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| <p>1. Hormonal Feedback Regulators MOA: Analogs of GnRH → activates GnRH release → Increased LH/FSH release (pulsatile)</p> <p><u>Leuprolide</u> <u>Goserelin</u> <u>Gonadotropins</u> – FSH, LH, hCG USE: Pulsatile: Ovulation/conception, DX of hypogonadism Continuous: Breast CA, Prostate CA, Uterine fibroids, endometriosis, precocious puberty SE: Pulsatile: Ovarian hyperstimulation, Continuous: Hot flushes, Impotence, osteoporosis</p> | <p>3. Estrogen Antagonists/Partial Agonists MOA: Occupies Estrogen receptor → Increased GnRH release <i>Clomiphene</i> – Partial Estrogen agonist <i>Fulvestrant</i> – Full Estrogen Antagonist USE: <i>Clomiphene</i>: Ovulation/conception <i>Fulvestrant</i>: Breast CA, Uterine fibroids, endometriosis SE: <i>Clomiphene</i>: Multiple births, Mood swings <i>Fulvestrant</i>: DVT, Hot flushes, osteoporosis</p> | <p>5. Selective Estrogen Receptor Modulators MOA: Modulates Estrogen Receptor → Tissue specific <i>Tamoxifen</i> Antagonist-breast, Agonist-bone/endometrium <i>Raloxifene</i> Antagonist-breast, uterus, Agonist-bone <i>Ospemifene</i> Agonist-Uterus, Antagonist-other tissues USE: ER(+) breast cancer, Osteoporosis, painful intercourse (Ospemifene) SE: <i>Tamoxifen</i>: Hot flushes, ↑ risk of DVT and endometrial CA <i>Raloxifene</i>: Hot Flushes, ↑ risk of DVT</p> | <p>7. Estrogens MOA: Bind to Estrogen receptors <i>Estradiol, ethinyl estrogen, DES</i> USE: Contraception, Hormone replacement SE: ↑ risk of DVT, endometrial CA,</p> <p>8. Progesterone agonists MOA: Bind to Progestin receptors <i>Levonorgestrel norethindrone medroxyprogesterone</i> USE: Emergency Contraception, Combined with Estrogens, abnormal uterine bleeding, Uterine cancer SE: acne, depression, weight gain, breast tenderness</p> |
| <p>2. GnRH receptor antagonists MOA: Suppresses Pituitary mediated FSH/LH release <u>Ganarelix</u>, <u>Cetorelix</u> USE: In vitro reproduction, Reproductive cancers SE: Edema, N/V/D</p> | <p>4. Aromatase inhibitors MOA: Prevents the conversion of testosterone to estrogen → decreased ER receptor activation <i>Anastrozole, Letrozole</i> USE: Postmenopausal ER(+) breast cancer (SERM resistant) SE: Osteoporosis, ↑ risk of DVT</p> | <p>6. Anti-progesterone agents MOA: Antagonize Progestin receptors <i>Mifepristone, Ulipristal</i> USE: Terminal of pregnancy, emergency contraception SE: acne, depression, weight gain, breast tenderness</p> | |
| <p>This diagram illustrates the hormonal feedback system. The Hypothalamus (Medial Preoptic Nucleus) releases GnRH, which acts on the Pituitary to stimulate LH and FSH release. The Pituitary then acts on the Ovary and Testis. The Ovary releases Androgen and Progestin, and the Testis releases TST. These hormones are converted to Estrogen and DHT respectively via aromatase and hydroxylases. Estrogen and DHT bind to ER-Receptors in Body Tissues, leading to Gene Transcription. The diagram also shows various feedback loops: 1. and 2. from pituitary to hypothalamus; 3., 5., and 7. from body tissues back to hypothalamus; 4. from pituitary to ovary/testis; 6. and 8. from body tissues back to pituitary; 9. and 10. from pituitary to testis; 11. from testis to pituitary; 12. and 13. from body tissues back to pituitary.</p> | <p>This diagram details steroidogenesis. In the Ovary, LH and FSH stimulate Androgen and Progestin release. Androgen is converted to Estrogen by aromatase. In the Testis, TST is converted to DHT by hydroxylases and 5-α-reductase. Estrogen and DHT bind to their respective ER-Receptors in Body Tissues to regulate Gene Transcription. Numbered boxes indicate specific feedback points: 4. from pituitary to ovary/testis; 11. from testis to pituitary; 12. and 13. from body tissues back to pituitary.</p> | | <p>This diagram shows the actions of Estrogen and Progestin across different tissues. Estrogen leads to inhibition of libido, osteoclast activity, LDL, and HDL levels, while promoting breast proliferation, weight gain, and endometrial maintenance. Progestin leads to depression, acne, weight gain, and cervical mucus changes. Numbered boxes indicate specific actions: 3., 5., and 7. from body tissues back to hypothalamus; 8. and 6. from body tissues back to pituitary; 12. and 13. from body tissues back to pituitary.</p> |
| <p>9. Hormonal Feedback Regulators MOA: Analogs of GnRH → activates GnRH release → Increased LH/FSH release (pulsatile)</p> <p><u>Leuprolide</u>, <u>Goserelin</u> <u>Gonadotropins</u> – LH, FSH, hCG USE: Pulsatile: Fertility, spermatogenesis Continuous: BPH, Prostate CA, SE: Gynecomastia, Impotence</p> | <p>11. Androgen synthesis inhibitors MOA: Inhibit enzymatic synthesis of testosterone <i>Ketoconazole</i>: inhibits 6β hydroxylase <i>Abiraterone</i>: inhibits 17α hydroxylase <i>Finasteride</i>: inhibits 5 α reductase USE: BPH, Prostate CA, Male pattern baldness SE: Gynecomastia, impotence</p> | <p>12. Anti-androgens MOA: Androgen receptor antagonist <i>Flutamide</i> <i>Bicalutamide</i> USE: BPH, Prostate CA – prevent androgen surge from Goserelin SE: Hot flushes, Gynecomastia, ↑ risk of Breast CA</p> | <p>13. Androgens, Anabolic steroids MOA: Androgen and anabolic mediated effects <i>Testosterone, Testosterone cypionate, testosterone esters</i> <i>Dromostanolone</i> – Anabolic steroid USE: AIDS patients USE: Hypogonadism, stimulate anabolism after injury/illness SE: Acne, edema, ↑Prostate size, adverse lipid panel, hypercalcemia, erythrocytosis, Liver toxicity – Alkylated testosterone,</p> |
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