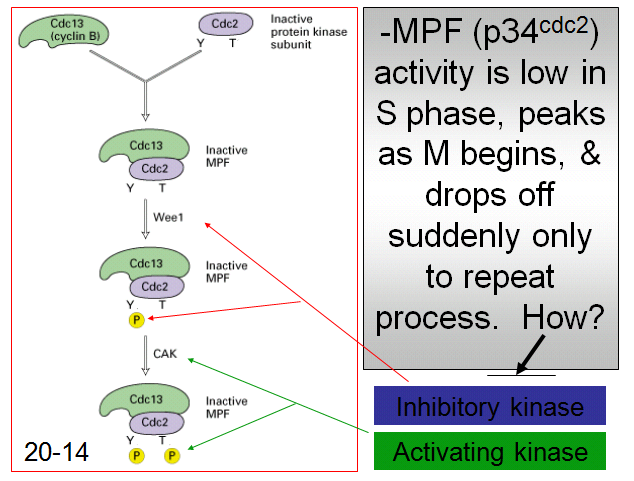
**Cell Cycling – Feb 1**

- some rote memorization and one liners

- thought questions -> change ingredients

- might not be pr/amt but the activation of pr

CDCs which changes in cell cycle

MPF = heterodimer

- cdc2 is transcribed and translated throughout

cell cycle and encodes a protein kinase

(p34cdc2) in all euks

- kinases phosphorylates Ser, Thr, Tyr

- inactive MPF complex forms and cell is

checking to make sure all conditions met

- repair needs to be done

- Wee1 (kinase) attaches phosphate onto Y (tyrosine)

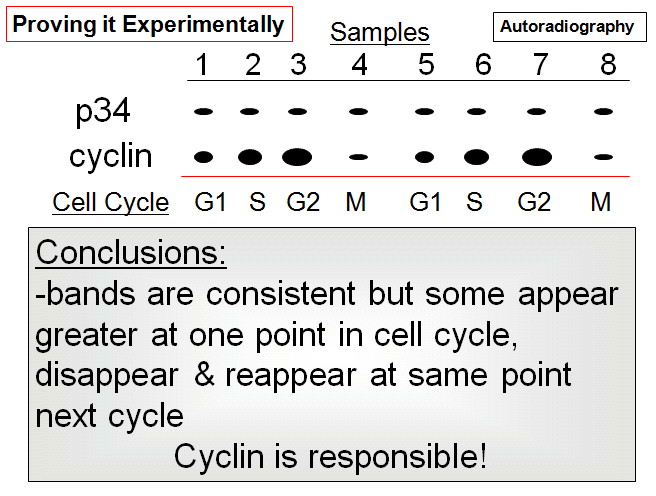
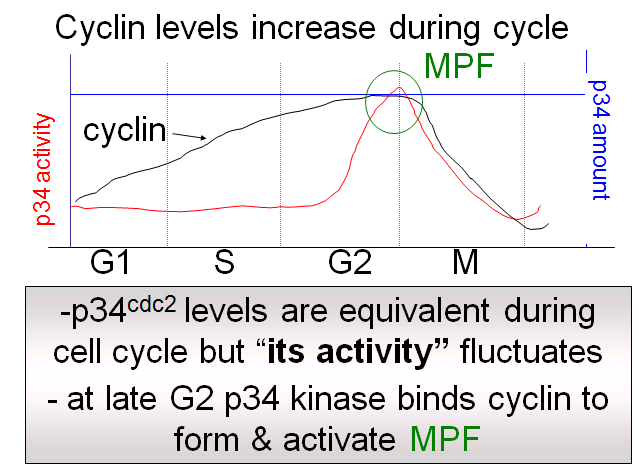
- mutate Wee1 and phosphorylation event will

