Large scale Event based model & Gene regulatory network models

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Last time:
CA as modeling tool
- generalizations of CA ss
- baseline expectations: "pattern default"

CA and ODE/MAP's as dynamical systems
- alternative simplifications
- common features (types of attractors etc.)
- Qualitative and quantitave different behaviors:
- Example birth death processes.
- Population based vs individual based
- Mean field approximation/assumption

Event based systems: Gillespie algorithm
- (average) cellcycle time and/vs growth rate

QUESTIONS?

TODAY

Event based systems cont.:
example: large scale "whole cell" modeling of translation

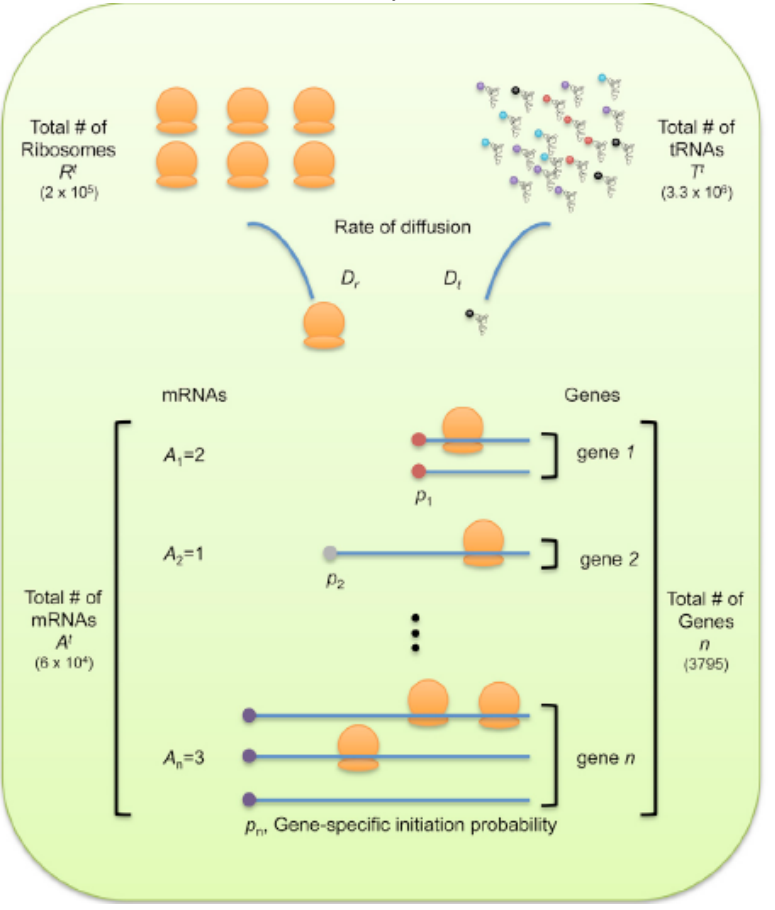
modeling in terms of subsystems (cont)
Network models
Boolean networks as model for gene regulatory networks:
- multiple attractors (= celltypes)
- domains of attraction, reachability, alternative transients.
- Understanding/interpreting gene knockouts
- inference of GRN from transcriptomics data
- minimal vs "real" networks
- non-network properties of GRN

Large scale, data driven EVENT based models: continuous time, discrete events Gillespie algorithm

seen as multi-entity - multistate decomposition

Example

Rate-Limiting Steps in
Yeast Protein Translation
P Shah, Y Ding, M Niemczyk,



Data, states, events

DATA

- fasta file of yeast mRNA + number of mol/cell
- yeast tRNA's (41) + number in cell + wobble
- number of ribosomes
- initiation prob of all mRNA types
- size of ribosome/tRNA's yeast cell
- diffusion constant ribosomes, tRNA's
- $--> $ characteristic times

STATES

- number of free ribosomes/tRNA's(of every type)
- Position of each bound ribosomes/tRNA's on each individual mRNA

EVENTS

- Initiation (binding of ribosome at free 5'end of mRNA)
- Elongation (change postion, free - bind tRNA)

Yeast data on cell content

Table 1. Summary of Model Parameters

Parameter	Description	Value or Range of Values	References
R^t	number of ribosomes	2×10^5	(Warner, 1999; von der Haar, 2008)
A^t	number of mRNAs	6×10^4	(Zenklusen et al., 2008)
T^t	number of tRNAs	3.3×10^6	(Waldron and Lacroute, 1975)
T_n	number of types of tRNAs	41	(Chan and Lowe, 2009)
T^t_j	number of tRNAs of type *j*	~12,000–190,000	(Chan and Lowe, 2009)
A_i	number of mRNAs of type *i*	1–1,254	(Ingolia et al., 2009)
p_i	gene-specific initiation probability	$\sim 3.5 \times 10^{-6}$–0.115	(Experimental Procedures)
n	number of genes	3,795	(Ingolia et al., 2009)
D_r	diffusion coefficient of ribosomes	3×10^{-13} m^2/s	(Politz et al., 2003)
D_t	diffusion coefficient of tRNAs	8.42×10^{-11} m^2/s	(Werner, 2011)
C_r	size of ribosome footprint in codons	10	(Ingolia et al., 2009)
s	tRNA competition coefficient	7.78×10^{-4}	(Experimental Procedures)
V	volume of the cell	4.2×10^{-17} m^3	(Siwiak and Zielenkiewicz, 2010)

$N_t = 1.24 \times 10^7$ and $N_r = 1.56 \times 10^6$,

Algorithm (pseudocode)

while *time* $< t$ *(total simulation time)* **do**

Calculate

Fraction of mRNAs of gene i that are *initiable*, f_i - i.e., those mRNAs with first 10 codons unbound.

Number of *elongatable* ribosomes waiting at codon j, $R^b(j)$ - ribosomes with next 10 codons unbound.

Rates of all possible events (see Table S2)

Total initiation rate: $\rho^t = \sum_{i=1}^{n} \frac{R^f f_i A_i p_i}{\tau_r N_r}$

Total elongation rate: $\epsilon^t = \sum_{j=1}^{61} \frac{R^b(j) T^f_{\phi(j)} w_j s}{\tau_t N_t}$

Probability of each possible event (see Table S2)

Randomly select an event based on its probability of occurrence (see Table S2)

Update the changes in the state of the cell (see $\Delta State$ in Table S2)

Increment *time* by $\frac{1}{\rho^t + \epsilon^t}$

Update the number of free ribosomes, R^f

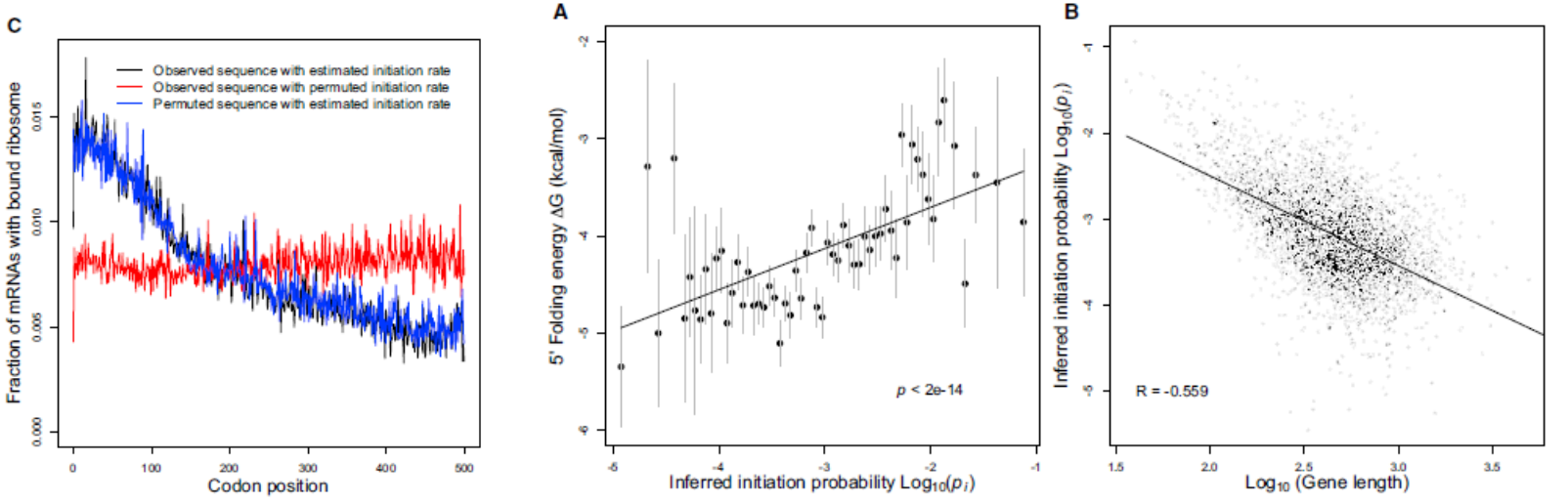
Update the number of free tRNAs of type $\phi(j)$, $T^f_{\phi(j)}$

end

Table S2. Markov States and Transition Rates, Related to Figure 1

Initiation	Gene #	mRNA # of gene			Initiation rate	Event probability	ΔState
	1	1			0	0	N/A
	1	2			0	0	N/A
	1	3			$\frac{R^f p_1}{\tau_r N_r}$	$\frac{R^f p_1}{\tau_r N_r(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f - 1$
	1	A_1			$\frac{R^f p_1}{\tau_r N_r}$	$\frac{R^f p_1}{\tau_r N_r(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f - 1$
	2	1			$\frac{R^f p_2}{\tau_r N_r}$	$\frac{R^f p_2}{\tau_r N_r(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f - 1$
	2	2			$\frac{R^f p_2}{\tau_r N_r}$	$\frac{R^f p_2}{\tau_r N_r(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f - 1$
	2	.	.	.	.	.	.
	2	A_2			0	0	N/A
	.	.	.	.	.	.	.
	n	.	.	.	.	.	.
	n	A_n			$\frac{R^f p_n}{\tau_r N_r}$	$\frac{R^f p_n}{\tau_r N_r(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f - 1$
				Total initiation rate	$\rho^t = \sum_{i=1}^{n} \frac{R^f f_i A_i p_i}{\tau_r N_r}$		
Elongation	**Gene #**	**mRNA # of gene**	**Codon position**	**Ribosome bound**	**Elongation rate**	**Event probability**	**ΔState**
	1	1	1	N	0	0	N/A
	1	1	2	Y	$\frac{T^f_{\phi(1,2)} w_{1,2} s}{\tau_t N_t}$	$\frac{T^f_{\phi(1,2)} w_{1,2} s}{\tau_t N_t(\rho^t+\epsilon^t)}$	Ribosome bound at codon 2 →N Ribosome bound at codon 3 →Y
	1	1	3	N	0	0	N/A
	1	1	.	.	.	.	.
	1	1	L_1	N	0	0	N/A
	1	2	1	Y	0	0	N/A
	1	2	.	.	.	.	.
	1	2	11	Y	$\frac{T^f_{\phi(1,11)} w_{1,11} s}{\tau_t N_t}$	$\frac{T^f_{\phi(1,11)} w_{1,11} s}{\tau_t N_t(\rho^t+\epsilon^t)}$	Ribosome bound at codon 11 →N Ribosome bound at codon 12 →Y
	1	2	.	.	.	.	.
	1	2	L_1	Y	$\frac{T^f_{\phi(1,L_1)} w_{1,L_1} s}{\tau_t N_t}$	$\frac{T^f_{\phi(1,L_1)} w_{1,L_1} s}{\tau_t N_t(\rho^t+\epsilon^t)}$	$R^f \rightarrow R^f + 1$
	1	.	.	.	.	.	.
	1	A_1	L_1	.	.	.	.
	2	.	.	.	.	.	.
	2	A_2	L_2	.	.	.	.
	.	.	.	.	.	.	.
	n	A_n	L_n	.	.	.	.
				Total elongation rate	$\epsilon^t = \sum_{j=1}^{61} \frac{R^b(j) T^f_{\phi(j)} w_j s}{\tau_t N_t}$		

Is protein production initiation or elongation limited in exponential growing yeast populations?



more ribosomes at 5énd BUT due to >> intiation prob. on short genes

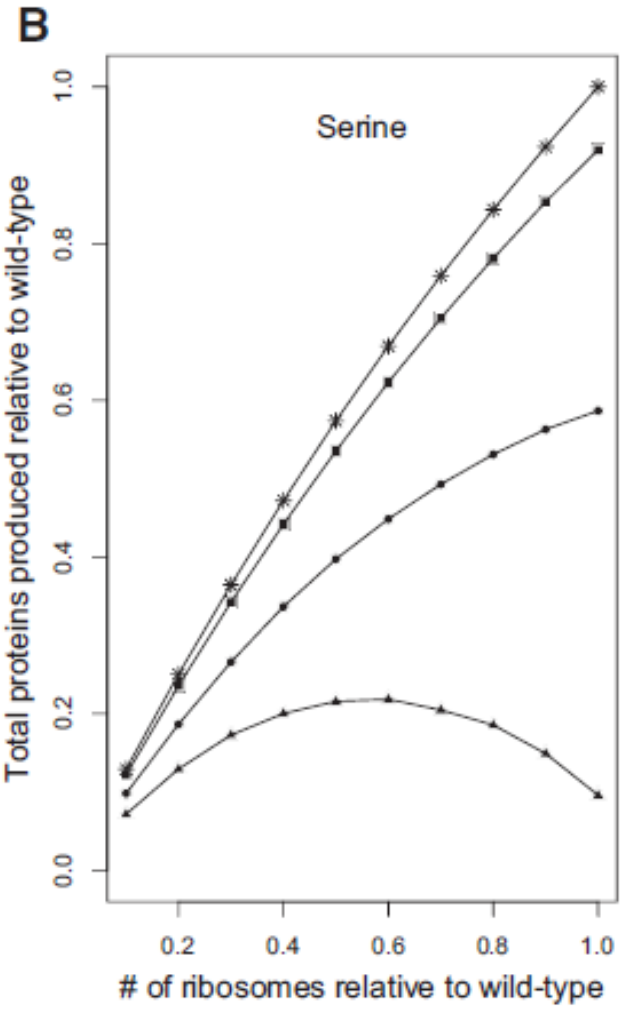
initiation limited

debugging of wrong inference from exp. data

Under amino-acid starvation down regulating ribosomes can increase protein production

because translation becomes elongation limited

reducing Ribosomes increases free TRNA'



conclusions event based modeling of stochastic reaction kinetics

Data intensive modeling

Quantitative conclusions

Upscaling to "whole cell modeling"

But note simplifications:

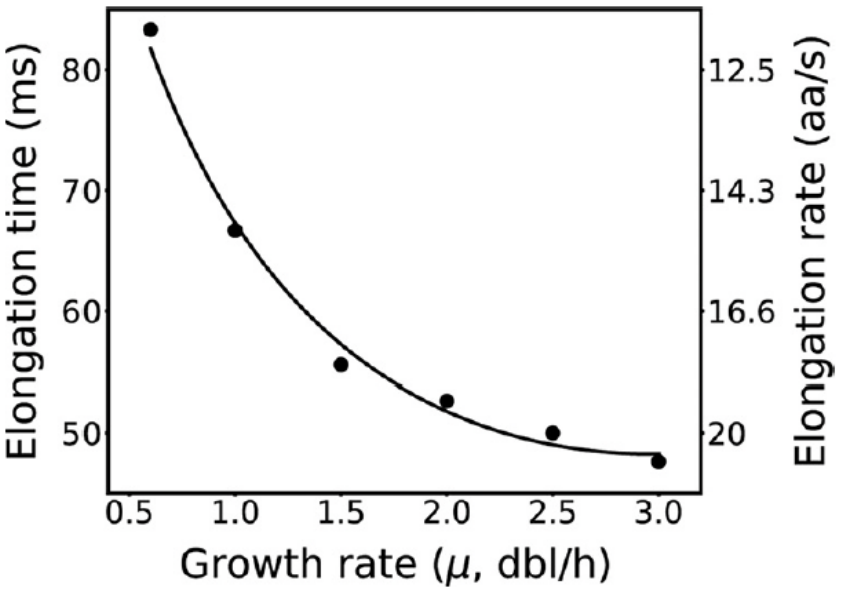
space but no spatial structure

fixed number of molecules

fixed conditions

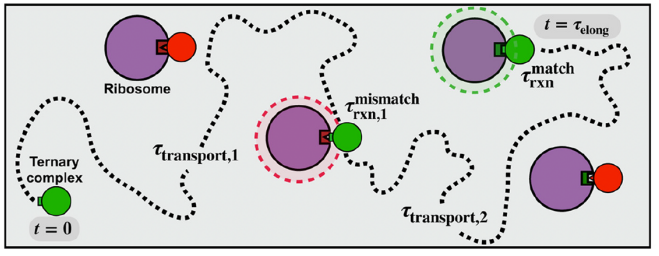
....

