ENDOCRINE PHARMACOLOGY - TABLES PACK

ملف الجداول الشامل - القسم الثاني والثالث

Section 1: Part 2 - Comprehensive Drug Tables | Section 2: Part 3 - High-Yield Comparisons

SECTION 1 - PART 2	SECTION 2 - PART 3
MOA | Why / Use | Adverse effects | Contraindications | Memory link	Side-by-side comparison tables for the drugs most likely to be confused

Study logic: MOA -> explains the use -> predicts adverse effects -> explains what to avoid.

Compiled from the tables prepared in this chat based on the uploaded endocrine pharmacology lectures.

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PART 2 - COMPREHENSIVE DRUG TABLES

القسم الأول: جداول الأدوية الشاملة

Core format: Drug/Class -> MOA -> Why/Main use -> Adverse effects -> Contraindications/Avoid -> Memory link

1. Hypothalamic & Pituitary Drugs

Drug / Class	MOA	Why / Main use	Adverse effects	Contraindications / Avoid	Memory link
Growth hormone / Somatropin	GH replacement -> promotes growth and anabolic effects	True GH deficiency	Excess/prolonged use may cause effects of GH excess; lecture mentions concern about excessive bone growth and increased BP	Do not give unless true GH deficiency	Deficiency -> replace GH
Octreotide	Synthetic somatostatin analog -> decreases GH, IGF-1 and inhibits VIP	Acromegaly; VIPoma-related secretory diarrhea	Flatulence, nausea, steatorrhea, delayed gallbladder emptying -> cholesterol gallstones	Not emphasized	Octreotide = universal inhibitor
Pegvisomant	GH receptor antagonist -> blocks GH action -> normalizes IGF-1	Refractory acromegaly	Not emphasized	Not emphasized	Pegvisomant = blocks GH effect
Leuprolide / Nafarelin	GnRH agonists. Initially increase LH/FSH; continuous administration -> receptor downregulation/desensitization -> decrease gonadotropins and sex hormones	Prostate cancer, endometriosis, precocious puberty	Hot flushes, sweating, decreased libido, depression, osteoporosis, ovarian cysts; in men initial testosterone flare -> bone pain, edema, gynecomastia	Pregnancy, breast-feeding	Continuous stimulation -> receptor gets tired -> DOWN
Oxytocin	Increases smooth-muscle contraction; uterus + myoepithelial cells of breast	Induce/reinforce labor, milk ejection, reduce postpartum bleeding	Hypertensive crisis, uterine rupture, water retention, fetal death reported	Not emphasized	OXY = uterus squeeze + milk squeeze
Vasopressin	V1: vasoconstriction. V2: increases renal water reabsorption	DI + control bleeding from esophageal varices/diverticula	Water intoxication, hyponatremia, bronchoconstriction, tremor	Caution: CAD, epilepsy, asthma	V1 = vessels; V2 = water
Desmopressin	Selective mainly for V2, minimal V1 activity	Preferred in central DI and nocturnal enuresis	Not emphasized in this section	Not emphasized	DESMO = ADH without major pressure effect

2. Thyroid Pharmacology

Drug / Class	MOA	Why / Main use	Adverse effects	Contraindications / Avoid	Memory link
Levothyroxine - T4	Replaces thyroid hormone; T4 is converted peripherally to active T3	Drug of choice for hypothyroidism	Excess -> nervousness, palpitations, tachycardia, heat intolerance, weight loss	Important interaction: calcium decreases absorption	T4 = chronic replacement
Liothyronine - T3	Direct active T3 replacement	Faster action; may be considered in severe situations such as myxedema coma	More cardiovascular effects due to higher potency/faster action	Not preferred routinely	T3 = faster + stronger
PTU	Inhibits thyroid hormone synthesis; additionally inhibits peripheral T4 -> T3 conversion	Hyperthyroidism; especially thyroid storm; preferred in first trimester in lecture	Agranulocytosis, rash, edema	Not emphasized here	PTU = synthesis + peripheral conversion

