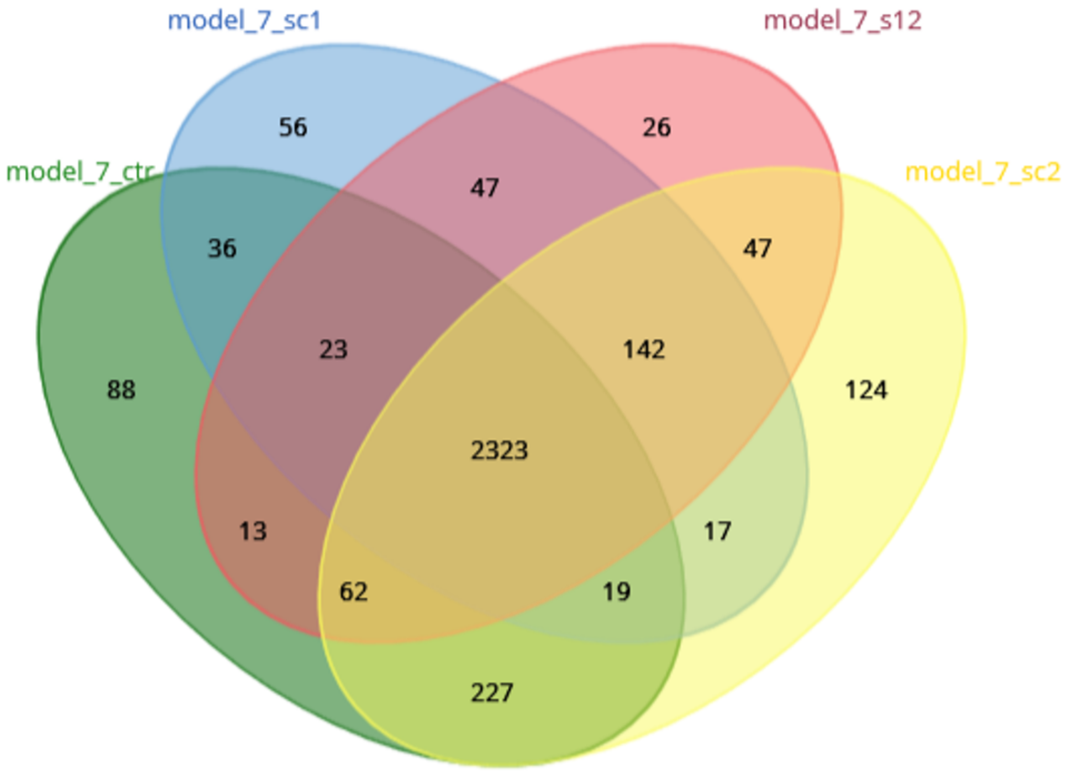


S synthesis
M metabolism
D degredation
Dt detoxification
O oxidation
P pathway
Ph phosphorylation
I interconversion
C cycle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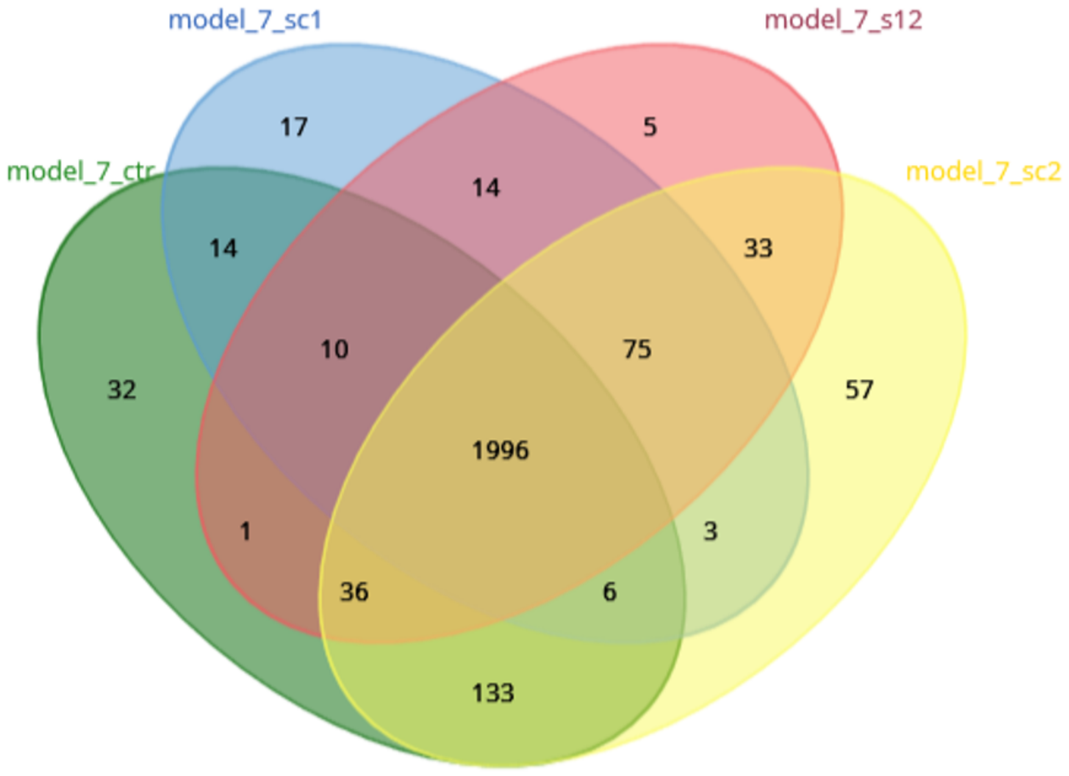
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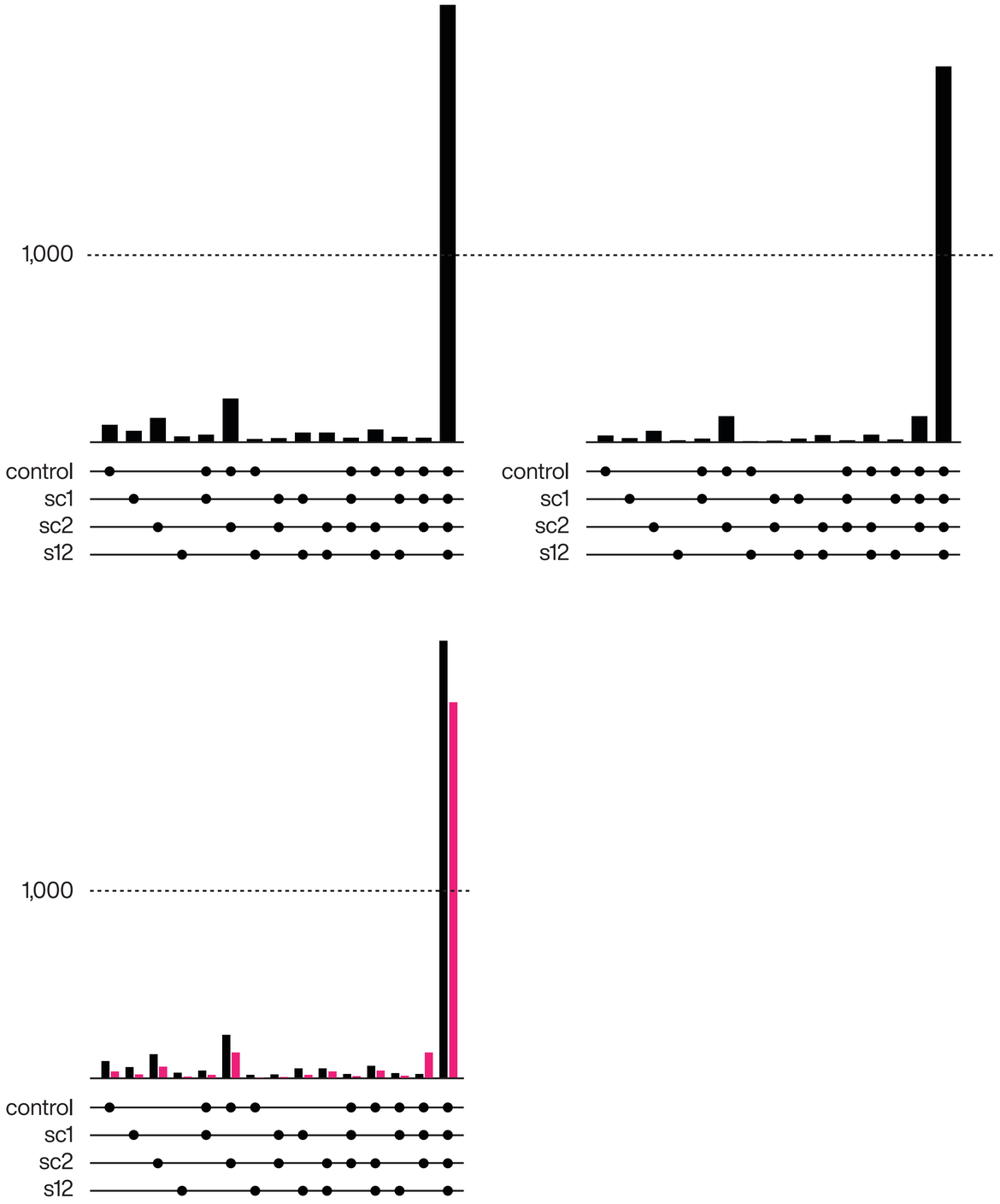
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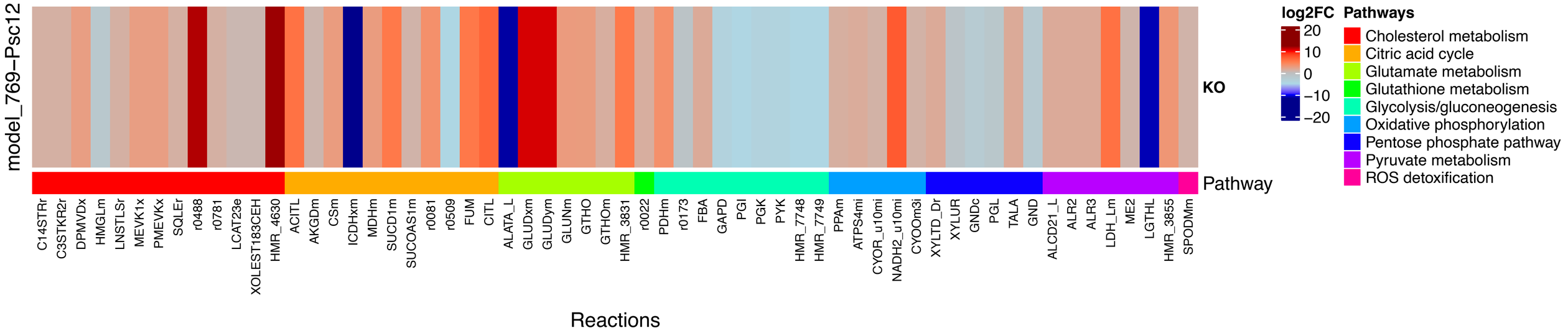
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HMR_4630
ACITL
AKGDm
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XYLUR
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PGL
TALA
GND
ALCD21_L
ALR2
ALR3
LDH_Lm
ME2
LGTHL
HMR_3855
SPODMm
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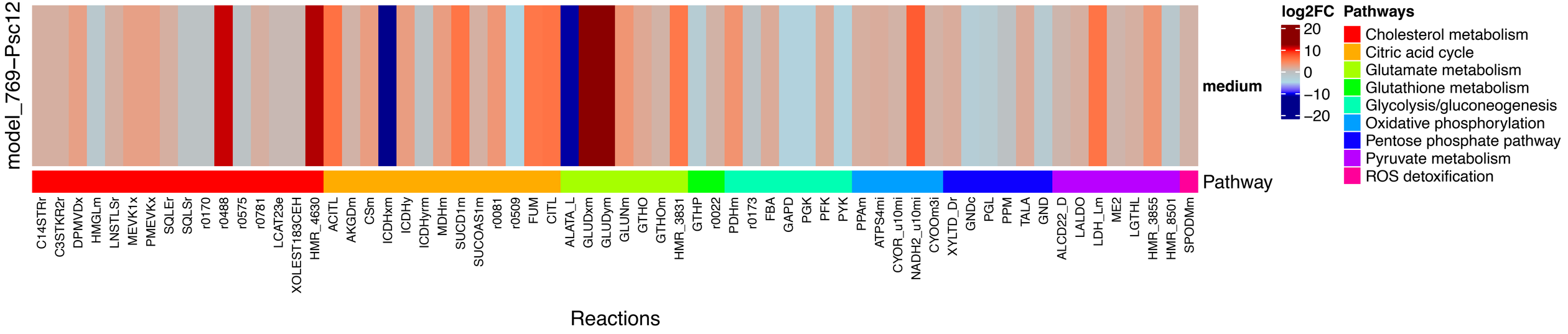
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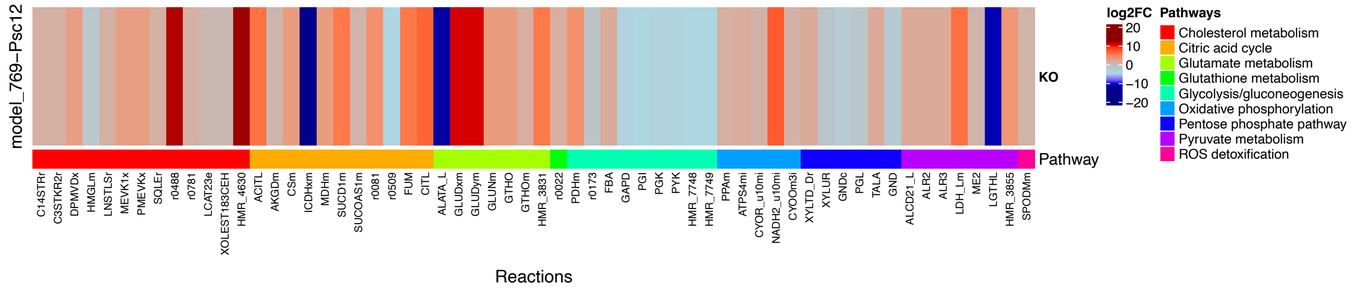
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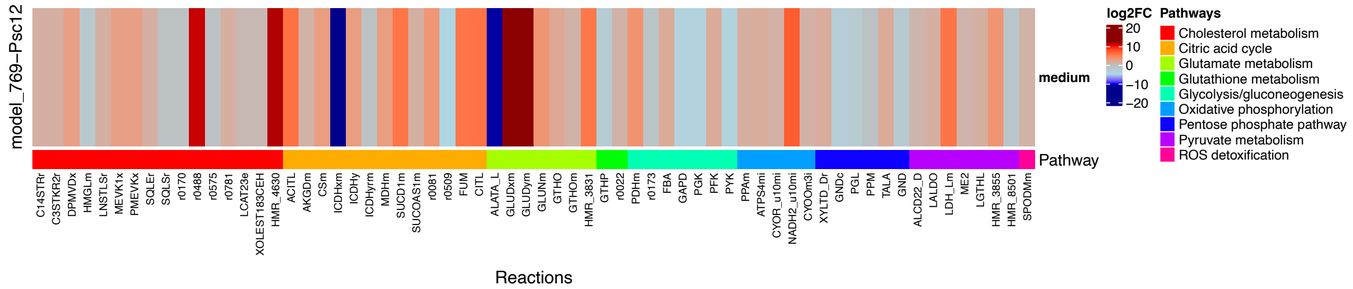
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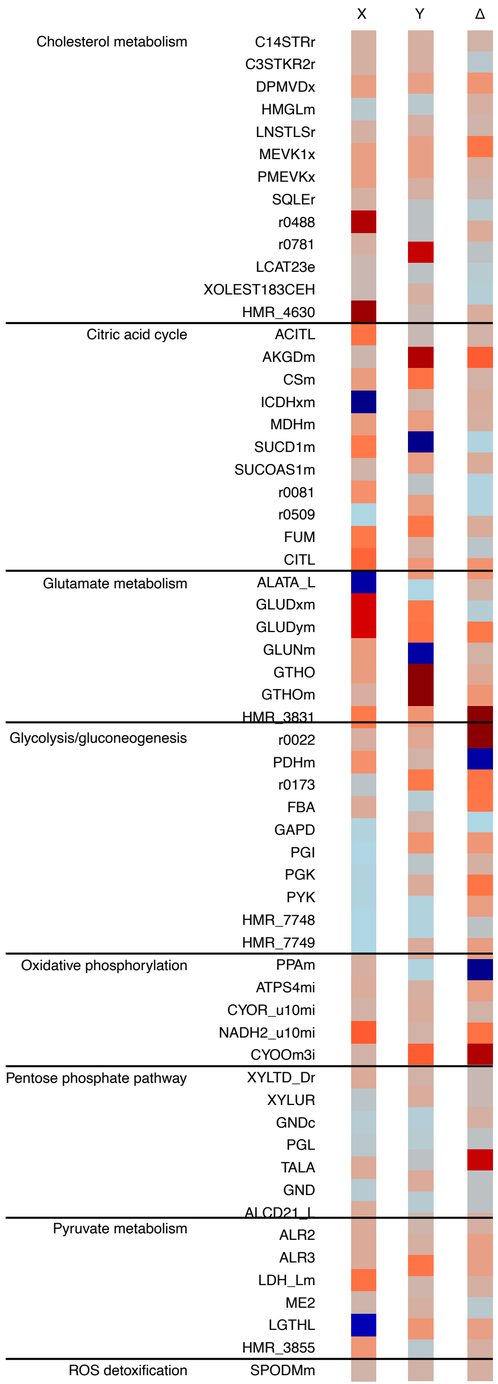
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SUCD1m
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r0509
FUM
CITL
Glutamate metabolism
ALATA_L
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Glycolysis/gluconeogenesis
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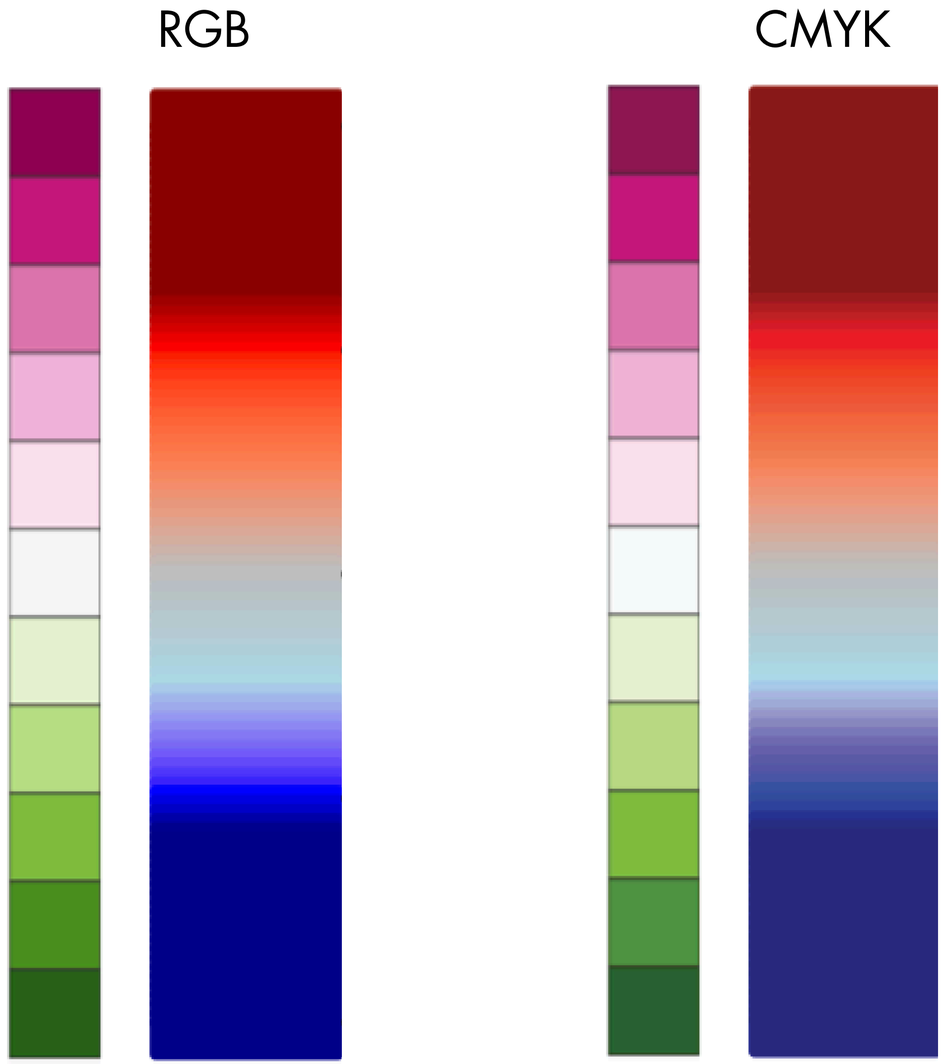
Oxidative phosphorylation
PPAm
ATPS4mi
CYOR_u10mi
NADH2_u10mi
CYOOm3i
Pentose phosphate pathway
XYLTD_Dr
XYLUR
GNDc
PGL
TALA
GND
ALCD21_L
Pyruvate metabolism
ALR2
ALR3
LDH_Lm
ME2
LGTHL
HMR_3855
ROS detoxification
SPODMm

