

GENERAL MEDICINE 1

Marrow SS Medicine





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General Medicine

Volume - 1

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Contents

Volume - 1

Endocrinology and Metabolism

1. Classifications of Hormones	1
2. Basics of Pituitary Gland	11
3. Prolactin	19
4. Growth Hormone	27
5. Congenital Hypopituitarism	36
6. Acquired Hypopituitarism	45
7. Management of Hypopituitarism	53
8. Basics of ADH	57
9. Disorders of Water Metabolism : I	63
10. Disorders of Water Metabolism : II	69
11. Management of Hyponatremia	77
12. Basics of Adrenal Gland	82
13. Pheochromocytoma	88
14. MEN & MEON Syndromes	95
15. Congenital Adrenal Hyperplasia	101
16. Polyglandular Autoimmune (PGA) Syndrome	106
17. Addison's Disease	109
18. Disorders of Sexual Development	115
19. Primary Hyperaldosteronism	120
20. Cushing Syndrome	125
21. Endocrine Hypertension	139
22. Basics of Thyroid	143
23. Thyroid Function Test	151
24. Thyroid Hormone	155

25. Thyroiditis	158
26. Hypothyroidism	163
27. Thyrotoxicosis	173
28. Basics of Bone and Mineral Metabolism	187
29. Calcium Metabolism : I	193
30. Calcium Metabolism : II	199
31. Hypocalcemia	206
32. Hypercalcemia	212
33. Osteoporosis	220
34. Phosphorus Metabolism	228
35. Magnesium Metabolism	235
36. Diabetes Today	239
37. Classification of DM : I	246
38. Classification of DM : II	253
39. Type-1 Diabetes Mellitus	261
40. Incretin	267
41. Acute Complications of DM	269
42. Glimpse of Complications of DM	276
43. Story of Resistance	281
44. Management of DM	290
45. Hypoglycemia	302

Renal System

46. Development of Kidney	307
47. Renal Anatomy	314
48. Nephron	318
49. Renal Physiology : I	328
50. Renal Physiology : II	333
51. Core Clinical Basics - Urine Analysis	337
52. Core Clinical Basics - Problem Solving	350

108. Development of Liver	602
109. Liver Physiology - LFT : I	607
110. Liver Physiology - LFT : II	616
111. Inherited Hyperbilirubinemias	622
112. Acute Liver Failure	626
113. Basics of Chronic Hepatitis / Cirrhosis	632
114. Autoimmune Hepatitis	641
115. Hepatitis A & E	644
116. Hepatitis C Virus	648
117. Hepatitis B Virus : I	656
118. Hepatitis B Virus : II	666
119. Biliary Disorders : PBC/PSC	673
120. Metabolic Diseases of Liver : I	676
121. Metabolic Diseases of Liver : II	683
122. Alcoholic Liver Disease	687
123. Nonalcoholic Liver Disease	692
124. Portal Hypertension	698
125. Ascites	707
126. Hepatorenal Syndrome	714
127. Hepatopulmonary Syndrome	717
128. Hepatic Encephalopathy	719
129. Vascular Disorders of Liver	726

Nervous system

130. Human Brain - Me, Myself : I	732
131. Human Brain - Me, Myself : II	738
132. Primary Headache	743
133. Dangerous Headache	751
134. Memory	760
135. Language Vs Speech	763

136. Myasthenia Gravis	767
137. Inflammatory Muscle Diseases	773
138. Guillain Barre Syndrome	779
139. Guillain Barre Syndrome Vs Chronic Inflammatory Demyelinating Polyneuropathy	782
140. LMN Approach : Muscle I	785
141. LMN Approach : Muscle II	796
142. Seizure : I (Semiology)	801
143. Seizure : II	807
144. Demyelinating Disorders of CNS	814
145. Cortex-Lobar Organisation I	821
146. Cortex-Lobar Organisation II	829
147. Lobar Dysfunction - Aphasia	834
148. Dementia : I	837
149. Dementia : II	843
150. Basics of Extrapyrarnidal System	849
151. Parkinson's Disease - Typical/Atypical	852
152. Management of Parkinson's Disease	861
153. Spinal Cord - Applied Anatomy	863
154. Spinal Cord : I (Compressive Myelopathy)	872
155. Spinal Cord : II (Non-Compressive Myelopathy)	875
156. Spinal Cord - Clinical Case Examples	880
157. Inflammatory Demyelination of CNS	883
158. Tubercular Meningitis	889
159. Viral Meningoencephalitis	893
160. Approach To UMN Lesion	896
161. Cranial Nerves : I	903
162. Cranial Nerves : II	911
163. Stroke : Introduction, Classification & Risk Factors	918

82. Skin Manifestations in Chronic Kidney Disease	472
83. Introduction To Potassium Metabolism	476
84. Diabetic Kidney Disease	481
85. Hypokalemia	486
86. Hypokalemia - Renal Tubular Acidosis	490
87. Hypokalemia & Alkalosis	494
88. Hyperkalemia	499
89. Hemodialysis	503
90. Renal Transplantation	510