Drug / Class	MOA	Why / Main use	Adverse effects	Contraindications / Avoid	Memory link
Methimazole	Inhibits thyroid hormone synthesis	Hyperthyroidism	Agranulocytosis, rash, edema	Lecture prefers PTU instead during first trimester	Methimazole = synthesis only
Iodides	Acute Wolff-Chaikoff effect -> inhibits iodination + inhibits release of thyroid hormone	Thyroid storm and before thyroid surgery because decreases vascularity	Sore mouth/throat, tongue/laryngeal swelling, rash, mucosal ulcers, metallic taste	Not for long-term therapy because thyroid escapes effect	Iodide = fast shut down, short term
Radioactive iodine I-131	Taken up by NIS -> emits beta particles -> destroys thyroid tissue	Hyperfunctioning thyroid / treatment of iodine-avid differentiated thyroid cancer	Hypothyroidism	Pregnancy	Radioactive iodine destroys thyroid
Propranolol	Nonselective beta blocker -> blocks adrenergic cardiac manifestations	Symptomatic control of hyperthyroidism; especially palpitations; IV may be used in thyroid storm	Not the focus of lecture	Severe asthma/heart failure -> lecture mentions diltiazem alternative	Does not fix thyroid; fixes symptoms

3. Bone & Calcium Drugs

Drug / Class	MOA	Why / Main use	Adverse effects	Contraindications / Avoid	Memory link
Teriparatide	Recombinant PTH analog. Intermittent/moderate exposure -> osteoblast activity > osteoclast -> bone formation	Osteoporosis, especially high fracture risk	Hypercalcemia emphasized	Not emphasized	Intermittent PTH BUILDs bone
Calcitonin	Inhibits osteoclasts -> decreases bone resorption	Conditions where reduction of bone resorption is desired	Not emphasized	Not emphasized	Calcitonin keeps Ca in bone
Bisphosphonates	Directly inhibit osteoclasts; inhibit farnesyl pyrophosphate synthase -> decrease osteoclast survival/activity	Osteoporosis	Specific adverse effects not retrieved in current lecture snippets	Not emphasized	Bisphosphonate = inhibit osteoclast
Cinacalcet	Calcimimetic -> activates Ca-sensing receptor on parathyroid -> decreases PTH -> decreases bone resorption	Hyperparathyroidism, parathyroid carcinoma	Not emphasized	Not emphasized	Pretends Ca is high -> PTH shuts off
Doxercalciferol	Vitamin D analog requiring activation	Secondary hyperparathyroidism in CKD	Not emphasized	Not emphasized	CKD secondary HPT
Paricalcitol	Active vitamin-D-related analog; lecture says no further liver/kidney activation needed	Secondary hyperparathyroidism with CKD; preferred in lecture if liver disease	Not emphasized	Not emphasized	Paricalcitol = already active

4A. Adrenal Pharmacology - Glucocorticoids & Mineralocorticoid

Drug / Class	Main feature / MOA	Why / Main use	Important adverse effects / key point
Hydrocortisone	Short acting; resembles endogenous cortisol most	Replacement in adrenal insufficiency; rapid glucocorticoid when needed	Divide dose to mimic circadian cortisol secretion
Prednisone	Intermediate acting; prodrug	Anti-inflammatory / immunosuppressive conditions	Requires hepatic activation
Prednisolone	Intermediate acting; active form	Similar uses; useful when liver activation is undesirable	Already active
Triamcinolone	Intermediate acting	Topical inflammatory skin diseases; inhaled forms for inflammatory airway disease	Local route reduces systemic effects
Dexamethasone	Long acting, potent anti-inflammatory, little mineralocorticoid activity	Chronic inflammatory conditions + dexamethasone suppression test	Long duration
Betamethasone	Long acting	Chronic inflammatory uses	Long duration
Fludrocortisone	Strong mineralocorticoid	Added in primary adrenal insufficiency because aldosterone is also deficient	Not routinely needed in secondary/tertiary insufficiency

4B. Major Adverse Effects of Prolonged Glucocorticoids

Adverse effect	WHY / Logic
Hyperglycemia / DM	Increased glucose production and anti-insulin metabolic effects
Hypokalemia	At high levels -> mineralocorticoid-like sodium retention + potassium loss
Edema	Sodium and water retention
Weight gain / increased appetite	Central stimulation of appetite
Infections	Immunosuppression
Growth suppression in children	Decreased GH/IGF-1 signaling
Cataracts	Long-term glucocorticoid effects on lens proteins
Adrenal suppression	Exogenous steroids suppress CRH/ACTH

Very important: prolonged glucocorticoids should NOT be stopped abruptly because the HPA axis is suppressed -> risk of acute adrenal crisis.

