PHARM.D • YEAR 3

Advanced Pharmacology

CHAPTER 3.13

# Chemotherapy of Cancer

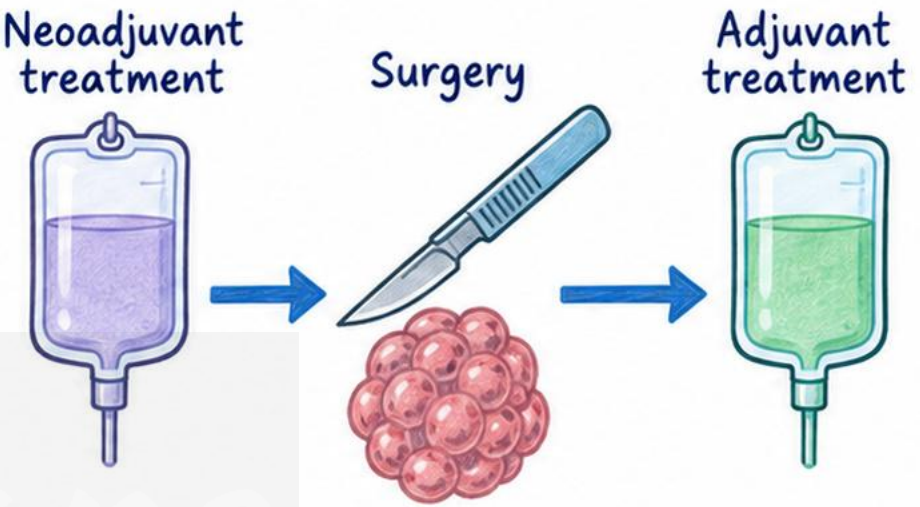
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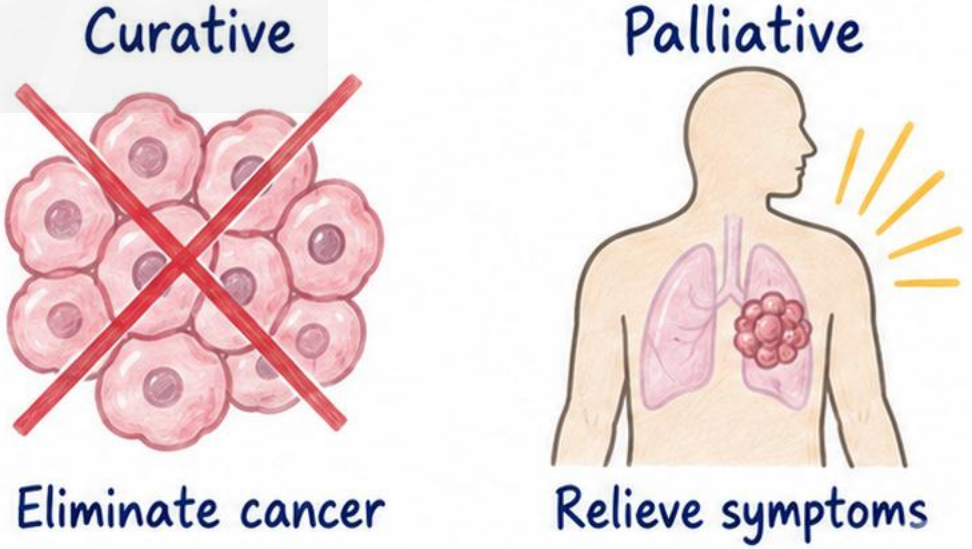


