growth

(of cells, tissues and organisms)

Resources: http://golgi.ana.ed.ac.uk/coursenotes/cto2/cto2.html

Please note that all of the text of the live lecture is here but some images are not (because I do not have copyright permission to show them). The missing images have no content you need specifically to know – they are just the ones I show to illustrate a concept).

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Features/ problems of growth:

1. Anisotropy
2. Proportionality
3. Adaptability
4. Discontinuous scaling

Structure of the growth section of this course:

Lecture 1: Cell-level controls

Lecture 2: Cellular sources of tissue anisotropy

Lecture 3: Body size and body shape

Lecture 4: Keeping different tissue components in proportion.

Practical: Emergence of gross anatomy from cell-level events.

Limits to cellular growth

Transport

Communication/ coordination

*** mRNA synthesis ***

“Tricks” to make big cells

(1) – vacuoles

e.g. plant cells

(2) – syncytia

e.g. skeletal muscle

(3) – polytene chromosomes

e.g. fruitfly salivary glands

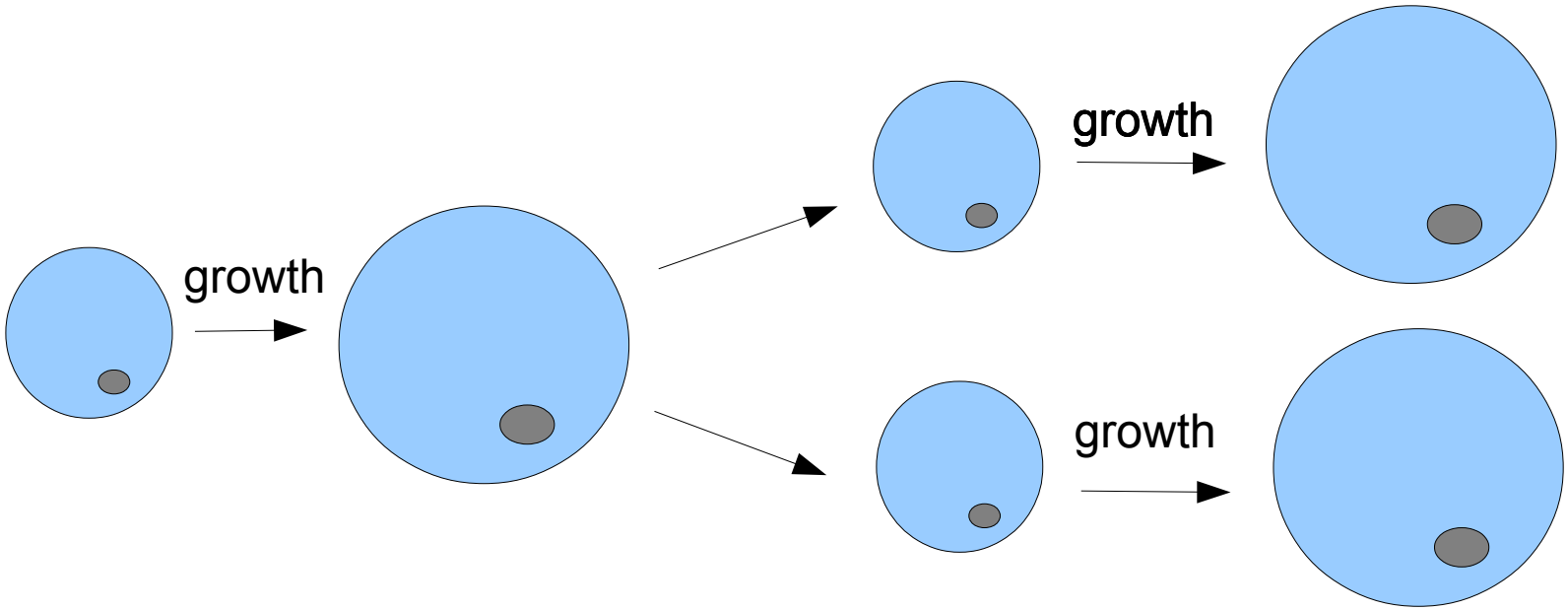
(4) – helper cells

e.g. granulosa cells in the ovarian follcile

But those are all odd, specialist tricks.

