Roberto Kolter - Stationary Phase

Features of Stationary Phase

Develop Stress Resistance
Produce Antibiotics & Reserves
Alter cell shape - rod to sphere

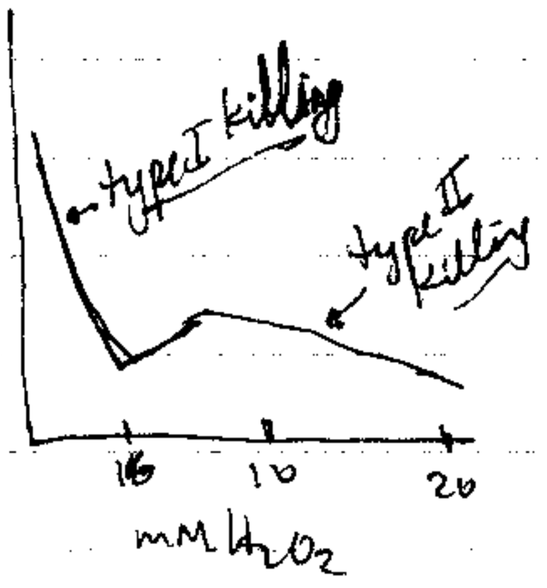
Induction

- rpoS = σ^S regulates 50+ genes
- including dps = pecB
  - discovered w/ 2D gels & S^{35} labelling
  - DNA binding protein

10/21/95

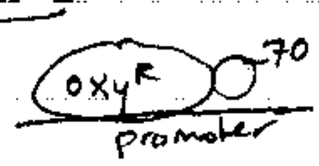
- KO -
  - hypersensitive to H_2O_2 in SP

mode - inducible operon
- in RecA⁻ cells
- apparently protects cells from damage even in log phase

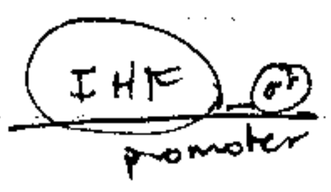


- induced by H_2O_2 also
- single promoter
  - same RNA made w/ H_2O_2 & SP treatment

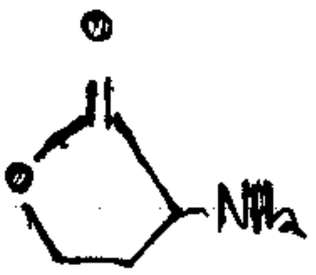
Log phase



SP



σ^S induction



- ppGpp
- UDP-Glc (repressin)
- HSL (homoserolactone)

rpo S → σ^S → induction

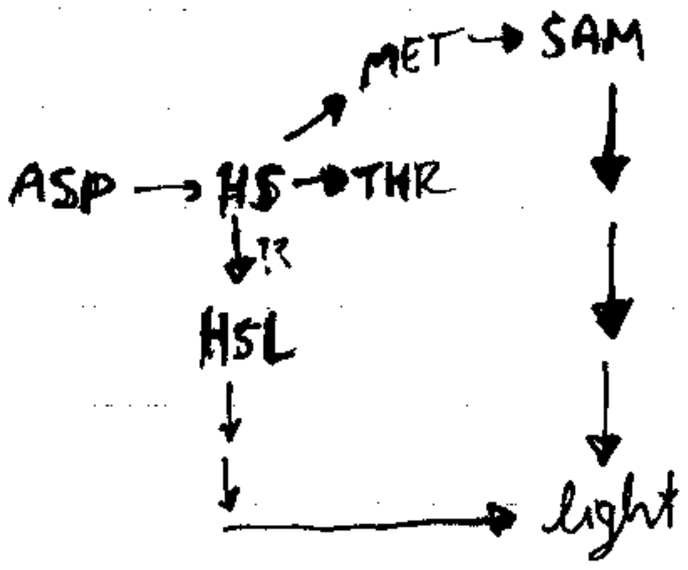
tx
tl

protein stabilization

induction → bolA (use bolA:lacZ)
→ dps
→ mcs
→ cfa (switch membrane composition)
→ KatE

Cell Density Signals (not required because cells still induce when at low density)