Volume - 2

Gastrointestinal and Hepatobiliary System

91. Physiology of Absorption	517
92. Gastric Anatomy / Gastritis	523
93. Symptoms & Tests for Malabsorption	529
94. Diarrhea and Malabsorption	534
95. Miscellaneous Causes of Malabsorption	540
96. Infections & Diarrhoea	547
97. Celiac Disease	554
98. Tropical Sprue	560
99. Whipple's Disease	563
100. Introduction To IBD	565
101. Pathology of IBD	570
102. Clinical Features of IBD	573
103. Management of IBD	580
104. Irritable Bowel Syndrome	586
105. H.pylori infection	589
106. Gastrinoma	594
107. Anatomy of Liver	597

53. Renal Artery Stenosis	353
54. Thrombotic Microangiopathy	358
55. Clinical Approach To Glomerular Diseases : I	364
56. Clinical Approach To Glomerular Diseases : II	367
57. Membranoproliferative Glomerulonephritis	371
58. Podocytopathies : I	376
59. Podocytopathies : II	382
60. Podocytopathies : III	387
61. Nephritic Syndrome : Post-Streptococcal Glomerulonephritis	391
62. Rapidly Progressive Glomerulonephritis	396
63. IgA Nephropathy	400
64. Alport's Syndrome	404
65. Goodpasture's Disease	406
66. Classification of Acute Kidney Injury	409
67. Causes of Acute Kidney Injury	412
68. Drug Induced Acute Kidney Injury	418
69. Sepsis & Acute Kidney Injury	422
70. Cholesterol Embolism and AKI	426
71. Tumor Lysis Syndrome	428
72. Summary of Acute Kidney Injury	432
73. Hereditary & Cystic Disorders	434
74. CTID	441
75. Chronic Kidney Disease	445
76. Anemia in Chronic Kidney Disease	448
77. Disease Progression of Chronic Kidney Disease	456
78. Chronic Kidney Disease - Mineral and Bone Disorder	458
79. Summary of Chronic Kidney Disease	464
80. Central Nervous System in Chronic Kidney Disease	465
81. Cardiovascular System in Chronic Kidney Disease	467

164. Blood Supply of Brain	924
165. Stroke : Vascular Localization	931
166. Posterior Circulation	937
167. Build Up To Stroke	942
168. Brainstem Syndromes	951
169. Stroke Management	959
170. LMN Approach : Part 1 (Radial Plexus)	965
171. LMN Approach : Part 2 (Nerve)	970
172. LMN Approach : Part 3 (Patterns)	975
173. LMN Approach : Part 4 (Inherited Neuropathies)	980
174. LMN Approach : Part 5 (Acquired Neuropathies)	983
175. LMN Approach - Anterior Horn Cell	986

Volume - 3

Infectious Diseases

176. Bacterial v/s Viral Meningitis	991
177. CNS Infections : Acute Bacterial Meningitis	993
178. Acute Vs Subacute Meningitis	1001
179. HIV 2022	1005
180. Infectious Diseases of The Kidney	1015
181. CVS Infections - Infective Endocarditis	1022
182. Tropical Infections - Synopsis	1031
183. Community Acquired Pneumonia	1039

Cardiovascular Disorders

184. Cardiology Basics : Pulse	1051
185. Cardiology Basics - JVP	1060
186. Cardiology Basics - Heart Sounds : I	1068
187. Cardiology Basics - Heart Sounds : II	1074
188. Aortic Valve Disorders - Aortic Stenosis	1082

189. Aortic Valve Disorders - Aortic Regurgitation	1090
190. Mitral Valve Disorders - Mitral Regurgitation	1094
191. Mitral Valve Disorders - Mitral Stenosis	1100
192. Rheumatic Heart Disease - Synopsis	1113
193. Heart Failure : I	1115
194. Heart Failure : II (Management)	1119
195. Acute Decompensated Heart Failure	1123
196. Systemic Hypertension 2022	1128
197. Pericardium and Acute Pericarditis	1137
198. Chronic Constrictive Pericarditis	1143
199. CCP Vs RCM/CT	1146
200. Cardiomyopathy : Part 1 (DCM)	1149
201. Cardiomyopathy : Part 2 (HCM)	1153
202. Cardiomyopathy : Part 3 (RCM - Unclassified)	1159

Hematology

203. Anemia - Basic Approach	1164
204. Hypoproliferative Anemia : Iron Deficiency Anemia	1169
205. Iron Deficiency Anemia : Treatment	1177
206. Hypoproliferative Anemia With MHA	1179
207. Macrocytic Anemia	1183
208. Hemolytic Anemia Classification	1192
209. Hemolytic Anemia - Inherited	1195
210. Hemolytic Anemia Acquired : Immune Mediated	1200
211. Hemolytic Anemia Acquired : PNH	1208
212. Hemolytic Anemia Acquired : Fragmentation	1211
213. Bone Marrow Failure Syndrome	1217
214. Myeloproliferative Neoplasms	1225
215. Plasma Cell Disorders : I	1232

216. Plasma Cell Disorders : II	1238
217. Introduction To Acute Leukemia	1242
218. Acute Myeloid Leukemia	1247
219. Acute Myeloid Leukemia Vs Acute Lymphoblastic Leukemia	1257
220. Chronic Myeloid Leukemia	1262
221. World of Lymphomas	1268
222. Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma	1272
223. Non Hodgkin's Lymphoma : I	1275
224. Non Hodgkin's Lymphoma : II	1279
225. T-cell Lymphoma	1283
226. Hodgkin's Disease	1285
227. Approach To Bleeding : I	1290
228. Approach To Bleeding : II	1294
229. Von Willebrand Disease	1299
230. Hemophilia	1302
231. Disseminated Intravascular Coagulation	1305