DOCTOR OF PHARMACY

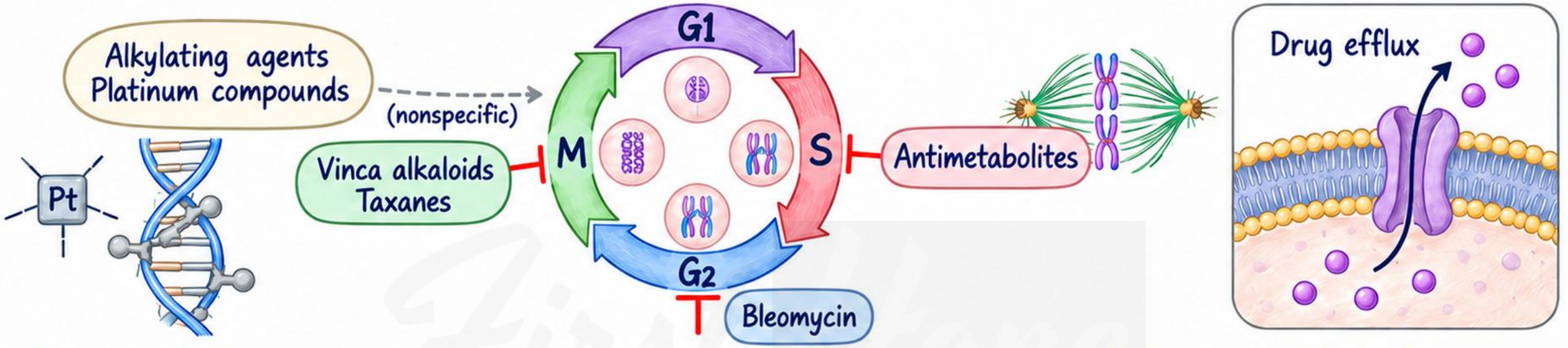
# Cancer chemotherapy: goals and selectivity

- Cancer treatment can aim for cure, prolong survival or relieve symptoms. Cytotoxic chemotherapy damages malignant cells but also affects susceptible normal tissues.
- Neoadjuvant treatment is given before definitive local treatment; adjuvant treatment follows it to reduce recurrence risk.
- Treatment can combine medicines with surgery, radiation, targeted therapy or immunotherapy.
- Not every cancer grows rapidly, and not every anticancer drug acts through general cytotoxicity.
- Histology, stage, molecular biomarkers and patient factors guide selection. Narrow therapeutic margins and resistance require planned schedules and monitoring rather than assuming that greater dose intensity always improves the benefit-risk balance.