4C. Drugs Against Aldosterone / Cortisol Synthesis

Drug	MOA	Why used	Adverse effects / key issue
Spironolactone	Mineralocorticoid receptor antagonist + androgen receptor antagonist	Hyperaldosteronism, HF, cirrhosis, hypertension; antiandrogen effect may help hirsutism	Gynecomastia due to antiandrogen action
Eplerenone	More selective mineralocorticoid receptor antagonist	Hypertension / aldosterone blockade	Much less gynecomastia than spironolactone
Metyrapone	Blocks 11-hydroxylation, reducing cortisol synthesis	Cushing syndrome; adrenal function testing; lecture mentions pregnancy use	Salt/water retention, hirsutism, dizziness, GI disturbance
Ketoconazole	Inhibits adrenal steroid biosynthesis	Excess cortisol / Cushing syndrome	Specific adverse effects not emphasized in retrieved passage

5. Diabetes Drugs - Master Table

Class / Drug	MOA	Why / Use	Major adverse effects	Contraindications / Avoid	Memory
Pramlintide	Amylin analog -> decreases gastric emptying + decreases postprandial glucagon	Adjunct to mealtime insulin in T1DM / advanced T2DM	Nausea, anorexia, vomiting, severe hypoglycemia with insulin	Gastroparesis, hypoglycemic unawareness	Slow stomach + stop glucagon
Sulfonylureas	Close beta-cell KATP channels -> depolarization -> Ca2+ influx -> increased insulin secretion	T2DM with functioning beta cells	Hypoglycemia, weight gain	Not useful in T1DM; consider sulfonamide allergy history	Squeeze insulin OUT
Metformin	Decreases hepatic gluconeogenesis + decreases lipogenesis + increases peripheral insulin sensitivity; does not stimulate insulin secretion	Drug of choice T2DM, PCOS, insulin resistance	GI upset, B12 deficiency, rare lactic acidosis	Renal/hepatic disease, acute MI/CHF, severe infection, DKA in lecture	Metformin stops liver making glucose
TZDs: Pioglitazone / Rosiglitazone	Activate nuclear PPAR-gamma -> altered gene transcription -> increased insulin sensitivity	T2DM, PCOS; second-line when metformin fails/cannot be used	Weight gain, osteopenia/fractures, rare liver toxicity, CV risk	MI/cardiac concern; nursing mothers not recommended	TZD = Tissue sensitivity
DPP-4 inhibitors: Sitagliptin etc.	Prevent incretin degradation -> glucose-dependent increased insulin / decreased glucagon	T2DM	Nasopharyngitis, headache; low hypoglycemia risk	Sitagliptin dose adjustment in renal dysfunction	Keep incretin alive

6. Insulin Types - Quick Table

Type	Drugs	Main role / special point
Ultra-rapid	Lispro, Aspart, Glulisine	Mealtime control; faster because they do not aggregate significantly
Short acting	Regular insulin	Only insulin emphasized in the lecture as suitable for IV; preferred IV in hyperglycemic emergency
Intermediate	NPH	Intermediate basal coverage
Long acting	Glargine, Detemir	Basal insulin with prolonged/flat effect
Detemir special	Detemir	Fatty-acid side chain -> binds albumin -> slow dissociation -> prolonged action

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PART 3 - HIGH-YIELD COMPARISON TABLES

القسم الثاني: جداول المقارنة عالية الأهمية

Use these tables for final revision: compare the feature, then memorize the one-line memory rule under each table.

1. PTU vs Methimazole

Feature	PTU	Methimazole
Class	Thioamide	Thioamide
Inhibits oxidation/iodination	YES	YES
Inhibits coupling of iodotyrosines	YES	YES
Blocks T4 -> T3 peripheral conversion	YES	NO
Effect on already stored T3/T4	No effect	No effect
Onset	Delayed until stored hormones are depleted	Delayed until stored hormones are depleted
Thyroid storm	Preferred	Not preferred
Why in thyroid storm?	Blocks synthesis + T4 -> T3	Blocks synthesis only
First trimester pregnancy	Lecture states PTU preferred	Not preferred in first trimester
Important AE shared	Agranulocytosis, rash, edema	Agranulocytosis, rash, edema

Memory: PTU has a Plus -> Peripheral T4 -> T3 conversion is blocked.