Volume - 4

Rheumatology and Immunology

232. SLE : Etiopathogenesis	1309
233. SLE : Immunological Basis	1313
234. SLE : Lupus Nephritis	1318
235. SLE : Clinical Presentation	1323
236. SLE : Organ System Manifestations	1328
237. SLE : Management	1331
238. Drug-Induced Lupus Erythematosus	1334
239. Antiphospholipid Syndrome	1335
240. Sjogren's Syndrome	1340
241. Systemic Sclerosis	1349

242. IgG4 Related Disease	1360
243. Sarcoidosis	1364
244. Overlap Syndrome	1376
245. Basics of Rheumatoid Arthritis	1378
246. Approach To Arthritis : I	1383
247. Approach To Arthritis : II	1386
248. Articular Features of Arthritis	1389
249. Extra Articular Features of Arthritis	1393
250. Treatment of Arthritis	1398
251. Spondyloarthritis	1401
252. Crystal Arthropathies	1418
253. Classification of Vasculitis	1435
254. Small Vessel ANCA Vasculitis	1437
255. Medium Vessel Vasculitis	1447
256. Large Vessel Vasculitis	1452
257. Variable Vessel Vasculitis	1464
258. Immune Complex Mediated Vasculitis	1468

Respiratory Disorders

259. Functional Anatomy of Lung	1475
260. Mechanics of Breathing	1488
261. Pulmonary Function Test - Interpretation	1495
262. Pulmonary Function Test - DLCO	1500
263. Flow Volume Curve	1503
264. Diagnosis of Tuberculosis	1508
265. Treatment of Tuberculosis	1515
266. Chronic Obstructive Pulmonary Disease	1518
267. Bronchial Asthma	1526
268. Allergic Bronchopulmonary Aspergillosis	1538

269. Hypersensitive Pneumonitis	1544
270. Eosinophilic Lung Disease	1548
271. Bronchiectasis	1555
272. Respiratory Failure	1562
273. Acute Respiratory Distress Syndrome	1565
274. Pulmonary Hypertension	1569
275. Venous Thromboembolism	1577
276. Interstitial Lung Disease	1589
277. Occupational Lung Disorders	1600
278. Obstructive Sleep Apnea Syndrome	1607
279. Effusion/Empyema	1610

Acid Base Regulation

280. Methodology and Interpretation of Abg Analysis	1616
281. Metabolic Alkalosis	1624
282. Case Scenarios on ABG	1626
283. Mineral Deficiencies	1637

ECG

284. Introduction To ECG	1639
285. Approach To Hypertrophy and Blocks	1648
286. AV Blocks	1663
287. SA Nodal Dysfunction	1674
288. Atrial Fibrillation and Flutter	1684
289. Tachyarrhythmias	1697
290. Ventricular Arrhythmias	1717
291. WPW Syndrome	1727
292. Introduction To ACS	1732
293. ACS : Coronary Circulation and Localization	1751
294. ACS - Evaluation and Management	1769

Biostatistics

295. Introduction To Data in Biostatistics	1783
296. Measures of Central Tendency and Variation	1785
297. Normal Distribution Curve	1788
298. Concept of Probability Value	1791
299. Tests of Significance	1794
300. Correlation, Regression and Skew	1795
301. Sampling Methods and Calculation	1799
302. Probability and Tiles	1803
303. Graphs	1806
304. Biostats Review and QA Round	1819
305. Analytical Epidemiology	1827
306. Advanced Analytical Study Designs	1829
307. Experimental Epidemiology	1832
308. Evidence Based Medicine	1838
309. Advanced Concepts in Screening of Diseases	1842

CLASSIFICATIONS OF HORMONES

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Classification based on structure

00:02:47

Derivatives :

Amino acid derivatives :

1. Tyrosine :

- T_3, T_4 .
- Catecholamines : produced by adrenal medulla, most abundant in order of **adrenaline (epinephrine)** > Nor-adrenalin (Nor-epinephrine) > Dopamine.

2. Tryptophan :

Serotonin & melatonin (Centrally acting neurotransmitters and peripherally acting hormones).

Serotonin :

- It is one of the **hormones of happiness**.
- Decreased serotonin linked to depression, anxiety & panic.
- It is produced on exposure to sunlight → Retinal ganglionic cells stimulated → Activate raphe nuclei in brain stem → Release of serotonin (High activity levels).
- It is part of **sleep wake cycle**.
- At around 9:30 pm → Serotonin stimulates → Pineal gland → melatonin released (Causes sleep) → Highest at 2-4 am, decreases by 7:30 am → Serotonin dominant hormone during the day (Same cycle repeats everyday).
- Abundant amount of serotonin is produced by GIT (Enterochromaffin cells).
- Note : 4 happy hormones : Dopamine, oxytocin, serotonin & endorphin.

Vitamin derivatives : A & D.

Peptide hormones :

- Small peptides (< 50 AA) : Hypothalamic hormones, posterior pituitary hormones, ACTH.
- Large peptides (> 50 AA) : Anterior pituitary hormones : **Prolactin (199 AA)**, **GH (191 AA)**, Insulin, Parathormone and renin.