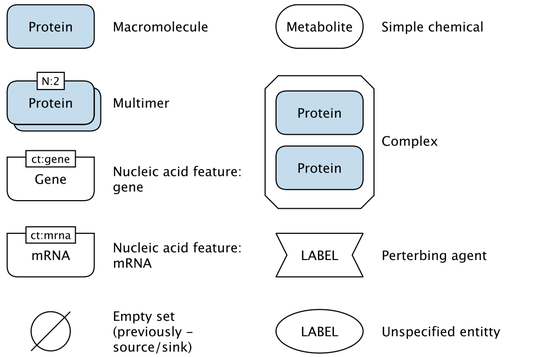
visible
RGB
CMYK


RGB
CMYK

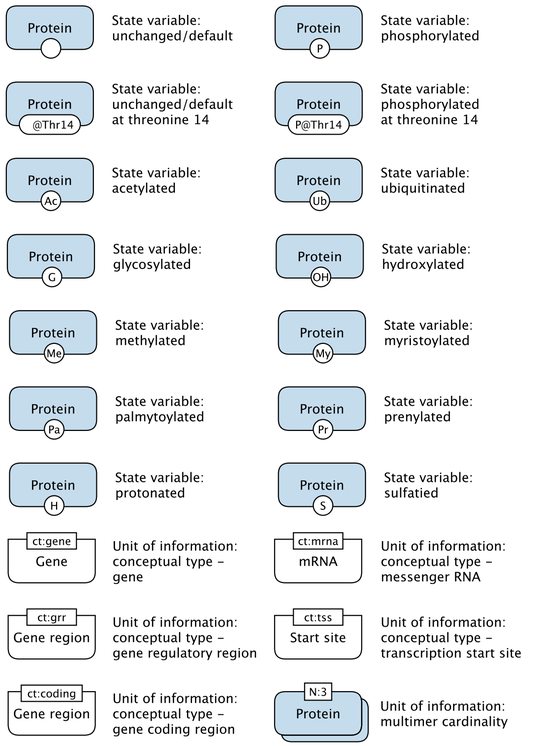
visual vocabulary

Gestalt

ENTITY POOL NODES



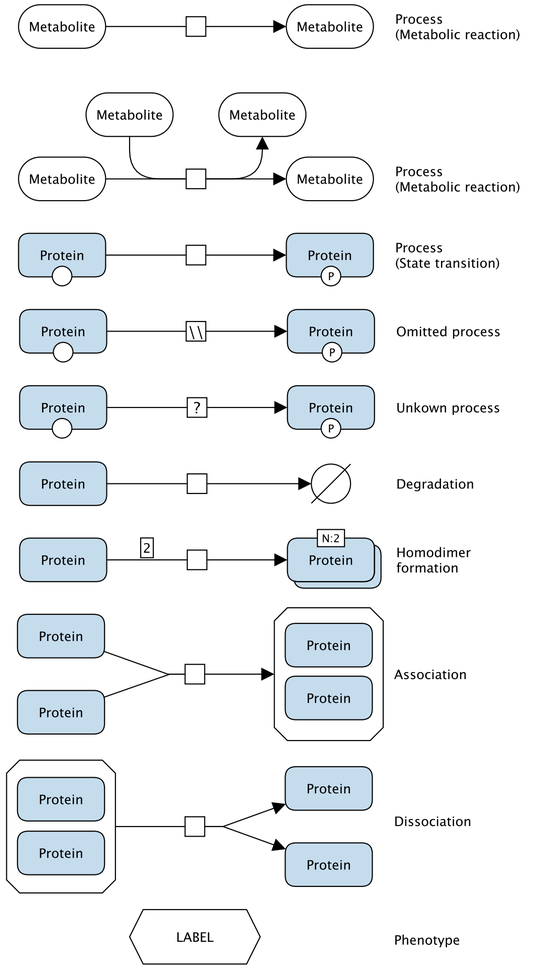
AUXUILIARY UNITS



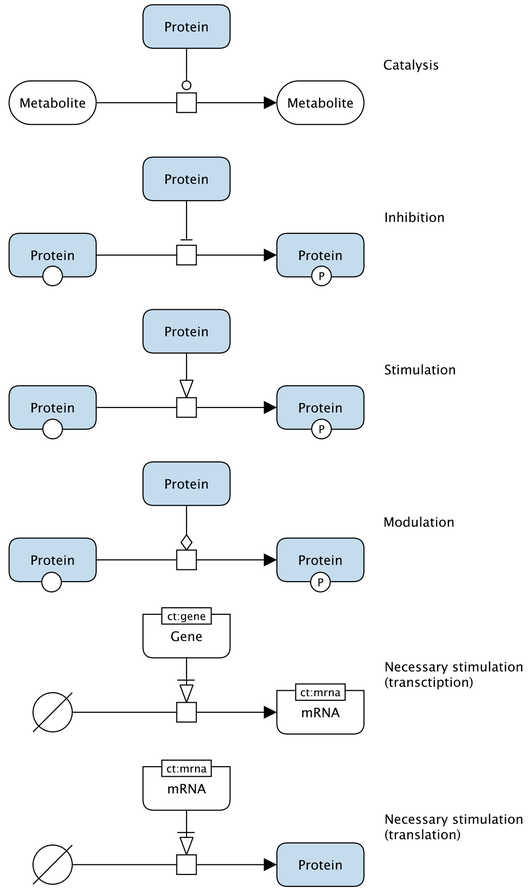
CONTAINER NODE

ENCAPSULATION NODE

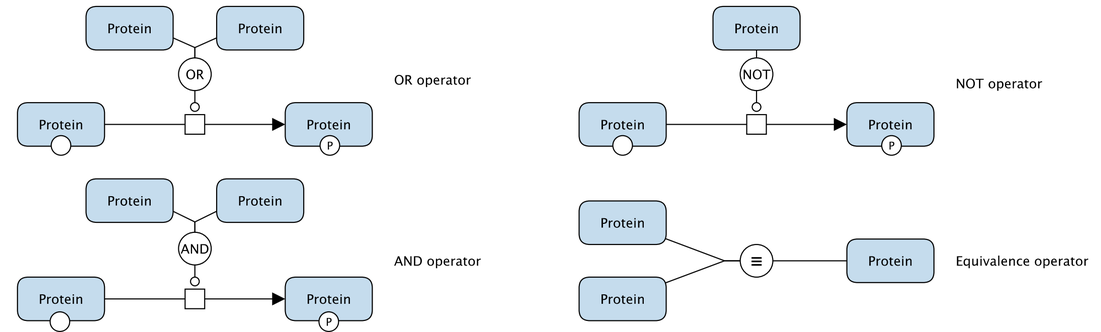
PROCESS NODES

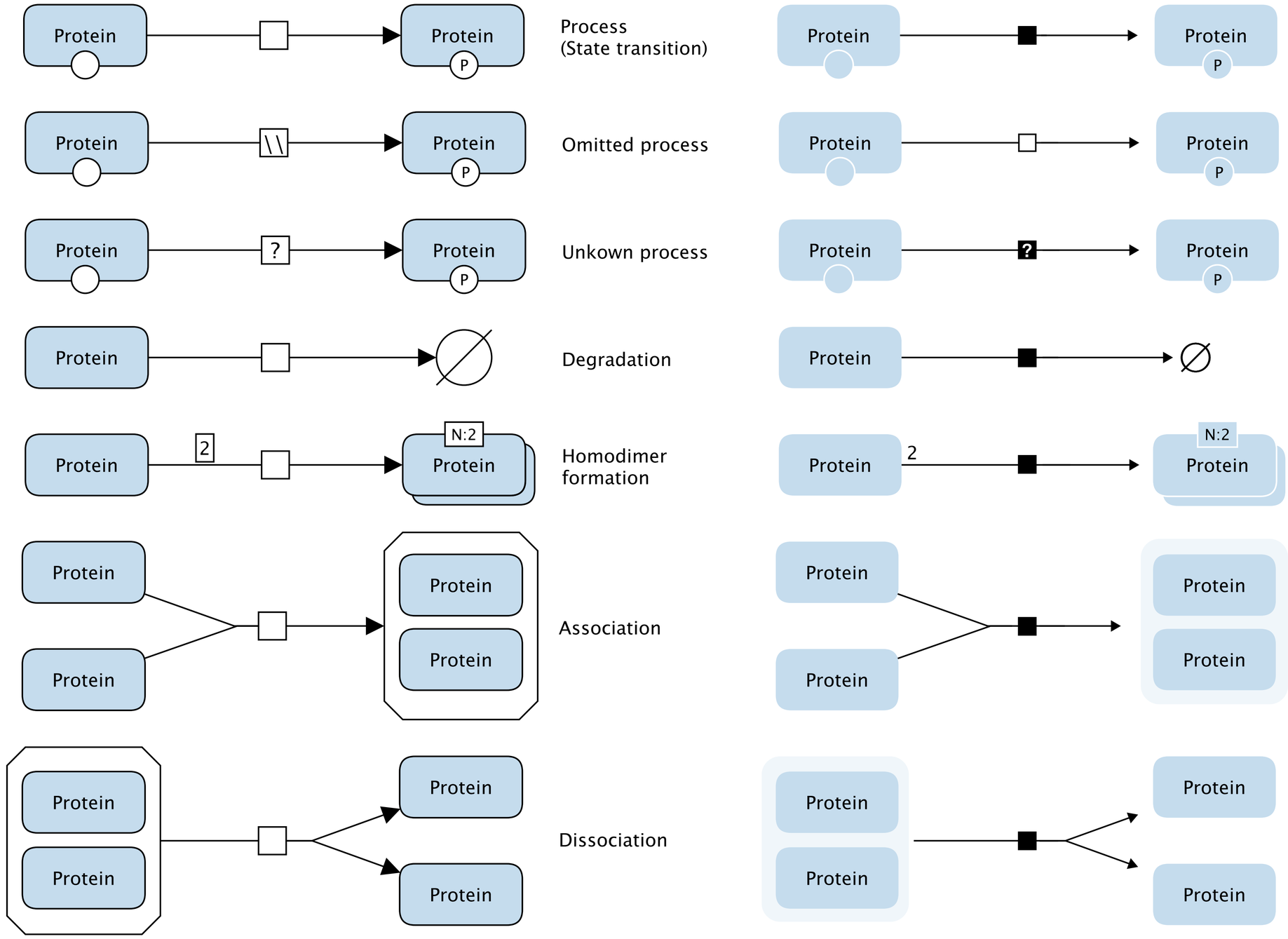


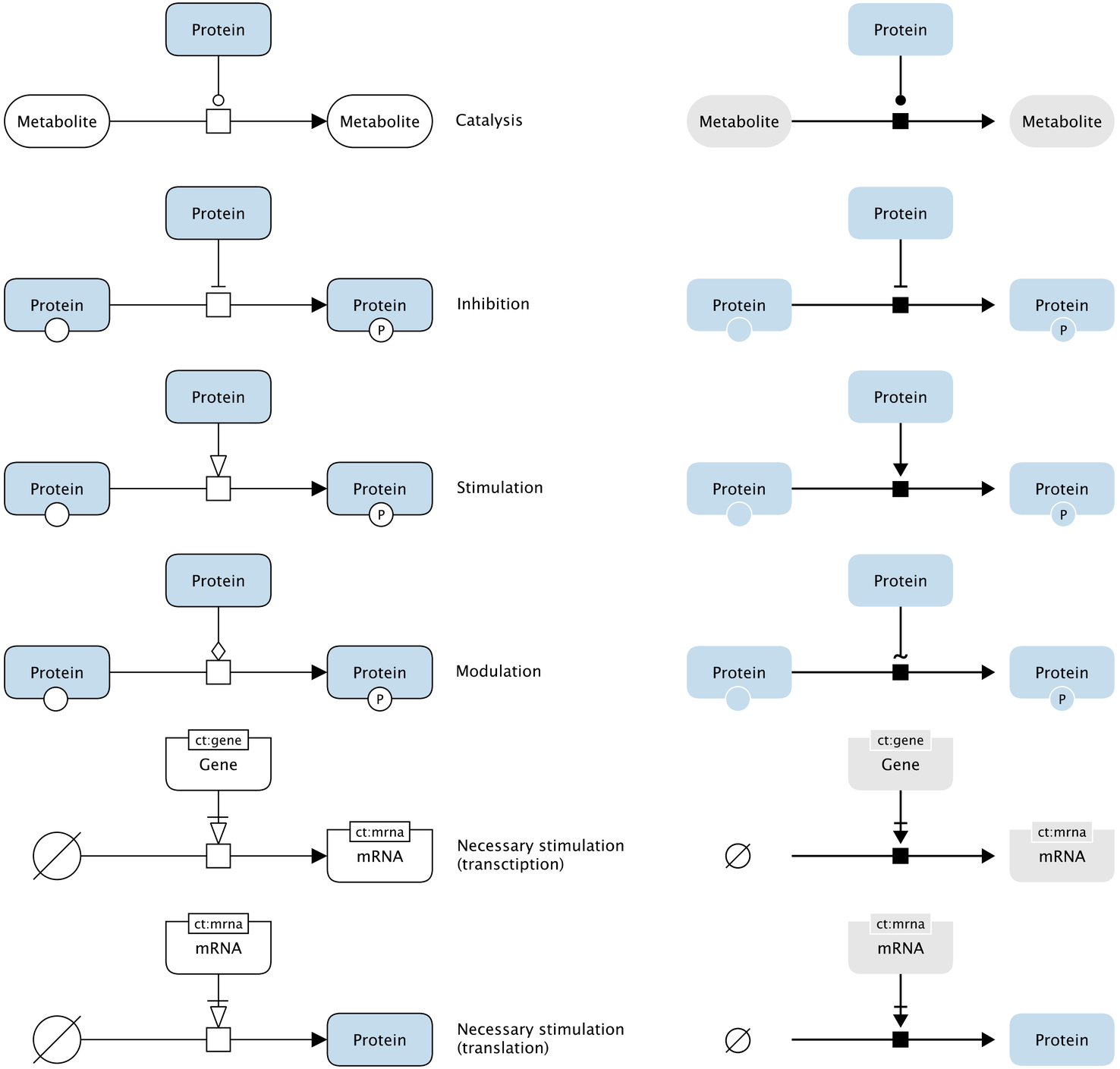
PROCESS REGULATION

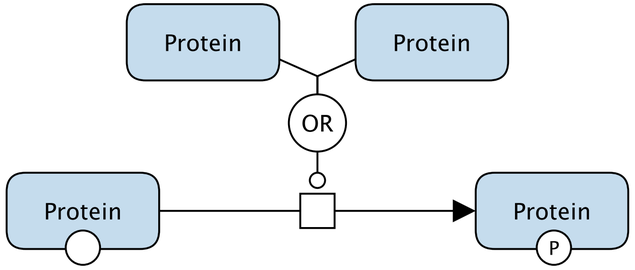


LOGIC OPERATORS

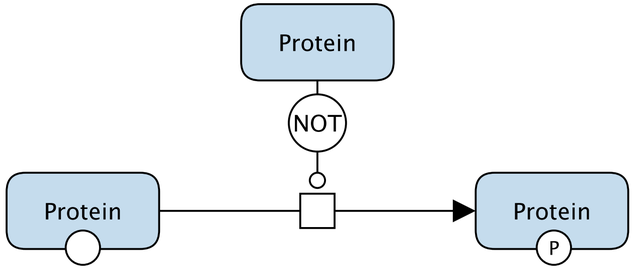



Protein
Protein
P
Process
(State transition)
Omitted process
Unkown process
?
Degradation
2
N:2
Homodimer
formation
Association
Dissociation

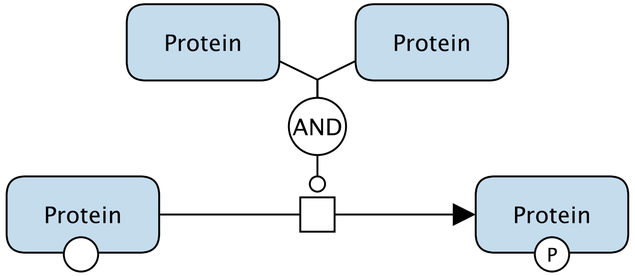

Protein
Metabolite
Metabolite
Catalysis
Protein
Protein
Protein
P
Inhibition
Protein
Protein
Protein
P
Stimulation
Protein
Protein
Protein
P
Modulation
ct:gene
Gene
ct:mrna
mRNA
Necessary stimulation
(transctiption)
ct:mrna
mRNA
Protein
Necessary stimulation
(translation)