- acyl homoserine lactones
- induces σ^S



Homoserine

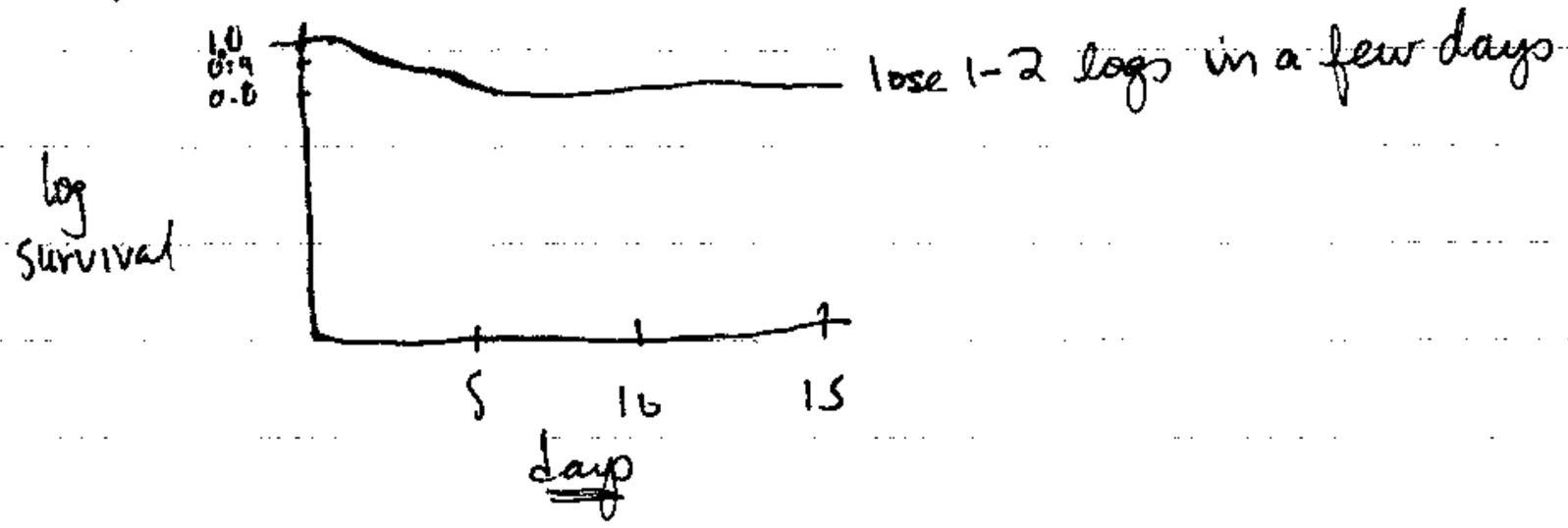
HS → adenylated HS → HSL

tRNA synthetases

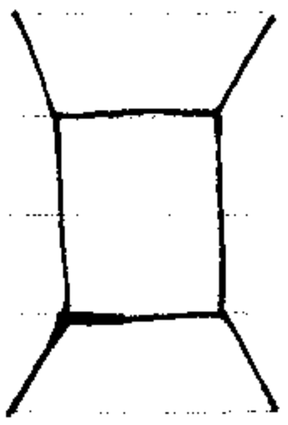
internal cyclization

- maybe when starved tRNA syn. accumulate

Population Behavior



- use vital stain acridine orange
  - stains RNA orange
  - cells that can't form colonies degrade ribosomes = not orange



① took cells that had survived 10 days
② mixed w/ normal cells
③ these cells take over when starved
④ the surviving strains are rpoS attenuated
  - most are rpoS mutants
  - these mutations cause growth advantage in SP (GASP)
  - mutation leads to inability to stabilize rpoS
  - these cells are better at competing for scarce nutrients than WT cells

- many strains in labs are rpoS mutants
- same with ~~GASP~~ samples in field

- GASP mutants → get 2nd mutation which KO GASP phenotype
  - onp^R - outer membrane protein
  - NHA - Na anti-porter
  - NADH dehydrogenase - electron tport

- maybe cells take something from dying cells

- GASP phenotype requires cell contact (well, suggested ... use a membrane to separate and it doesn't happen)
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