💊 Antitumor Antibiotics

🔑 Key Concept

- Derived from microbial sources but used as anticancer drugs 🧫
- Mechanism: DNA damage via free radicals or intercalation

⏱️ Cell Cycle Specificity

- Bleomycin → G2 phase specific
- Anthracyclines → G2/M phase specific
- Dactinomycin → Cell cycle NON-specific ❗

💡 Core Mechanism (Big Picture)

Antitumor antibiotics damage DNA by:

- Free radical formation 💥
- DNA intercalation 🧬
- Topoisomerase inhibition

🔄 Flowchart: How Antitumor Antibiotics Kill Cells

Drug enters cell → Generates free radicals OR intercalates into DNA → DNA strand breaks / replication block → Cell cycle arrest → Apoptosis 💀

📊 High-Yield Drug Table

Drug	Mechanism	Clinical Use	Adverse Effects
Bleomycin	Free radical formation → DNA strand breaks	Testicular cancer, Hodgkin lymphoma	Pulmonary fibrosis, skin changes
Dactinomycin	DNA intercalation → inhibits RNA synthesis	Wilms tumor, Ewing sarcoma	Myelosuppression
Doxorubicin, Daunorubicin	Free radicals + DNA intercalation + Topo II inhibition	Solid tumors, leukemias	Dilated cardiomyopathy

🧪 Drug-by-Drug Notes

🅱️ Bleomycin

💡 Mechanism

- Generates free radicals → Causes DNA strand breaks → Cell cycle arrest (G2 phase) → Tumor cell death

📌 Clinical Uses

- Testicular cancer 🎯
- Hodgkin lymphoma

❗ Adverse Effects

- Pulmonary fibrosis (VERY high-yield ⚠️)

- Skin hyperpigmentation

🎯 Exam Pearls

- Bleomycin is unique because → Minimal myelosuppression (contrast with most chemo drugs)
- Think: "Bleo = Breath (lung toxicity)"

2️⃣ Dactinomycin (Actinomycin D)

💡 Mechanism

- Intercalates into DNA → Blocks RNA polymerase → Inhibits RNA synthesis

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🔄 Flowchart: Dactinomycin Action

Dactinomycin → Inserts between DNA base pairs (intercalation) → Prevents RNA polymerase movement → ↓ RNA synthesis → ↓ protein synthesis → Cell death

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📌 Clinical Uses

- Wilms tumor 🎯
- Ewing sarcoma
- Rhabdomyosarcoma

❗ Adverse Effects

- Myelosuppression 💉

🎯 Exam Pearl

- Think: "Dactino = DNA transcription blocked"

3️⃣ Anthracyclines

(Doxorubicin, Daunorubicin)

💡 Mechanism (MULTIPLE)

- Generate free radicals
- Intercalate into DNA
- Inhibit topoisomerase II

🔄 Flowchart: Anthracycline Action

Anthracycline → DNA intercalation → Topoisomerase II inhibition → Free radical formation → DNA strand breaks → ↓ replication → Cell death

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📌 Clinical Uses

- Solid tumors
- Leukemias

- Lymphomas

❗ Adverse Effects

- Dilated cardiomyopathy ❤️ (often irreversible ⚠️)
- Myelosuppression

🔄 Flowchart: Cardiotoxicity Mechanism

Anthracycline → Free radical formation → Oxidative damage to cardiac myocytes → Myocardial cell death → Dilated cardiomyopathy

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⚠️ Prevention

Cardiotoxicity risk → Give Dexrazoxane → Chelates iron → ↓ free radical formation → Protects heart ❤️

🎯 Exam Pearls

- Doxorubicin = "DOX dilates heart" ❤️
- Cardiotoxicity is dose-dependent
- Often tested with heart failure symptoms after chemo

🔥 Comparison Table

Feature	Bleomycin	Dactinomycin	Anthracyclines
Mechanism	Free radicals	DNA intercalation	Free radicals + Topo II inhibition
Cell cycle	G2	Non-specific	G2/M
Key toxicity	Pulmonary fibrosis	Myelosuppression	Cardiomyopathy
Unique feature	No myelosuppression	RNA synthesis block	Dexrazoxane protection

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🔑 Must-Know Points

- Bleomycin → pulmonary fibrosis, no myelosuppression
- Dactinomycin → inhibits RNA synthesis
- Anthracyclines → cardiotoxicity (prevent with dexrazoxane)
- Mechanisms:
    - Free radicals 💥
    - DNA intercalation 🧬
    - Topo II inhibition

-> The End <-