Anti-TB drugs

	Isoniazid (INH)	Ethambutol	PAS (4-aminosalicylic acid)	Rifampin	Cyclo serine	Streptomycin
Class:	Hydrazide	--	--	Rifamycin(ansamycins)		Aminoglycoside
MOA:	1. Inhibits synthesis of mycolic acid	Mycolic acid competitor on cell wall	PABA inhibitor for folic acid	Inhibits DNA-directed RNA polymerase		Inhibit protein synthesis
	2. Anti-metabolite of NAD					
Uses:	Anti-TB	Only for dividing mycobacteria	2nd line anti-TB that can be given orally as Na salt.	**Anti-TB and Anti-lepral drug.**		
Notes:			Synthesis involves Kolbe reaction then reduction.	Semi-synthetic from rifampicin.		Kanamycin, gentamycinamikacin can also be anti-TB.

Anti-tubercular combination **"Rimactazide"**: Isoniazid (INH) + Rifampin

Anri-lepral drugs

Dapsone (DDS)	Acedapsone	Sulfoxone sodium
Not water soluble or long acting so prodrugs were produced.	Orally bioavailable and long acting.	Can be injected (water soluble)
	From acetylation of dapsone.	Prodrug of dapsone.

Sulphonamides

Sulphanilamide	Sulfisoxazole	Sulfdiazine & Sulfametoxazole	Succinyl sulfathiazole	Sulfasalazine	Mafenide acetate
Original sulphonamide form from prodrug dye, prontosil, in-vivo activation by **azoreductase.**	Short-acting	Intermediate-acting	For GIT	For ulcerative colitis	Topical for burn therapy.
	It's rapidly excreted so concentrates in urine.	Silfadiazine: burn therapy	They're poorly absorbed so they concentrate in GIT.	Designed to be poorly absorbed from GIT to concentrate there.	Not a typical sulfonamide.
	UT antiseptic.	Sulfamethoxazole is chosen in the cotrimoxazole combination to have the same t1/2 as trimethoprim.	Prodrug hydrolyzed by amidase enzyme.	A prodrug that's activated by bacterial azoreductase enzyme into 5-ASA (not absorbed/remain in large intestine) & antibacterial sulfapyridine.	

MOA: Anti-metabolite of PABA during THFA synthesis.
- Inhibition of enzymes DHFS & DHFR can provide safety and selectivity.
Synergistic combination: **sulphamethoxazole + trimethoprim =Cotrimoxazole/septrin**