Generally, the problem is solved by cells remaining small but becoming more numerous.

Generally, cell division alternates with growth:



There are other patterns of cell division : see Dr Wilson's lectures later in the course.

This is normally drawn as a 'cell-cycle' so:

G1 → S → G2 → M

'G' = 'growth' (originally 'gap'); S = DNA synthesis, M = mitosis.

Early embryos decouple them, though (cleavage), and this can make analysis easier.

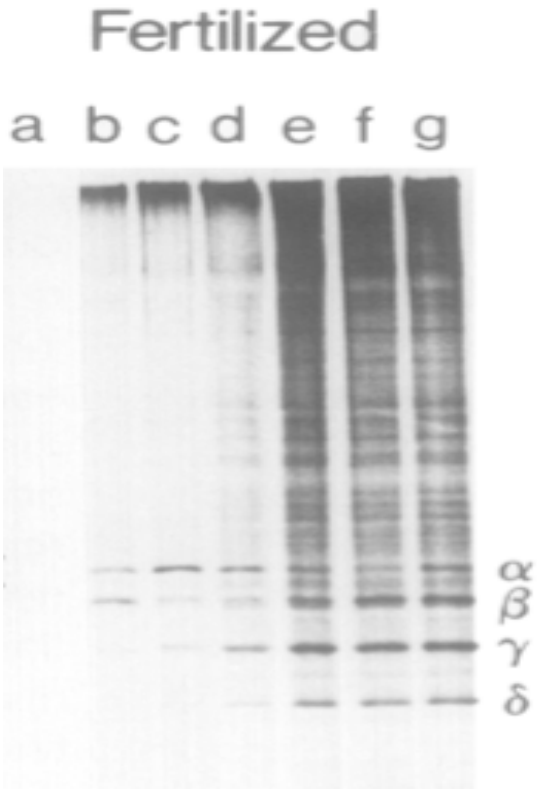
Analysis of proteins in cleaving sea urchin eggs showed periodicity of some:

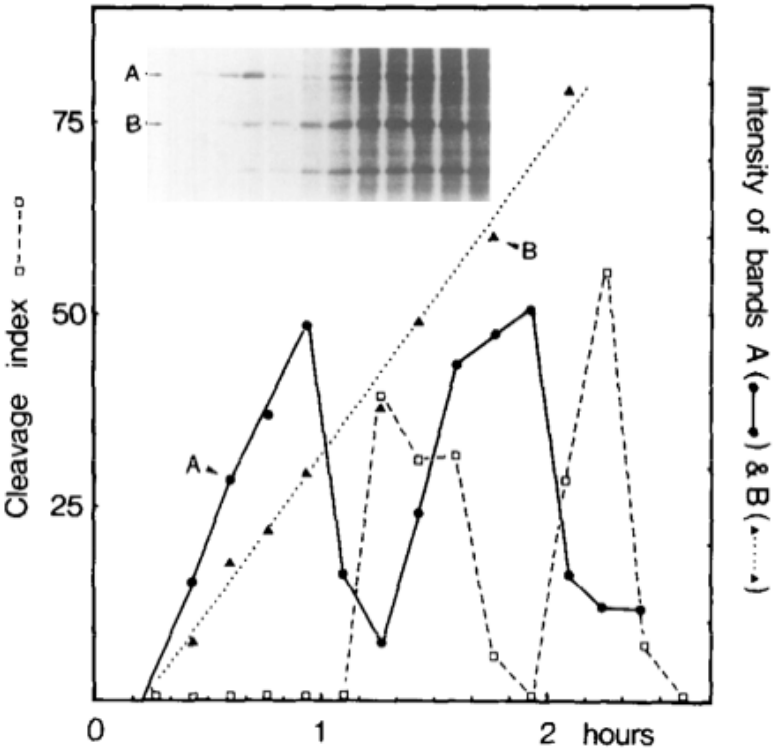
Cell, Vol. 33, 389–396, June 1983, Copyright © 1983 by MIT

