

Regulation of Cell Cycle

Learning objective 4.6.A Describe the events that occur at checkpoints in the cell cycle.

Name:

Date:

Period:

Use the tool **Permission to Divide**. Part 1 uses **Try to get through**, Part 2 uses **Cyclin and CDK** and Part 3 uses **When a checkpoint fails**. The idea to hold on to throughout: a cell does not divide because it **can**. It divides because nothing stopped it.

Part 1 • Where does the cell get stuck?

Open **Try to get through** and press **Everything in order**. Then switch off one condition at a time, putting it back before the next. Predict where the cell will stop before you look.

Condition switched off	Predicted stop	Actual stop	Why that checkpoint asks about it
The cell has grown large enough to divide			
Nutrients and energy are available			
A growth signal from other cells has arrived			
The DNA is undamaged			
Every chromosome has been completely and correctly replicated			
Every chromosome is attached to spindle fibers from both poles			

1. One condition appears on two checkpoints' lists. Identify it and explain why it is worth checking twice.

2. Switch off a condition that no checkpoint before it asks about, and explain why the earlier gates stay open.

Part 2 · Which line decides the timing?

Switch to **Cyclin and CDK**. Leave the threshold at **0.70** and drag **Where in the cycle you are looking** to each tick below.

Tick	Cyclin	CDK	Complex activity
0			
20			
42			
60			
70			
85			

3. Compare tick 20 with tick 60. State what is the same, what is different, and what that tells you about which molecule sets the timing.

4. Now set the threshold to 0.30, then to 0.90, and record the size of the active window each time. Describe the pattern and explain it from the shape of the cyclin curve.

Part 3 · Four things that can happen next

Switch to **When a checkpoint fails**. A cell reaches the G1 checkpoint with damaged DNA. Step through all four outcomes and complete the table for **10 generations**.

Outcome	Cells	Carrying the damage	Fault kept out of the tissue?
The damage is repaired			
The cell stops dividing			
Programmed cell death			
The checkpoint is ignored			

5. Two of the four outcomes end with the same number of cells. Identify them and state what actually differs between them.

6. Explain why apoptosis is a useful outcome for the organism rather than a failure.

Part 4 · Putting it together

These do not need the tool.

7. Describe the roles of cyclins and of cyclin-dependent kinases, and explain how they work together to move a cell from one phase to the next.

8. Explain why the cyclin is destroyed after the transition it drives, and what that has to do with the cycle running in one direction.

9. A drug stops spindle fibers from forming. State which checkpoint would detect this, and predict what happens to a cell treated with the drug.

10. Explain how the failure of a single G1 checkpoint can lead to a tumour, and why the consequence grows so much faster than the original event would suggest.

What the model simplifies

Six conditions are shown. A real cell monitors many more, and the checks are carried out by networks of proteins rather than by a single yes/no reading; the conditions here are the ones the course asks for.

The checkpoints are drawn as gates that are either open or shut. A real checkpoint is a delay rather than a wall — the cell holds, tries to fix the problem, and continues if it succeeds, which is what the 'repaired' outcome represents.

One cyclin is drawn. A real cell uses several, each partnered with its own CDK and each peaking at a different point, so there is a different complex driving each transition. The shape of one is the shape of all of them.

The cyclin curve is a straight rise and a straight fall in arbitrary units and arbitrary ticks. Real cyclin concentrations are measured relative to a peak, and the fall is faster than the rise, which is the feature the drawing keeps.

The lineage model divides every cell at every generation and adds no new damage. Real tissue has cells that stop dividing, cells that die, and further mutations accumulating — all of which make the untreated case worse, not better, than the one drawn.