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Glycoprotein hormones (>200 AA) :

- Lots of protein + less carbohydrates
- Examples : Thyroid Stimulating Hormone, Follicle Stimulating Hormone, Luteinizing Hormone.
- α subunit is common to all.
- β subunit is the differentiating factor.

Steroid hormones :

Adrenal cortical hormones :

- Mineralocorticoid : Aldosterone.
- Glucocorticoids : Cortisol.
- Sex steroids/adrenal androgens : DHEAS (Dihydro Epiandrosterone sulphate), Androstenedione.
- Gonadal hormones : Oestrogen, progesterone, testosterone.

Classification based on mechanism of action

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Classification :

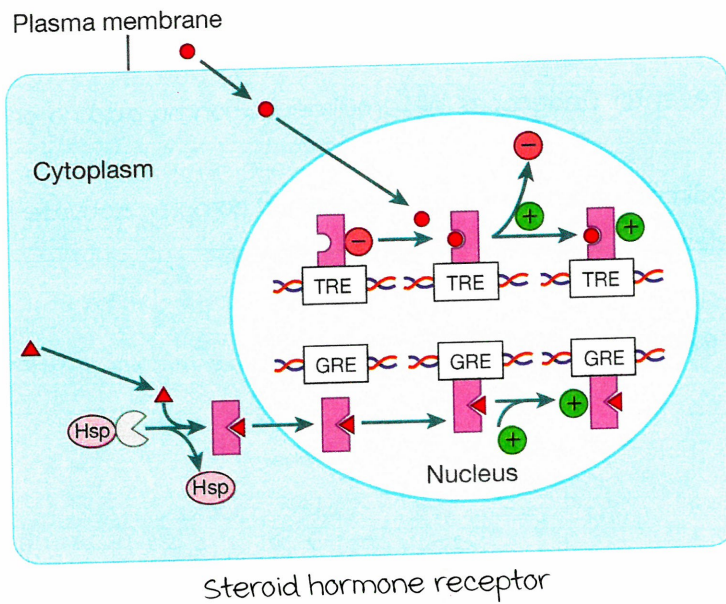
- Group 1 hormones : They have intracellular receptors.
- Group 2 hormones : They have cell membrane (extracellular receptors).

Group 1 hormones :

- Lipophilic.
- Require transport proteins.
- Long TI/a.
- Receptor hormone complex : Effects are mediated through gene transcription (Rarely transport channels).

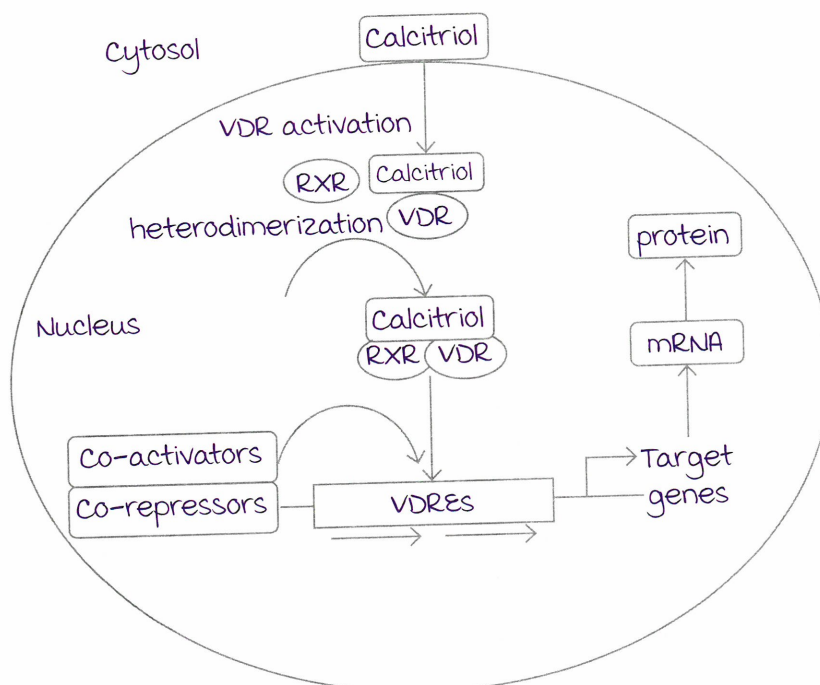
Types :

- Cytoplasmic receptors/type 1 receptor : Steroid hormones.
- Nuclear receptors/type 2 receptors : Vitamin A & D, T3 & T4.



- In the inactive state : Steroid receptor (Homodimer) is bound to **HSP** (Heat Shock Protein)-90/chaperones.
- Once ligand (Steroid hormones) arrives, steroid receptor changes configuration.
- HSP is released.
- And **receptor hormone complex** is formed and it migrates to nucleus.
- Binds to DNA at Glucocorticoid Response Element (GRE).
- End result is **gene transcription**.