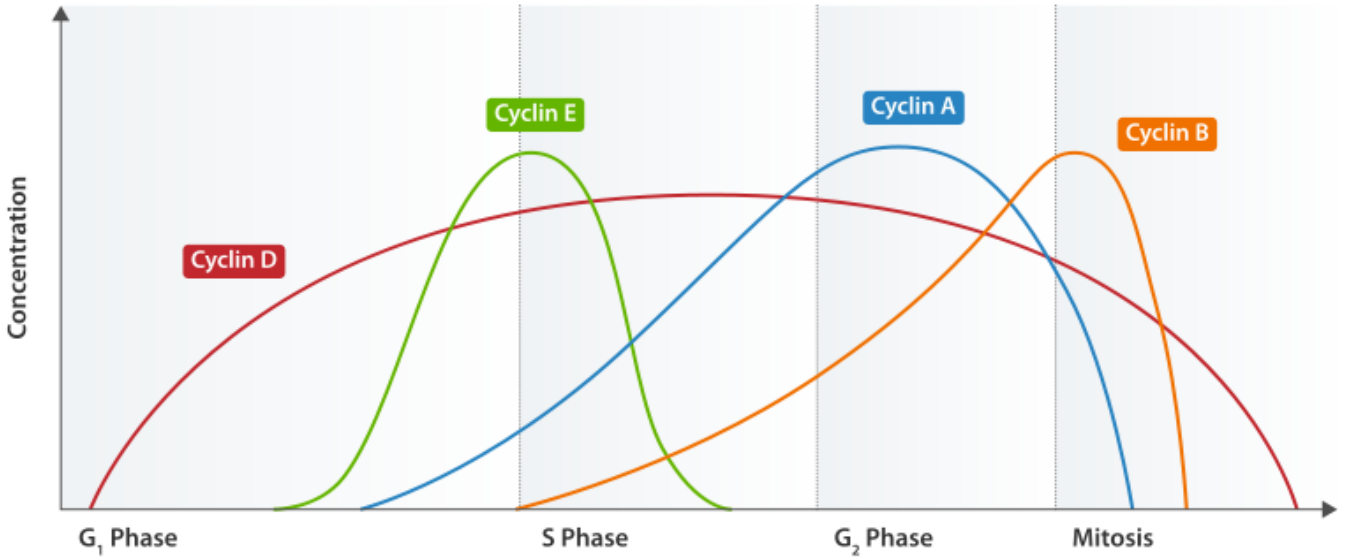
Cyclin: A Protein Specified by Maternal mRNA in Sea Urchin Eggs That Is Destroyed at Each Cleavage Division

Tom Evans,* Eric T. Rosenthal,† Jim Youngblom,‡ Dan Distel,§ and Tim Hunt‖

Tim Hunt






Concentration
Cyclin D
Cyclin E
Cyclin A
Cyclin B
G1 Phase
S Phase
G2 Phase
Mitosis

Cyclins work by controlling cyclin-dependent kinases (cdks)