Further scruteny of traslation dynamics
Higher efficiency at higher growth rates
How??



AJ Maheshwari, AM Sunol, E Gonzalez, D Endy 2023:
Colloidal physics modeling reveals how per-ribosome productivity increases with growth rate in escherichia coli

Detailed dynamics of elongation in ribosomes
Analyse dynamics per "voxel"

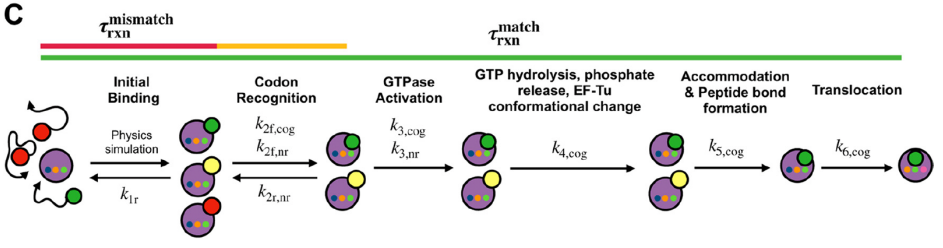


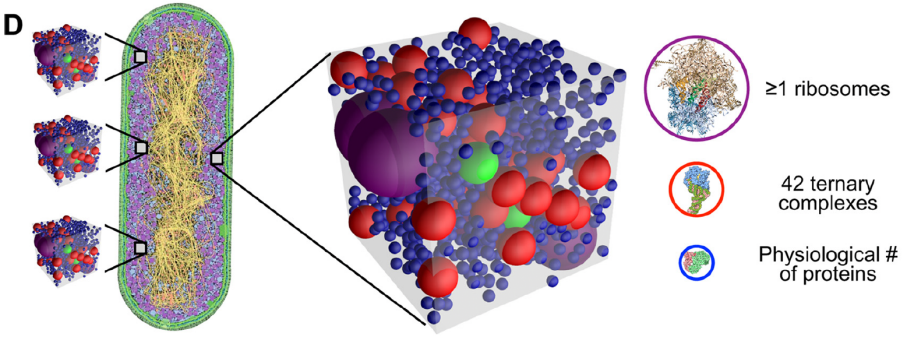
$$\tau_{elong} = \tau_{transport} + \tau_{rxn}$$

$$\tau_{transport} = \sum_i \tau_{transport,i}$$

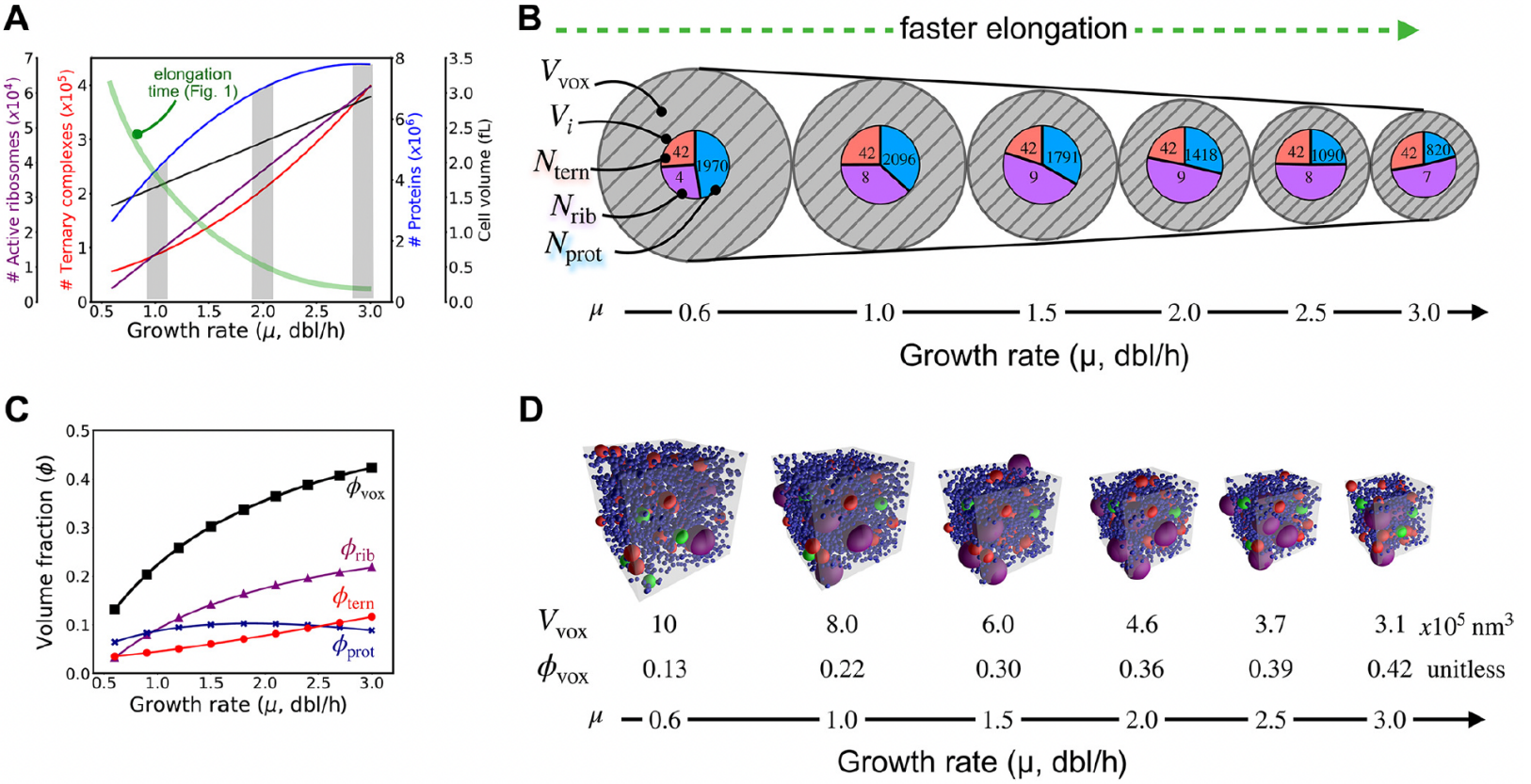
$$\tau_{rxn} = \tau_{rxn}^{match} + \tau_{rxn}^{mismatch}$$

$$\tau_{rxn}^{mismatch} = \sum_i \tau_{rxn,i}^{mismatch}$$

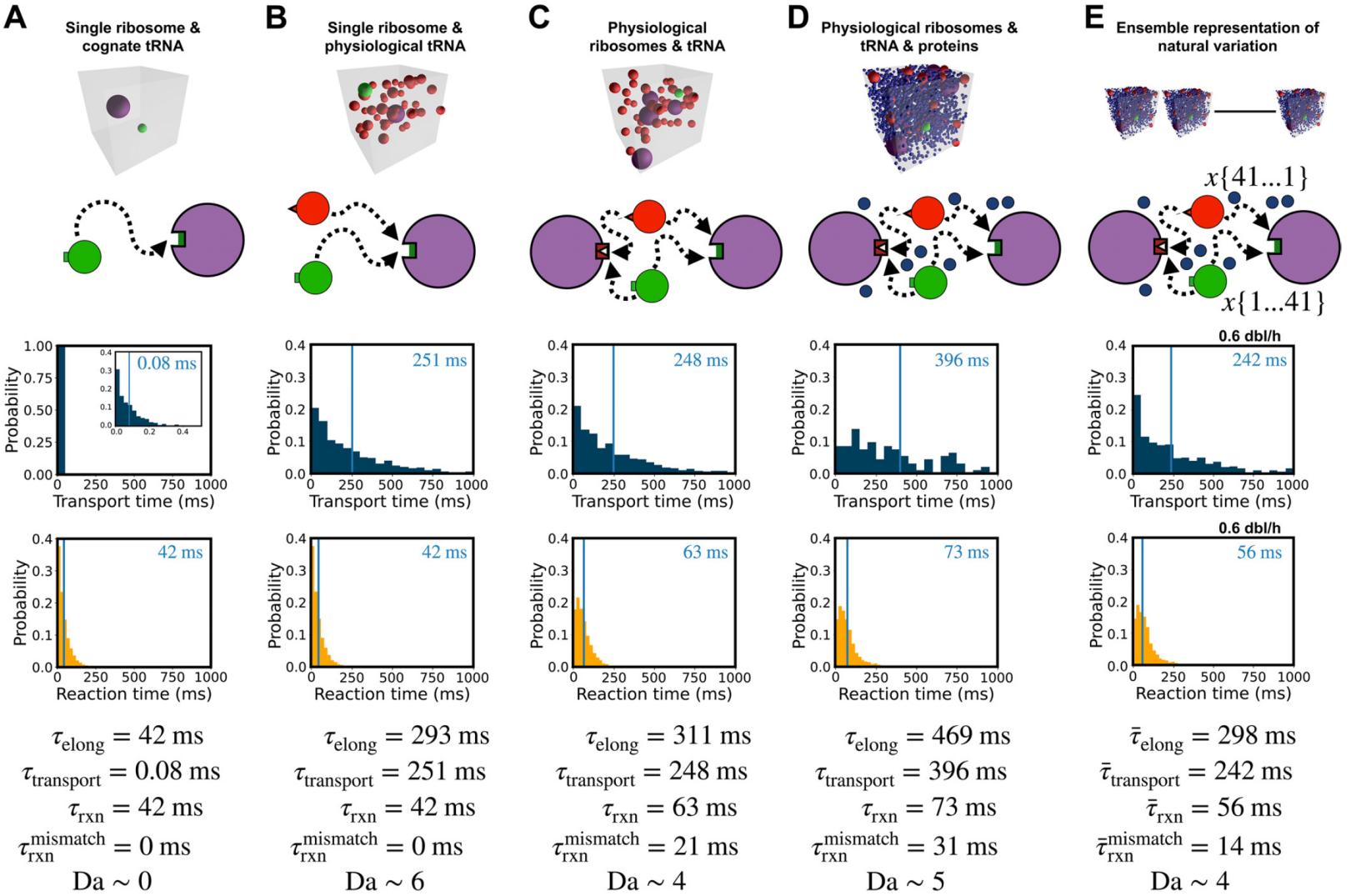




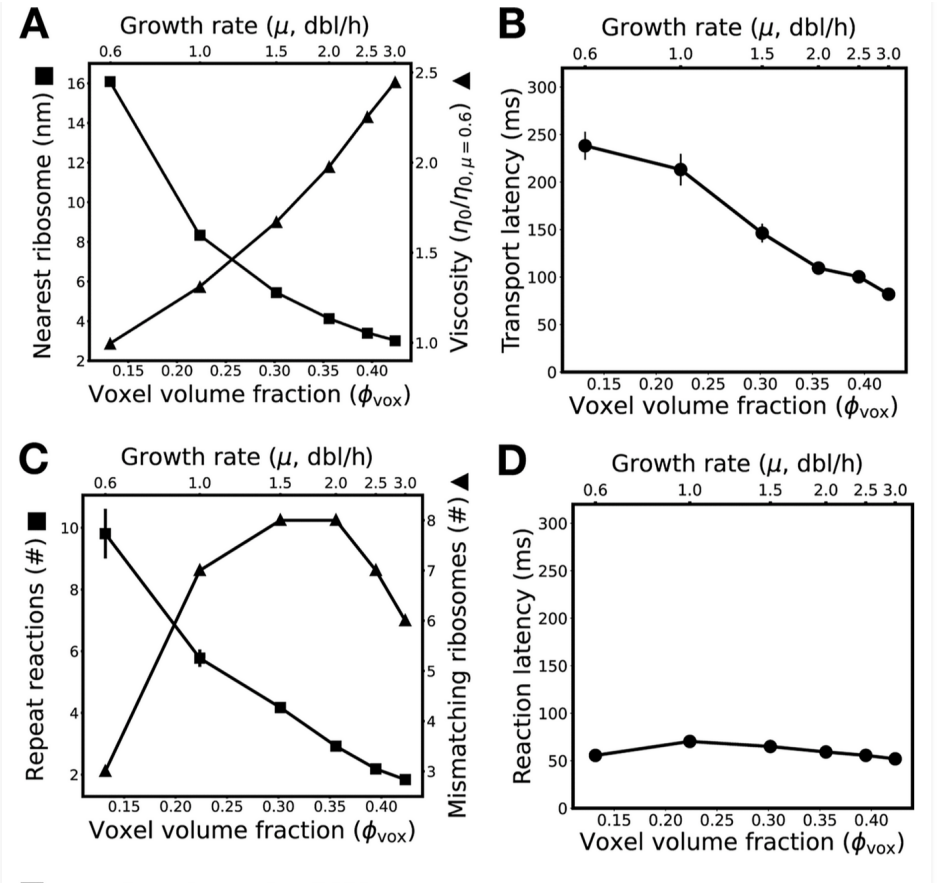
Molecular composition of E.coli cell dependent on growthrate

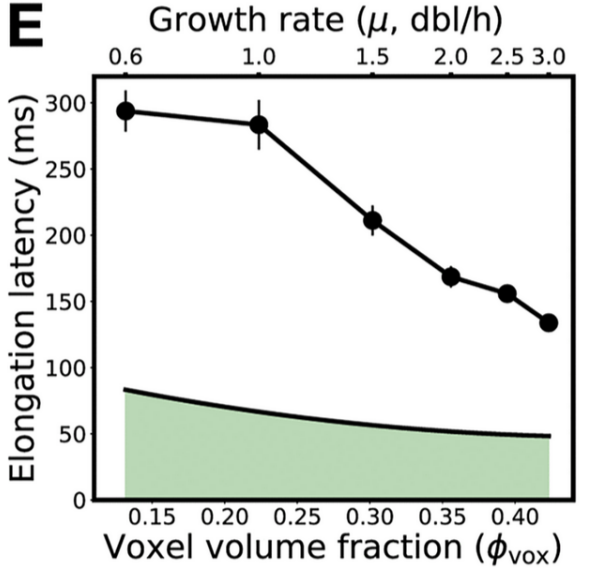


Charateristic times dependent on voxel composition: transport time dominate in "realistic compositions