2. Glucocorticoids: Short vs Intermediate vs Long

Feature	Short acting	Intermediate acting	Long acting
Examples	Hydrocortisone, Cortisone	Prednisone, Prednisolone, Methylprednisolone, Triamcinolone	Dexamethasone, Betamethasone
Onset according to lecture	Fastest	Intermediate	Slowest
Duration	Short	Intermediate	Long
Frequency	More frequent	Intermediate	Less frequent
Best logic	Rapid effect / replacement	Many inflammatory conditions	Chronic inflammation / less frequent dosing
Important example	Hydrocortisone	Prednisone / Prednisolone	Dexamethasone
Mineralocorticoid effect	Relatively more	Less	Very little/none emphasized
Anti-inflammatory potency	Lower	Higher	Highest

Memory: Short = replacement / quick. Long = powerful inflammation control.

3. Prednisone vs Prednisolone

Feature	Prednisone	Prednisolone
Duration	Intermediate	Intermediate
Active when administered?	No	Yes

Feature	Prednisone	Prednisolone
Nature	Prodrug	Active drug
Needs liver activation?	Yes	No
Liver impairment	Less suitable	Preferred according to lecture logic
Main difference	Needs activation	Ready to act

Memory: PredniSONE needs someone to turn it ON; PredniSOLONE is already ON.

4. Hydrocortisone vs Fludrocortisone

Feature	Hydrocortisone	Fludrocortisone
Main activity	Glucocorticoid	Strong mineralocorticoid
Replaces mainly	Cortisol	Aldosterone effect
Primary adrenal insufficiency	YES	YES - usually added
Secondary adrenal insufficiency	YES	Usually not needed
Tertiary adrenal insufficiency	YES	Usually not needed
Why?	Cortisol deficient in all 3	Aldosterone mainly deficient in primary
Special dosing	Larger AM dose, smaller later	-

Key exam rule: Primary Addison -> Hydrocortisone + Fludrocortisone. Secondary/Tertiary -> usually Hydrocortisone alone.

5. Spironolactone vs Eplerenone

Feature	Spironolactone	Eplerenone
Blocks mineralocorticoid receptor	YES	YES
Blocks aldosterone effect	YES	YES
Selectivity	Less selective	More selective
Antiandrogen effect	Yes	Very little / none
Gynecomastia	Can occur	Much less likely
Hirsutism	Can be useful because antiandrogen	Not emphasized for this
Hypertension	Used	Used

Memory: Spironolactone spills over to androgen receptors -> gynecomastia. Eplerenone is more selective.

6. Insulin Types

Feature	Ultra-rapid	Regular	NPH	Glargine
Examples	Lispro, Aspart, Glulisine	Regular insulin	Isophane / NPH	Glargine
Class	Ultra-rapid	Short	Intermediate	Long
Onset	5-10 min	30-60 min	Delayed	Slower
Duration	2-3 h	7-8 h	~12 h	Prolonged
Main role	Mealtime	Mealtime + emergencies	Basal/intermediate coverage	Basal
IV?	Not emphasized	YES	Never	SC only
SC?	YES	YES	YES	YES
Peak	Has mealtime peak	Has peak	Has peak	No peak / flat
DKA / emergency	Not main lecture choice	Regular insulin	Not for emergency	Not for emergency
Why prolonged?	-	-	Protamine -> less soluble -> slow absorption	Precipitates after injection -> prolonged release

Memory: Rapid = Food | Regular = Emergency | NPH = Protamine | Glargine = Flat basal.

7. Sulfonylureas vs Meglitinides

Feature	Sulfonylureas	Meglitinides
Examples	Glimepiride, Glipizide, Glyburide	Repaglinide, Nateglinide
Need functional beta cells?	YES	YES
General MOA	Close KATP channel -> depolarization -> Ca2+ -> insulin release	Similar pathway; bind different site on sulfonylurea receptor
Main effect	Increased insulin secretion	Increased insulin secretion
Duration	Longer	Shorter
Onset	Slower relative to meglitinides	Rapid
Best glucose target	General	Postprandial glucose
Hypoglycemia	Higher	Lower than sulfonylureas
Weight gain	Important	Less of a problem
Combine together?	NO	NO
Why not combine?	Same final effect -> severe hypoglycemia	Same

Memory: Meglitinides = meal drugs -> rapid ON + short duration.