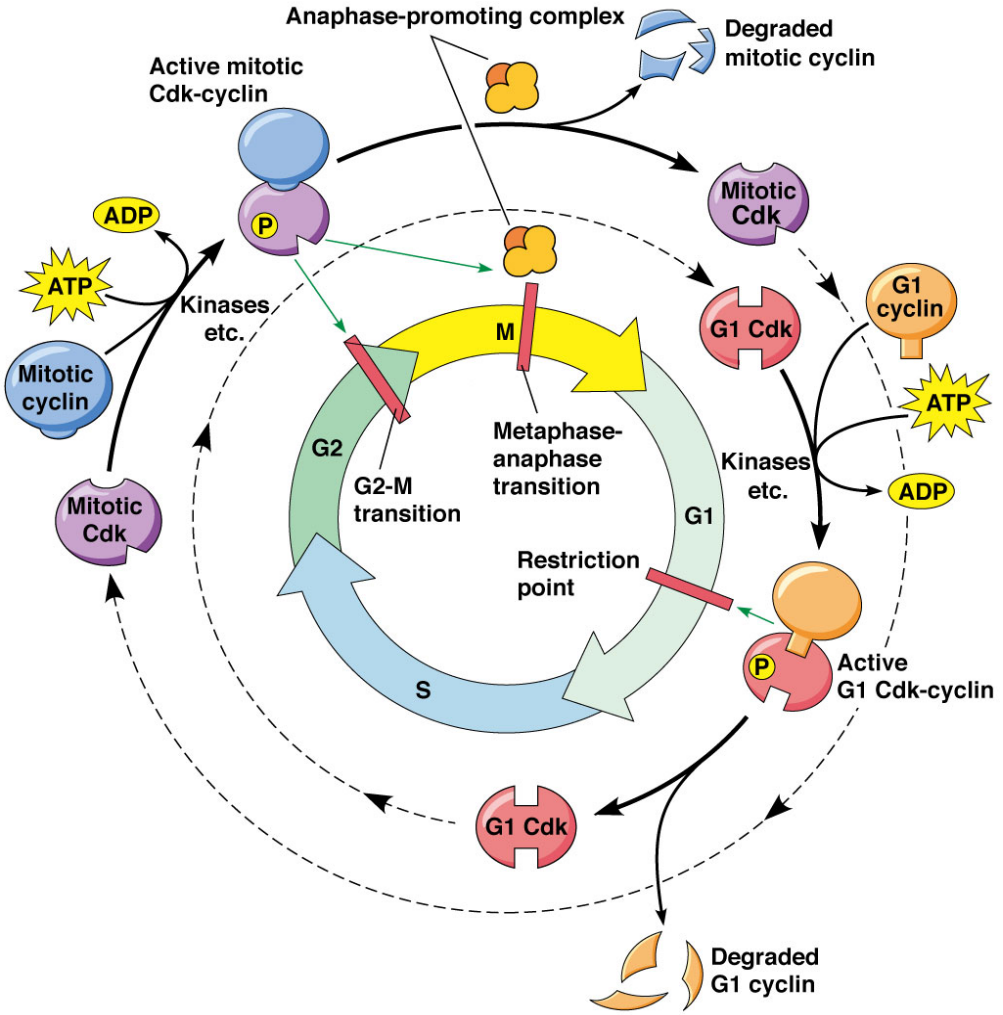


Image: Pearson education (with permission).

DO NOT feel you have to learn which cyclin goes with which cdk, and which cdk controls what: learning lists like that is pointless. If we set any exam questions on this topic, we will give you the necessary cyclin-cdk information in the question.

There are checkpoints:

G2 → M Progression blocked if DNA damaged

G2

S

M

G1 → S transition blocked unless:

G1

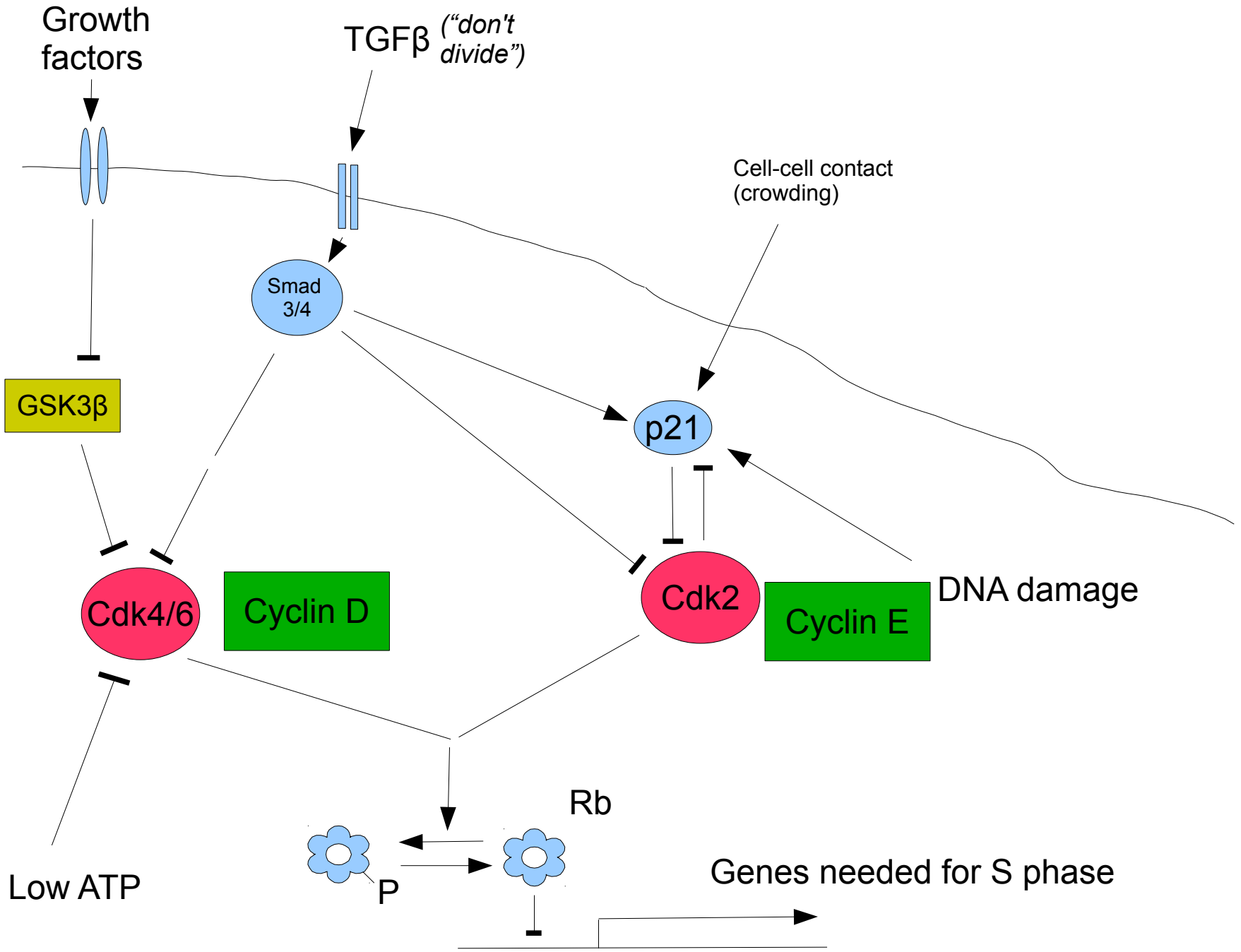
1) The cell has enough resources

2) The cell has enough room

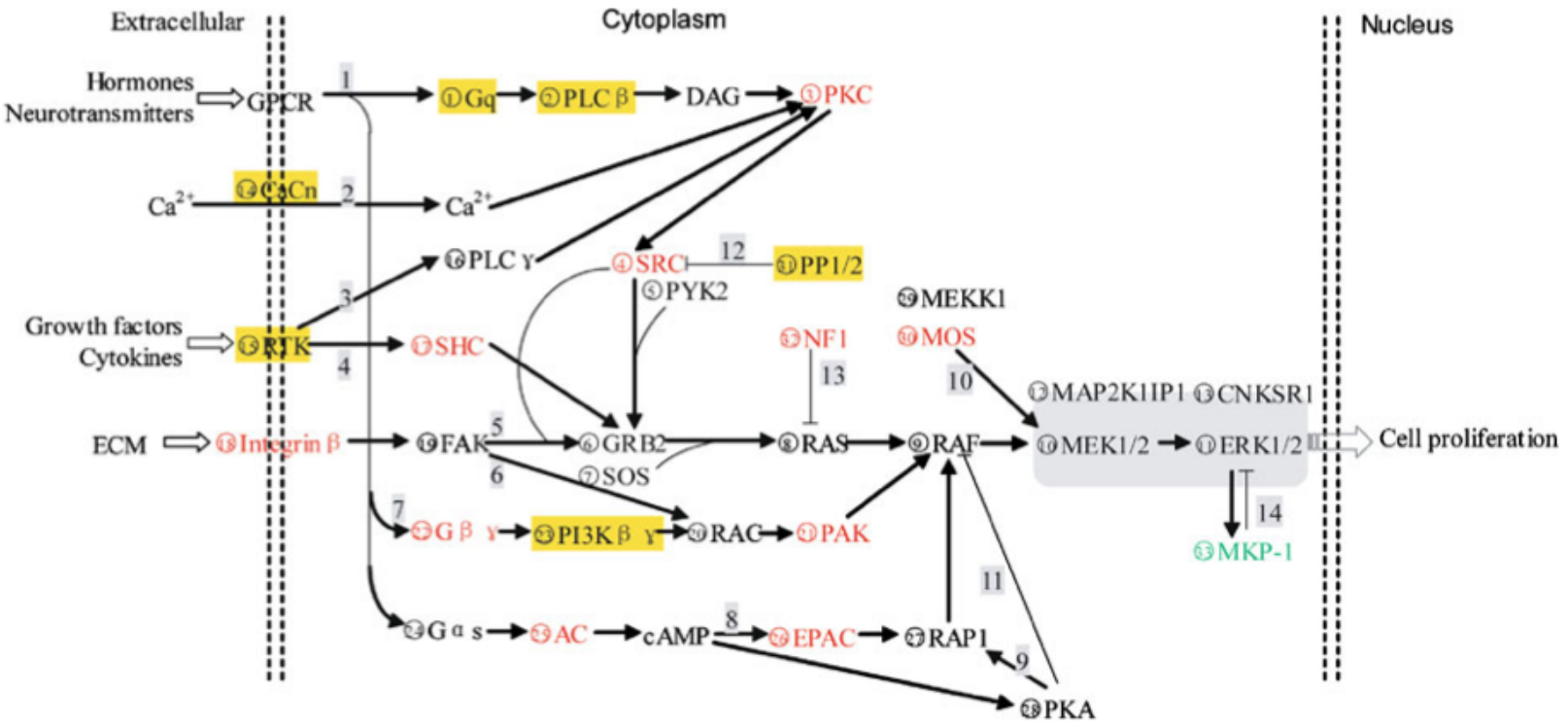
3) The cell has external signals asking it to divide.

4) There are no signals saying 'do not divide'.

Data source:
http://www.biocarta.com/pathfiles/h_g1Pa
(although this image is my own)