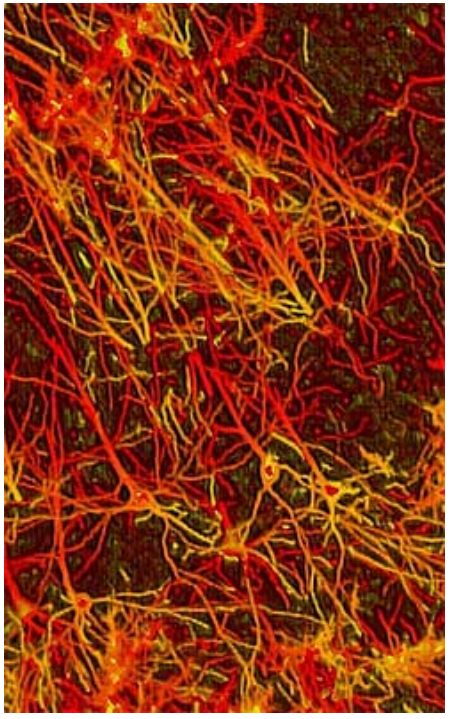

A
Single ribosome & cognate tRNA
B
Single ribosome & physiological tRNA
C
Physiological ribosomes & tRNA
D
Physiological ribosomes & tRNA & proteins
E
Ensemble representation of natural variation
x{41...1}
x{1...41}
0.08 ms
251 ms
248 ms
396 ms
0.6 dbl/h
242 ms
Probability
Transport time (ms)
42 ms
42 ms
63 ms
73 ms
0.6 dbl/h
56 ms
Reaction time (ms)
τelong = 42 ms
τtransport = 0.08 ms
τrxn = 42 ms
τrxnmismatch = 0 ms
Da ∼ 0
τelong = 293 ms
τtransport = 251 ms
τrxn = 42 ms
τrxnmismatch = 0 ms
Da ∼ 6
τelong = 311 ms
τtransport = 248 ms
τrxn = 63 ms
τrxnmismatch = 21 ms
Da ∼ 4
τelong = 469 ms
τtransport = 396 ms
τrxn = 73 ms
τrxnmismatch = 31 ms
Da ∼ 5
τ̄elong = 298 ms
τ̄transport = 242 ms
τ̄rxn = 56 ms
τ̄rxnmismatch = 14 ms
Da ∼ 4

Model explains speedup at higher growthrates shorter distances, but slower diffusion by crowding But not elongation rate, (parameter uncentainty??)

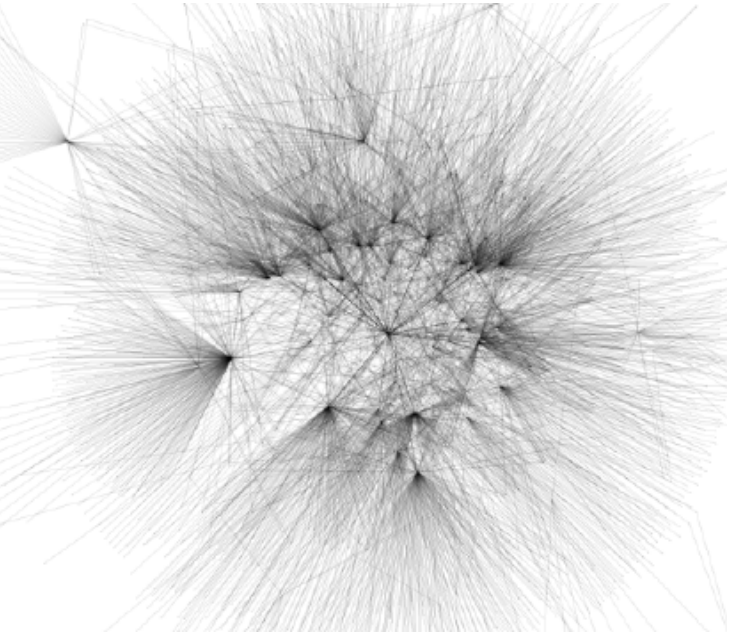




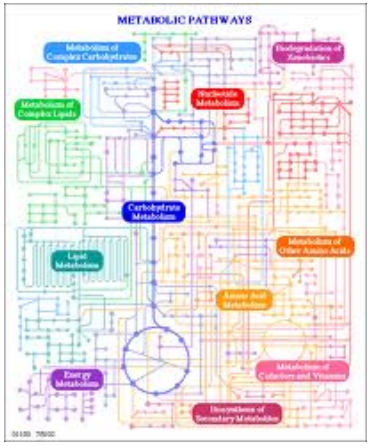
dynamical systems:
decomposition in many simple systems cont.,
NETWORKS



Neural net
connected

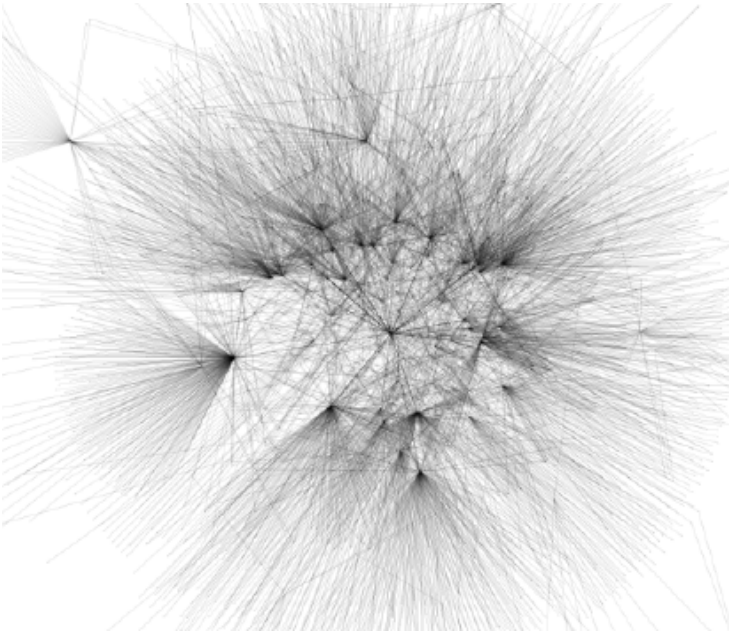


yeast transcription net
information transfer



Keg metabolic net
mass conservation
stochiometric

Gene regulation Networks:
"full" transcription network of yeast



How does it behave? *how special is it?* *(evolution)*

Boolean Networks

Proposed by S.Kauffmann (1969)
as model for gene regulation

Like binary CA but
specific network structure (IO relations)
each node own transition rule
(Boolean function with k inputs)
studied: NK networks

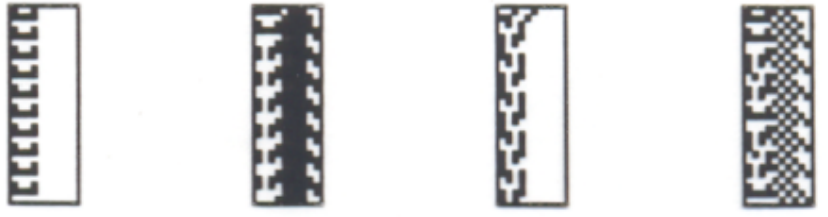
Boolean network : special cases can be mapped into CA (homogeneous network structure, "rule-layer")

Multiple attractors

rules

1	2	3	4	5	6	7	8	9	10
8	6	4	12	5	5	1	7	2	1

attractors

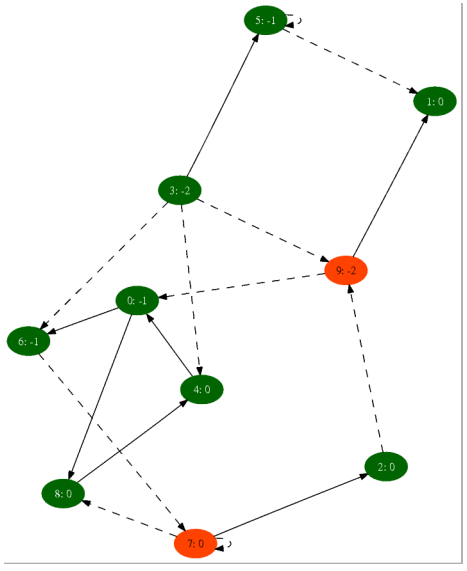


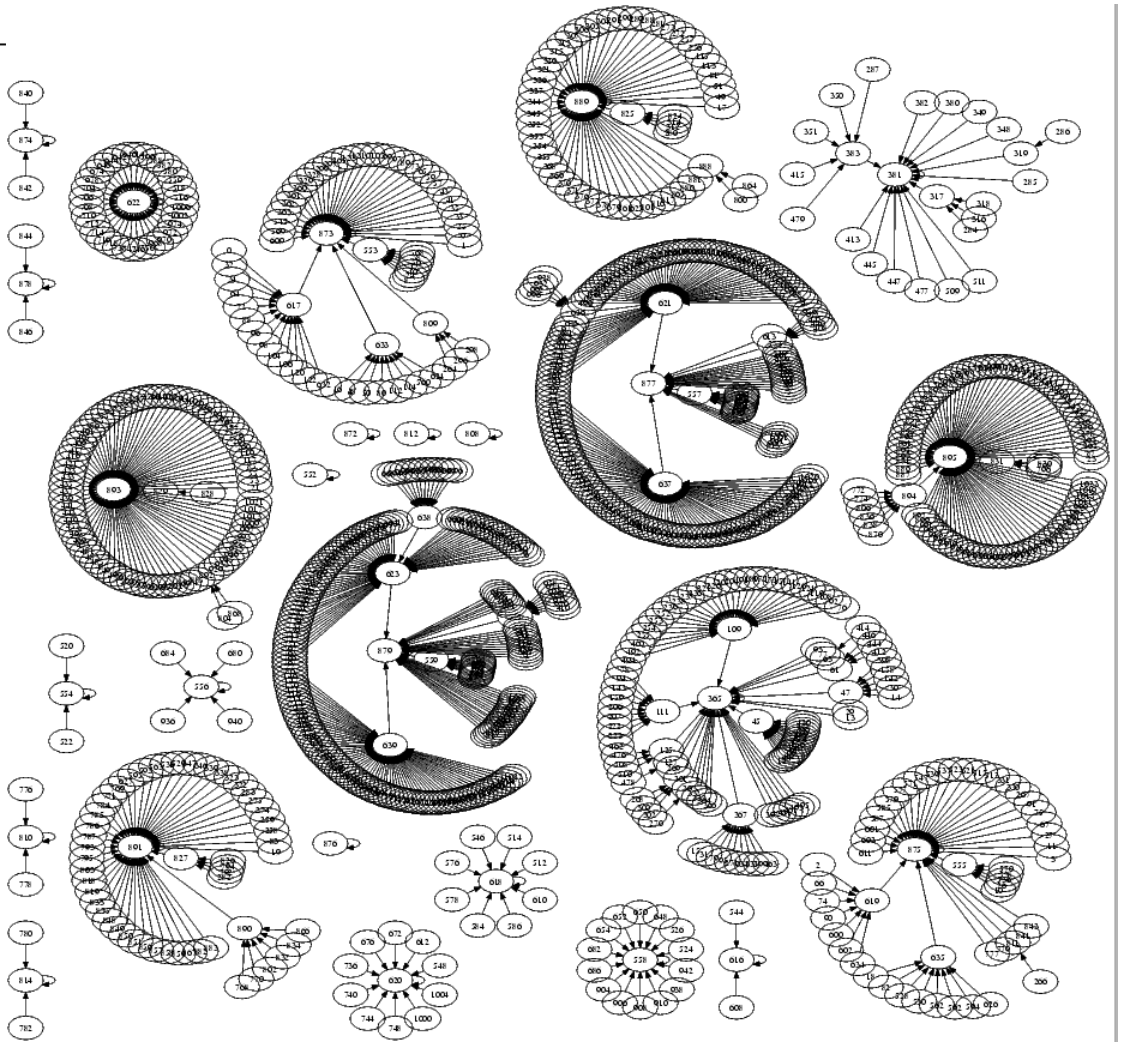
basin of attraction	112	128	272	512
cyclelength	3	4	6	12

simple random network (threshold dynamics)

$$V_j(t+1) = \begin{cases} 1, & \text{if } \Sigma_k T_{jk} V_k(t) + \\ 0, & \text{otherwise} \end{cases}$$

(Anton Crombach)
MULTPLE ATTRACTORS





ONLY 10 nodes (=genes)!

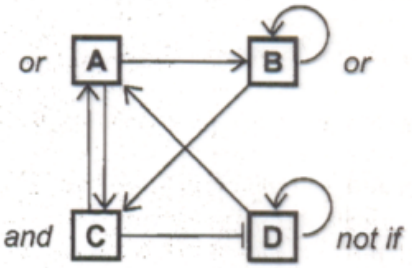
STATESPACE

What kind of behavior do we expect from gene regulation networks?

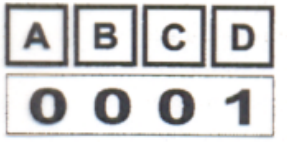
multiple attractors (cell types)
alternative trajectories from A' and A'' to B
multiple causes
robustness (knockouts)

Huang S, Ingber D. Exp Cell Res. 2000

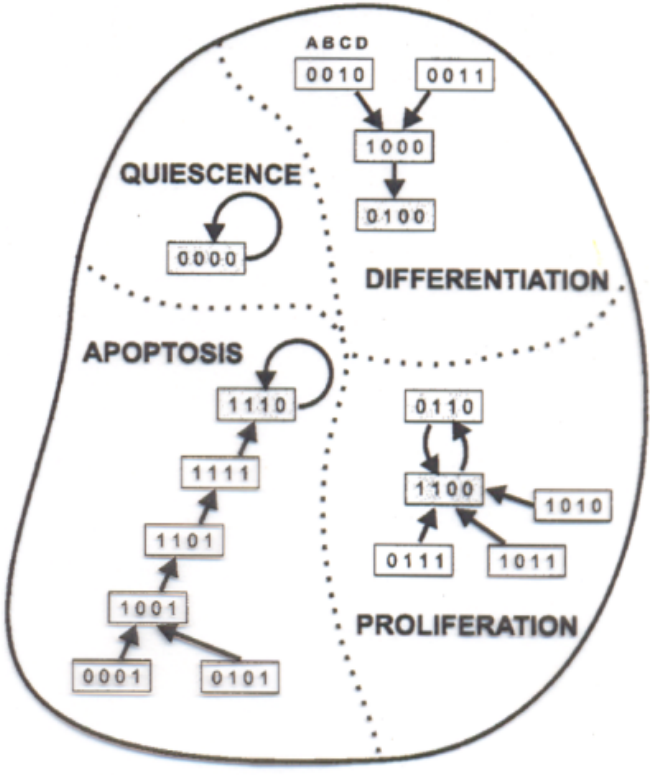
a Network wiring diagram



b A network state



c Protein activity state space



Boolean functions:

A: " *or* "

INPUTS C	INPUTS D	OUTPUT A
0	0	0
0	1	1
1	0	1
1	1	0

B: " *or* "

