Interaction of Hormones at Target Cells

1) **Permissiveness** –
one hormone cannot exert its effects without another hormone being present

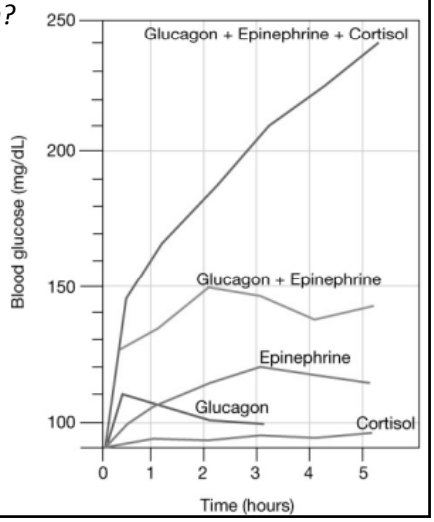
2) **Synergism** –
more than one hormone produces same effects on a target cell

3) **Antagonism** –
one or more hormones opposes action of another hormone

Hormone Interactions

Synergism & Antagonism:
- multiple synergistic stimuli more than additive
- insulin opposes glucagon

Is insulin a competitive inhibitor of glucagon?



Permissiveness:
- need 2nd hormone to get full expression
- e.g. reproductive development

Adrenal Androgens

Source: zona recticularis
physiological role??
very low titers compared to gonadal production
females: contribute to pubertal changes:
adrenarche
presence of pubic hair < 8 years old
development of axillary (armpit) hair & adult axillary odor
premature production ↑ dehydroepiandosterone (DHEA)
↑ growth : advancing bone age & clitoral enlargement
↑ incidence in African American females
benign condition that requires no treatment
abnormal growth acceleration
development secondary traits

pregnancy: abnormal production due to tumor or steroidogenesis
morphogenetic consequences:
mammary carcinomas
cigarette smoking
dehydroepiandosterone (DHEA)
antiestrogenic effect
breast cancer/endometrial cancer risks
cardiovascular disease & osteoporosis

postmenopausal:
substrate for extragonadal estrogen production

males: contribute prepurbertal changes
1) adrenarche
pubic hair < 9 years old
penal enlargement
2) prostate carcinomas

Common Inherited Disorders of Steroid Hormone Metabolism

↓ 21-hydroxylase enzyme: synthesis of glucocorticoids/mineralocorticoids

↓ glucocorticoids → ↑ ACTH anterior pituitary gland
normal feedback mechanism

↑ adrenal glands enlarge:

↑ pregnenolone synthesis

↑ progesterone

↑ 17α-Hydroxyprogesterone

↑ androgens

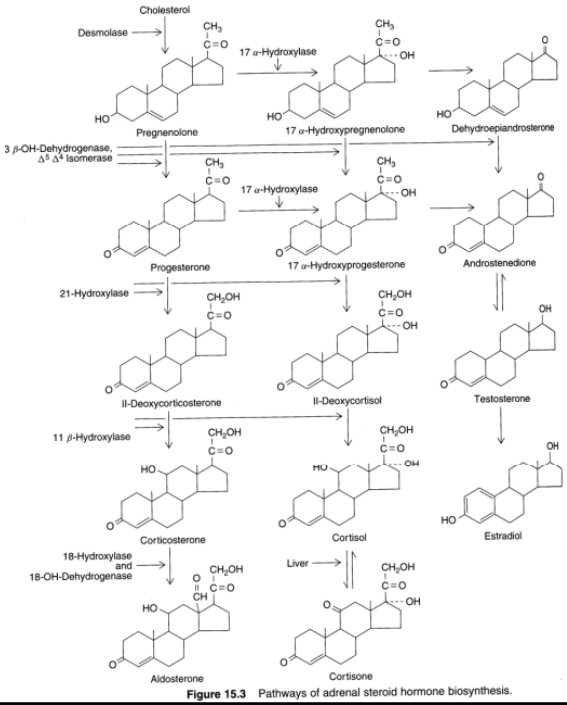


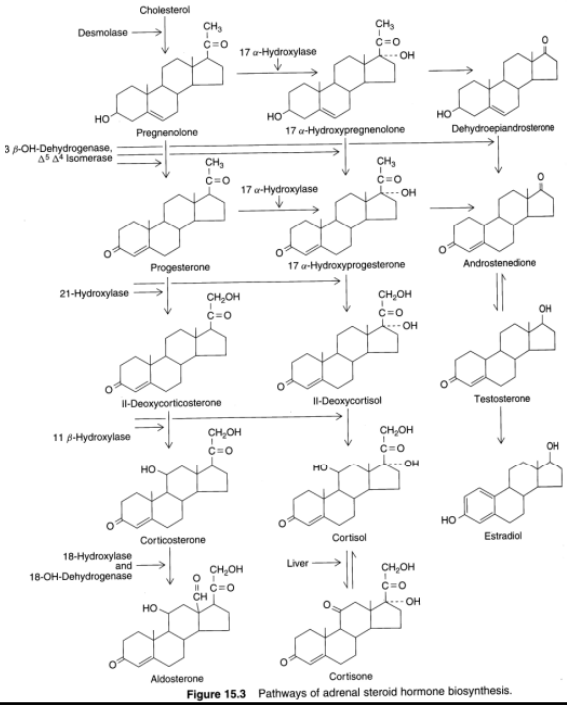
Figure 15.3 Pathways of adrenal steroid hormone biosynthesis.

A) Effects of high levels of androgens
- 1) Development of female fetus → masculinization of external gentalia
- 2) Development of male fetus
  - a) sexual organs normal at birth
  - b) sexual precocity apparent several months later
  - c) ↑↑ growth & ↑↑ early bone maturations → short stature
- 3) 50% with 21-Hydroxylase deficiency lose Na^+ in urine
- 4) ↓ titers aldosterone (mineralocorticoid)
  - loss of salts → dehydration & hypotension

5) Therapy
- a) administration of glucocorticoid hormone: ↓ ACTH secretion
- b) mineralocorticoid for those who lose salts
- c) symptoms reversed: therapy started within first 2 years after birth

B) Congenital adrenal hyperplasia (inherited disease)
1) affect enzymes required for steroidogenesis
11 β- Hydroxylase virilization*
17 α-Hydroxylase
3 β-OH-Dehydrogenase
Desmolase
2) all above enzymes deficiency →
enlargement of adrenal gland

**development of exaggerated masculine characteristics, usually in women, often as a result of adrenal glands overproducing androgens*



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