8. First vs Second Generation Sulfonylureas

Feature	1st generation	2nd generation
Example	Tolbutamide	Glimepiride, Glipizide, Glyburide
Potency	Less potent	More potent
Required dose	Higher	Lower
Duration	Tolbutamide = shortest	Generally longer
Strength trap	Not stronger	More potent

Exam trap: First generation is NOT stronger. Second generation is more potent -> smaller dose gives the effect.

9. Metformin vs Sulfonylureas

Feature	Metformin	Sulfonylureas
Main mechanism	Decreases hepatic gluconeogenesis + increases insulin sensitivity	Increases pancreatic insulin secretion
Stimulates insulin release?	NO	YES
Requires endogenous insulin?	Requires insulin for its sensitizing action	Requires functional beta cells
Hypoglycemia alone	Very low	Important
Weight	Tends toward weight loss / decreased appetite in lecture	Weight gain
T1DM	Not main therapy	Ineffective because beta cells destroyed
Main logic	Insulin sensitizer	Insulin secretagogue

Core idea: Metformin makes insulin work better. Sulfonylurea makes the pancreas release more insulin.

10. Metformin vs TZDs

Feature	Metformin	TZDs: Pioglitazone / Rosiglitazone
Both are	Insulin sensitizers	Insulin sensitizers
Stimulate insulin secretion?	NO	NO
Main MOA	Mainly decreases hepatic gluconeogenesis + increases sensitivity	Activates nuclear PPAR-gamma
Main target emphasized	Liver + peripheral tissues	Adipose, liver, skeletal muscle
Weight	Often decreases / appetite decreases in lecture	Weight gain
Cardiovascular concern in lecture	More favorable	CV toxicity concern emphasized
Place in therapy	Preferred	Second-line alternative if metformin fails / contraindicated
PCOS	Used	Can also be used

Memory: Metformin -> liver glucose production DOWN. TZD -> PPAR-gamma -> insulin sensitivity UP.

11. Pioglitazone vs Rosiglitazone

Feature	Pioglitazone	Rosiglitazone
Class	TZD	TZD
MOA	PPAR-gamma agonist	PPAR-gamma agonist
Insulin sensitivity	Increases	Increases
HDL	Increases	Increases
LDL according to lecture	Not affected	Increases
Weight gain	YES	YES
CV concern	Class concern	More emphasized with rosiglitazone in lecture

12. GLP-1 Agonists vs DPP-4 Inhibitors

Feature	GLP-1 agonists	DPP-4 inhibitors
Examples	Exenatide, Semaglutide	Sitagliptin
What do they do?	Mimic GLP-1	Prevent breakdown of endogenous incretin
Target	GLP-1 receptor	DPP-4 enzyme
Increase insulin	YES - glucose-dependent	YES - glucose-dependent
Decrease glucagon	YES	YES
Hypoglycemia risk	Low	Low
Gastric emptying	Decreased emphasized	Not emphasized as major direct effect in comparison
Appetite	Decreases	Not emphasized
Weight loss	Important, especially semaglutide	Not emphasized
Route	GLP-1 analogs discussed as injections	Sitagliptin = oral
Common sitagliptin AE	-	Nasopharyngitis, headache
Renal issue	-	Sitagliptin dose adjustment in renal dysfunction

Memory: GLP-1 agonist gives the incretin EFFECT directly. DPP-4 inhibitor protects your OWN incretin from destruction.

13. Hypoglycemia Risk Logic

Drug / Class	Insulin stimulation	Hypoglycemia logic
Metformin	Does not directly stimulate insulin	Very low
GLP-1 / DPP-4	Glucose-dependent	Low
Sulfonylurea	Glucose-independent stimulation	Higher
Exogenous insulin	Direct glucose-lowering regardless of glucose	High risk

FINAL REVISION RULE

When you see a drug name in an MCQ, first identify its CLASS, then recall the MOA, then use the MOA to predict the use, adverse effect, and contraindication.