INPUTS A	INPUTS B	OUTPUT B
0	0	0
0	1	1
1	0	1
1	1	0

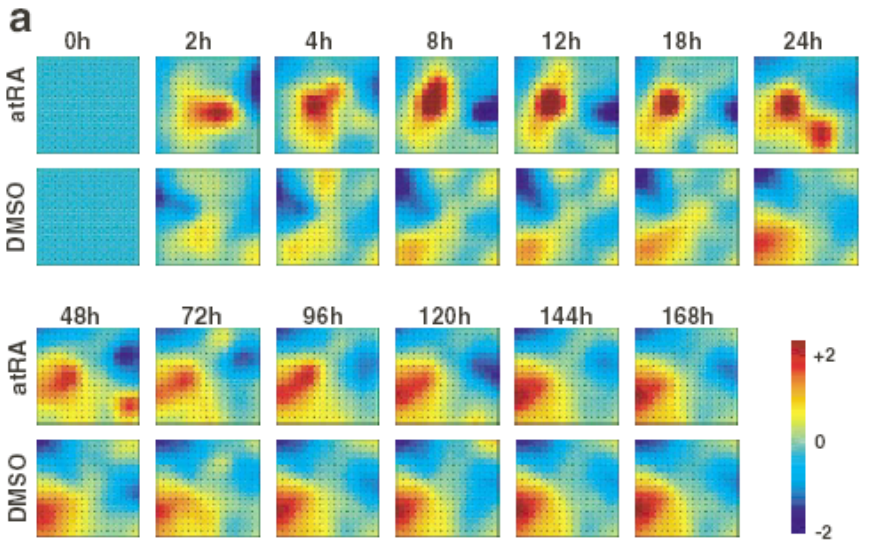
C: " *and* "

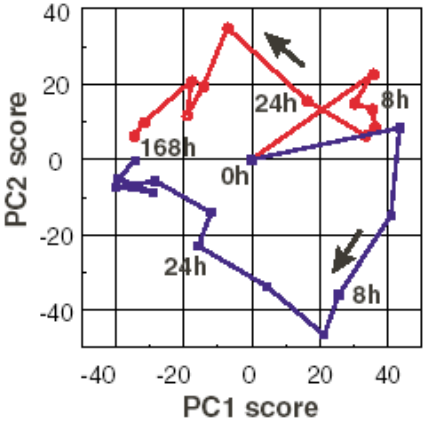
INPUTS A	INPUTS B	OUTPUT C
0	0	0
0	1	0
1	0	0
1	1	1

D: " *not if* "

INPUTS C	INPUTS D	OUTOUT D
0	0	0
0	1	1
1	0	0
1	1	0

2 pathways to Neutrophyl differentiation
Huang et all 2005 (Phys Rev Letters)



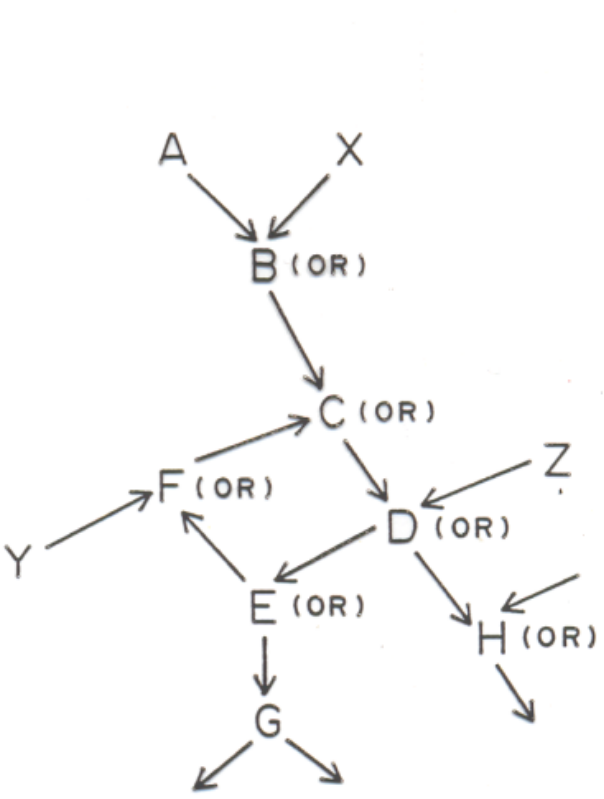


gene expression through time

2773 dim state-space, trajectories in 2D projection

n^{2773} states!

Robustness: Forcing structures



OR

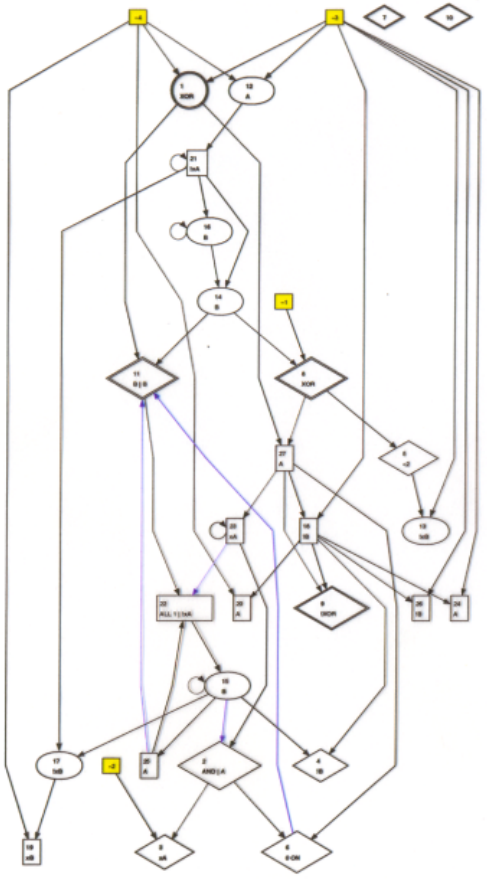
A	X	B
0	0	0
0	1	1
1	0	1
1	1	1

FIGURE 8 Forcing structure among binary elements governed by the Boolean OR function. The forcing "1" value propagates down structure and around forcing loop which eventually is "frozen" into the forced state with "1" values at all elements around the loop. Loop then radiates fixed forced values downstream. From *Origins of Order: Self Organization in Evolution* by S. A. Kauffman. Copyright © 1990 by Oxford University Press, Inc. Reprinted by permission.

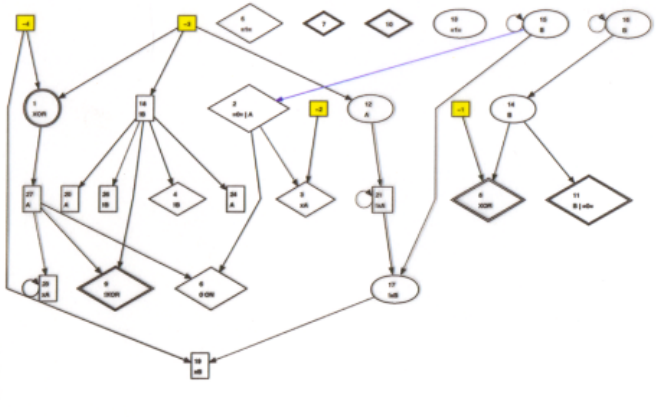
Evolved gene regulation networks

"existing" vs "functioning"

?? "false positives" ??



53 links



32 links

16 celltypes

18 reg. genes

Properties of Random Boolean Networks (depending on K)

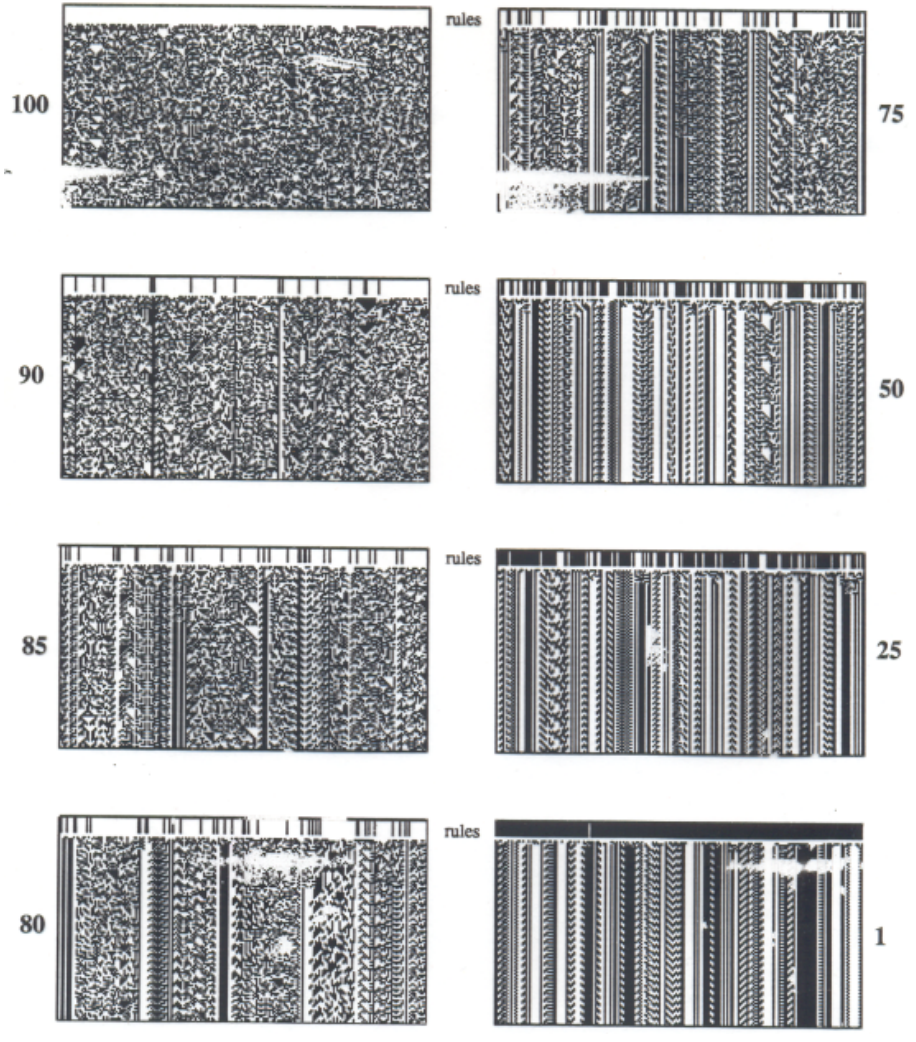
TABLE 1 Properties of Random Boolean Nets for Different Values of K[1]

	State Cycle Length	Number of State Cycle Attractors	Homeostatic Stability	Reachability Among Cycles After Perturbation
$K = N$	$0.5 \times 2^{N/2}$	N/e	Low	High
$K > 5$	0.5×2^{BN} $(B > 1)$	$\sim N\left[\frac{\log\left(\frac{1}{1/2 \pm \alpha}\right)}{2}\right]$; $\alpha = p_{(K)} - 1/2$	Low	High
$K = 1$	Very Long	Very Many	Low	High
$K = 2$	$\sqrt{N}$	$\sqrt{N}$	High	Low

[1] Column 1: state cycle length is median number of states on a state cycle. Column 2: number of state cycle attractors in behavior of one net. ($\alpha = P_K - 1/2$, where P_K is mean internal homogeneity of all Boolean functions on K inputs; see text.) Column 3: homeostatic stability refers to tendency to return to same state cycle after transient reversal of activity of any one element. Column 4: reachability is number of other state cycles to which net flows from each state cycle after all possible minimal perturbations, due to reversing activity of one element.

Importance of sampling method: Dependence on K is dependence on fraction (non) forcing rules!

Non forcing rules in 1D CA (k=2)



conclusion: Boolean Kaufman Networks

Important:

Identification of cell state with attractor of gene regulation network

Multiple attractors in simple networks

alternative trajectories to attracotr

Domain of attraction: i.e. “robusteness”

forcing functions i.e. “robustness”

NOT IMPORTANT (WRONG!) connectivity of 2 “ideal”

Gene expression data $--> $ Boolean networks

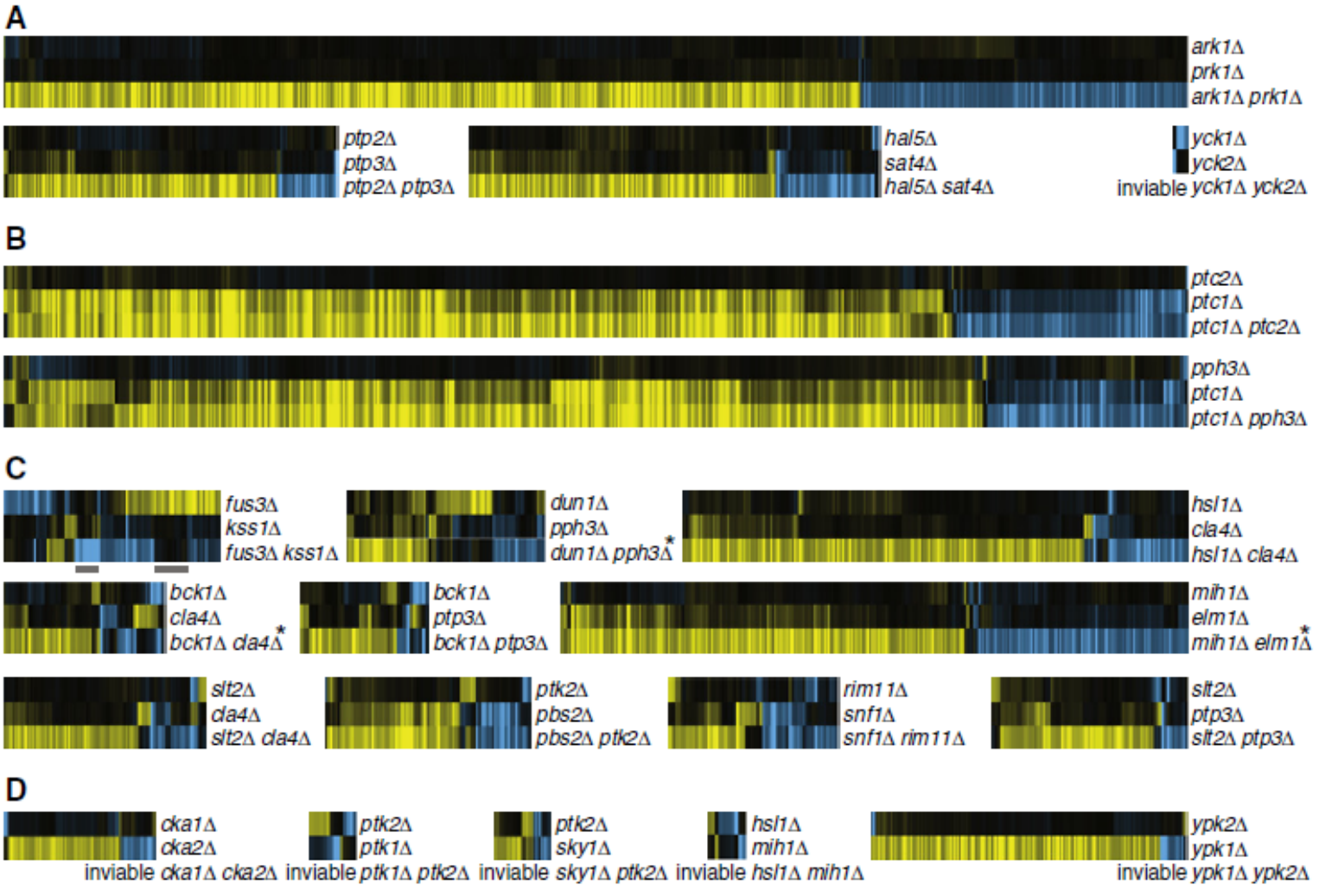
Functional Overlap and Regulatory Links Shape Genetic Interactions between Signaling Pathways
Sake van Wageningen, Patrick Kemmeren,..... Berend Snel and Frank C.P. Holstege Cell Dec 2010

141 kinases, 38 phosphatases in Yeast.

