Study Guide — MI Unit 3: How to Conquer Cancer (Mid-Unit Test)

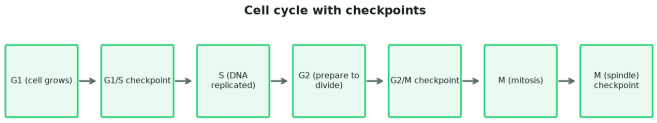
Medical Interventions (Genetics of Disease) · Unit 3 — How to Conquer Cancer · Modeled on the PLTW Guided Review format

Vocabulary to know cold

Term	Definition
Cell cycle (G1, S, G2, M)	G1 = growth, S = DNA replication, G2 = preparation, M = mitosis (division).
Checkpoint	A control point (G1/S, G2/M, spindle) that pauses the cycle to verify conditions before proceeding.
TP53 / p53	A tumor suppressor 'guardian of the genome' that halts the cycle for repair or triggers apoptosis on severe
Apoptosis	Programmed cell death; a normal way to remove damaged or dangerous cells.
Proto-oncogene	A normal gene that promotes controlled cell division; can mutate into an oncogene.
Oncogene	A gain-of-function (overactive) version of a proto-oncogene that drives excess division.
Tumor suppressor gene	A gene that restrains division/repairs DNA; cancer-linked mutations cause LOSS of function.
Missense mutation	A single base change that swaps one amino acid for another.
Nonsense mutation	A base change that creates a premature stop codon, truncating the protein.
Frameshift mutation	An insertion/deletion (not a multiple of 3) that shifts the reading frame of all downstream codons.
Benign vs. malignant	Benign tumors stay localized; malignant tumors invade and can metastasize.
Metastasis	Spread of cancer cells: invade → enter vessels → travel → exit → grow a secondary tumor.
Angiogenesis (VEGF)	New blood-vessel formation; tumors secrete VEGF to recruit vessels for oxygen and nutrients.

Worked examples (follow the reasoning)

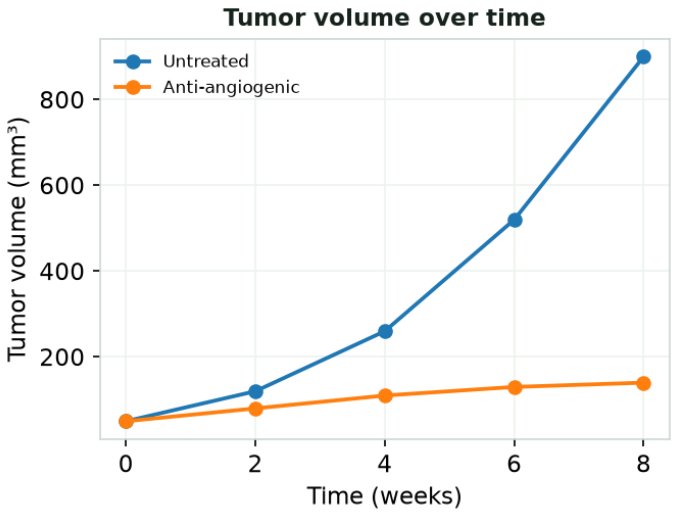
Example 1. Order the cell cycle and identify which checkpoint stops a cell with un-repaired DNA damage from entering mitosis.



- List interphase in order: G1 → S → G2, then M (mitosis).
- Locate the checkpoint that sits just before M: the G2/M checkpoint.
- Recall its job: confirm DNA was replicated correctly and is undamaged.
- If damage remains, p53 and the G2/M checkpoint hold the cell out of mitosis until repair (or trigger apoptosis).

Answer: Order: G1 → S → G2 → M. The G2/M checkpoint blocks a damaged cell from entering mitosis.

Example 2. An anti-angiogenic drug blocks new blood-vessel growth. Using the graph, compare tumor growth with and without the drug and explain the biology.



- Both tumors start at 50 mm³ at week 0.
- Untreated grows steeply to ~900 mm³ by week 8.
- The treated tumor nearly plateaus near 140 mm³.
- Without new vessels, the tumor cannot get enough oxygen/nutrients, so its growth stalls.

Answer: Blocking angiogenesis starves the tumor of its blood supply, so growth slows dramatically compared with the untreated tumor.

Practice questions (with reasoning)

1. A mutation makes an accelerator gene 'stuck on.' This describes:

A. A tumor suppressor loss
B. An oncogene (gain of function)
C. A silent mutation
D. Apoptosis

Answer: B. Gain-of-function in a proto-oncogene creates an oncogene that constantly signals division.

2. Inserting one base into a coding sequence causes:

A. A silent mutation
B. A frameshift mutation
C. No change
D. A duplication of a whole chromosome

Answer: B. A single-base insertion shifts the reading frame for all downstream codons.

3. Which is an early step of metastasis?

A. Staying inside the original tissue
B. Invading nearby tissue and entering a blood or lymph vessel
C. Losing the ability to divide
D. Becoming a red blood cell

Answer: B. Metastasis begins with local invasion and intravasation into vessels.

4. p53 detects irreparable DNA damage. The protective outcome is to:

A. Force immediate division

B. Trigger apoptosis so the damaged cell dies instead of becoming cancerous

C. Build new blood vessels

D. Make the cell larger

Answer: B. By triggering apoptosis, p53 removes dangerous cells before they can form a tumor.