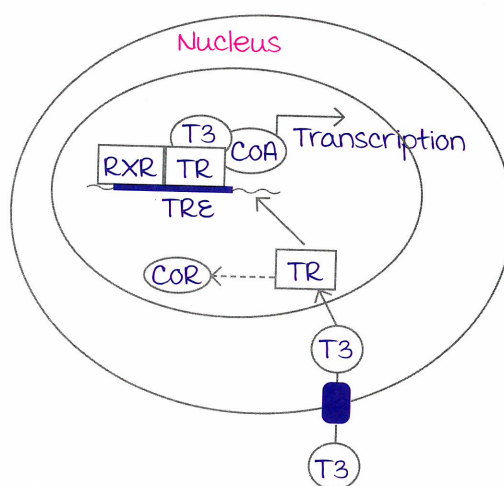
mechanism of action of calcitriol :



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- Calcitriol (1,25 OH₂ D₃) : active form of vitamin D₃ binds to vitamin D receptor in nucleus.
- Vitamin D receptor undergoes heterodimerisation by adding on retinoic acid receptor.
- The heterodimer receptor & ligand (calcitriol) complex activate vitamin D response element (VDRE) → gene transcription.

mechanism of action of thyroid hormones :

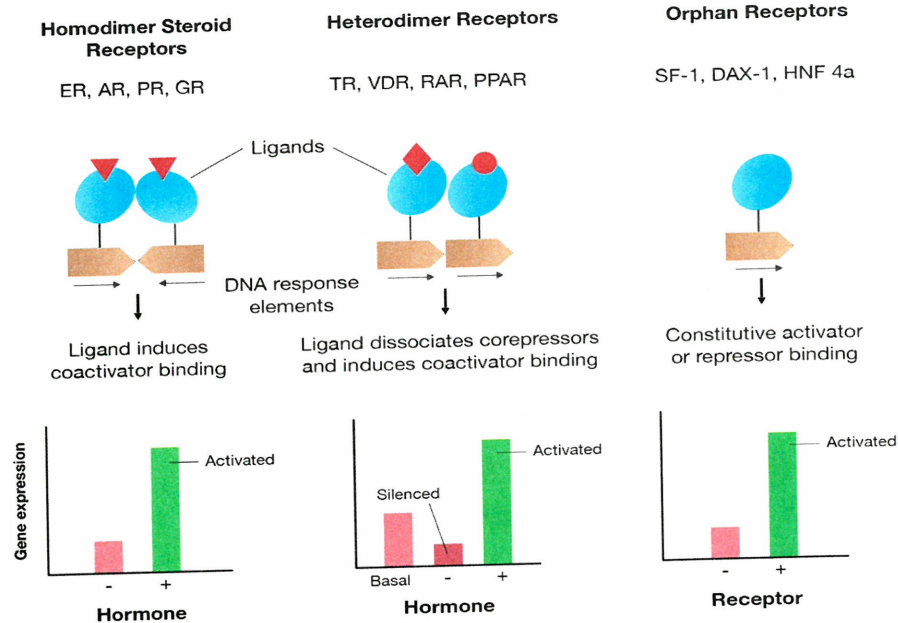


- T₃ (ligand) binds to the thyroid receptor in the nucleus.
- In the thyroid receptor, there is the dissociation of the co - repressor.
- The thyroid receptor binds to retinoic acid receptor to form a heterodimer and activate Thyroid Response Element (TRE).
- Then gene transcription occurs.

Group I	Type I receptor	Type 2 receptor
Type	Homodimer	Heterodimer
Location	Cytoplasm	Nucleus
Mechanism	migration to the nucleus & direct gene transcription.	Dissociation of co repressor & hetero dimerisation & then gene transcription.
Examples	Steroid	T ₃ , T ₄ , Vitamin A & D.

Nuclear Receptor Signaling

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Other Heterodimer receptors :

PPAR (peroxisome proliferator activator receptor) :

PPAR α : Agonist is clofibrate (used when triglycerides > 440 mg/dL).

PPAR γ : Agonist is pioglitazone (thiazolidinediones) (associated with Carcinoma bladder).

Dual PPAR α and γ agonists : Saroglitazal is DOC for diabetic dyslipidaemia (acts against oxidised LDL & increases HDL).

Orphan receptors :

No ligand is required.

It is constitutive.

Example :

- SF-1
 - DAX-1
 - HNF-4 α (MODY-1)
- Required for gonadotroph cell development

Group 2 hormones

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General features of hormone classes :

	Group I	Group II
Solubility	Lipophilic	Hydrophilic
Transport proteins	Yes	No
Plasma half-time	Long (hours to days)	Short (minutes)