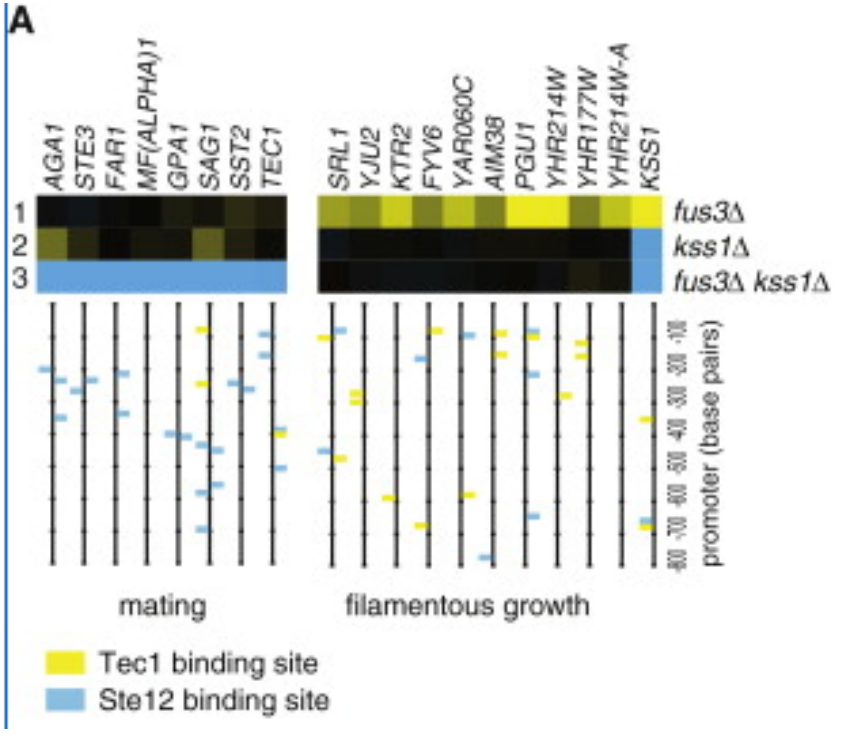
60% single knockouts "no phenotype"
(== <8 genes different of WT) (single growth condition)

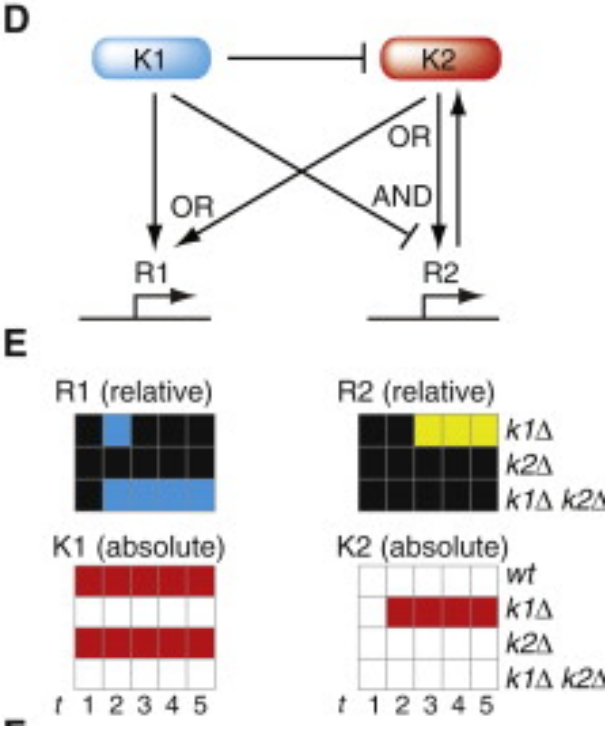
Double knockouts: 21 "buffering" s with other kinase/phosphatasse v

double knockout expression profiles

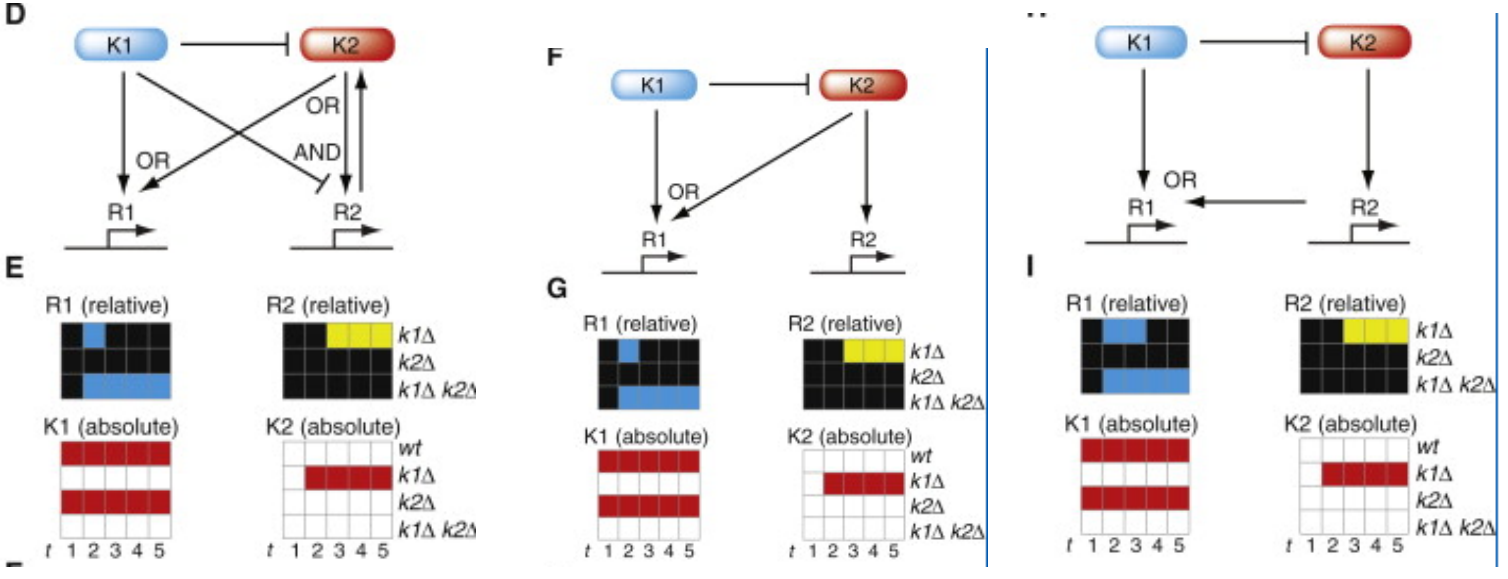

A
ark1Δ
prk1Δ
ark1Δ prk1Δ
ptp2Δ
ptp3Δ
ptp2Δ ptp3Δ
hal5Δ
sat4Δ
hal5Δ sat4Δ
yck1Δ
yck2Δ
inviable yck1Δ yck2Δ
B
ptc2Δ
ptc1Δ
ptc1Δ ptc2Δ
pph3Δ
ptc1Δ
ptc1Δ pph3Δ
C
fus3Δ
kss1Δ
fus3Δ kss1Δ
dun1Δ
pph3Δ
dun1Δ pph3Δ*
hsl1Δ
cla4Δ
hsl1Δ cla4Δ
bck1Δ
cla4Δ
bck1Δ cla4Δ*
bck1Δ
ptp3Δ
bck1Δ ptp3Δ
mih1Δ
elm1Δ
mih1Δ elm1Δ*
slt2Δ
cla4Δ
slt2Δ cla4Δ
ptk2Δ
pbs2Δ
pbs2Δ ptk2Δ
rim11Δ
snf1Δ
snf1Δ rim11Δ
slt2Δ
ptp3Δ
slt2Δ ptp3Δ
D
cka1Δ
cka2Δ
inviable cka1Δ cka2Δ
ptk2Δ
ptk1Δ
inviable ptk1Δ ptk2Δ
ptk2Δ
sky1Δ
inviable sky1Δ ptk2Δ
hsl1Δ
mih1Δ
inviable hsl1Δ mih1Δ
ypk2Δ
ypk1Δ
inviable ypk1Δ ypk2Δ

example of mixed epistasis
filamentous growth vs mating





2 simpler networks with same effect (complexer network most similar to exp. inferred network)



Many networks (max 2 inputs per node) with same effect!

Edges	Models	Correct models	Root models
2	0	0	0
3	352	0	0
4	4,960	2	2
5	32,896	6	1
6	129,280	24	8
7	294,912	28	7
8	331,776	46	10

all buffering pairs: Many non-homologs!; many mixed

Table 1. Buffering Relationships between Kinases and Phosphatases

Gene 1	Gene 2	Type	Duplication	Time (Years Ago)	Buffering Relationship
HAL5	*SAT4*	kk	old	600 M – 2 G	complete redundancy
ARK1	*PRK1*	kk	whole genome	125 M	complete redundancy
PTP2	*PTP3*	pp	recent	125 M – 600 M	complete redundancy
YCK1	*YCK2*	kk	whole genome	125 M	complete redundancy[a]
PTC1	*PTC2*	pp	old	600 M – 2 G	quantitative redundancy
PTC1	*PPH3*	pp	not homologous		quantitative redundancy
PBS2	*PTK2*	kk	ancient	>2G	mixed epistasis
CLA4	*SLT2*	kk	ancient	>2G	mixed epistasis
CLA4	*HSL1*	kk	ancient	>2G	mixed epistasis
SNF1	*RIM11*	kk	ancient	>2G	mixed epistasis
BCK1	*PTP3*	kp	not homologous		mixed epistasis
SLT2	*PTP3*	kp	not homologous		mixed epistasis
FUS3[b]	*KSS1*	kk	recent	125 M – 600 M	mixed epistasis
ELM1	*MIH1*	kp	not homologous		mixed epistasis[c]
CLA4	*BCK1*	kk	ancient	>2G	mixed epistasis[c]
DUN1	*PPH3*	kp	not homologous		mixed epistasis[c]
CKA2	*CKA1*	kk	recent	125 M – 600 M	not classified[a]
YPK1[b]	*YPK2*	kk	whole genome	125 M	not classified[a]
PTK1	*PTK2*	kk	whole genome	125 M	not classified[a]
HSL1	*MIH1*	kp	not homologous		not classified[a]
SKY1	*PTK2*	kk	ancient	>2G	not classified[a]

regulatory network via mixed response networks

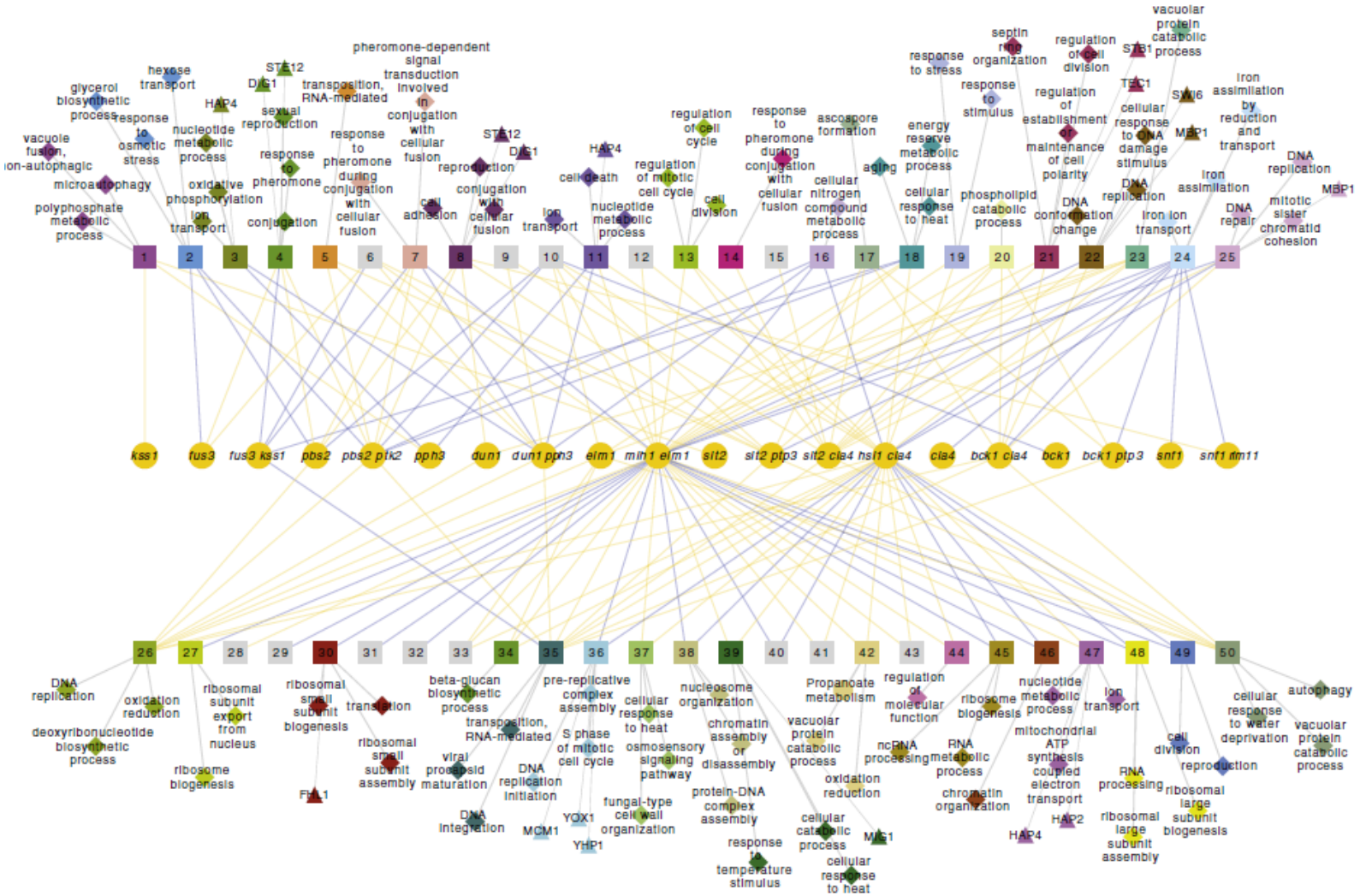
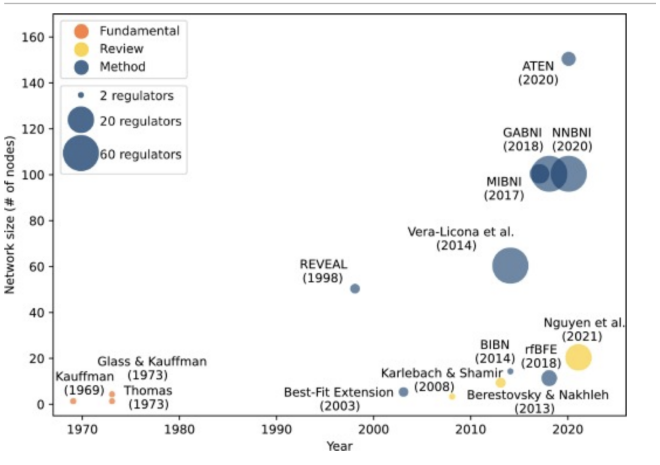


Figure 6. Multiprocess Control through Signaling Components with Mixed Epistasis

Inference of (Boolean) GRN from timeseries dynamic and static "accuracy"

VERY Active research:
Google Scholar 2023: *"gene regulatory network inference"*
4930 hits: Baysian, deep NN, random forests
heuristics: conform to known structural features