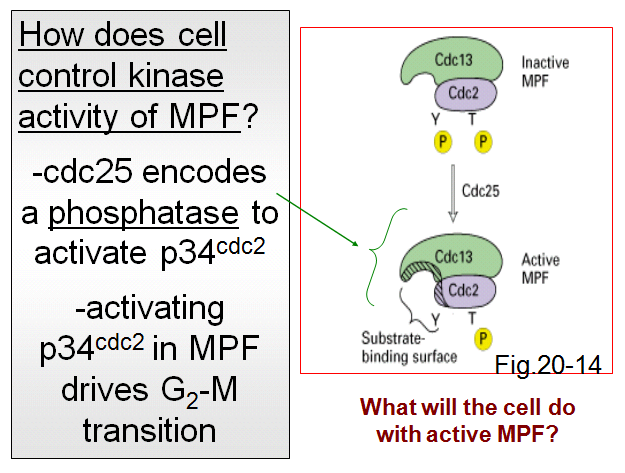
not occur -> you get smaller cells

- CAK (activating kinase) attaches phosphate

onto threonine

-> **STILL INACTIVE MPF**



- autoradiography at consistent time pts

- p34 is expressed at equal lvls throughout cell cycle

- cyclin lvls start increasing

- if cyclin lvls go up, it is a good predictor of what

the enzyme p34 will do

-> more cyclin produced which associates with p34

and MPF complex forms

How do you activate MPF? ------------------------------🡪

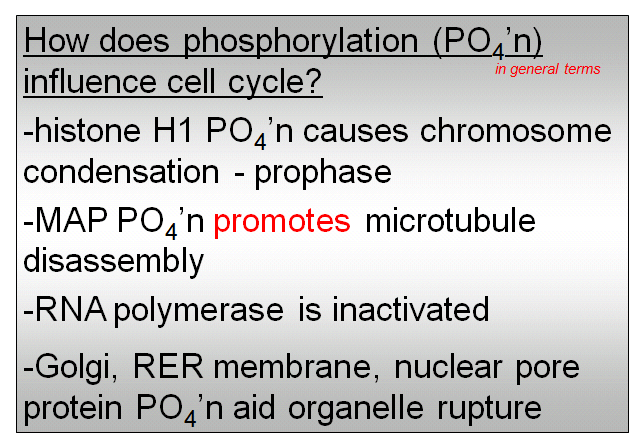
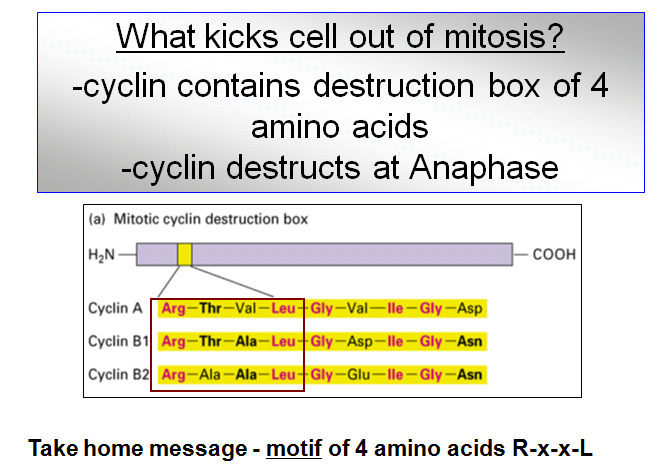
Q: What happens if you mutate wee1? \*\*\*

What happens if mutate CAK? CAK doesn’t do anything

to cyclin!

If CAK mutated, mitosis will be hindered

- mutating wee1, CAK puts on P and premature division



- phosphorylation of H1 causes chrs to condense

- MAP: mt associator pr

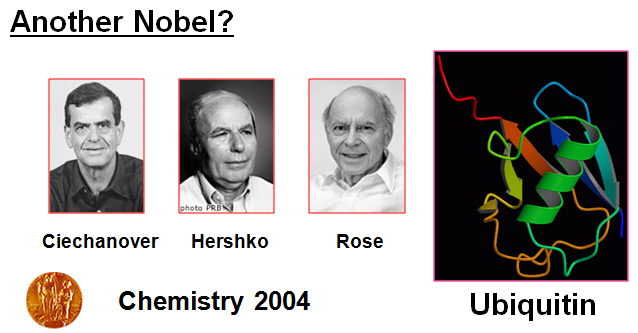
- where are mts used in mitosis and why would u

want to promote disassembly?