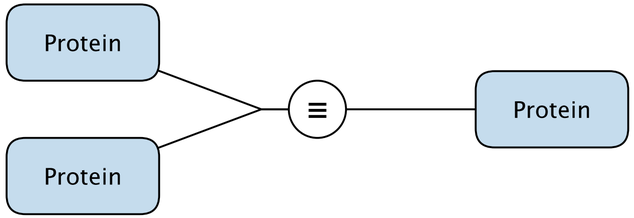
OR operator



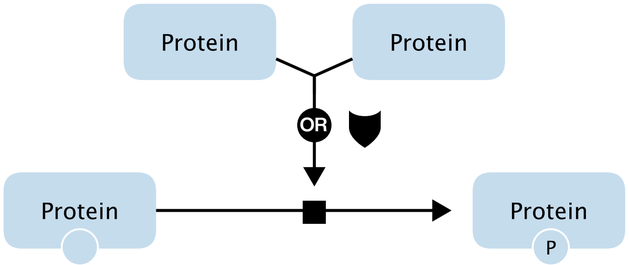
NOT operator

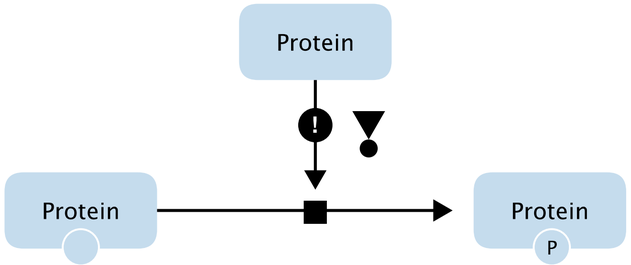


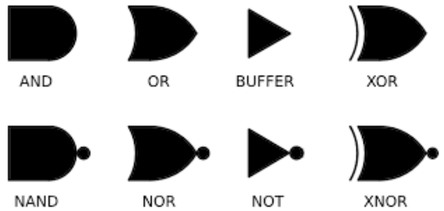
AND operator

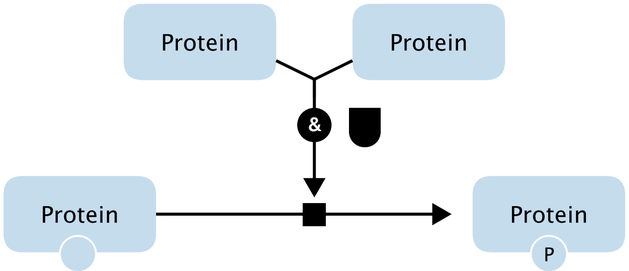


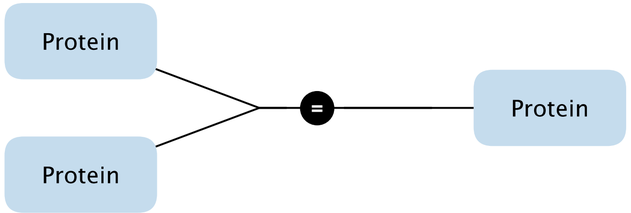
Equivalence operator

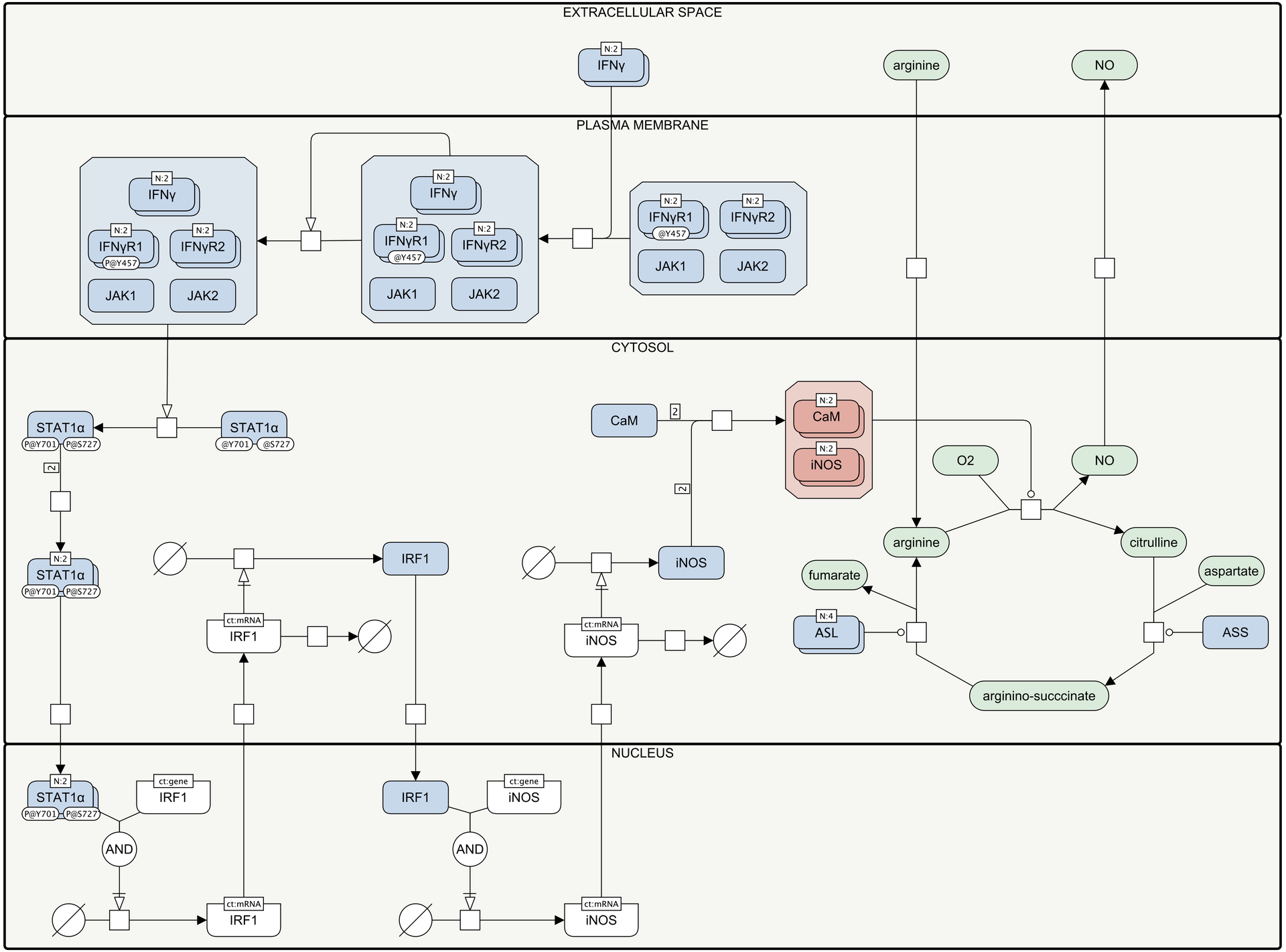


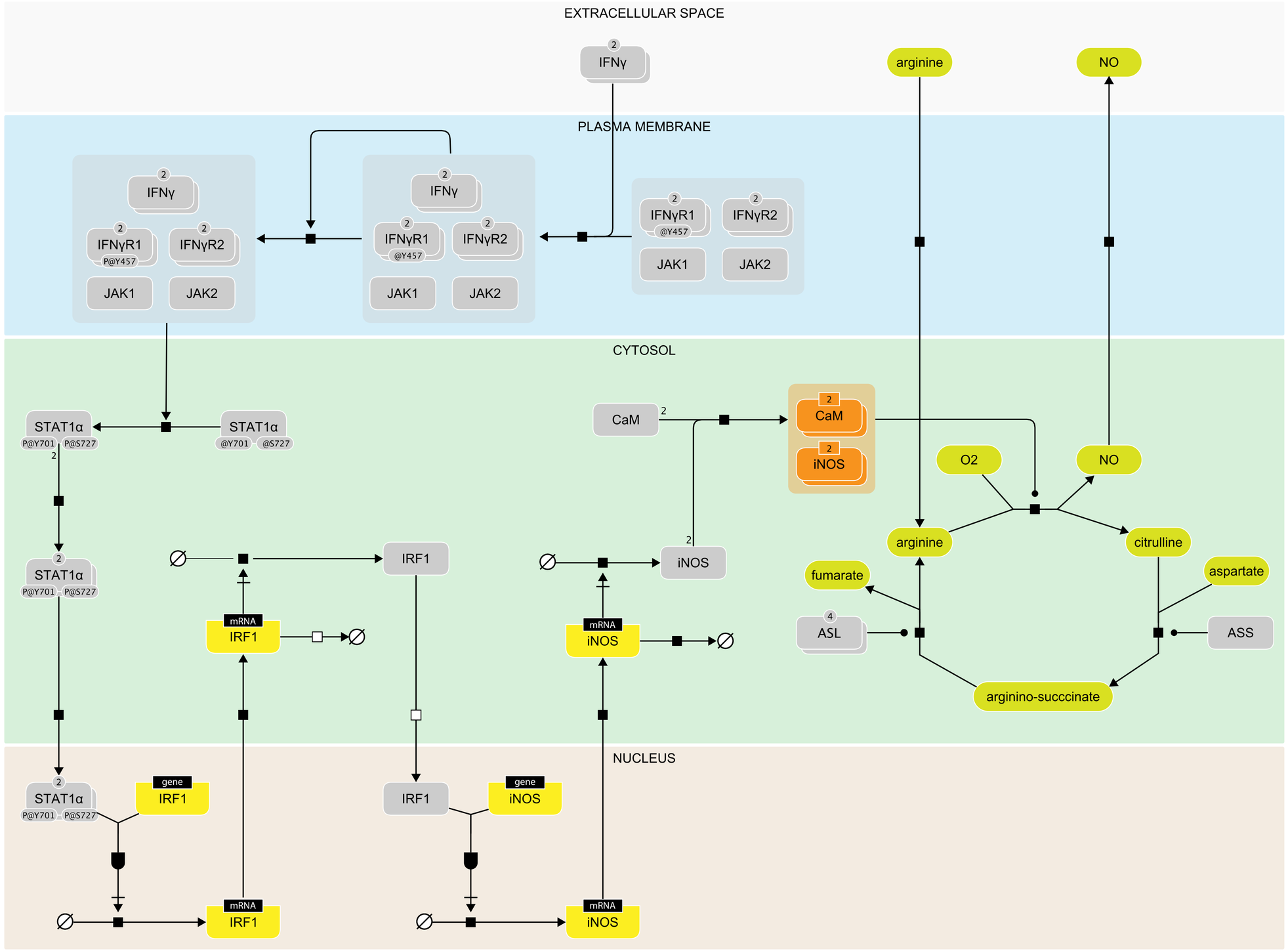


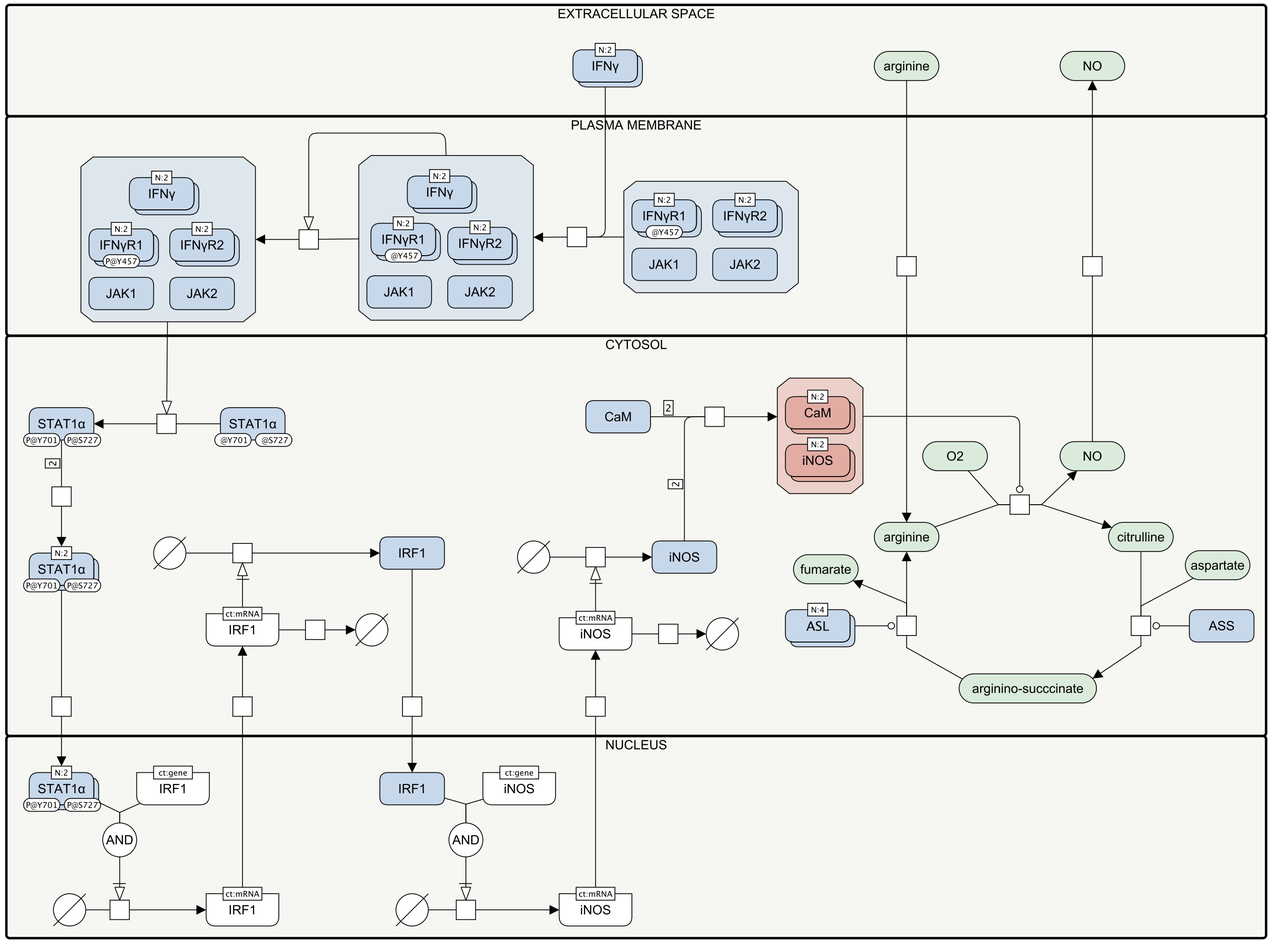


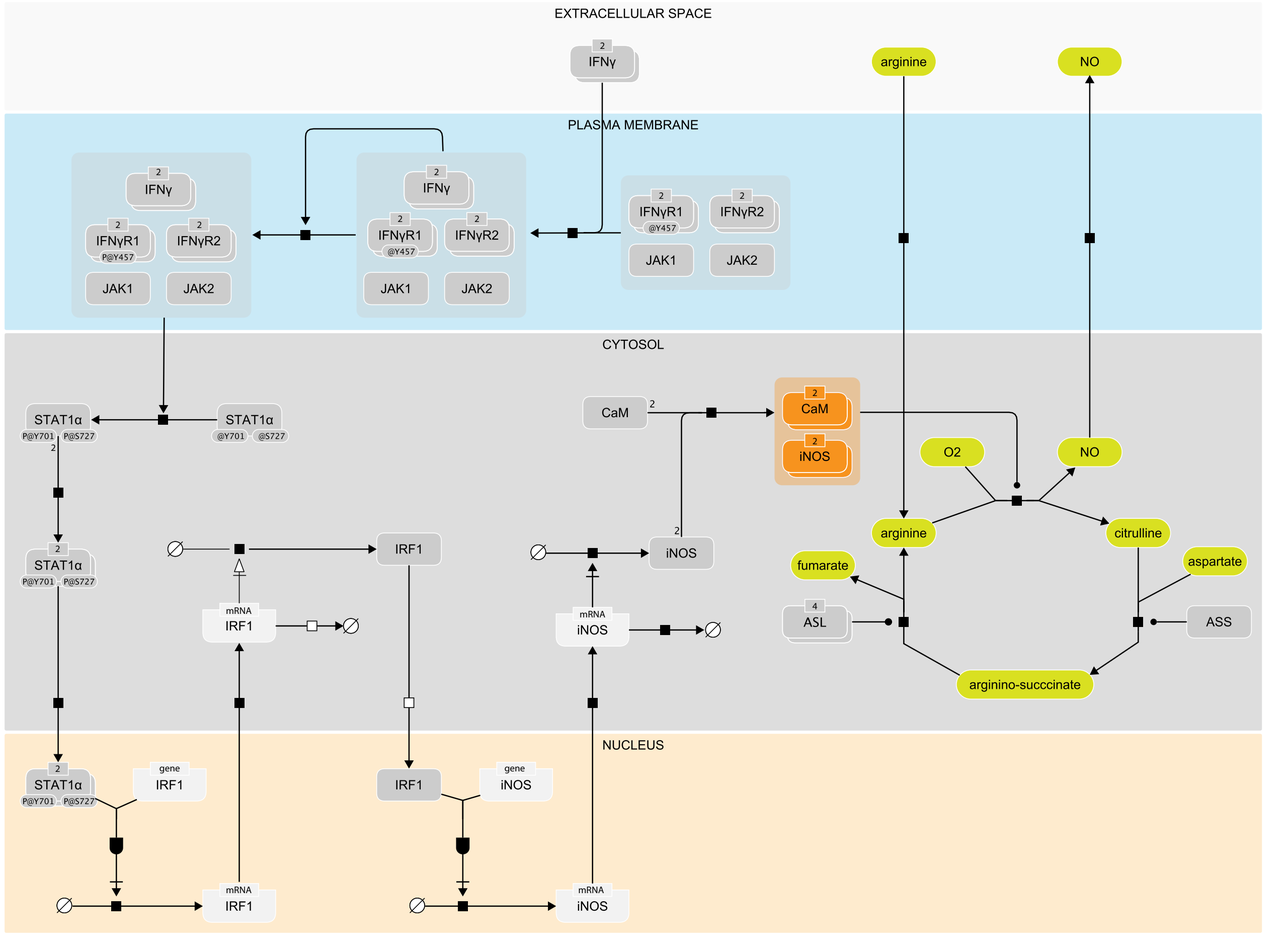


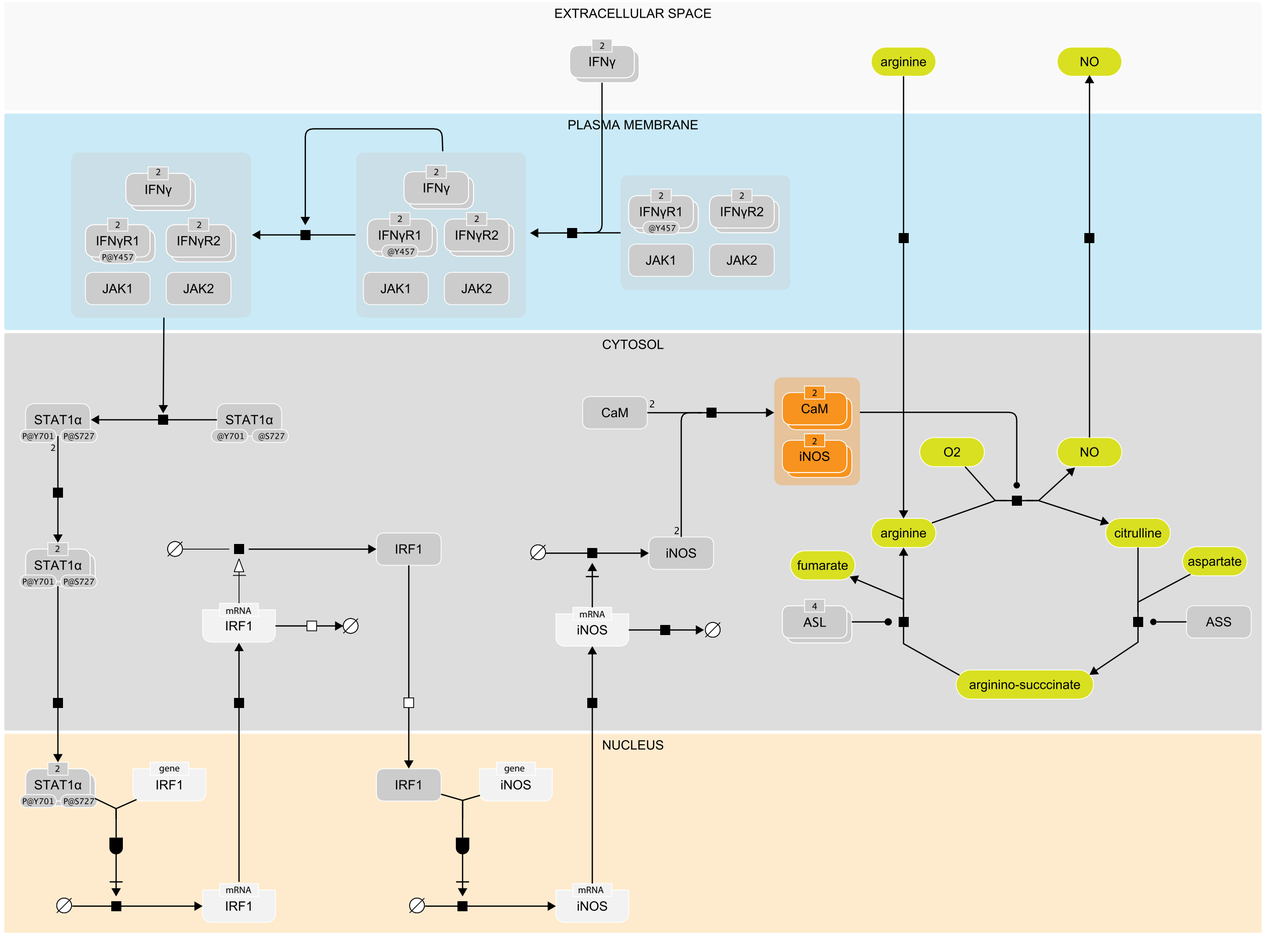


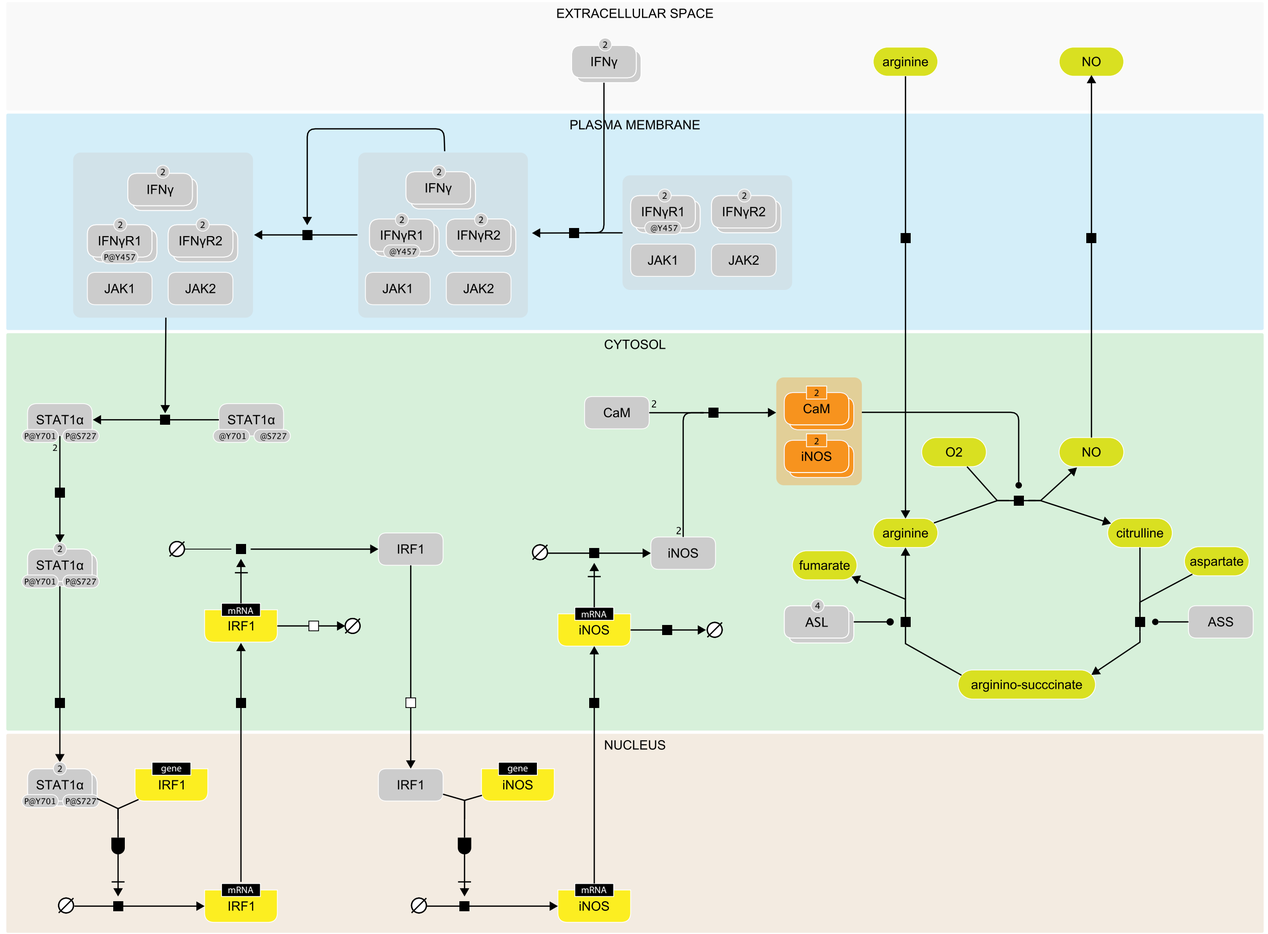

EXTRACELLULAR SPACE
N:2
IFNγ
arginine
NO
PLASMA MEMBRANE
N:2
IFNγ
N:2
IFNγR1
P@Y457
N:2
IFNγR2
JAK1
JAK2
N:2
IFNγ
N:2
IFNγR1
@Y457
N:2
IFNγR2
JAK1
JAK2
N:2
IFNγR1
@Y457
N:2
IFNγR2
JAK1
JAK2
CYTOSOL
STAT1α
P@Y701
P@S727
STAT1α
@Y701
@S727
2
N:2
STAT1α
P@Y701
P@S727
IRF1
ct:mRNA
IRF1
CaM
2
N:2
CaM
N:2
iNOS
2
iNOS
ct:mRNA
iNOS
O2
NO
arginine
citrulline
fumarate
aspartate
N:4
ASL
ASS
arginino-succcinate
NUCLEUS
N:2
STAT1α
P@Y701
P@S727
ct:gene
IRF1
AND
ct:mRNA
IRF1
IRF1
ct:gene
iNOS
AND
ct:mRNA
iNOS