E.G. performance assessed from simulated boolean networks
(extracted from E.coli GRN database)
. Pušnik,M.Mraz,N.Zimicand M.Moškon 2022



Fairly good Dynamic accuracy

Poor static accuracy
(many different measures)
TP, FP, TN, FN in different combi's

hard because:

stochasticity, missing data, very large search space
AND
"redundant regulation": minimal or "true" network? multiple regulators with different Boolean functions produce the same results

some measurements of prediction quality

$$Precision = \frac{TP}{TP+FP}. \tag{8}$$

On the other hand, recall presents a fraction of correctly inferred edges among all edges in the initial network:

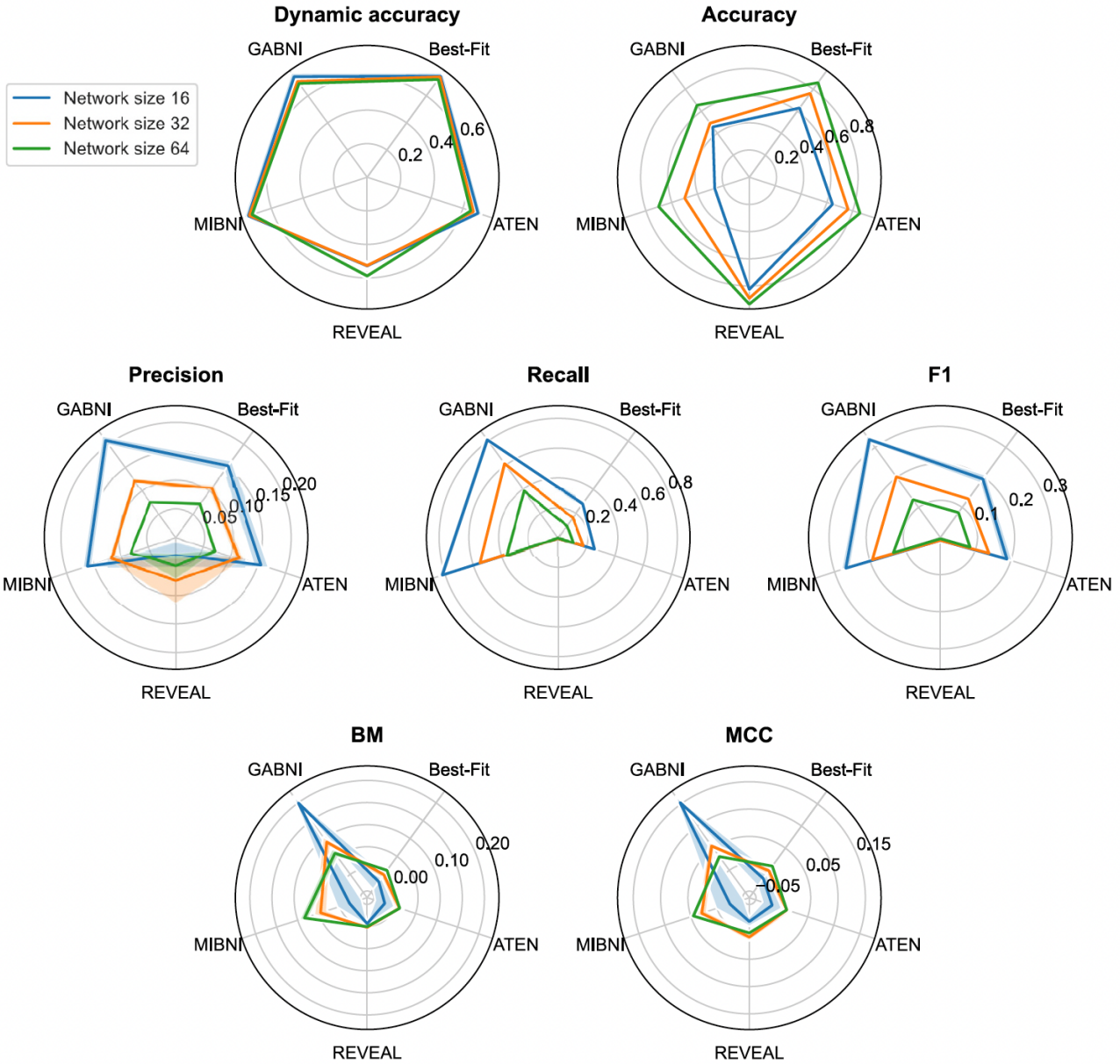
$$Recall = TPR = \frac{TP}{TP+FN}. \tag{9}$$

Precision and recall are important metrics, since we are more interested in edges than in non-edges. Accuracy is the proportion of all correct predictions, including edges and non-edges:

$$Accuracy = \frac{TP+TN}{TP+TN+FP+FN}. \tag{10}$$

F1 score is defined as a harmonic mean of the precision and recall:

$$F1 = 2 \cdot \frac{Precision \cdot Recall}{Precision+Recall}. \tag{11}$$


Dynamic accuracy
Accuracy
Precision
Recall
F1
BM
MCC
Network size 16
Network size 32
Network size 64
GABNI
Best-Fit
ATEN
REVEAL
MIBNI

Analysis of network structure for:

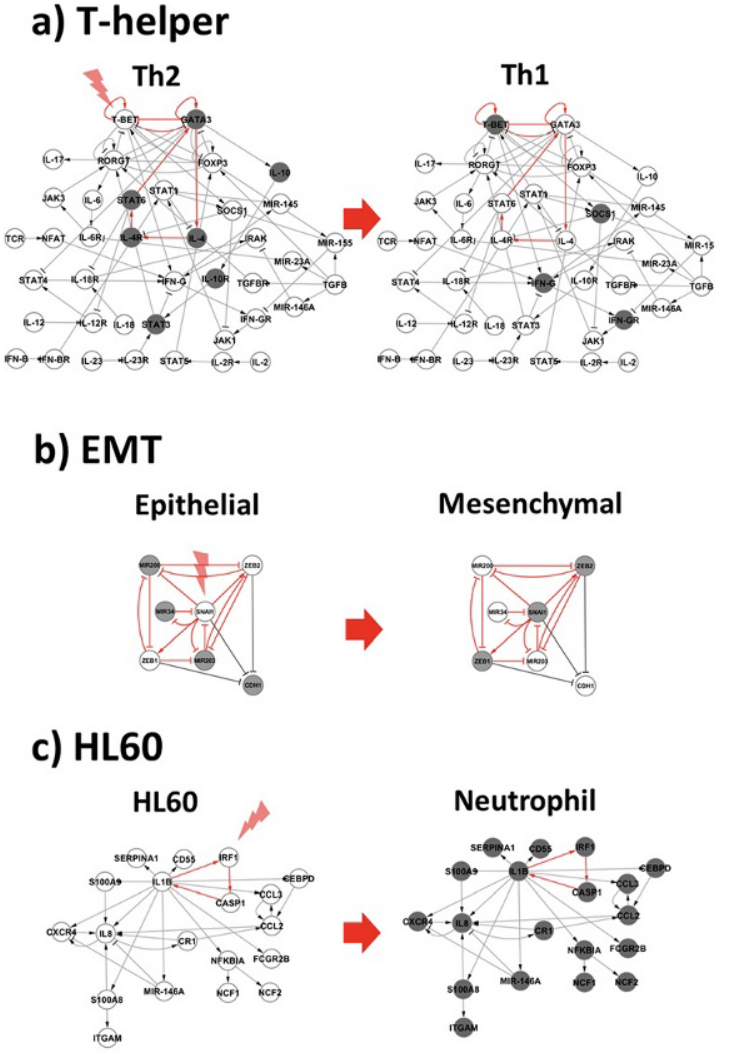
Finding attractor (stable cell states)
positive feedback loops

Finding "hubs"

HOW TO SWITCH CELL STAT == how to switch attractors

Examples of minimal set of perturbations needed to switch cell states calculated from Boolean network models and verified in experiments.

Despite simplifications usefull predictions



Crespo et al. BMC Systems Biology 2013

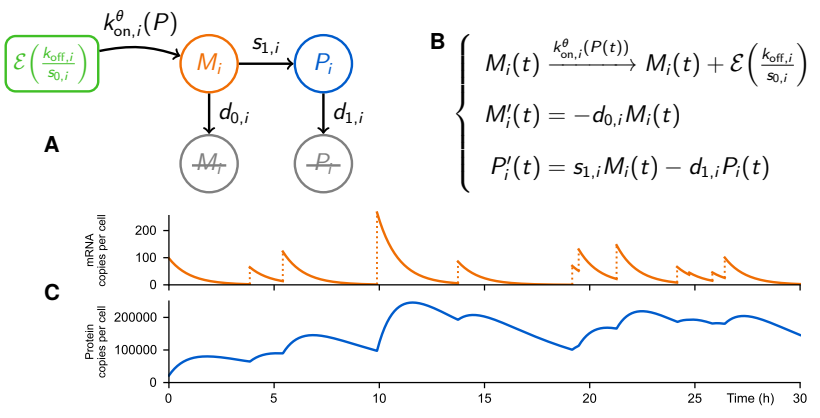
"ARE" GRN boolean networks"
no....

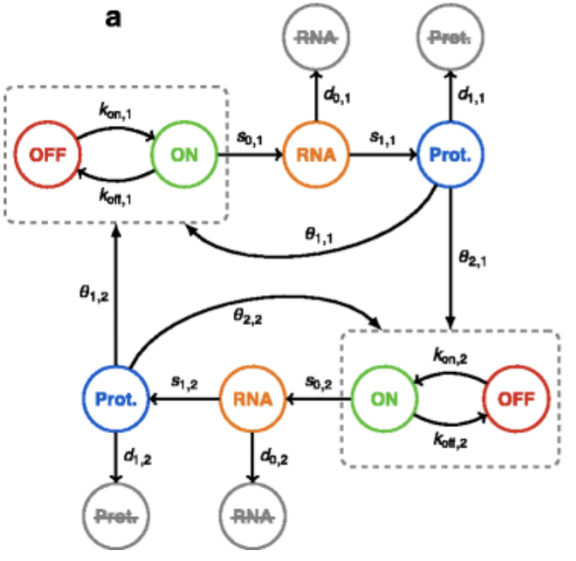
- not on/off but quantitative response. e.g. modeled as set of ODE's with sigmoid response sizes
- mRNA $--> $ protein
- Stochastic binding/unbinding of TF on DNA binding sites; gene expression in bursts
- Competition for binding sites
  overlap of binding sites for different TF
  and competition for TF
- .....

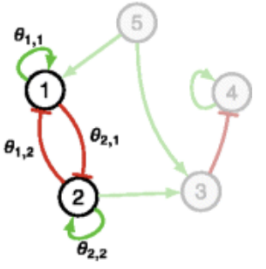
insights are obtained by multiple (wrong) models (Caricatures)

Stochastic gene expression
single cell gene expression signatures

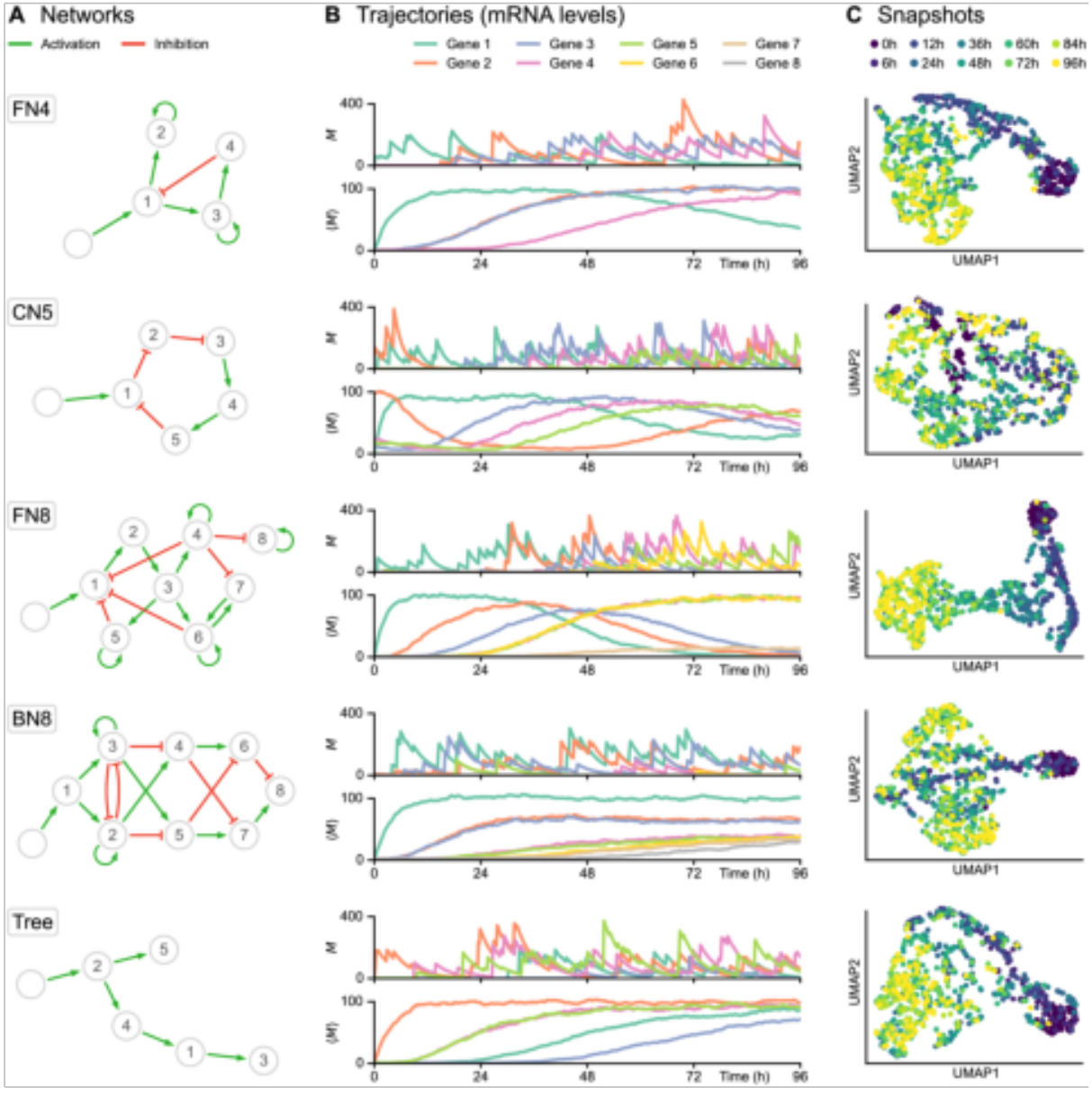
gene expression often in bursts, folowed by slow defradation of mRNA and buildup/decrease of protein







Elias Ventre, Olivier Gandrillon 2023, PLOS Comp Bio.