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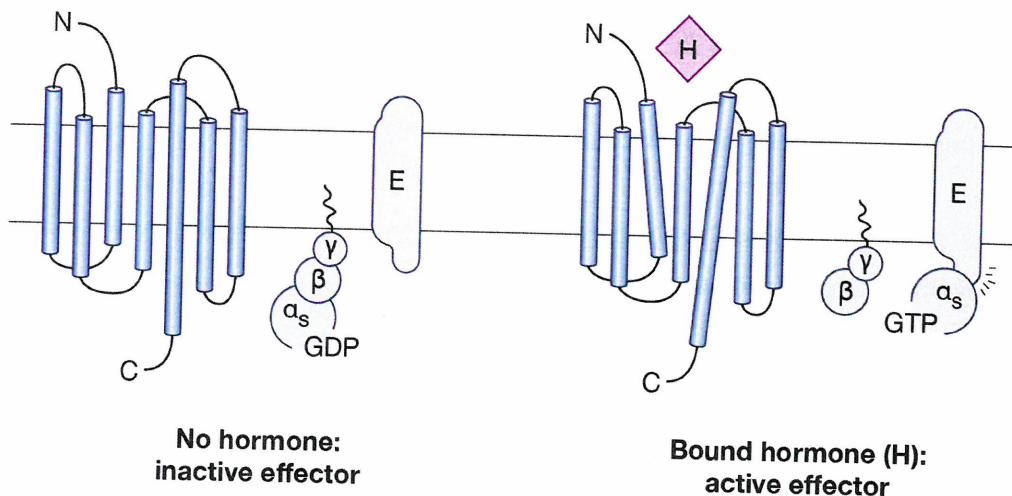
Signals are generated by **second messengers**. They are responsible for protein modification or translocation followed by transport channel regulation.

Receptors :

- G-protein coupled receptor (GPCR).
- Tyrosine kinase receptor.
- JAK (Janus kinase or cytokine) receptor.
- Serine threonine kinase receptor.

Hormones acting via GPCR :

- It is called as transmembrane receptor.
- They have 7 membrane spanning domains.
- It has α , β , γ subunits bound to GDP in inactive form.
- When hormone binds to receptor; $\text{GDP} \rightarrow \text{GTP}$.
- Alpha subunit & GTP dissociate from complex & bind to the effector.



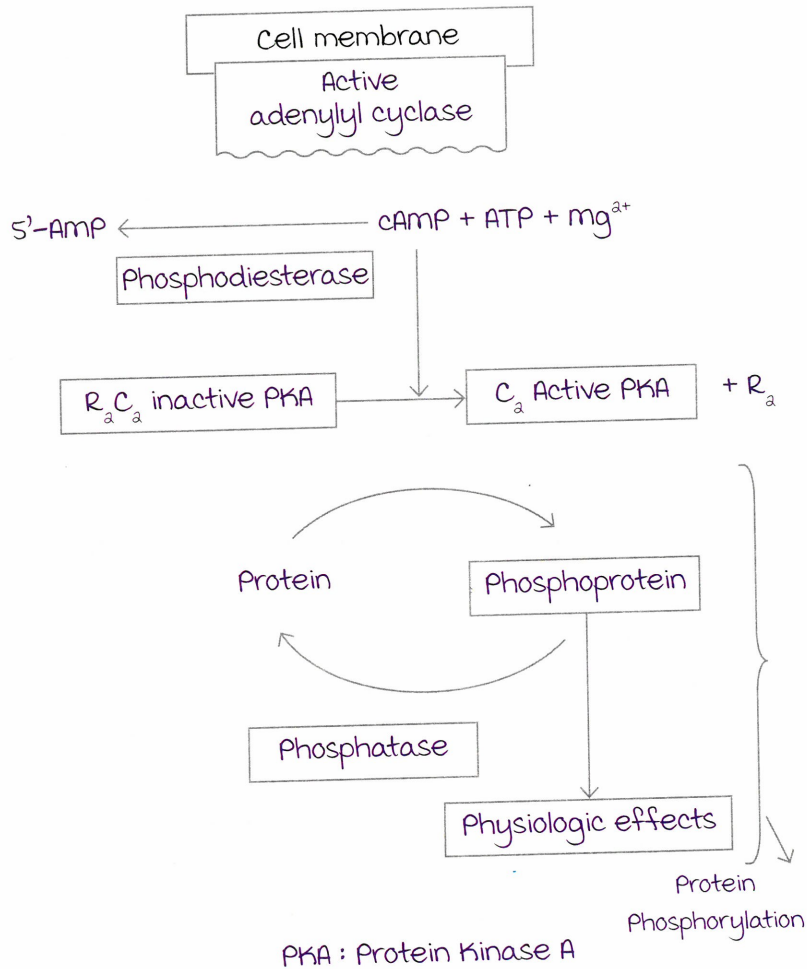
effector is 2nd messengers; It can be,

- cAMP (G_s : stimulatory or G_i : inhibitory).
- IP₃/DAG (G_q).
- cGMP (transducin mediated G_t).

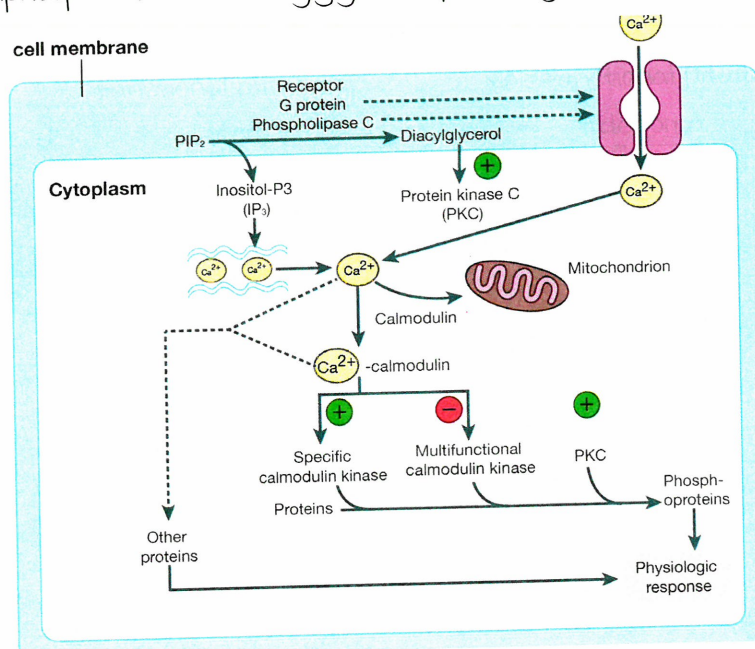
cGMP pathway :

vasodilators like NO and ANF act via this pathway.