-> MFP finds MAP and causes mts to fall off

- MFP phosphorylates prs and organelles collapse - cyclin has destruction box; pr binds to another pr and and reassemble in 2 daughter cells degraded

- destruction boxes in cyclins is near amino terminus

- shut down RNAP (transcription) to conserve E,

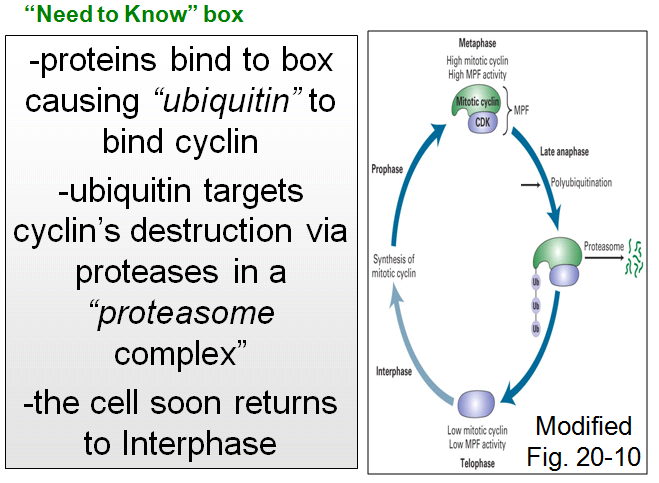
stop gene expr, loosen up DNA

- no transcription during mitosis since DNA tightly packed

- phophorylate RNAP (and dephosphorylated after rep)

- in a ribbon diagram what does the barrel denote

How about the arrows



- ubiquitin ligase, pr-pr interaction

recognizes destruction box at amino terminus

and during late anaphase, UL starts

adding ubiquitin to carboxy terminus of cyclin

- UL

- any pr that has ubiquitin moves to

proteasome

recognizes destruction box

- destruction box and ubiquitin go hand in

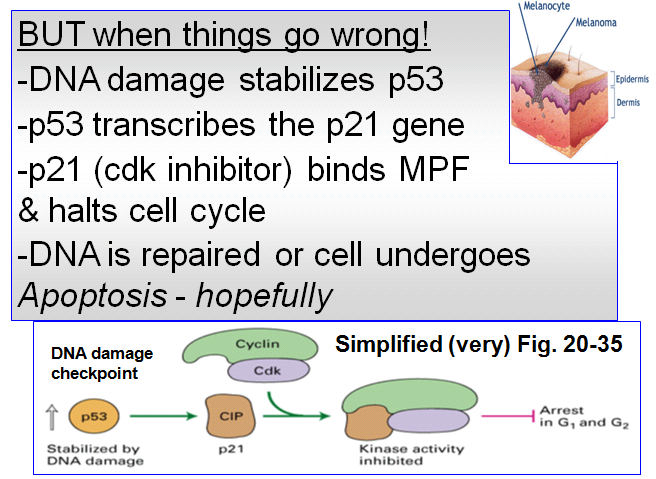
hand with proteosome

- any pr w. ubiquitin

- 80% of prs enter proteasome and are

Degraded and aa’s reused for next round

of translation

- p53 is a TF (bind unique regions of promoter)

- p53 is a tumour suppressor which ensures

that tumour doesn’t develop

- TF has short half life

- p53’s half life increases: gets stabilized in

cytoplasm and gets into nucleus (after DNA

Damage) and turns on p21 gene

- p21 encodes pr called cdk inhibitor which

binds to MPF -> MPF INACTIVE

- cell undergoes repair mechs or apoptosis

- 2 hallmark features of apoptosis:

1. characteristic fragmentation of DNA (nucleases)

2. blebbing -> bulging in the plasma membrane

caused by cell's cytoskeleton breaking up

-> final checkpoint is p53 action

\*\*\*KNOW THE NOTES!