A Networks
Activation
Inhibition
B Trajectories (mRNA levels)
Gene 1
Gene 2
Gene 3
Gene 4
Gene 5
Gene 6
Gene 7
Gene 8
C Snapshots
0h
6h
12h
24h
36h
48h
60h
72h
84h
96h
FN4
CN5
FN8
BN8
Tree
M
⟨M⟩
Time (h)
UMAP1
UMAP2

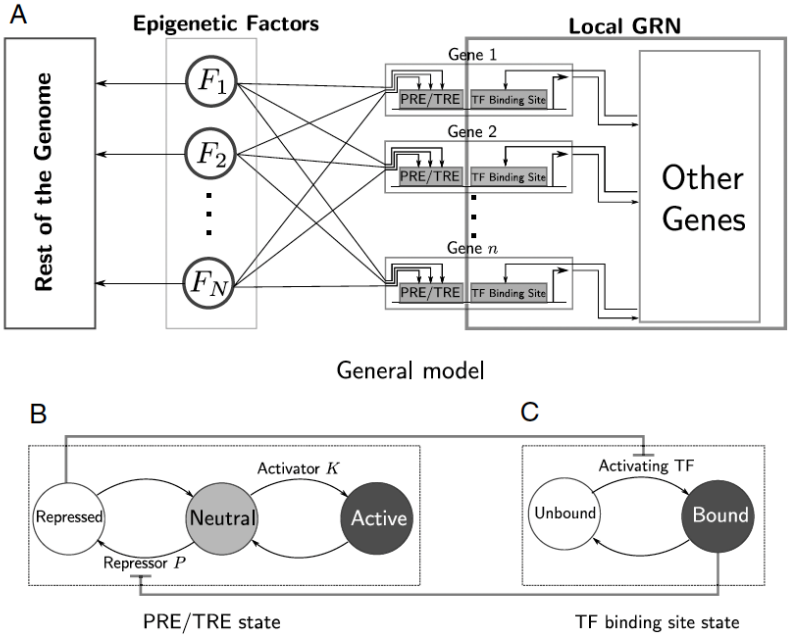
Coding structure of gene regulatory networks
e.g. global epigenetic factors and competition for binding sites

Competition for binding sites

Global epigenetic factors
bind at very many places

but they are in limited numbers

Model by distinguishing
bound/unbound states
of (overlapping) binding sites,
and EF and TF



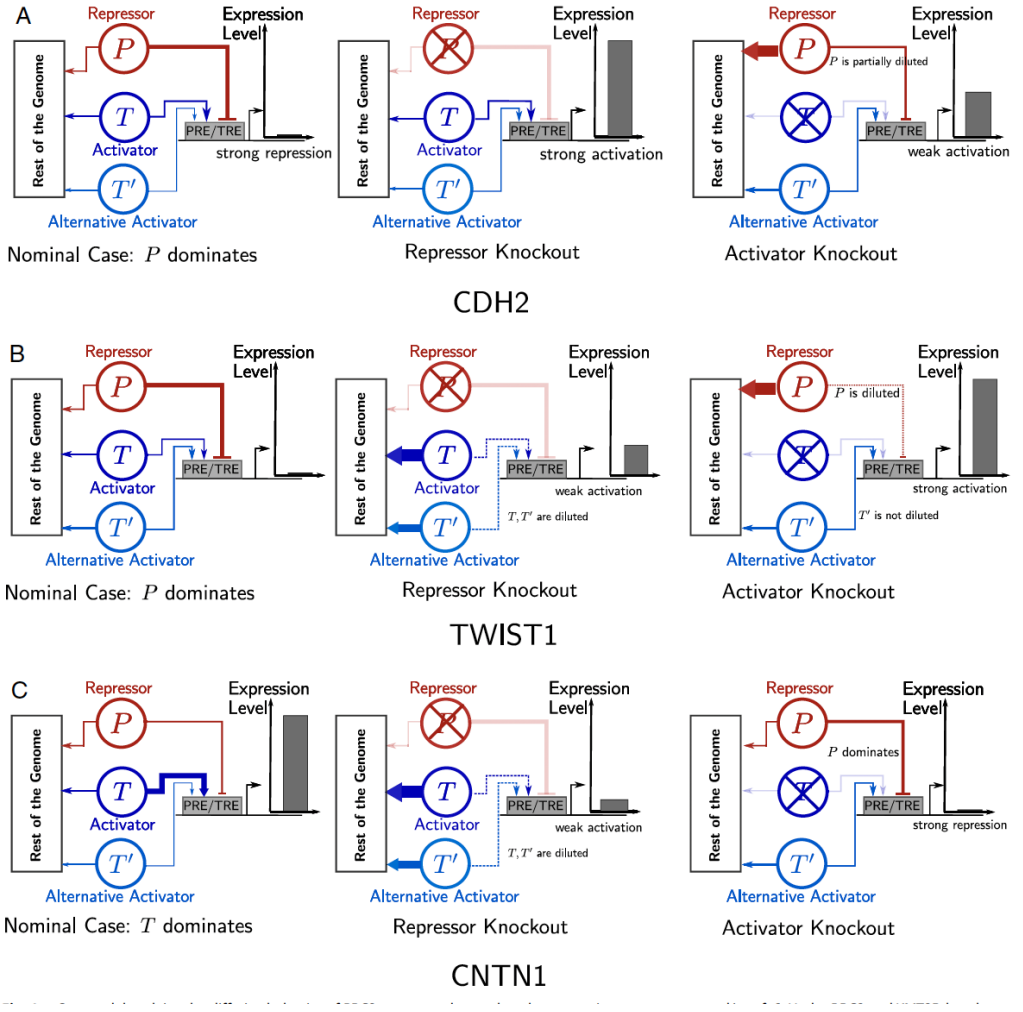
Can lead to very counter intuitive outcomes.

Examples from the The Polycomb (repressors)
and Trithorax Groups (activators) of EFs which modulate histon tails.
Very many targets (e.g. PRC3 10% of genes in embryonic stem cells)

Epigenetic factor competition reshapes the EMT landscape

M. Ali Al-Radhawi... Herbert Levine PNAS 2022

some modeled and experimentally observed examples
everything possible by tuning the parameters...



Event based models (2)
Individual based models (IBM,IOM,agent based models)

simple rules -> complex behavior

Simplest form:

Individual simple (in)finite state machine
"schedules" its own next "event" in continuous time Interacts with (potentially complex) environment
and other individuals like it

LOCAL information determines behavior

Variable structure: not invariant set of interaction partners
(in contrast to CA, Boolean nets)

distributed (shared) environmental "memory"
$--> $ flexible behaviour from rigid rules / automatic adaptation

The best model of the world is the world
internal model of the world as "crux" for information shortage

contexts
Ethology; Ecology, Evolution, (also transcription) swarm intelligence
'intelligence without reasoning (Brooks)'

simple rules to complex behavior

cf " Simon (1969)

"an ant seen as a behavioral system is quite simple -
the apparent complexity of its behavior is due to the complexity of the environment in which it finds itself"

" a human seen as a behavioral system is quite simple -
the apparent complexity of his behavior is due to the complexity of the environment in which it finds himself "

'

TODO as alternative explanation for observed behavior
Social structure as side-effect of foraging

Question: social structure of chimpanzees
Why all males groups in Chimpanzees?
Why single females?
Why do males travel further?

Modeling strategy: Make model WITHOUT behavior we are interested in
(but include some basic structure of system under consideration)
and OBSERVE

individuals: (CHIMPS)
- go to nearest fruit tree and eat until satisfied or fruit exhausted - rest
- males : search for receptive females - females eat protein food not eaten by males

environment: GOMBE-like

$- - - - - >$ Social structure of Chimpanzees

opportunity vs optimality based explanation

. Hogeweg & Hesper 1990; te Boekhorst H. 1994

MCHIMP

```
if        DARK then SLEEP

elseif    thereis a CHIMP at 15<DIST<100 (ANGLE <120)then
           if TUMESCENT FCHIMP then   FOLLOW
           else GO TO the CHIMP most in front and
                if there is FRUIT at DIST <10 then EAT
                                  else REST (.RAND .02 .03)

elseif    thereis a FRUIT at 5 < DIST < 100 then
          GO TO FRUIT most in front and EAT

else      FORWARD (RAND 25 40)
          if just eaten then REST (.3 * amount eaten)
          else REST (RAND .02 .03)
```

FCHIMP

```
if        DARK then SLEEP

elseif    thereis a FRUIT at 5 < DIST < 100 then
          GO TO FRUIT most in front and EAT

elseif    thereis a PROT at 5 < DIST < 100 ANGLE<120 then
          GOT TO FRUIT most in front and EAT

else      FORWARD (RAND 25 40)
          if just eaten then REST(.3 * amount eaten)
          else REST(RAND .02 .03)
```

FRUIT

number	1200 (variable --> ca 250) (600 1800)
size	1 - 35 chimphours (2-70, 1-23)
renewel	5 - 10 days

PROT

number	250 fixed (125 275)
size	.03 * FRUIT
renewel	when eaten

MCHIMP

```
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```

FCHIMP

```
if        DARK then SLEEP

elseif    thereis a FRUIT at 5 < DIST < 100 then
          GO TO FRUIT most in front and EAT

elseif    thereis a PROT at 5 < DIST < 100  ANGLE<120 then
          GOT TO FRUIT most in front and EAT