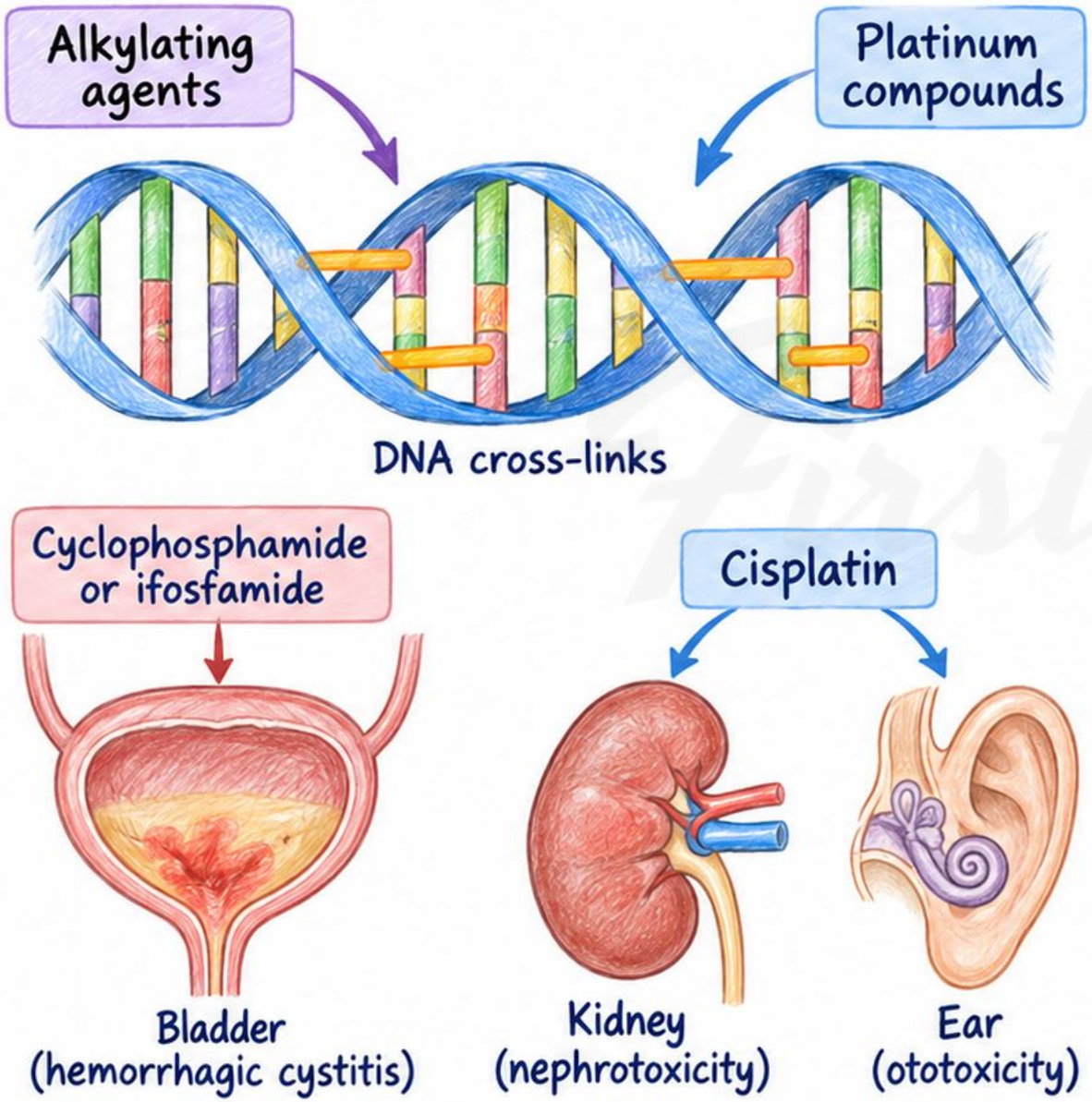



# Cell-cycle specificity and resistance




- Antimetabolites are commonly associated with S-phase activity, while vinca alkaloids and taxanes disrupt mitotic function.
- Alkylating agents and platinum compounds are generally cell-cycle nonspecific, although proliferative state still affects response.
- Bleomycin is a notable G2-associated agent; all antitumor antibiotics should not be assigned one identical cycle pattern.
- Repeated cycles balance tumor killing with recovery of normal tissues.
- Resistance can involve altered targets, enhanced repair, metabolic changes or drug efflux. Combining agents can improve activity, but overlap in toxicity and interactions must be planned. A classification label does not alone determine a safe schedule.

# DNA injury: alkylators and platinum drugs




- Alkylating agents such as cyclophosphamide and ifosfamide form covalent DNA damage, including cross-links.
- Platinum compounds such as cisplatin also create DNA cross-links that impair replication and promote cell death.
- Marrow and gastrointestinal toxicity are important class concerns, with additional agent-specific effects.
- Cyclophosphamide or ifosfamide can cause hemorrhagic cystitis. Hydration and mesna use depend on the particular regimen; mesna is not universal for every cyclophosphamide dose.
- Cisplatin causes nephrotoxicity and ototoxicity. Monitor organ function and protocol-specific supportive measures; carboplatin and cisplatin cannot be treated as identical toxicity or dosing profiles.

# Antimetabolites: three complementary examples

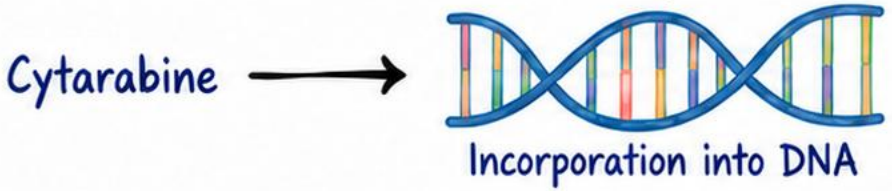
- Methotrexate is a folate antagonist that inhibits dihydrofolate reductase, limiting nucleotide synthesis.




- 5-Fluorouracil metabolites inhibit thymidylate synthase and can affect RNA or DNA pathways.




- Cytarabine is a nucleoside analogue that interferes with DNA synthesis.




- These mechanisms differ; not every antimetabolite simply mimics a DNA base.



Methotrexate (folate antagonist)



5-Fluorouracil (metabolite)



Cytarabine (nucleoside analogue)

- Marrow suppression and mucosal toxicity are important. Methotrexate also requires attention to renal clearance and third-space fluid accumulation.

- High-dose methotrexate protocols use monitored folinic-acid rescue and other supportive measures. NSAIDs, selected antimicrobials and other interacting drugs can increase toxicity; follow the actual oncology protocol.

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