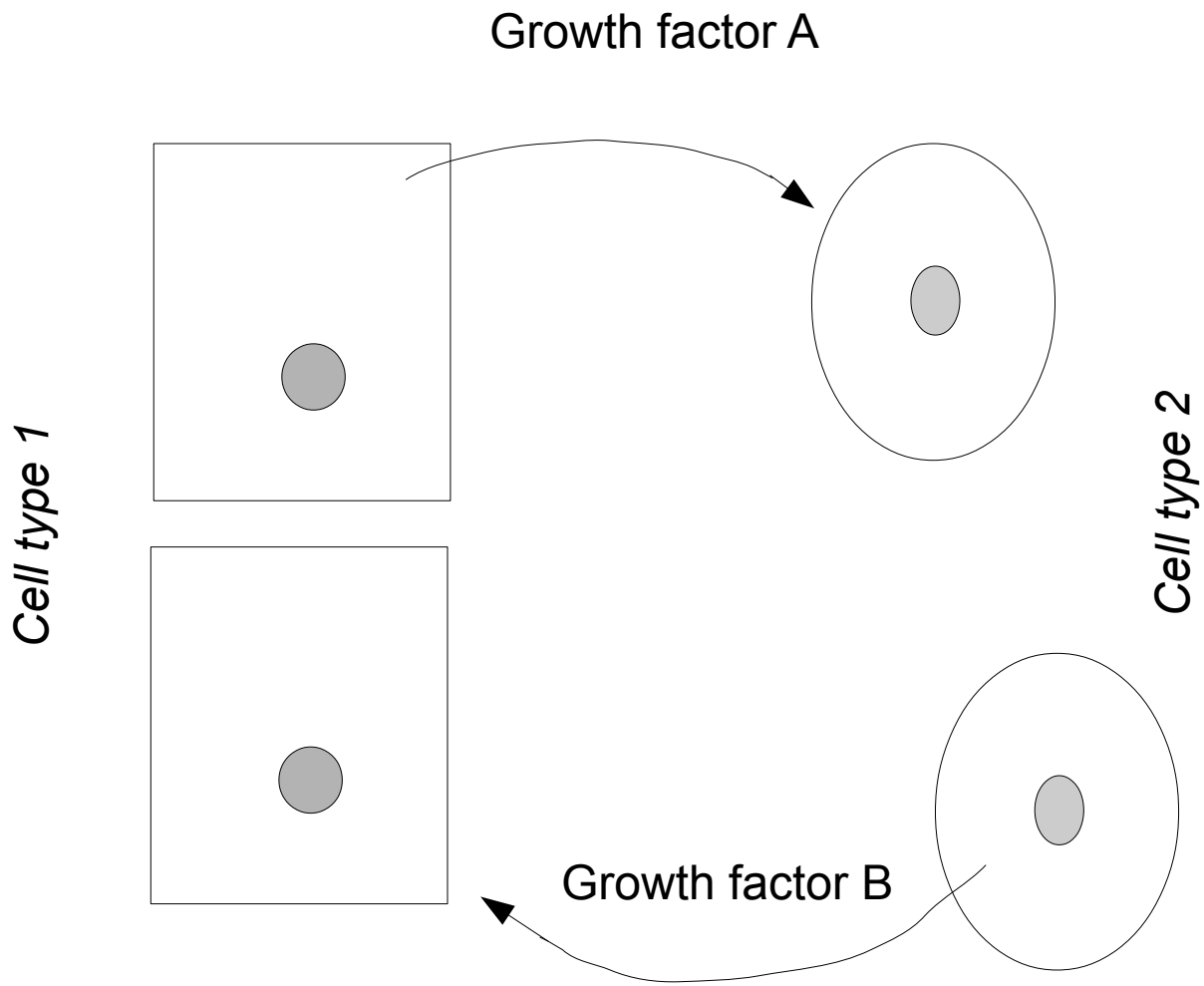
Warning: real cells pay attention to many growth signals and combine them in very complicated ways:



Eight paths of ERK1/2 signalling pathway regulating hepatocyte proliferation in rat liver regeneration

J. W. LI[1], G. P. WANG[1,2], J. Y. FAN[2], C. F. CHANG[1,2] and C. S. XU[1,2*]

Generally, cells make growth factor signals for one another (paracrine signalling)



We will explore the consequences of this in lecture 4

Cancer cells pay less attention to external signals

"Self-sufficiency makes me feel high:",
said the cancer cell. "I will not die!
Autocrine as I am,
I don't give a damn
that there's no growth factor supply".

This lecture has stayed at the scale of a cell.

In the next session, we will consider how growth of tissues relates to behaviours of cells.