EXTRACELLULAR SPACE
IFNγ
2
arginine
NO
PLASMA MEMBRANE
IFNγR1
IFNγR2
P@Y457
@Y457
JAK1
JAK2
CYTOSOL
STAT1α
P@Y701
P@S727
@Y701
@S727
CaM
iNOS
O2
citrulline
aspartate
fumarate
ASL
4
ASS
arginino-succcinate
IRF1
mRNA
NUCLEUS
gene


EXTRACELLULAR SPACE
IFNγ
N:2
arginine
NO
PLASMA MEMBRANE
IFNγR1
IFNγR2
P@Y457
@Y457
JAK1
JAK2
CYTOSOL
STAT1α
P@Y701
P@S727
@Y701
@S727
CaM
iNOS
O2
citrulline
aspartate
fumarate
ASL
ASS
arginino-succcinate
IRF1
ct:mRNA
NUCLEUS
ct:gene
AND


EXTRACELLULAR SPACE
IFNγ
arginine
NO
PLASMA MEMBRANE
IFNγR1
IFNγR2
JAK1
JAK2
CYTOSOL
STAT1α
CaM
iNOS
O2
citrulline
aspartate
fumarate
ASL
ASS
arginino-succcinate
IRF1
mRNA
NUCLEUS
gene

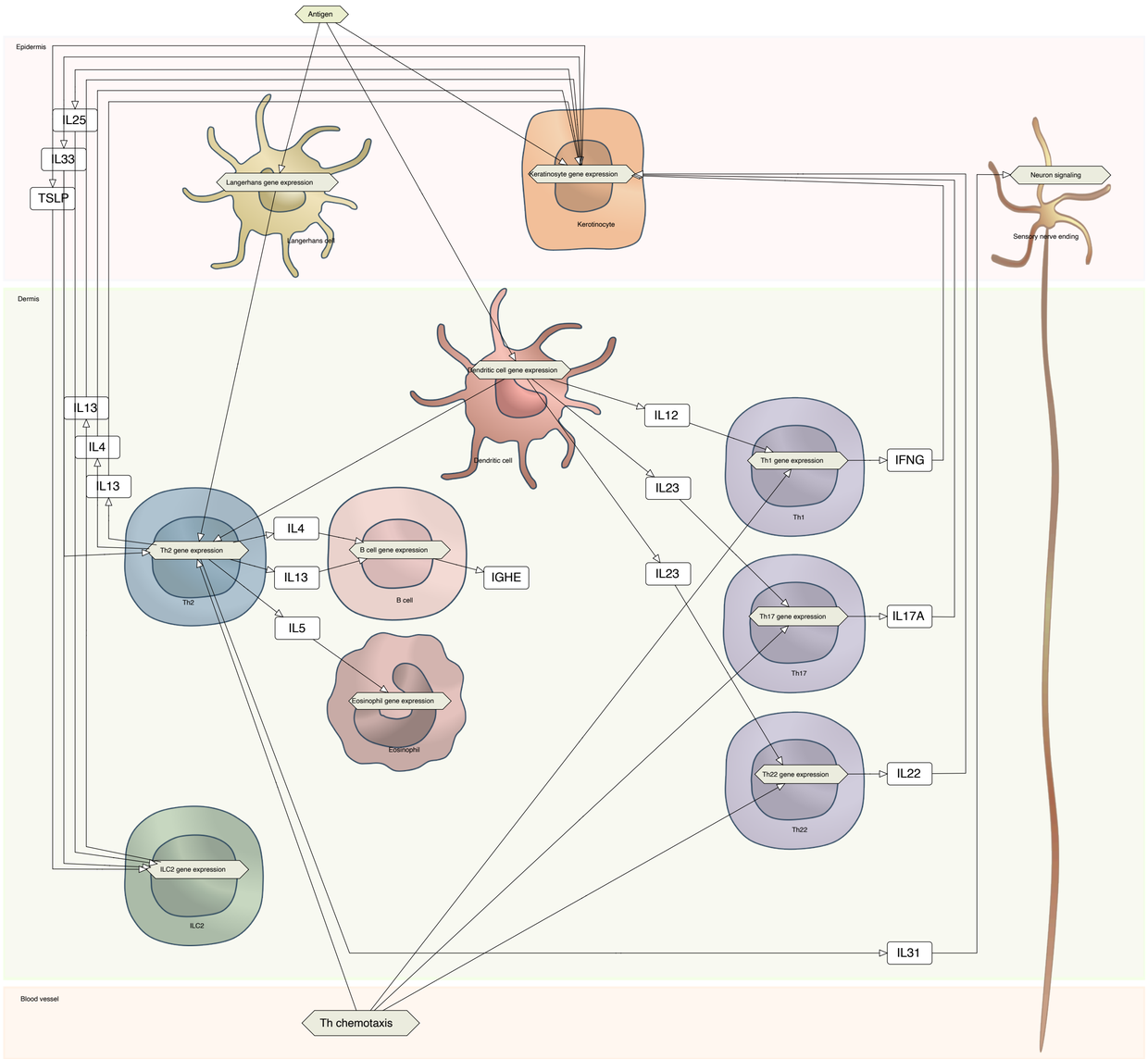

EXTRACELLULAR SPACE
IFNγ
arginine
NO
PLASMA MEMBRANE
IFNγR1
IFNγR2
P@Y457
@Y457
JAK1
JAK2
CYTOSOL
STAT1α
P@Y701
P@S727
@Y701
@S727
CaM
iNOS
O2
arginine
citrulline
aspartate
fumarate
ASL
ASS
arginino-succcinate
IRF1
mRNA
NUCLEUS
gene


EXTRACELLULAR SPACE
IFNγ
arginine
NO
PLASMA MEMBRANE
IFNγR1
IFNγR2
P@Y457
@Y457
JAK1
JAK2
CYTOSOL
STAT1α
P@Y701
P@S727
@Y701
@S727
CaM
iNOS
O2
arginine
citrulline
aspartate
fumarate
ASL
ASS
arginino-succcinate
IRF1
mRNA
NUCLEUS
gene

