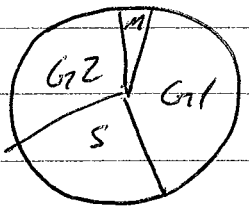
CH 12 - Cell Cycle

I. Cell Cycle



G1 - Gap 1

S - DNA synthesis

G2 - Gap 2

M - Mitosis - nuclear division

C - cytokinesis - cell seperation / ÷

II. Mitosis

A. Mitotic Spindle

- composed of protein tubulin (microtubules)
  in centriole 9 sets of 3 tubes
  located in center of centrosome

(asters)
① centrosome replicates + hangs out near nuc.
② move apart to opposite ends of cell
③ pair of chromatids join @ centromere
which contains 2 kinetochores

(premetaphase)
- spindle microtubes attach @ kin
④ microtube pulls kinetochore toward pole

(metaphase plate)
which lines them up in CTR of cell
⑤ shortening of tubes seperates chromo's
⑥ non-kin tubes elongate stretching cell

B. Phases

(1) Prophase - chromatin coils
- nucleoli disappear
- sister chromatids join
- mitotic spindle forms w/ lengthen asters from centrosomes
- centrosomes move away to poles

(2) Prometaphase - nuc. envelope disappears
- tubes interact w/ chromo
- kinetochores form
- spindles begin to connect to kin
- non-kin. tubes connect w/ opposites

(3) Metaphase - centrosomes opposite
- metaphase plate forms w/ sis. tids on either side of plate

(4) Anaphase - chromo's seperate along tubes releasing tubulin subunits
- walk towards pole spitting out ends
- non-kin tubes lengthen

(5) telophase - nuc. envelopes form
- chromos → thin (uncoil)

C. Cytokinesis

(animals) ① cleavage furrow forms
- Ring of actin + myosin @ meta plate contract
- pinches in cell

(plants) ② - vesicles from golgi move to middle of cell joining to form cell plate
- vesicles contain material for new cell wall membrane from golgi joins to form new membrane for each new cell

D. Other Org's
① Binary fission

II. Regulation

A. Molecular control

- cycle progresses due to chem signals in cyto

① Checkpoints

- point in time when go/no go signals
- typically no go until go is given
  - if cellular processes adequate go goes

a) G1 - Restriction point

- go divides
- No go cell stays in G0

G2→b) Kinases that drive cell [constant]

- activated by joining to cyclins
  - a.k.a cdk (cyclin-dependent kinase)

- need ↑ MPF (M phase promoting factor) to pass G2 checkpoint
- MPF = cdk + cyclin

  as cyclin ↑, MPF ↑

  when MPF reaches level mitosis begins
- after mitosis MPF split + cyclin degraded by proteolytic enzymes

(FEEDBACK)

Metaphase: if Anaphase is halted until all spindles are attached to kinetochores

After procedin prevents odd separation or no separation

② Signals for checkpoints

a) internal - M phase checkpoint - prevents disjunction by insuring kinetochores bound to spindle

b) External - growth factors

i) - platlet derived growth factor (PDGF)

- Required for division of fibroblasts
- have PDGF receptors in membrane that triggers signal transduction path

ii) density - dependant inhibition

- cell div. stops when too crowded

iii) anchorage dependance

- must attachte to extracellular matrix to divide (like an oyster)

III. Cancer

- do not listen to normal signals
  ex. - don't exhibit density dep. inhib
- immortal - continue to rep. (telomerase)
  - normal go through 20-50 cycles

(A) Tumor

- mass of abnormal cells

1) benign - Remain @ original site
2) malignant - impairs function of 1 or more organs
3) metastasis - spread through body

~~[redacted]~~

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B. Molecular Regulation

- Δ in DNA causes regulation to stop

(1) Onco genes - cancer causing genes
proto-onco genes - genes for normal cell growth + ÷

- Δ from proto ⇒ onco caused by
  a) movement of DNA w/i genome
  b) amplification of proto gene: ↑ # copies of gene
  c) point mutation
     a { - DNA broken + rejoined incorrectly
       - translocation near active promotor

(2) Δ in genes which inhibit cell div.

- tumor-suppressor genes
  - by Δing these cells continue to grow
  - stimulate growth due to lack of suppression

accelerator genes

braking genes

- ① + ② example

a) - Ras proto-oncogene mutated ≈ 30% human cancers

- p53 ≈ 50%

- Ras = G protein relays growth signal to stimulate cell cycle - normally only works w/ growth factor however mutated works w/o

b) - p53 or gaurdian angel gene - Rec. Δ in DNA

- activates ~~production of~~ gene p21 which halts cell cycle by binding to CDK's ∴ allowing cell to Repair DNA

- can turn on Repair of DNA

- can activate apoptosis

∴ p53 prevents mutations from passing on

G2 checkpoint

③ Most cancers due to accumulation of mutations

a) several tumor-suppressor genes

- most Recessive ∴ need to affect both alleles

b) 1 oncogene

c) telomerase activated

④ Build new blood supply