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IP₃ (inositol triphosphate)/DAG (diacylglycerol) pathway :



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Broad terminology : vasoconstrictors : IP3 pathway, vasodilators : cAMP pathway.

	cAMP	IP3/DAG/Ca ²⁺
Hypothalamus	CRH	TRH, GnRH
Pituitary	ACTH, FSH, LH, TSH + V2 (ADH)	Oxytocin V1, V3 (ADH)
Vasoconstrictors	-	AT-II, Substance P
Pancreas	Glucagon, Somatostatin (Gi)	-
GIT	Secretin	CCK, Gastrin
ANS	α 2(Gi), Beta	α 1, Muscarinic Ach
Miscellaneous	PTH, Calcitonin	-

most potent vasoconstrictor is **urotensin**.

Subclassification of Group II A Hormones :

Hormones that inhibit adenylyl cyclase (Ha)
Acetylcholine
α -2-adrenergics
Angiotensin II
Somatostatin

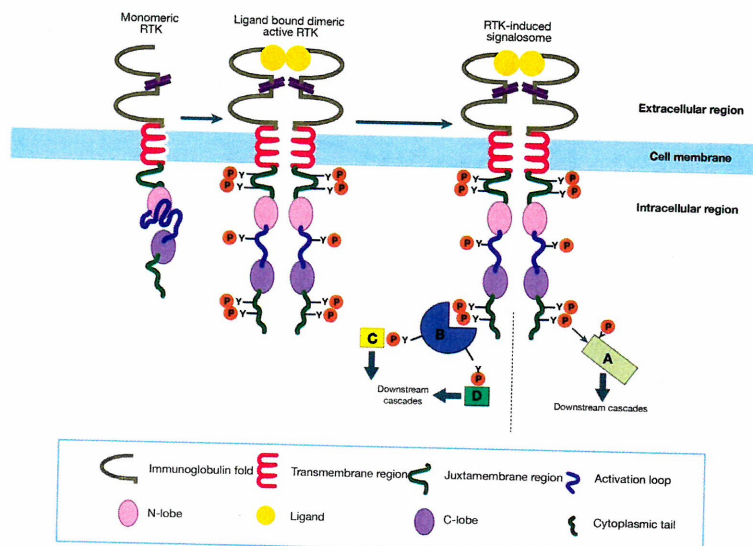
Acetyl choline and angiotensin II decrease cAMP, but their hormonal actions are due to IP3/DAG pathway.

α -2 adrenergics and Somatostatin hormonal action mediated by $\downarrow$ in cAMP.

Tyrosine kinase pathway

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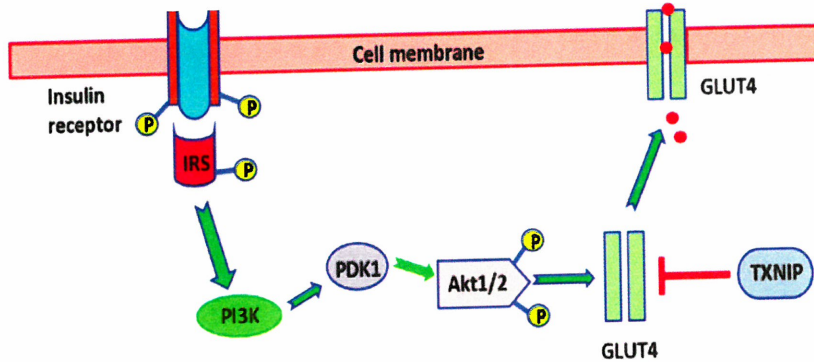
Insulin, Insulin like Growth Factor, Nerve growth factor, Epidermal growth factor, Fibroblast growth factor, TGF α .



Inactive tyrosine kinase receptor (monomeric state) $\rightarrow$ Ligand binding $\rightarrow$
 Intracellular dimerisation of receptor $\rightarrow$ Receptor autophosphorylation $\rightarrow$
 Activate signalling molecules.

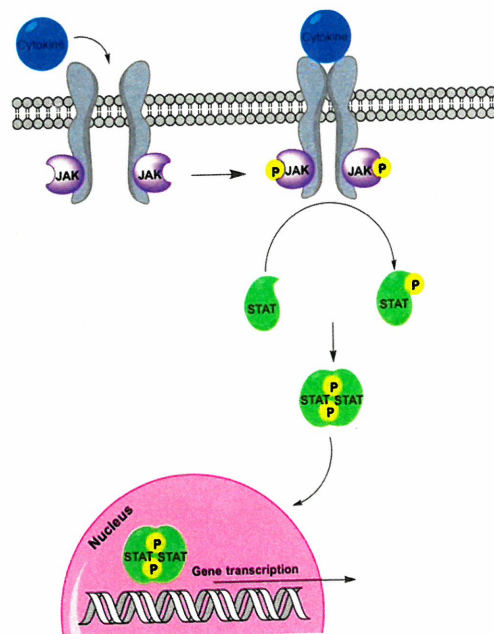
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mechanism of action of insulin :



Insulin receptor substrate undergoes phosphorylation $\rightarrow$ Phosphoinositide 3-kinase (PI3K) / Akt 1/2 activated $\rightarrow$ Activates GLUT 4 $\rightarrow$ Glucose uptake.
 GLUT 4 : seen in Heart, skeletal muscles, Adipose tissue.