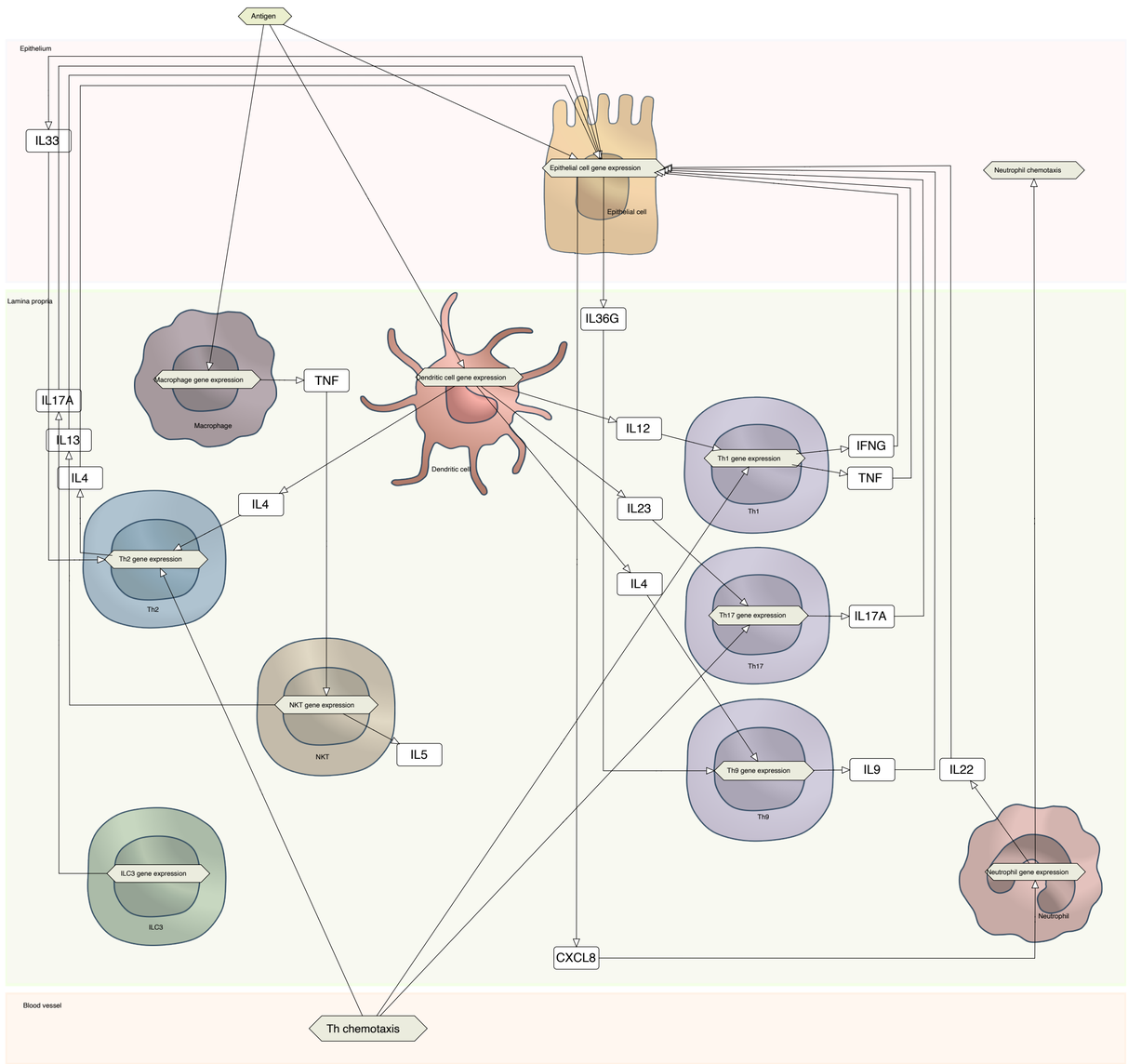
layout

man vs machine

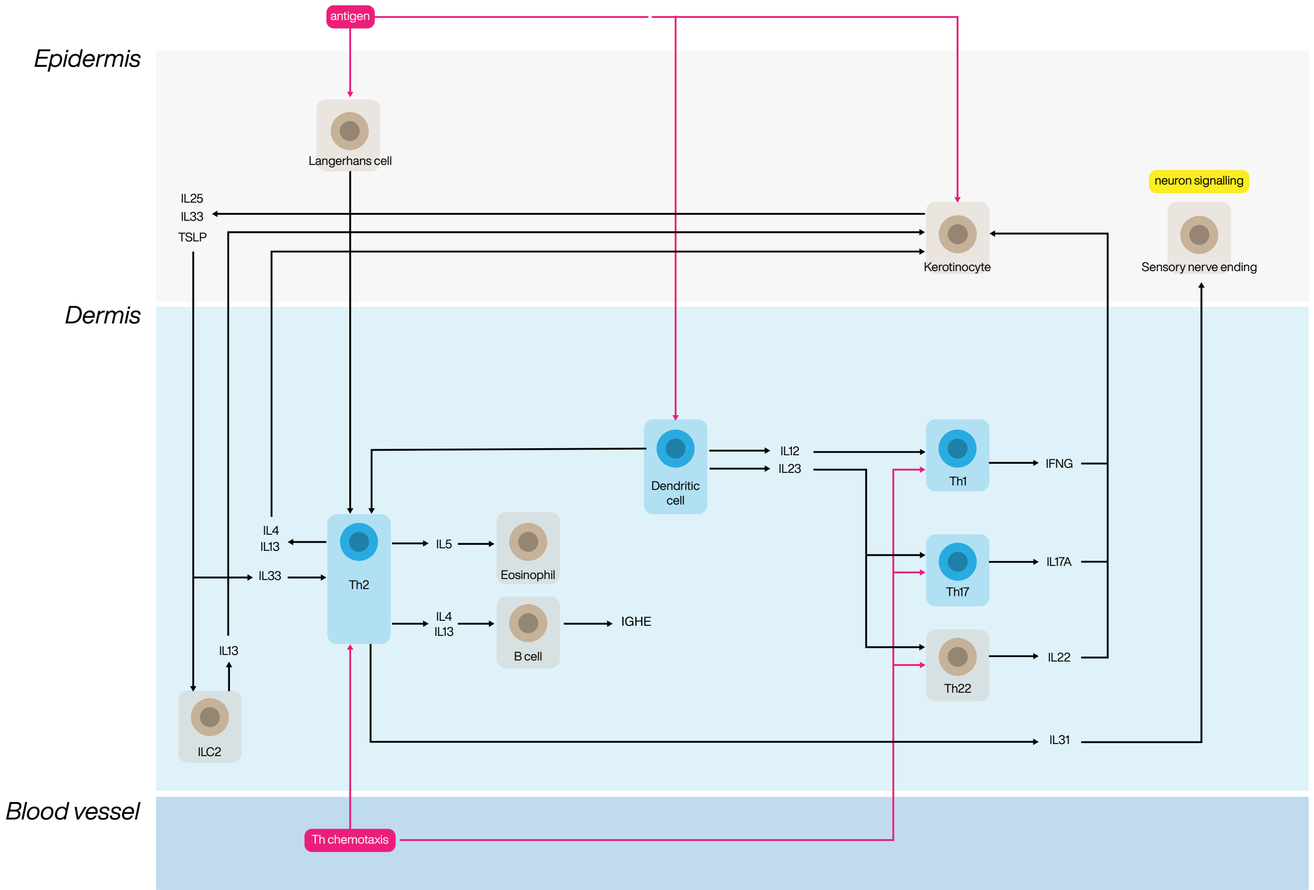
ATOPIC DERMATITIS



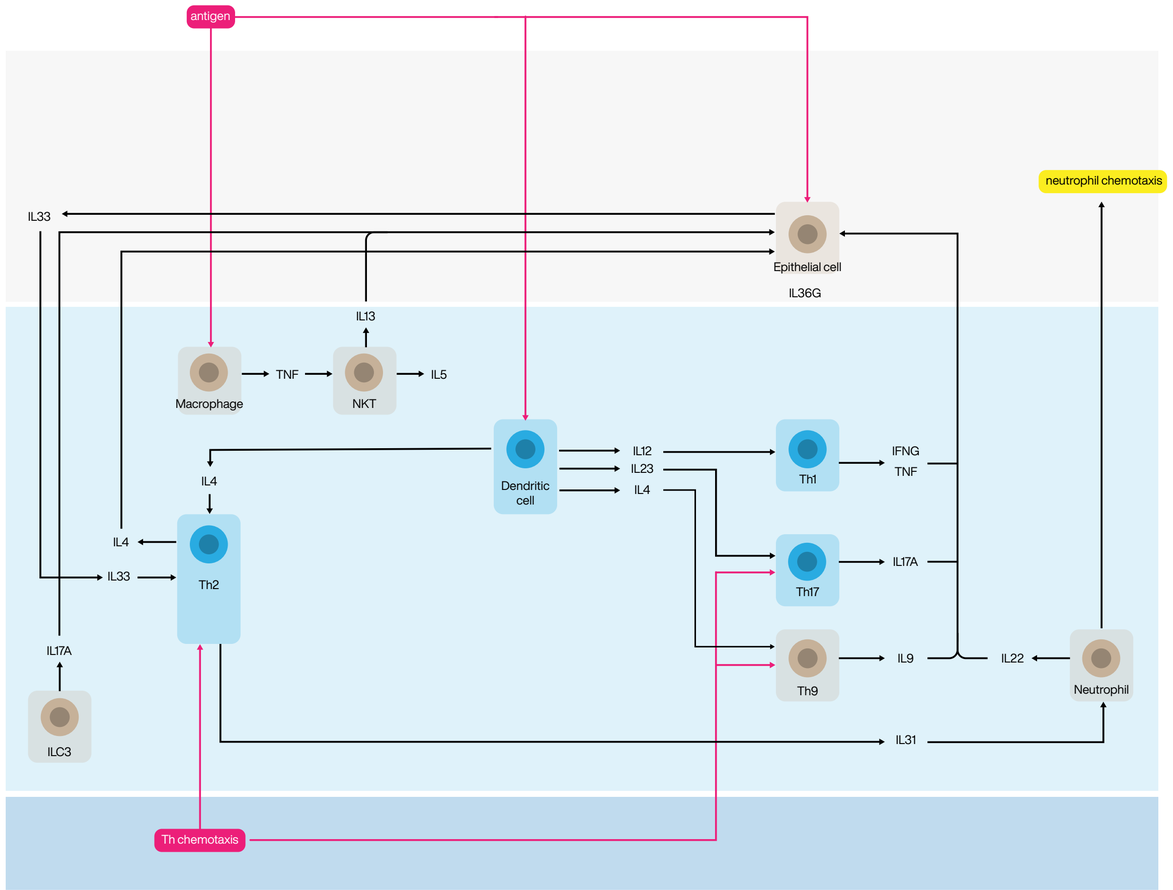
ULCERATIVE COLITIS



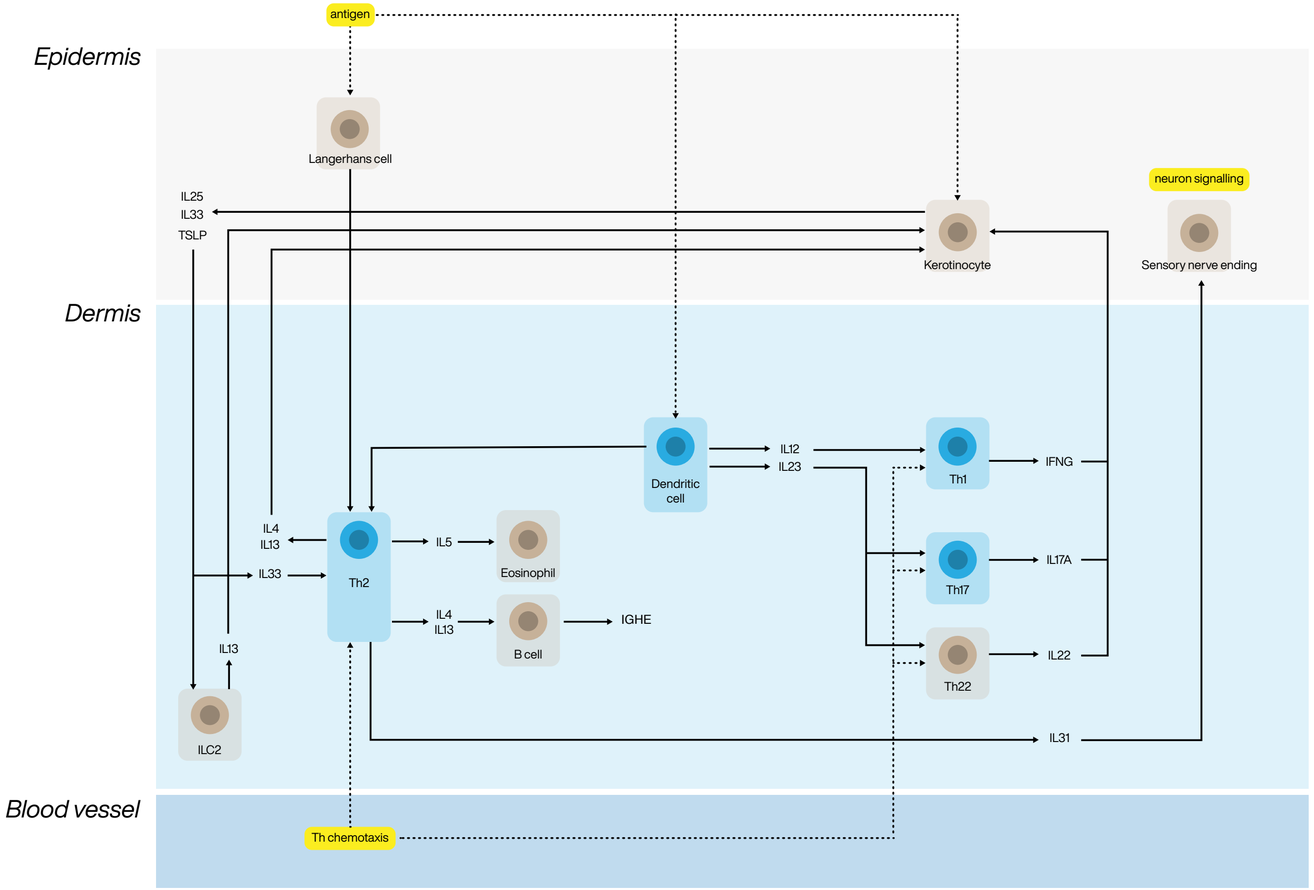
ATOPIC DERMATITIS



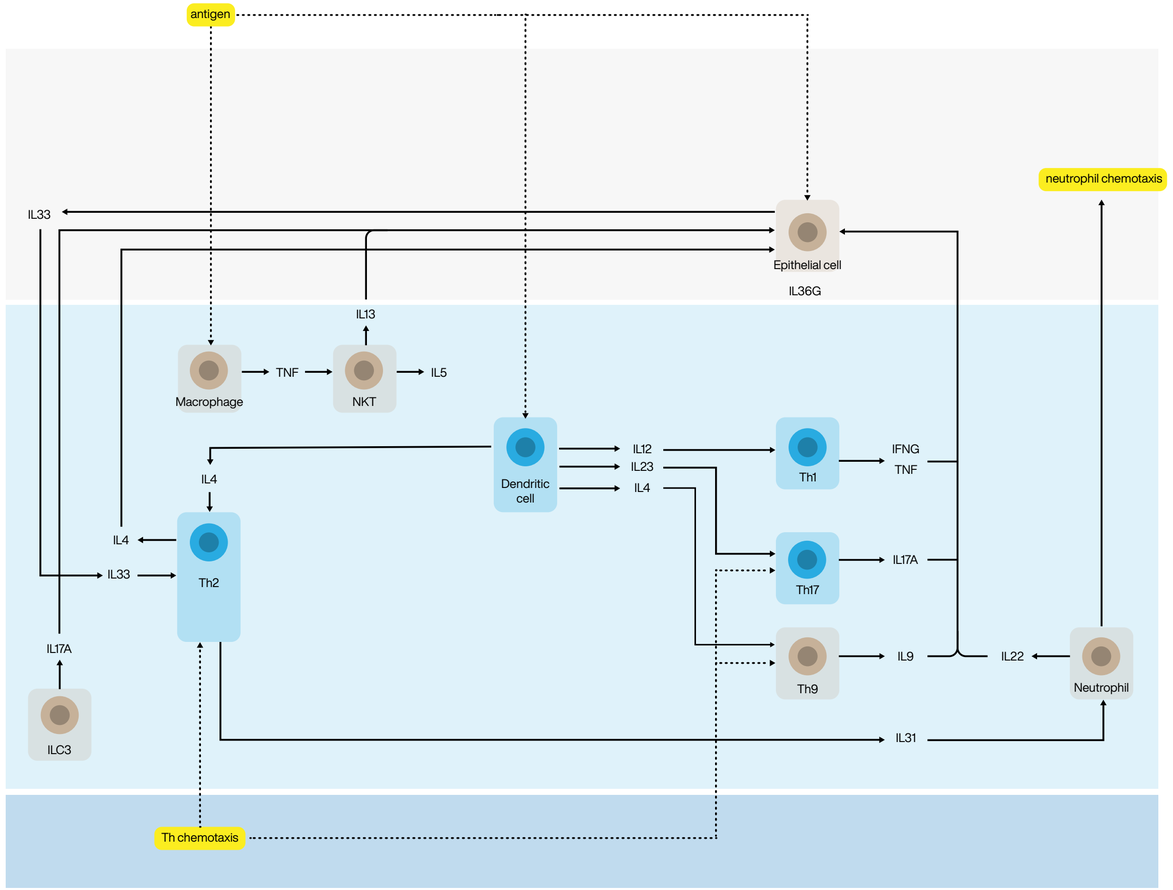
ULCERATIVE COLITIS



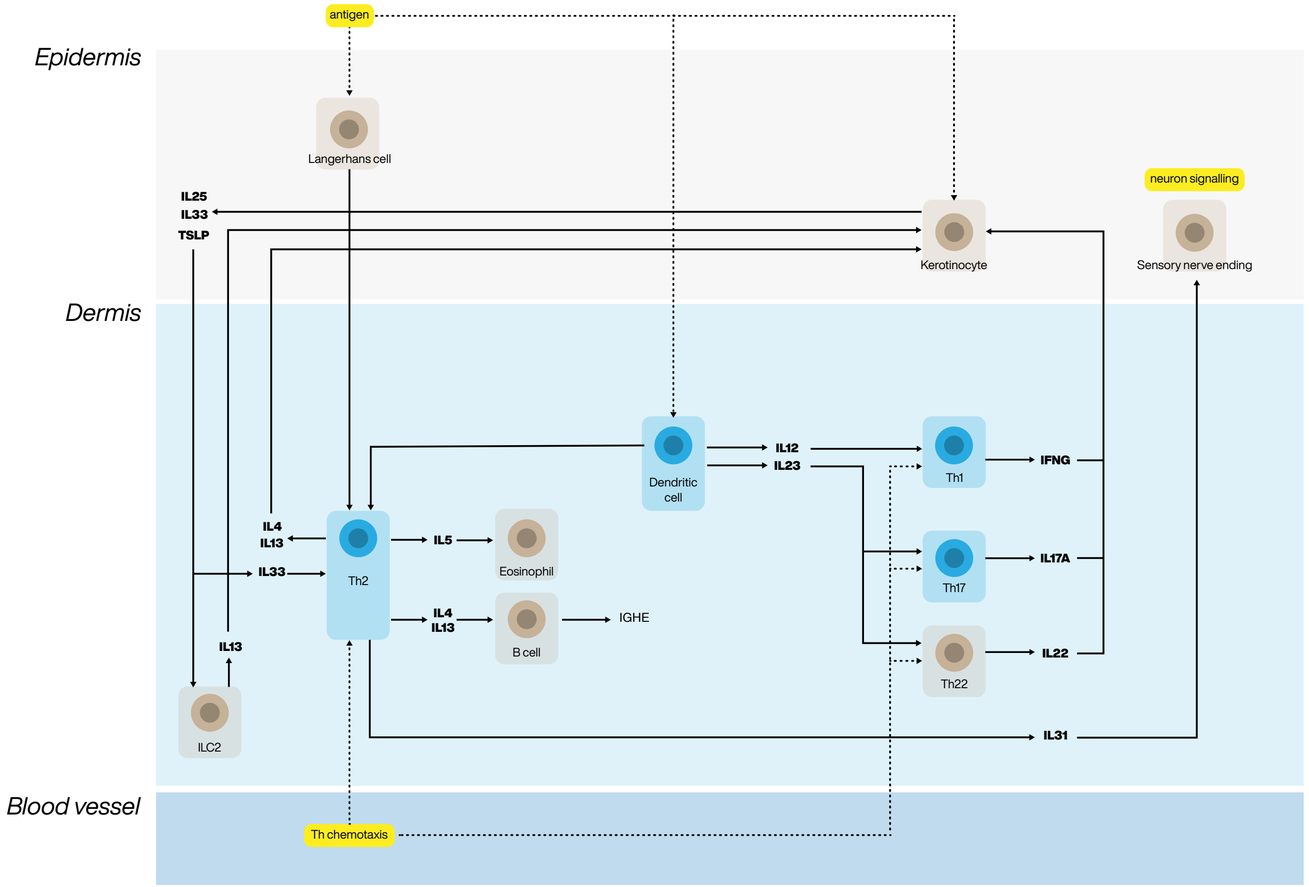
ATOPIC DERMATITIS



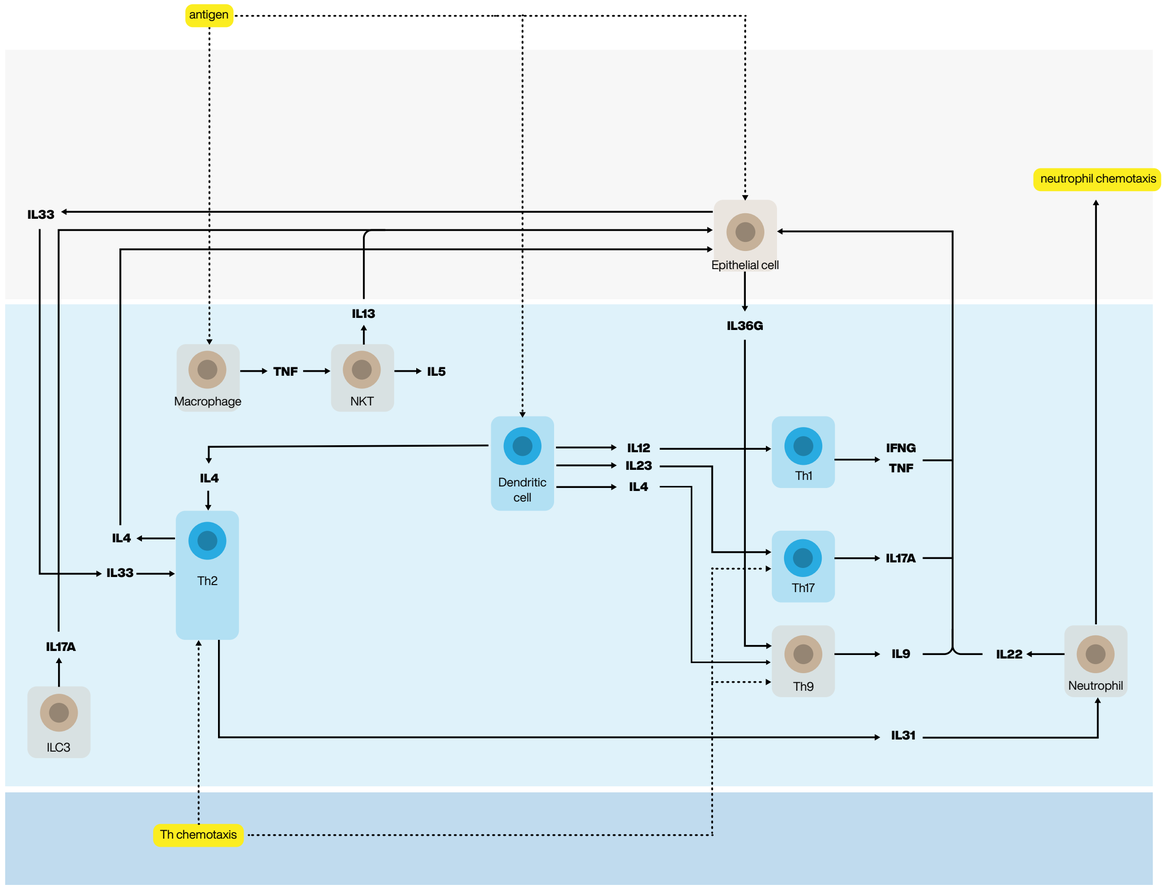
ULCERATIVE COLITIS

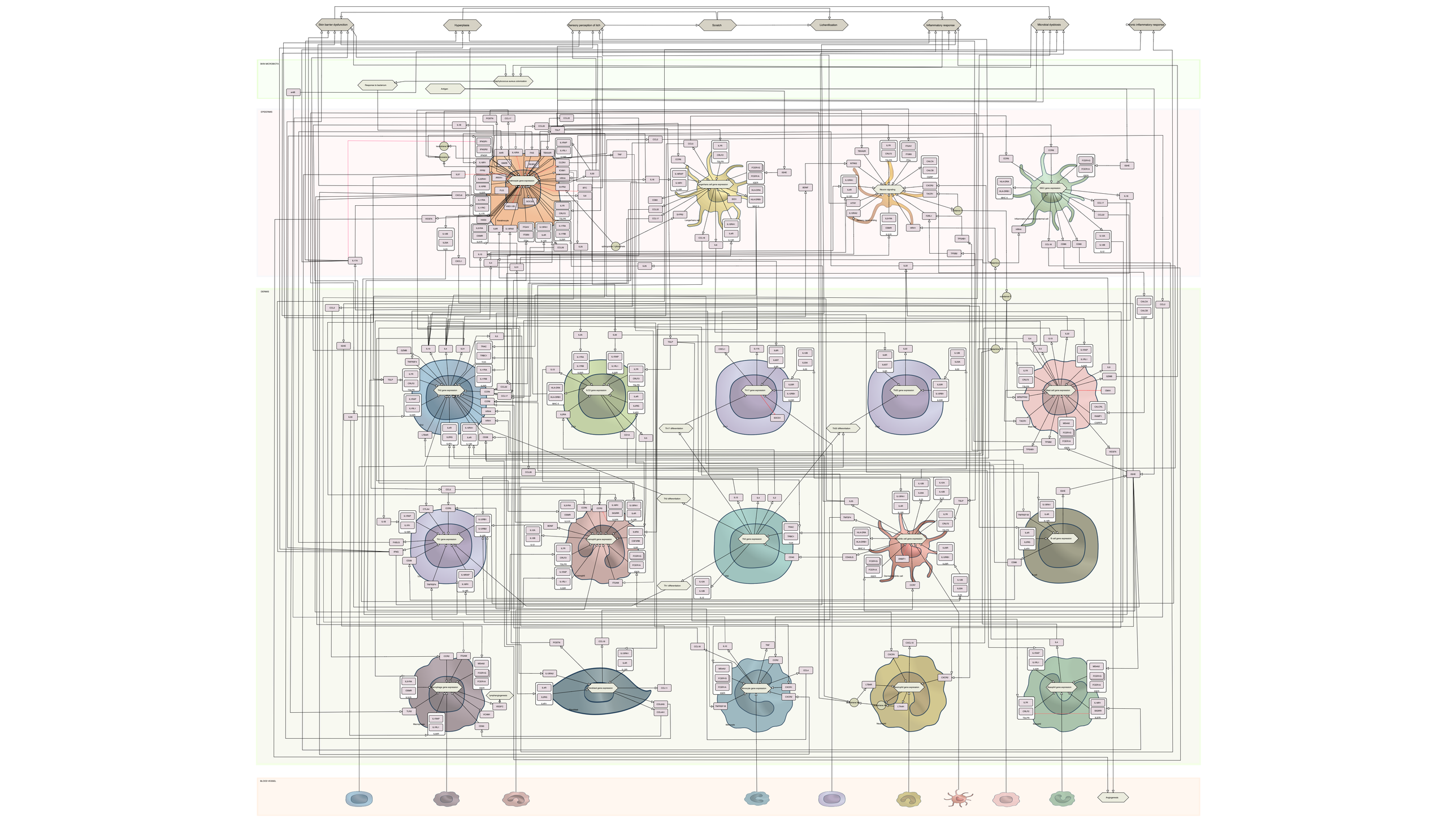


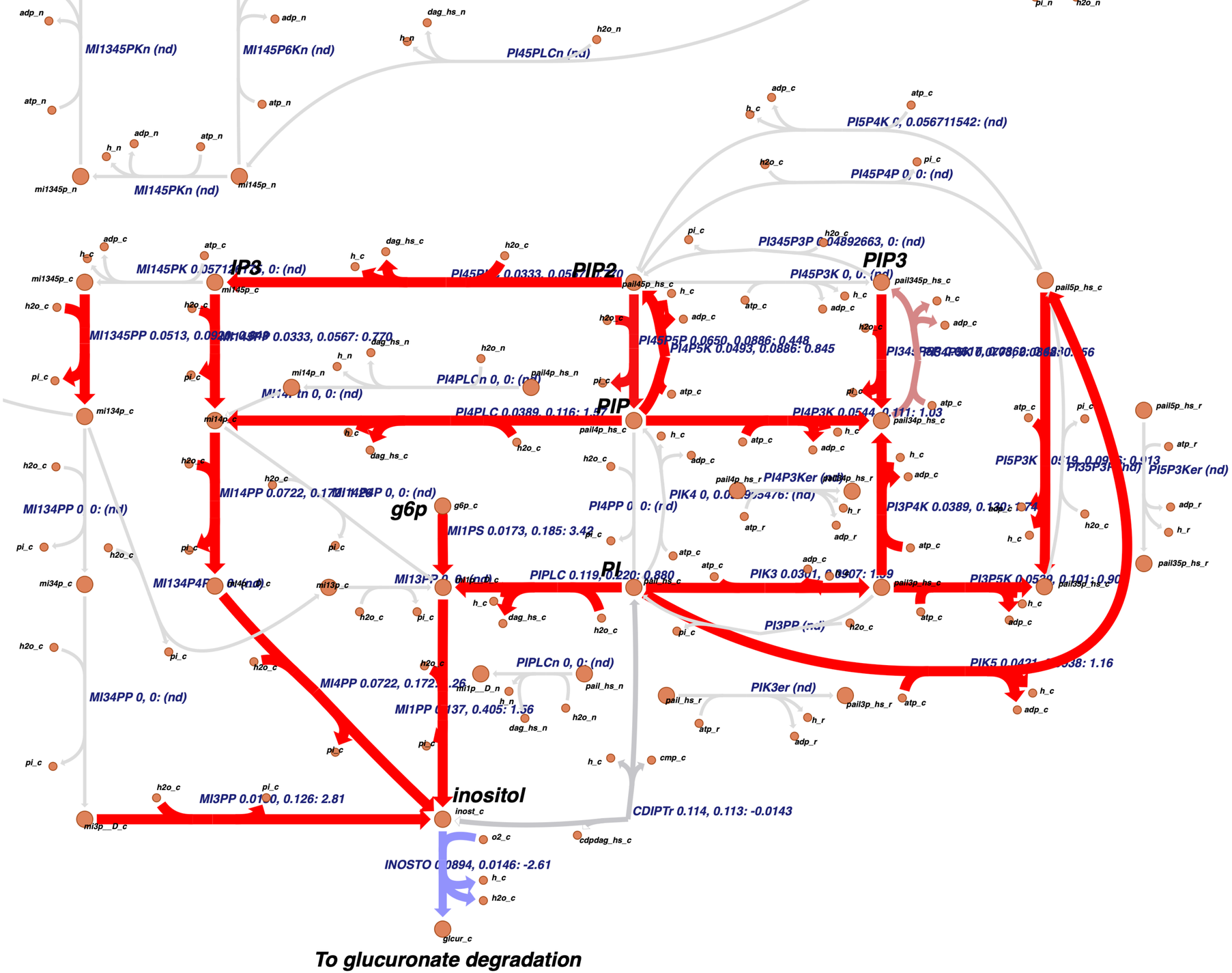
ATOPIC DERMATITIS



ULCERATIVE COLITIS




Skin barrier dysfunction
Hyperplasia
Sensory perception of itch
Scratch
Lichenification
Inflammatory response
Microbial dysbiosis
Chronic inflammatory response
SKIN MICROBIOTA
EPIDERMIS
DERMIS
BLOOD VESSEL
Response to bacterium
Antigen
Staphylococcus aureus colonization
Angiogenesis