else      FORWARD (RAND 25 40)
          if just eaten then REST(.3 * amount eaten)
          else REST(RAND .02 .03)
```

FRUIT

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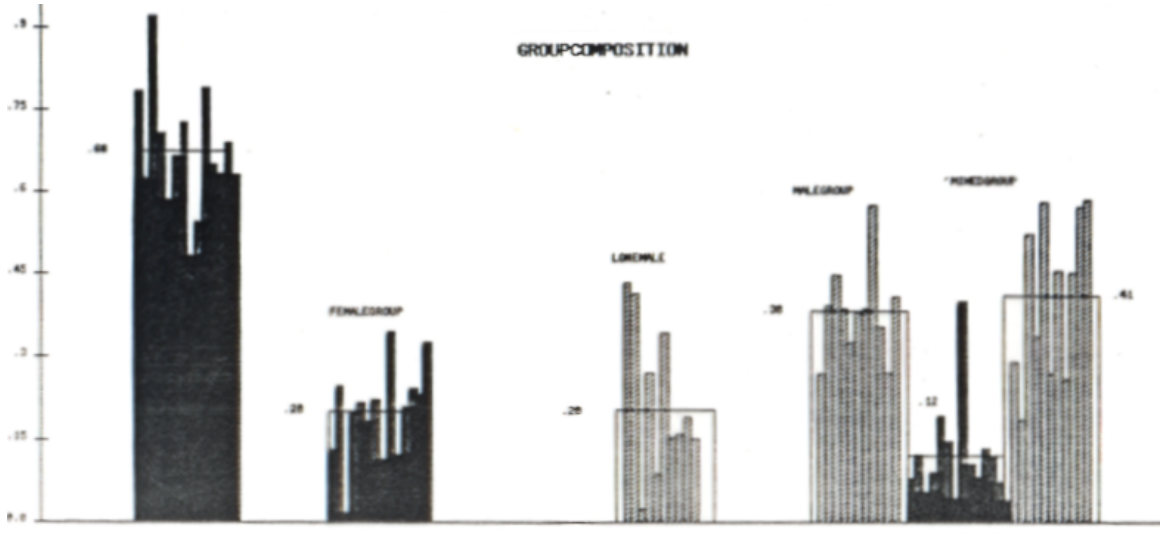
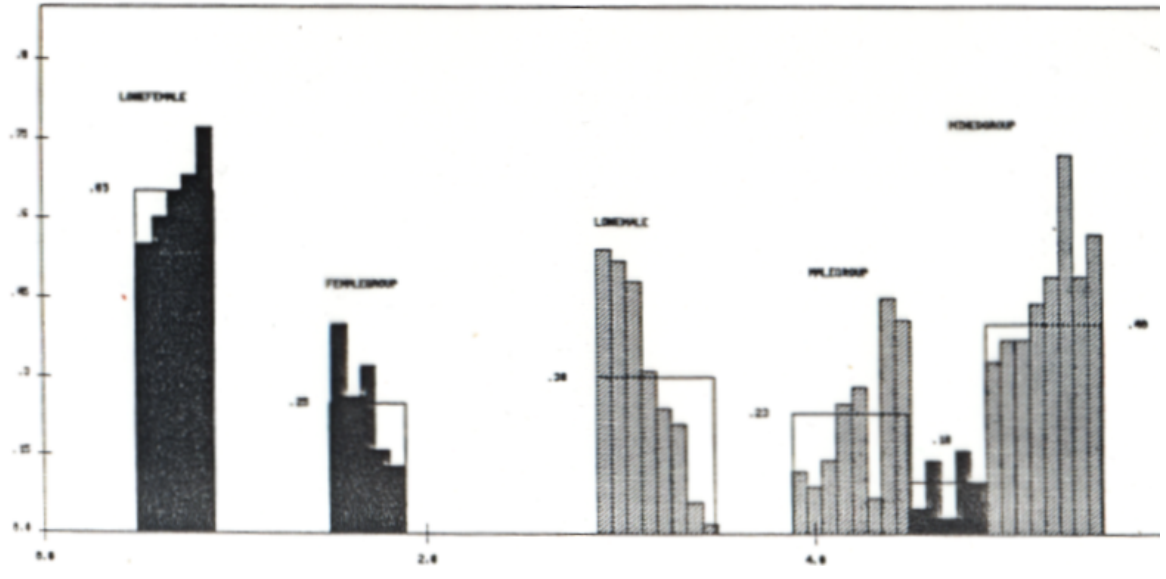
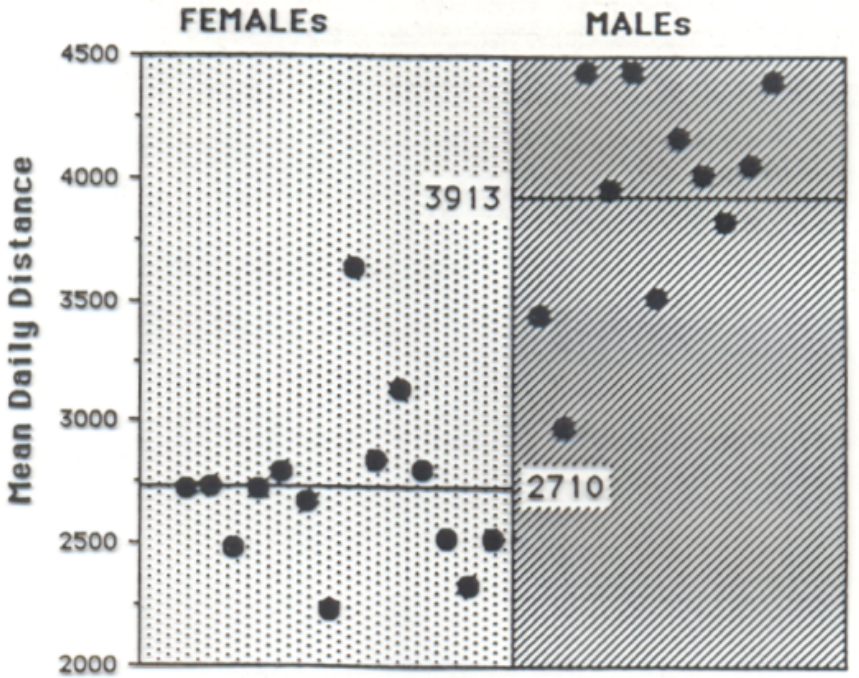
PROT

number	250 fixed (125 275)
size	.03 * FRUIT
renewel	when eaten



GROUPCOMPOSITION IN GOMBE 1972-1973 (HAPLERIN 1979)





exp: Females 2.7 Km
Males 4 Km

Fig. 5. Distance travelled by CHIMPs in the standard environment. Average values of individual CHIMPs are shown data from MALEs are in the dark shaded area.

IBM: continuous models of Collective behaviour: information integration

Much studied prototypes:

flocking birds (BOIDS, Reynolds)
schooling fish (e.g. Hemelrijk) migrating herds (locusts (Couzin), wildebeest (Levin))

Mostly (continuous) force-based models:
attraction, repulsion, aligning relative to
neighboring 'beasts' (variable set)

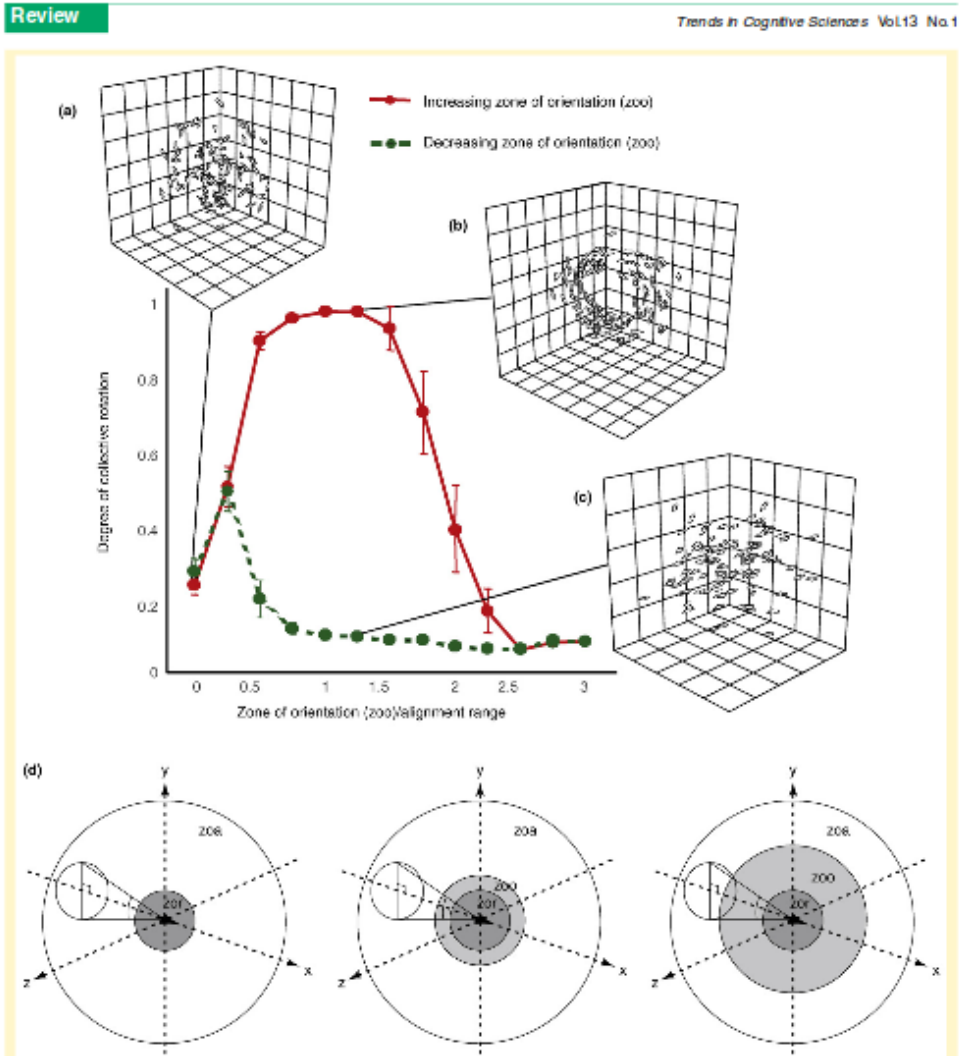
Different behaviours by different ranges
of vision (angle)
repulsion/attraction/aligning

minimal models
or more physics of environment
(air/water/)

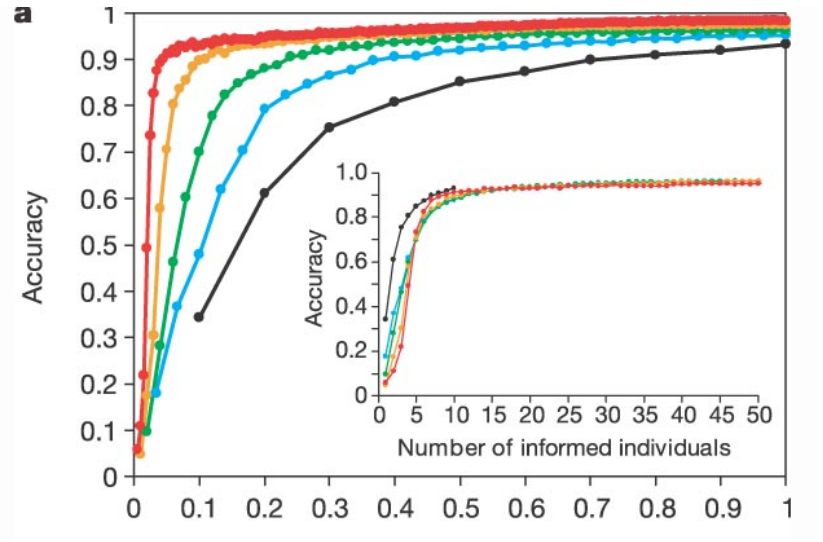
NO environmental memory

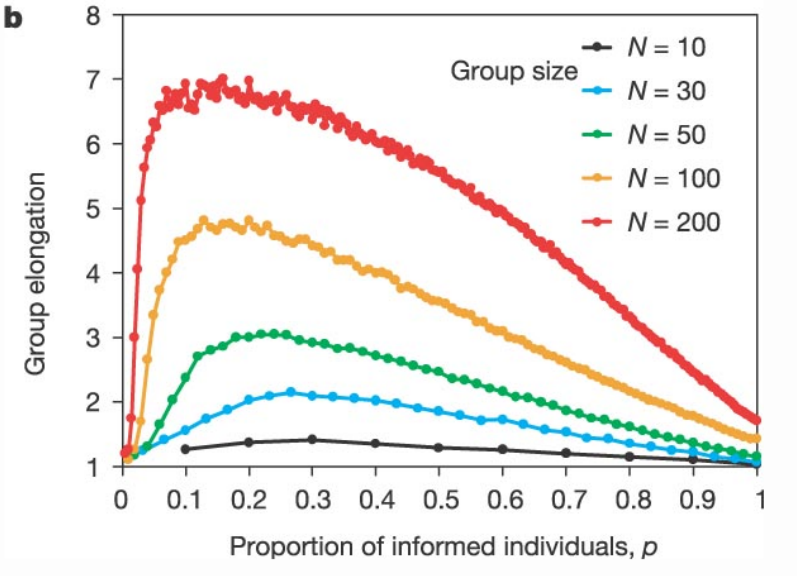
inert environment
basic grouping modes;
bistability

from Couzin 2009,
Trend cogn science



Effective decision making in flocks and schools
Couzin, Nature 2005





$$\mathbf{d}_i(t+\Delta t) = -\sum_{j \neq i} \frac{\mathbf{c}_j(t) - \mathbf{c}_i(t)}{|(\mathbf{c}_j(t) - \mathbf{c}_i(t))|}$$

repulsion

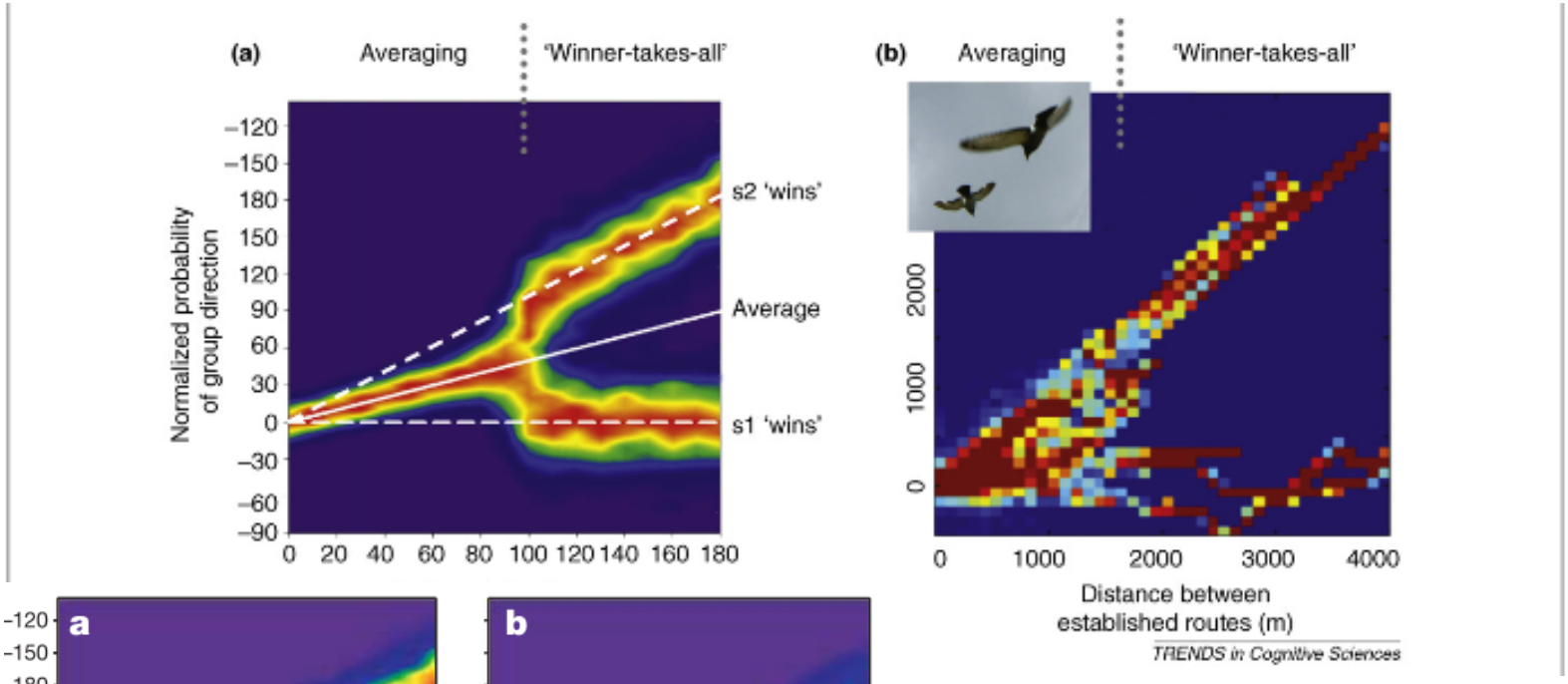
$$\mathbf{d}_i(t+\Delta t) = \sum_{j \neq i} \frac{\mathbf{c}_j(t) - \mathbf{c}_i(t)}{|(\mathbf{c}_j(t) - \mathbf{c}_i(t))|} + \sum_{j=1} \frac{\mathbf{v}_j(t)}{|\mathbf{v}_j(t)|}$$

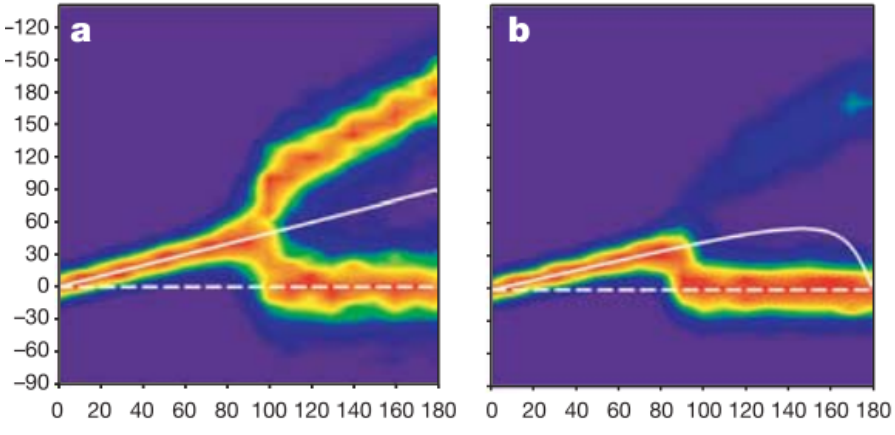
attraction + alignment

purpose:

$$\mathbf{d}_i{}'(t+\Delta t) = \frac{\hat{\mathbf{d}}_i(t+\Delta t) + \omega \mathbf{g}_i}{|\hat{\mathbf{d}}_i(t+\Delta t) + \omega \mathbf{g}_i|}$$

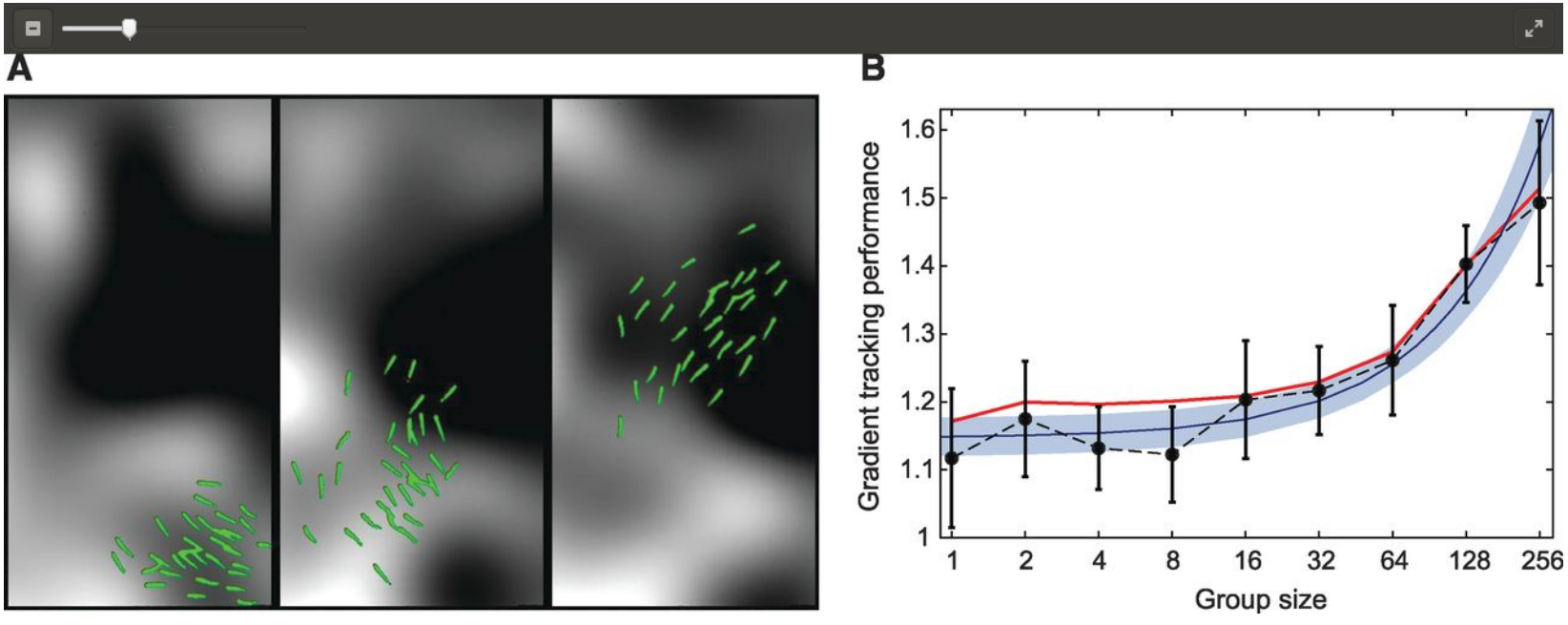
conflict resolution in schools and flocks





one more in group1

photo-taxis in fish groups, Berdahl,..,Couzin, Science 2013



Fish move slower in the dark

Larger schools can find dark places better