JAK stat pathway :



- Phosphorylation of STAT proteins seen.
- GH (Growth hormone), prolactin (Twin hormones) and EPO (erythropoietin) act via this pathway.

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Serine-threonine kinase pathway

00:56:40

Inhibin, Bone morphogenic Protein -7, activin, TGF-beta (most potent fibrogenic cytokine).

Smad proteins activate the pathway.

Exception: TGF-beta → Serine Threonine Kinase pathway.

All other growth factors + TGF alpha → Tyrosine Kinase pathway.

BASICS OF PITUITARY GLAND

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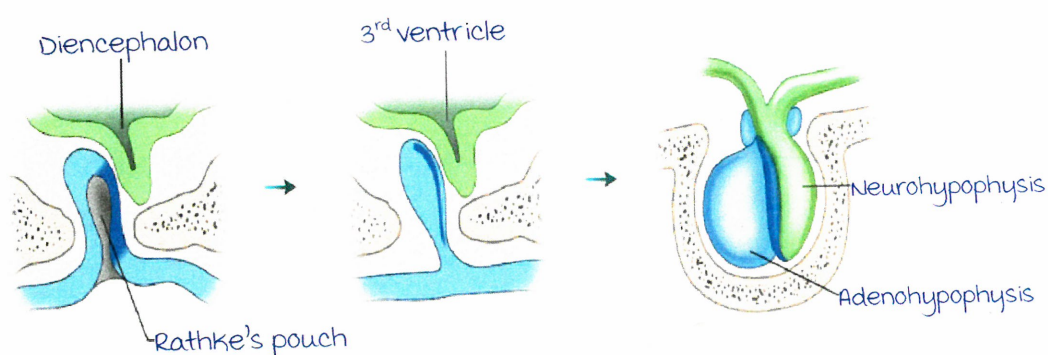
master gland of the endocrine system (regulates everything).

Size = 600 mg, enlarges itself to 1 g during pregnancy.

Divided into :

- Anterior pituitary gland.
- Posterior pituitary gland.

	Anterior pituitary gland	Posterior pituitary gland
Developed from	Rathke's pouch (upgrowth from the roof of the oral cavity)	Downgrowth from the floor of the 3rd ventricle
Derivative of	Surface ectoderm	Neuroectoderm



Congenital hypopituitarism :

- m/c cause : Pituitary Dysplasia.
- Pituitary dysplasia → Anterior pituitary hormones not produced, posterior pituitary gland is intact.

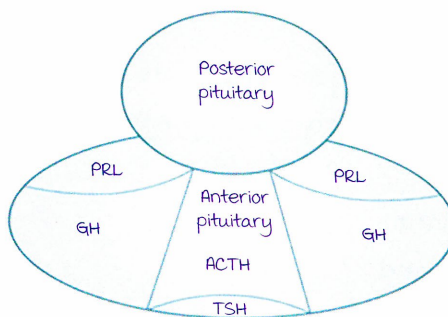
Clinical features :

- midline cranio-facial abnormalities (single central incisor, cleft lip etc)
- Anterior pituitary formed from → Rathke's pouch → Located in the nasopharynx → From there cells migrate across the midline → reach the pituitary. Because of this migration, pituitary dysplasia causes midline cranio-facial abnormalities.

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Anterior pituitary

00:06:14



Cells	Development	Hormones produced	Amount of cells seen	Notes	Produced from which part
Somatotrophs	12 weeks	Growth Hormones (Polypeptide hormones)	most abundant cells (50%)	(191 amino acids)	Produced from lateral portion of anterior pituitary
Lactotrophs	Last cells to develop (34 weeks of intrauterine life)	Prolactin (Polypeptide hormones)	15% of cells in anterior pituitary	(199 amino acids)	Produced from lateral portion of anterior pituitary
Corticotrophs	Earliest cells to develop (6 weeks of intrauterine life)	POMC (Pro opiomelanocorticotrophic hormone)		POMC = ACTH + MSH + Beta-lipotrophins (endorphins are derived from them)	Produced from central portion of anterior pituitary
Gonadotrophs	12 weeks	FSH, LH	15% of cells in anterior pituitary		Scattered throughout the pituitary
Thyrotrophs	12 weeks	TSH	5% of cells (least)		Produced from central portion of anterior pituitary

Note : 4 hormones of happiness :

- Dopamine (hormone of pleasure).
- Oxytocin (hormone of love).
- Serotonin (hormone of mood stabilization).
- Endorphins (natural pain killer) (Endorphins inhibit μ -receptor $\rightarrow$ Decreased substance P $\rightarrow$ Decreased pain.)