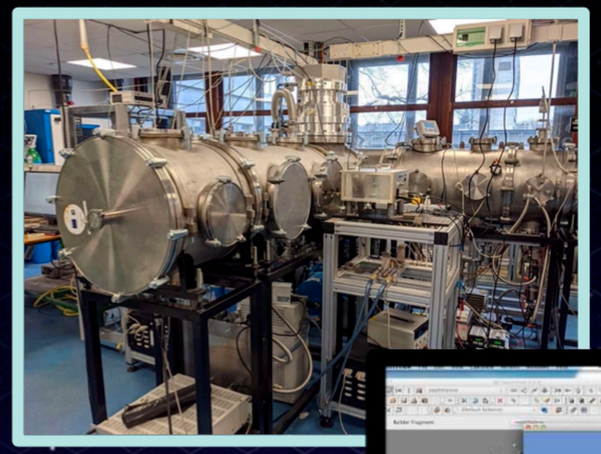

MI1345PKn (nd)
MI145P6Kn (nd)
PI45PLCn (nd)
MI145PKn (nd)
PI5P4K 0, 0.056711542: (nd)
PI45P4P 0, 0: (nd)
PI345P3P 0.04892663, 0: (nd)
PI45P3K 0, 0: (nd)
IP3
PIP2
PIP3
PIP
PI
g6p
inositol
MI145PK 0.05712[illegible], 0: (nd)
MI1345PP 0.0513, 0.0[illegible]
MI145PP 0.0333, 0.0567: 0.770
PI45P5P 0.0650, 0.0886: 0.448
PI4P5K 0.0493, 0.0886: 0.845
PI4PLCn 0, 0: (nd)
MI14[illegible]tn 0, 0: (nd)
PI4PLC 0.0389, 0.116: 1.47
PI4P3K 0.0544, 0.111: 1.03
PI4P3Ker (nd)
PI5P3K 0.0519, 0.09[illegible]: 0.913
PI35P3Pn (nd)
PI5P3Ker (nd)
MI14PP 0.0722, 0.172[illegible]
MI134PP 0, 0: (nd)
MI134PP 0, 0: (nd)
PI4PP 0, 0: (nd)
PIK4 0, 0.0[illegible]: (nd)
PI3P4K 0.0389, 0.130: 1.74
MI1PS 0.0173, 0.185: 3.42
MI134P4P 0, 0: (nd)
MI13P2Pn 0, 0: (nd)
PIPLC 0.119, 0.220: 0.880
PIK3 0.0301, 0.0907: 1.99
PI3P5K 0.0529, 0.101: 0.90
PI3PP (nd)
PIK5 0.0421, [illegible]038: 1.16
PIPLCn 0, 0: (nd)
MI4PP 0.0722, 0.172[illegible].26
MI1PP [illegible]137, 0.405: 1.56
PIK3er (nd)
MI34PP 0, 0: (nd)
MI3PP 0.0[illegible]0, 0.126: 2.81
CDIPTr 0.114, 0.113: -0.0143
INOSTO [illegible]0894, 0.0146: -2.61
To glucuronate degradation

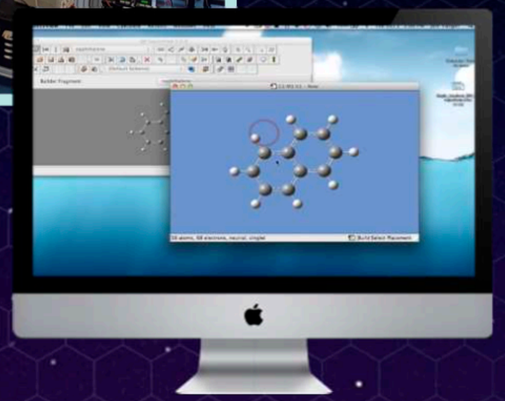
delight the eye — meaningfully

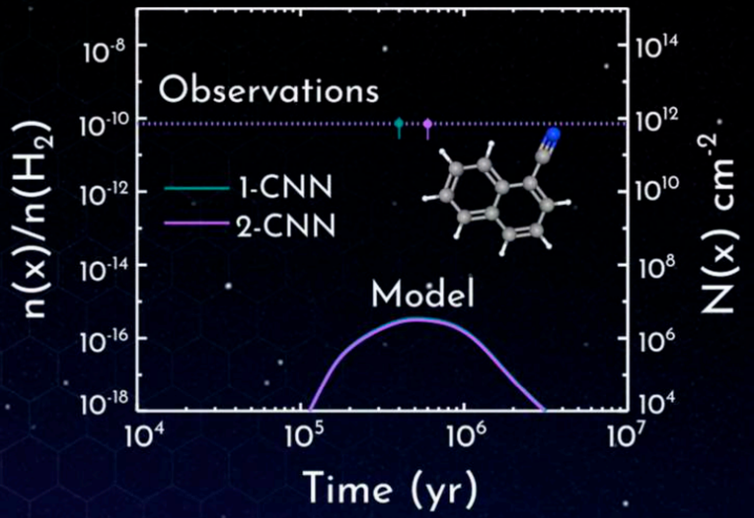
find a way to be playful without shouting

Astrochemistry goals: Detect new molecules in interstellar space and explain how they form

Radio Observations:
Detect new molecules
(>200 detected so far!)







Chemical kinetics modelling:

How, When and Where are these molecules formed?

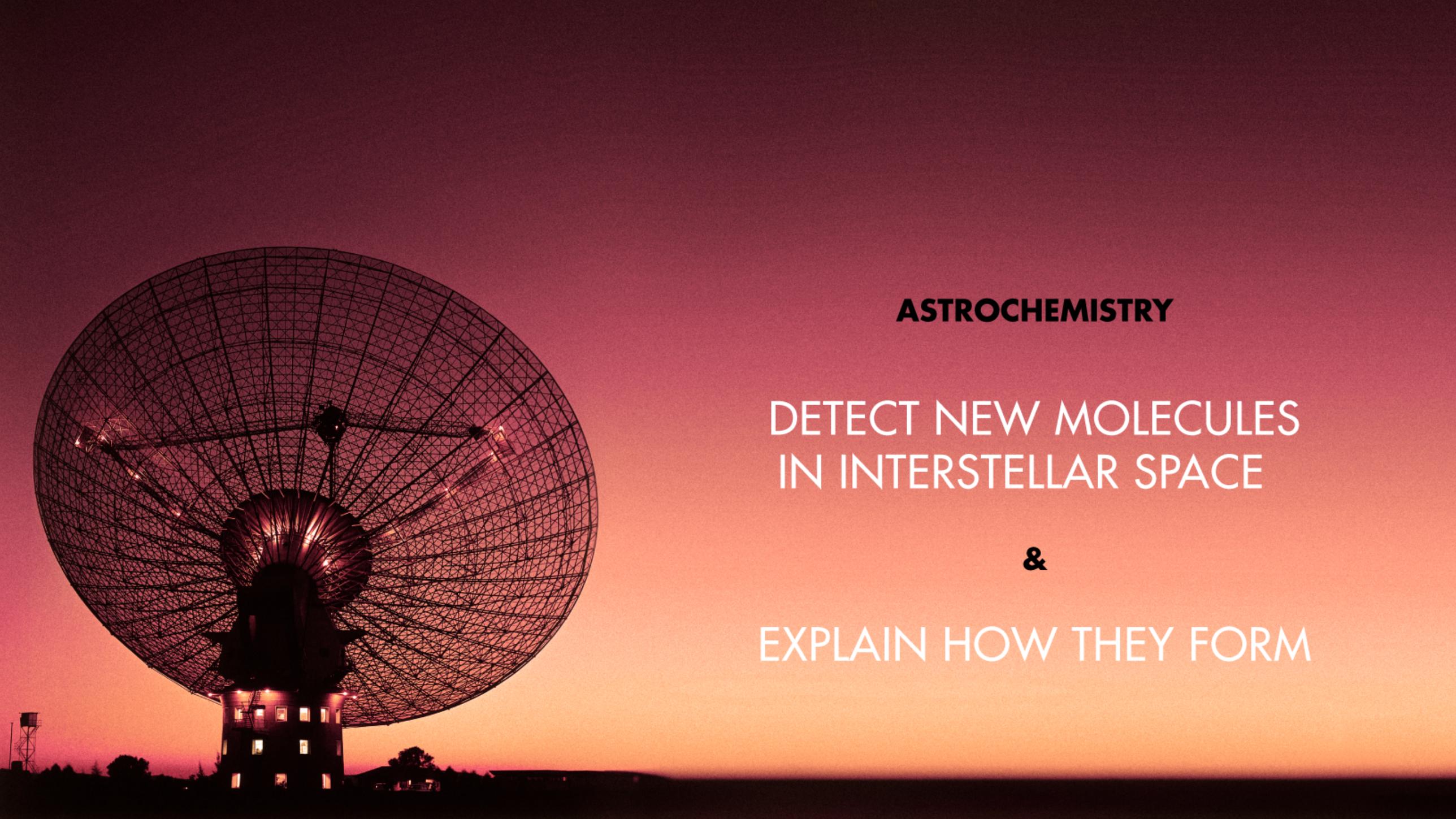
Experiments:
- *Spectroscopy*
- Kinetic parameters
- Reaction dynamics/mechanisms

Image: David Fernadez

The 64-meter radio telescope at Parkes Observatory as seen in 1969, when it was used to receive live televised video from Apollo 11.

This photo, taken in 1969, shows the telescope as it was around the time of the first manned Moon landing. CSIRO's Parkes radio telescope was officially opened on the 31 October 1961 by the Governor-General, Viscount De L'Isle forty years on, it is still one of the most advanced telescopes of its kind. A gigantic structure of steel and concrete, the telescope soars nearly 55 metres into the sky near Parkes NSW. It played a crucial role in receiving signals during the Apollo 11 Moon landing in 1969, relaying them for broadcast to an audience of 600 million around the world.

https://en.wikipedia.org/wiki/Radio_telescope



ASTROCHEMISTRY

DETECT NEW MOLECULES
IN INTERSTELLAR SPACE

&

EXPLAIN HOW THEY FORM

Implications of digestive dynamics of protein and starch for chicken-meat production in the future

Sonia Y Liu and Peter H Selle

Poultry Research Foundation,
Camden, NSW

THE UNIVERSITY OF SYDNEY

THE UNIVERSITY OF SYDNEY

PROTEIN DIGESTION RATES IMPACT FEED CONVERSION EFFICIENCY

Implications of digestive dynamics of protein and starch for chicken-meat production in the future

Sonia Y Liu
Peter H Selle

Poultry Research Foundation
Camden NSW

Phylogenomics of Land snails

Luisa Teasdale

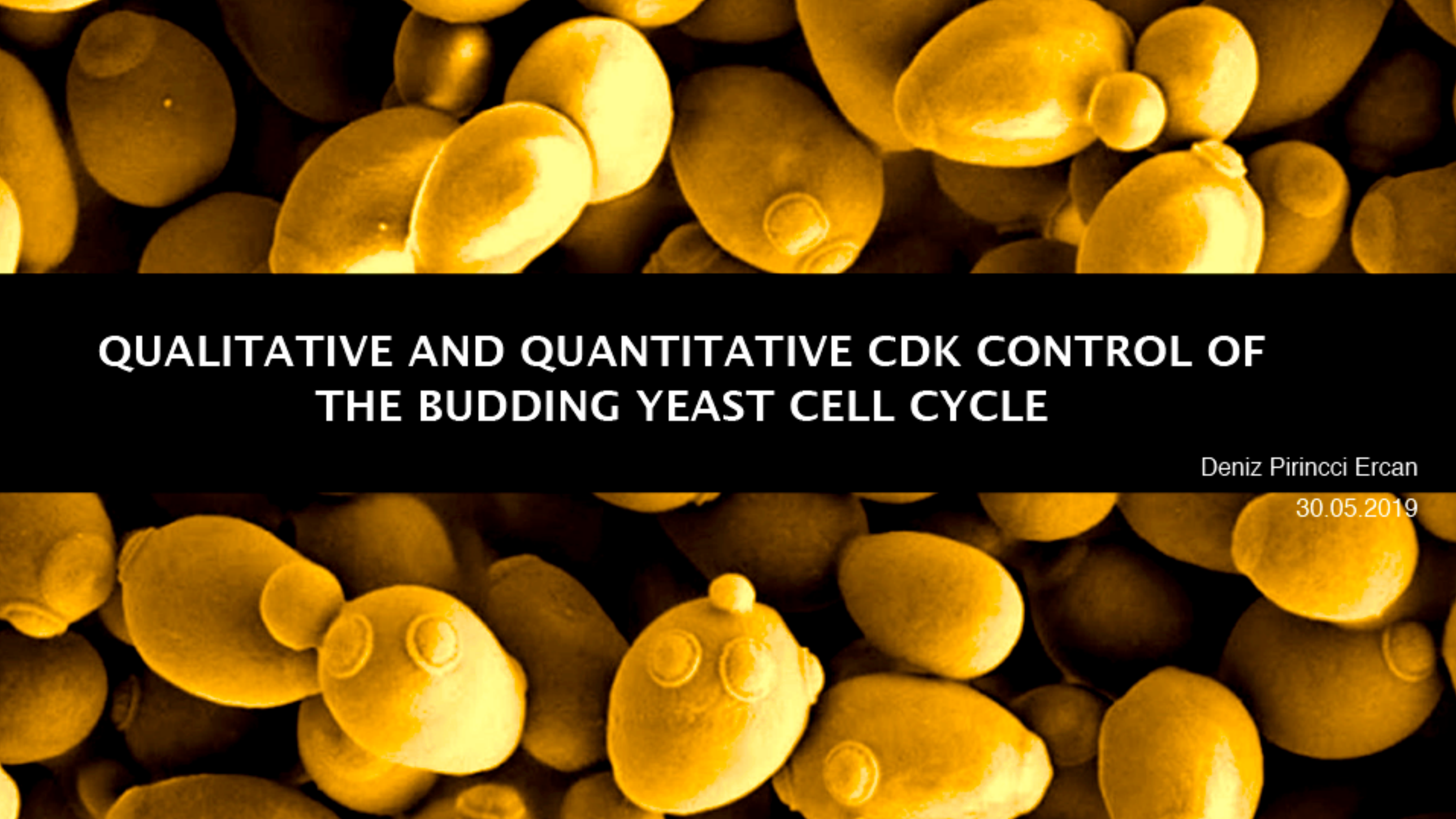
Museum Victoria and

The University of Melbourne

PHYLOGENOMICS OF LAND SNAILS

The role of cyclin-Cdk complexes in the budding yeast cell cycle

Deniz Pirincci Ercan



QUALITATIVE AND QUANTITATIVE CDK CONTROL OF THE BUDDING YEAST CELL CYCLE

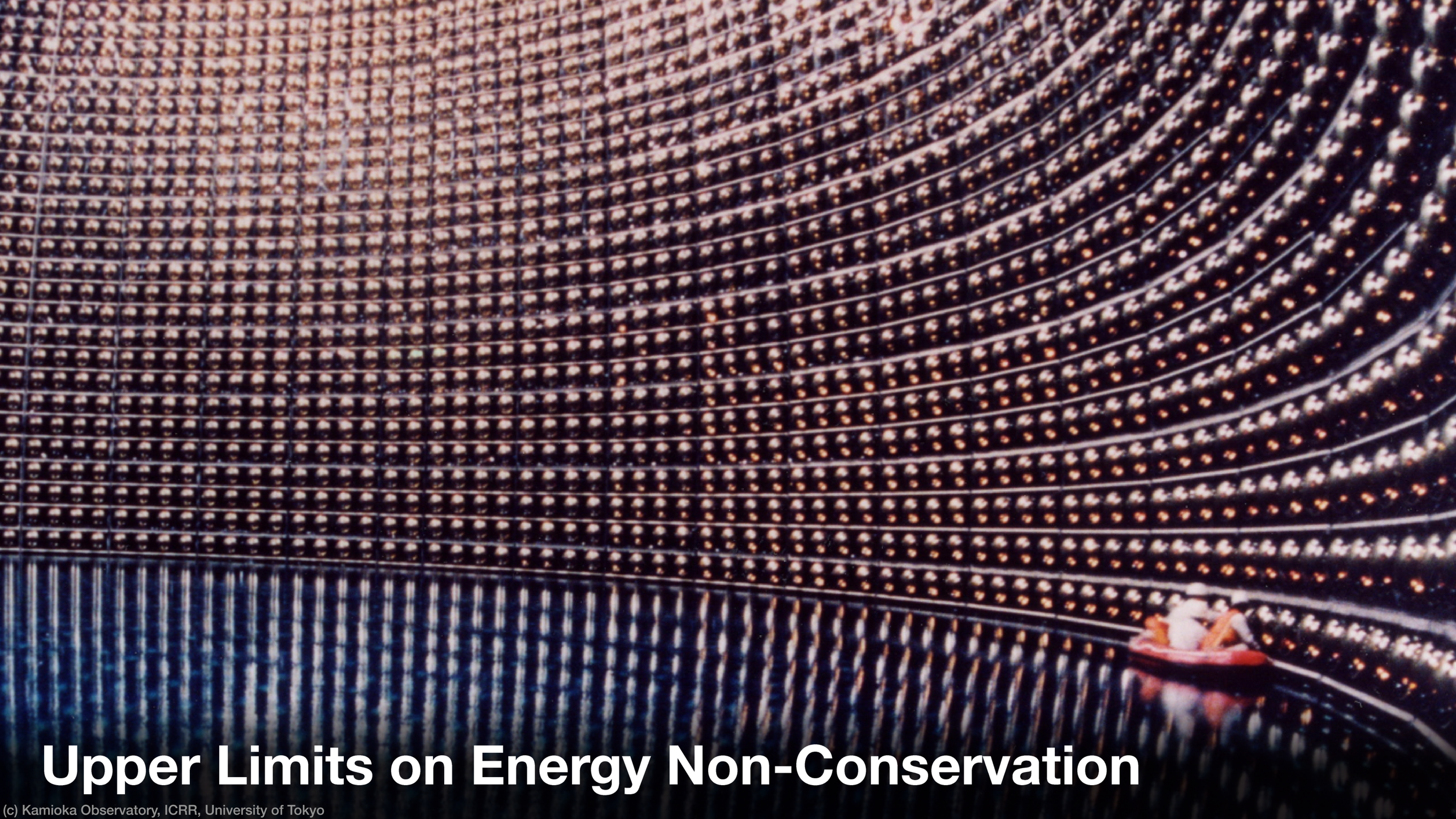
Deniz Pirincci Ercan

30.05.2019

Upper Limits on Energy Non-Conservation

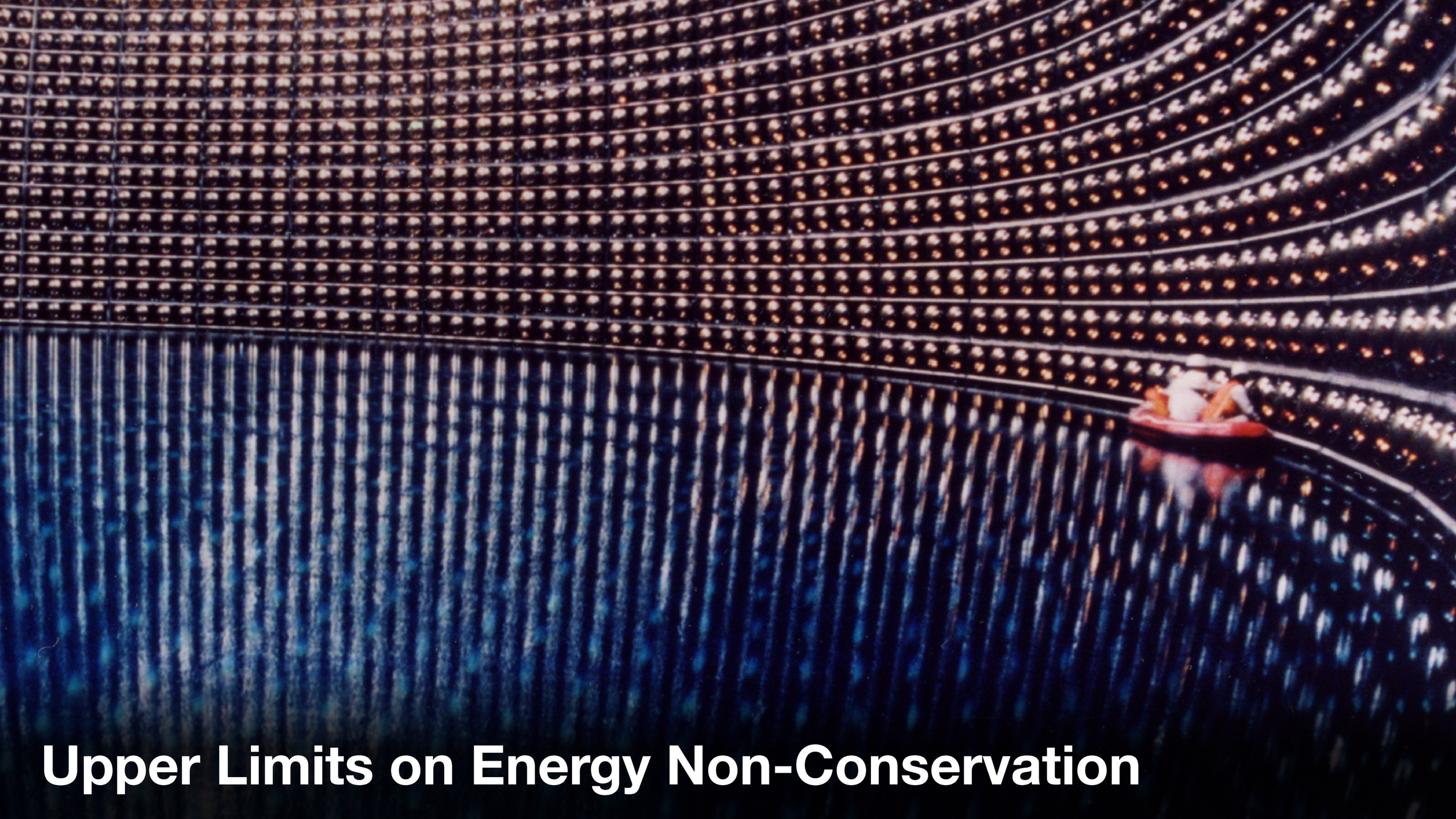
Peter Moen

(c) Kamioka Observatory, ICRR(Institute for Cosmic Ray Research), The University of Tokyo



Upper Limits on Energy Non-Conservation

(c) Kamioka Observatory, ICRR, University of Tokyo



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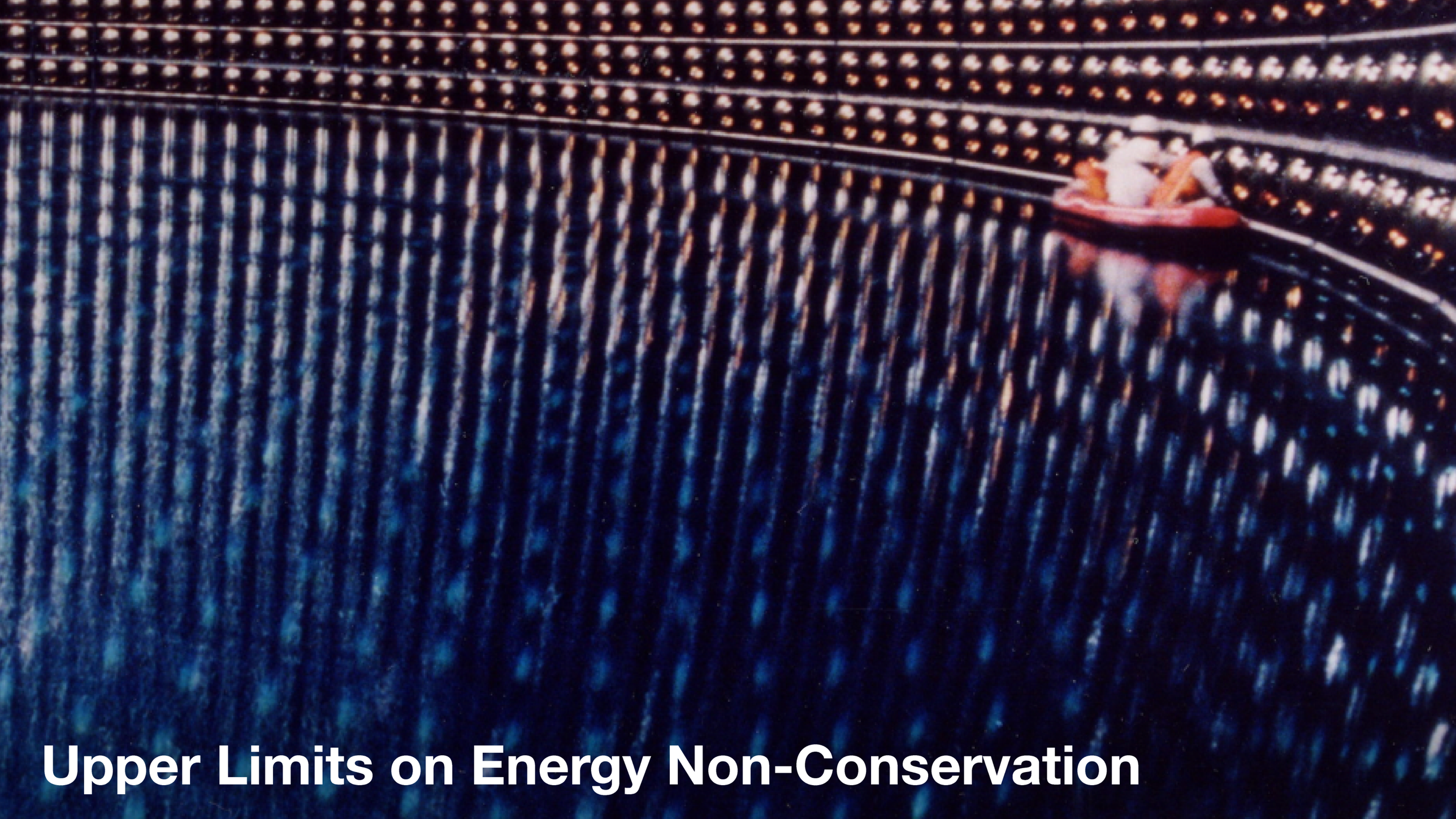
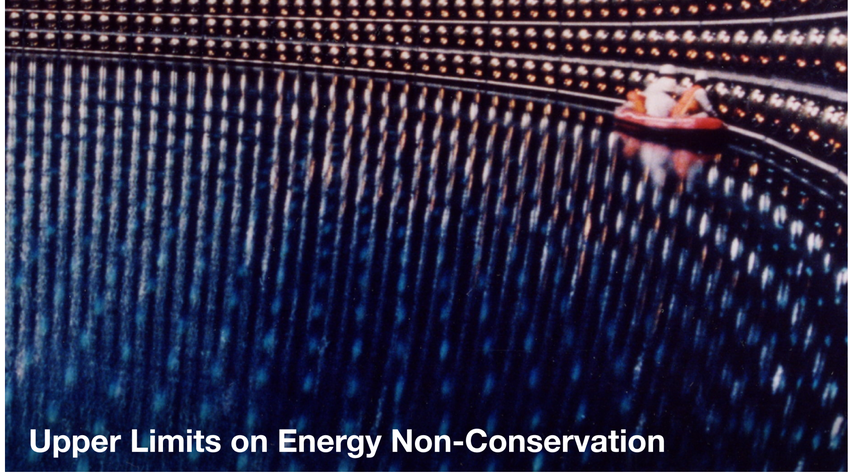
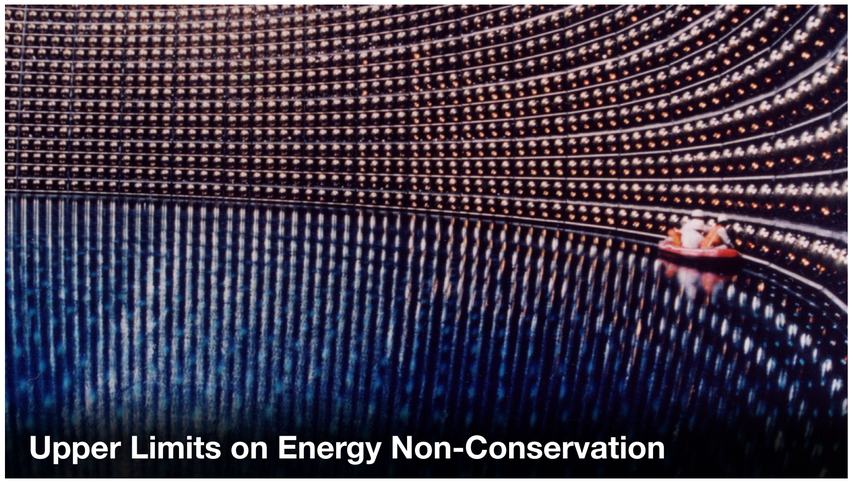
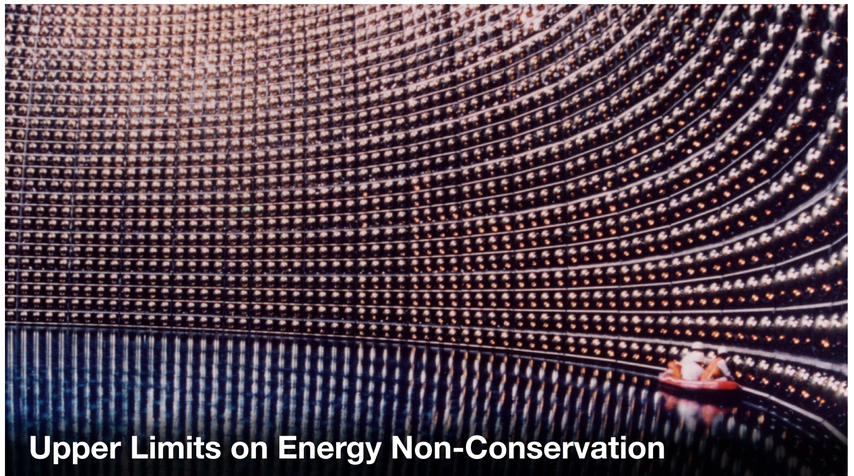


Upper Limits on Energy Non-Conservation







(c) Kamioka Observatory, ICRR, University of Tokyo

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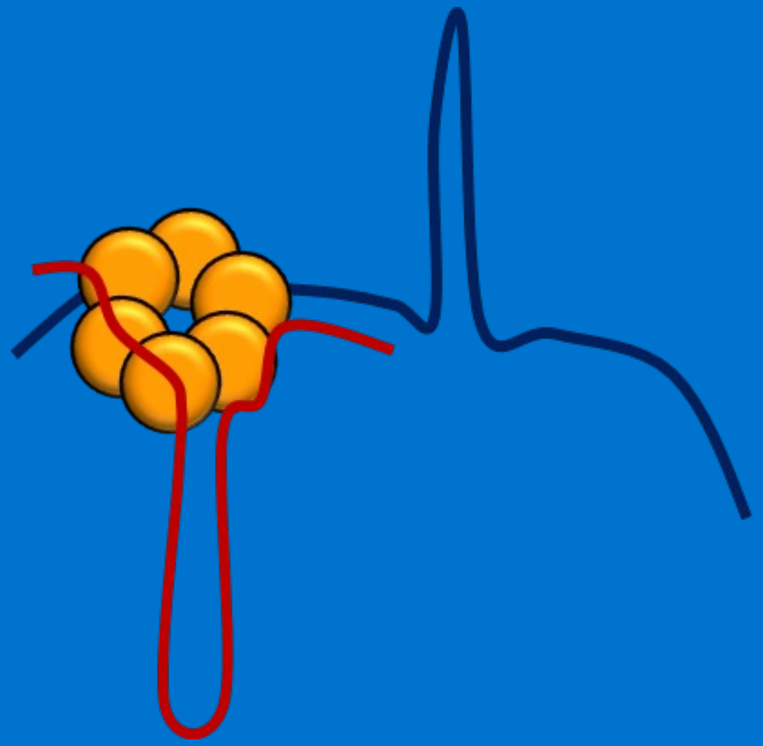
Mechanisms of non-coding RNA mediated regulation in *Escherichia coli*



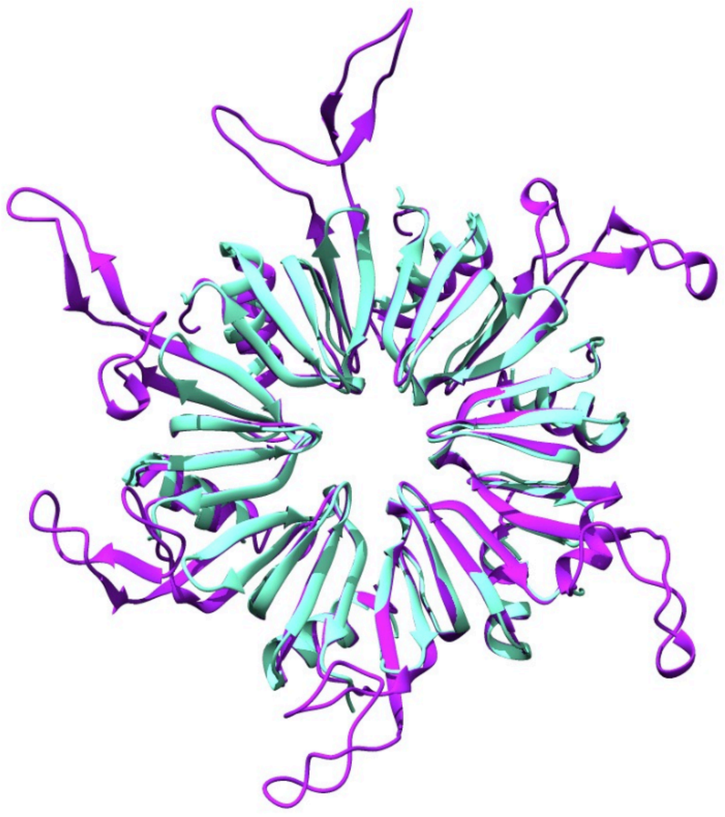
PhD in Biochemistry

Kai Katsuya Gaviria

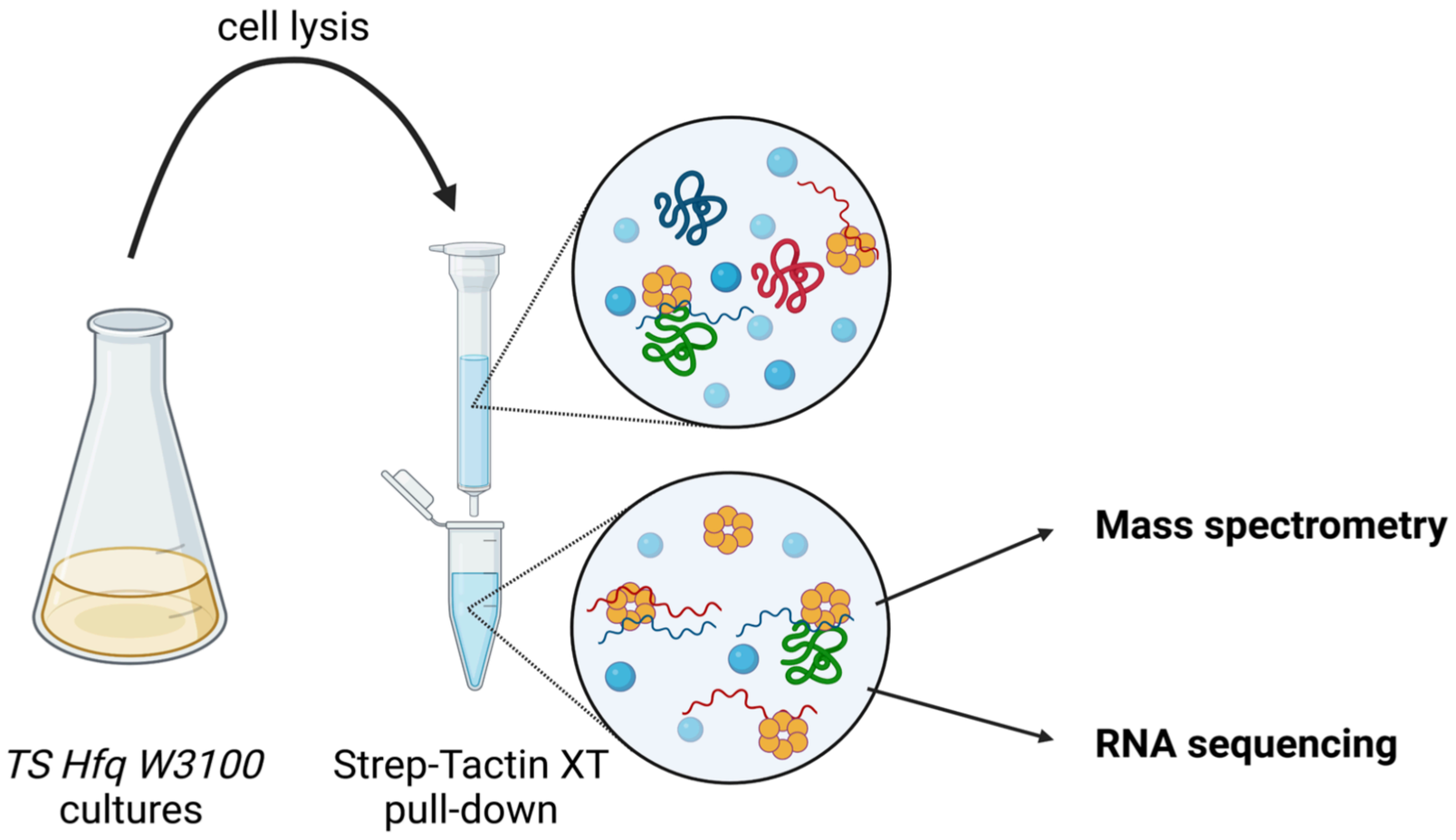
Prof Ben Luisi's group

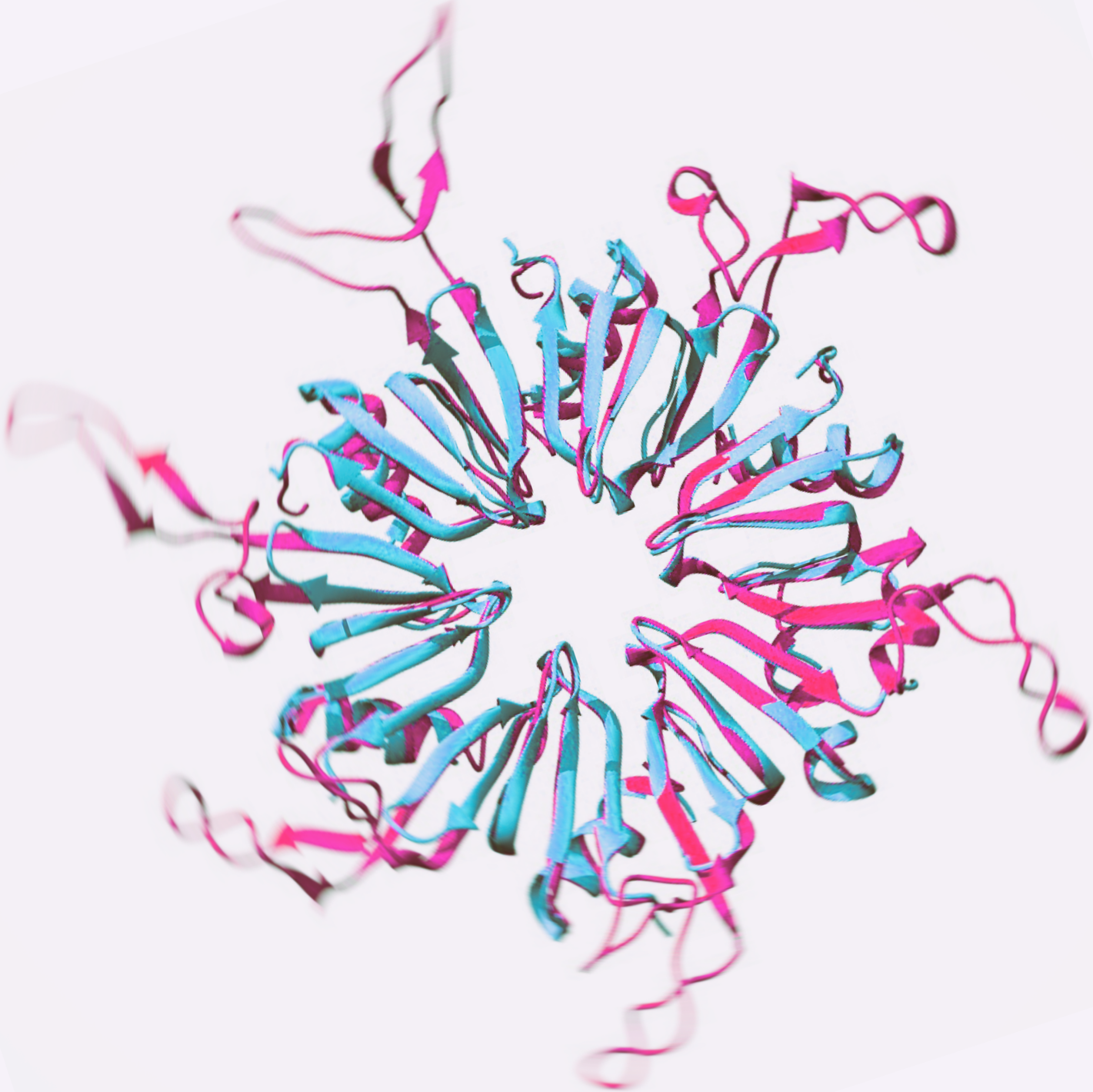
Department of Biochemistry – University of Cambridge

Boehringer Ingelheim Fonds – PhD fellowships



Wt Hfq
TwinStrep Hfq





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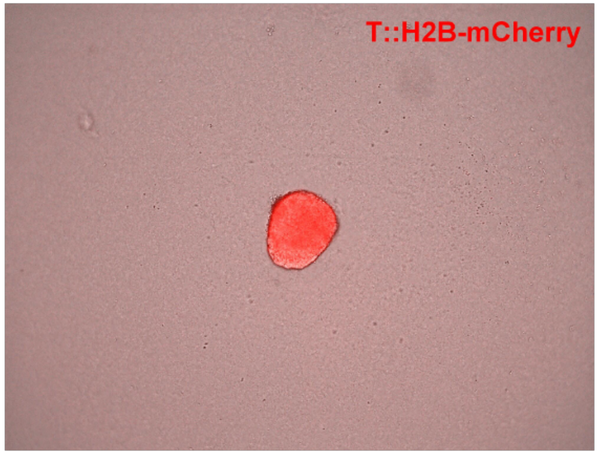
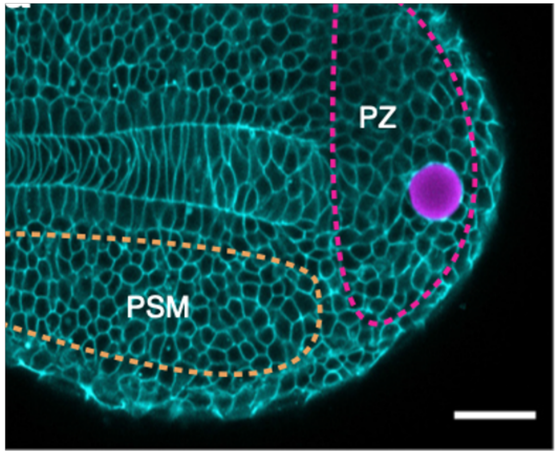
Understanding the mechanical basis of mammalian body axis formation and elongation with self-organized embryo-like structures



Marc Trani Bustos

Jesse Veenvliet Lab

Otger Campàs Lab



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CBG
Max Planck Institute
of Molecular Cell Biology
and Genetics

PoL
Physics of Life
TU Dresden

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DRESDEN

MECHANICAL BASIS OF

MAMMALIAN BODY AXIS FORMATION

MODELED BY SELF-ORGANIZED EMBRYO-LIKE STRUCTURES

MARC TRANI BUSTOS

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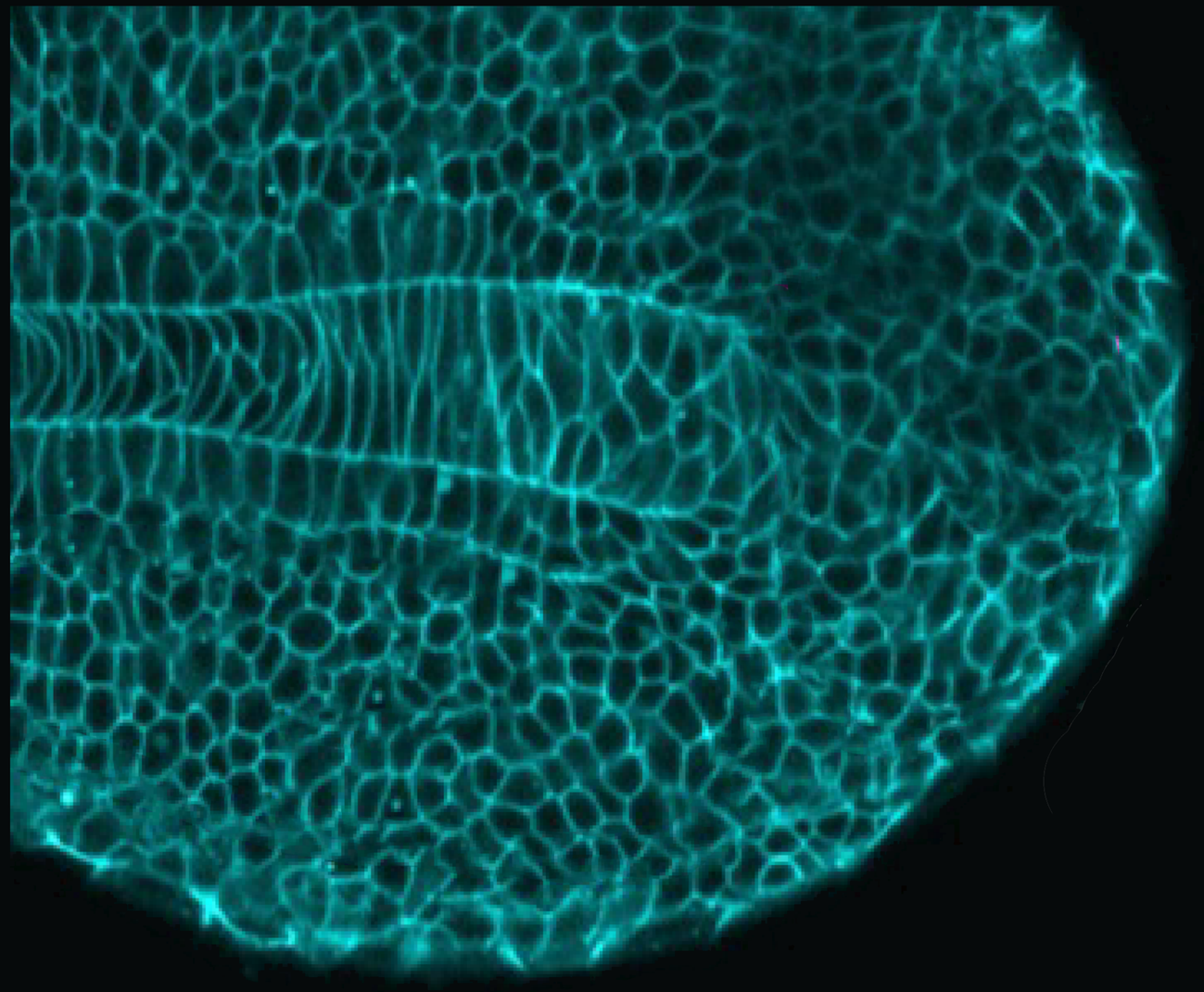

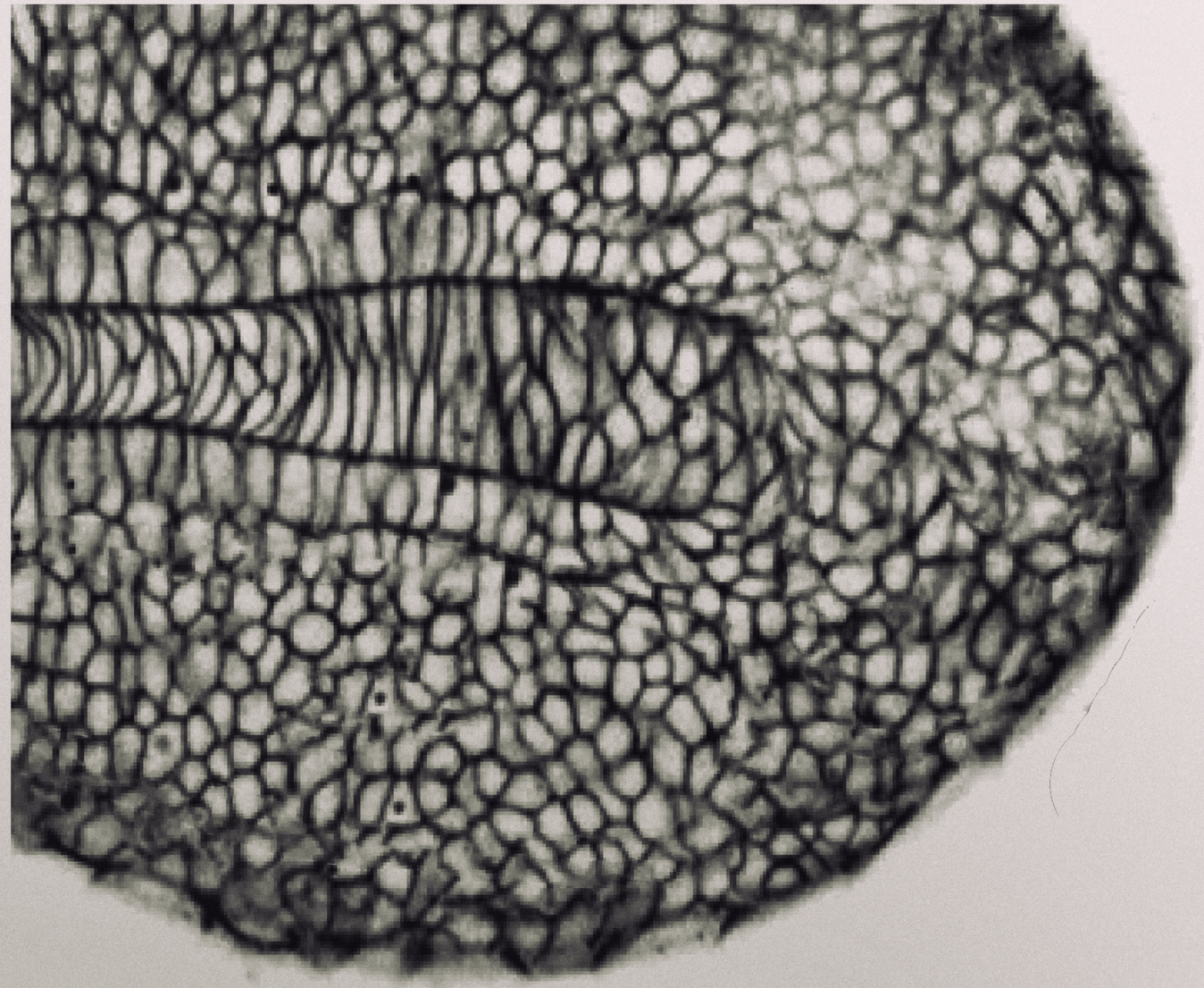

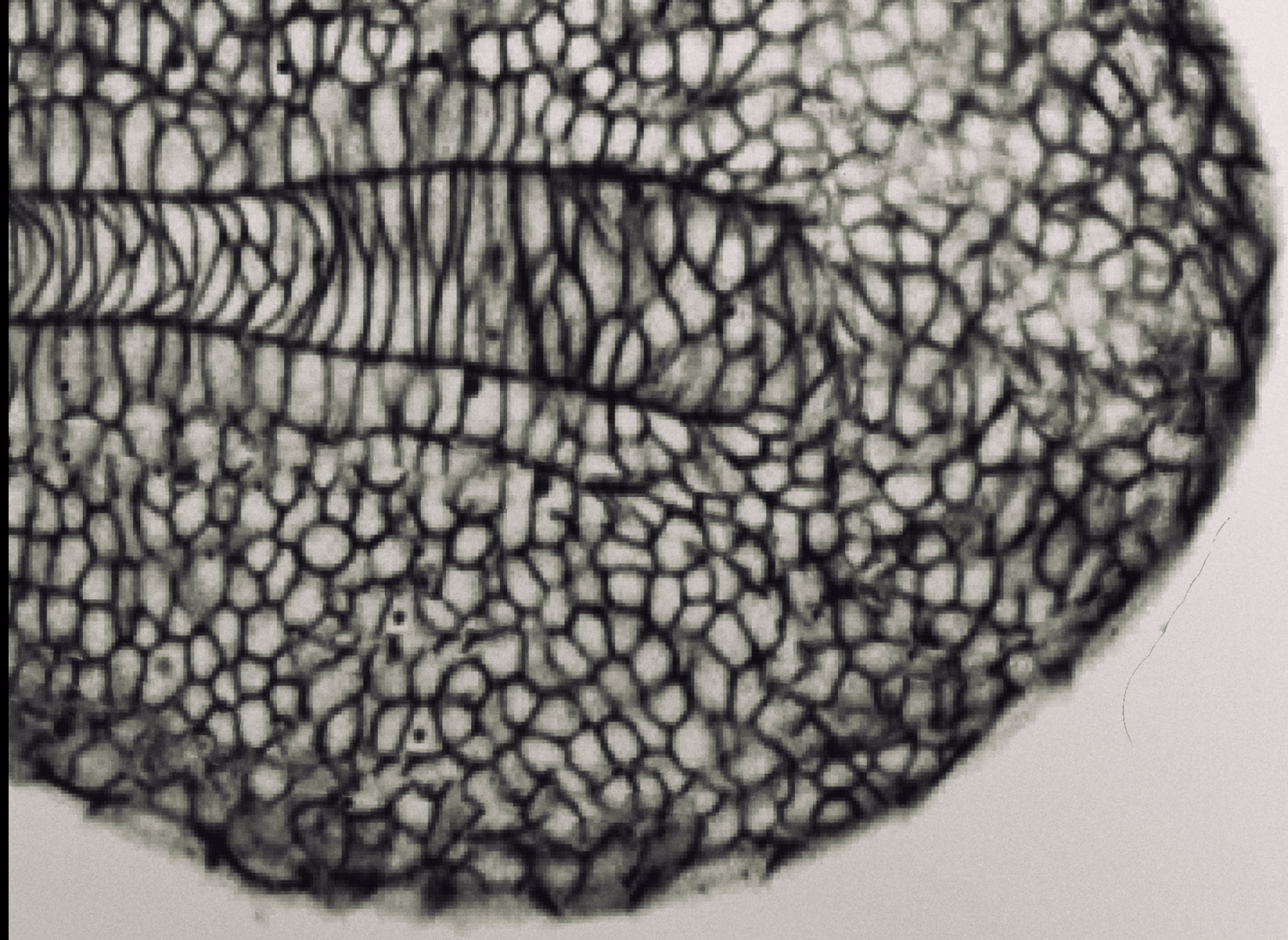

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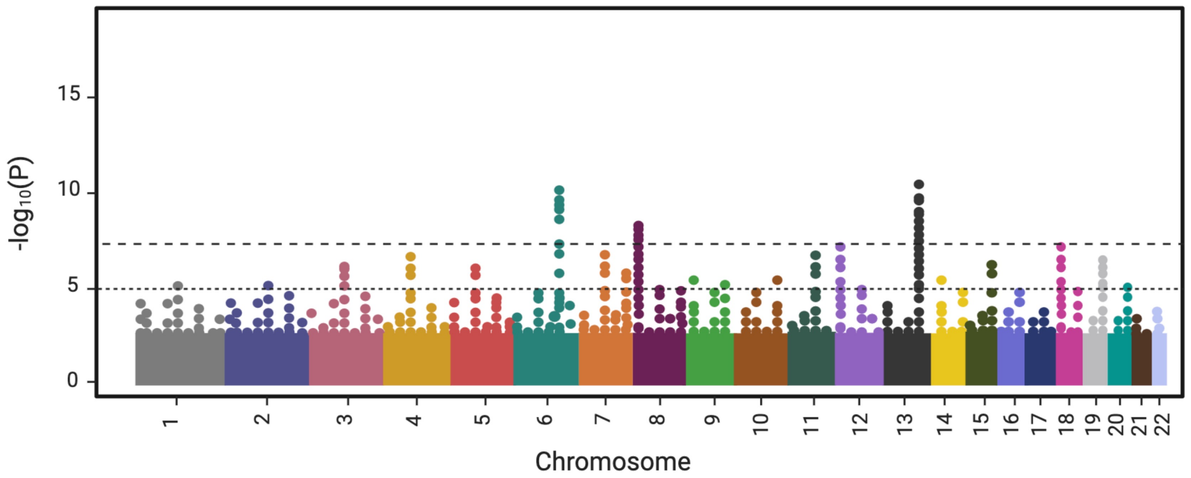
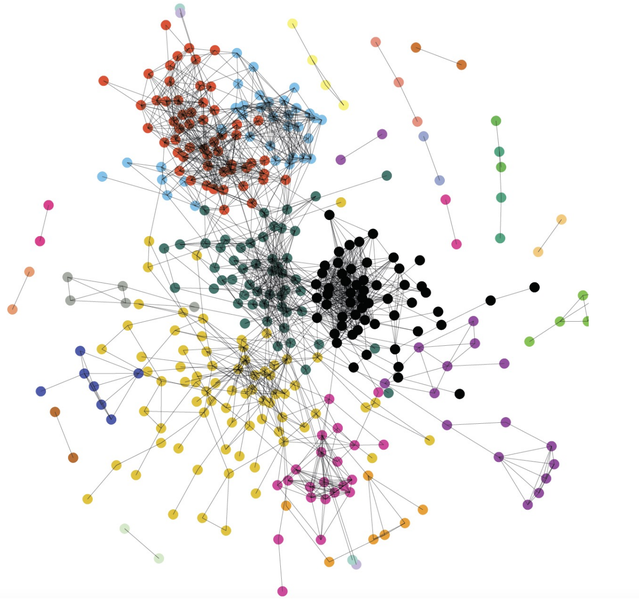
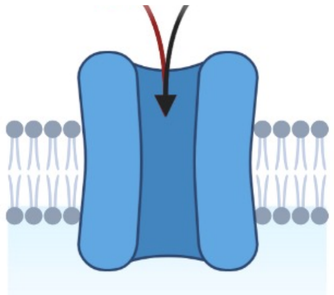
Systematic Discovery of Metabolic Gene Functions



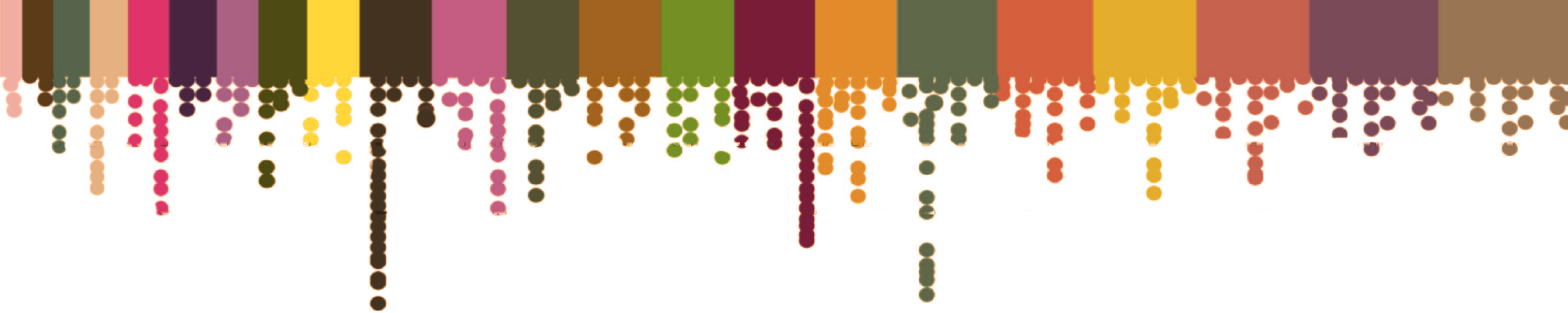




Artem Khan

Advisor: Dr. Kivanc Birsoy

October 13th, 2023



Systematic Discovery of Metabolic Gene Functions

Artem Khan

Dr. Kivanc Birsoy (advisor)
13 Oct 2023

The Rockefeller University 1901

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Dr. Kivanc Birsoy (advisor) — 13 Oct 2023

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Grundlagenforschung

Artem Khan

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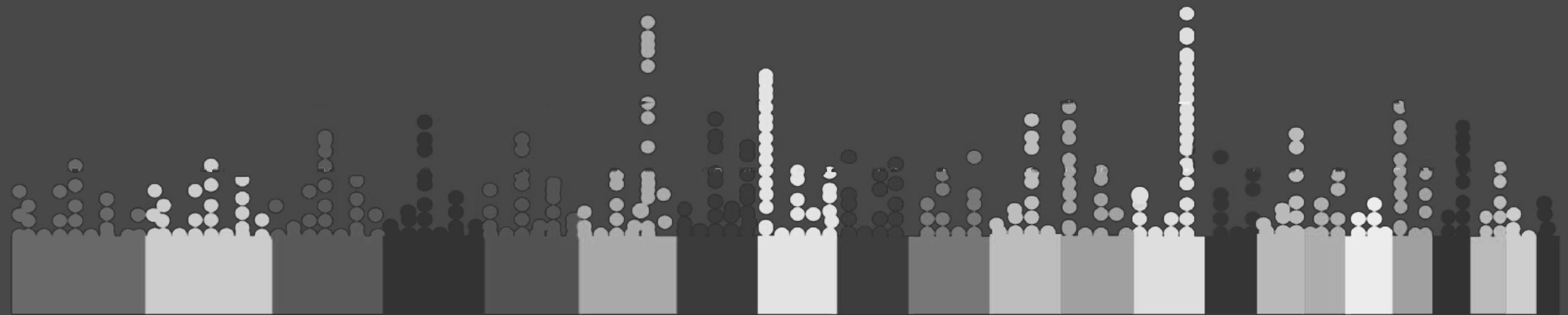

Gordon Andrews (designer)
Gazelle chair (c. 1950) designed, 1957 manufactured
plywood, aluminium, wool
74.0 x 48.0 x 55.0 cm
Museum of Applied Arts and Sciences, Sydney
Purchased, 1989 (89/499)