Chemical Signals

[Note: This is the text version of this lecture file. To make the lecture notes downloadable over a slow connection (e.g. modem) the figures have been replaced with figure numbers as found in the textbook. See the full version with complete graphics if you have a faster connection.]

Two classes of receptors: membrane and intracellular receptors

[See Fig. 45.3]

Response to most chemical signals through membrane receptors involves second messengers

(e.g. cAMP, cGMP, IP_3, Ca^{2+})

[See Fig. 11.12]

Some hormones (especially steroids) have intracellular receptors ("nuclear receptors") that regulate gene expression

[See Fig. 45.5]

One chemical signal can have different effects

1) different receptors: nicotinic acetylcholine receptors depolarize skeletal muscle; muscarinic acetycholine receptors activate G proteins and hyperpolarize cardiac muscle
2) different intracellular pathways: acetylcholine receptors can trigger intracellular release or influx of Ca^{2+} and hormone secretion (tropic hormones trigger release of second hormone)

[See Fig. 45.4]

One chemical signal can have different effects

Thyroxine secreted from human thyroid gland regulates metabolic rate but stimulates metamorphosis of tadpole into frog

[See Fig. 45.6]

[See Fig. 45.8]

Chemical signal modes of action

1) pheromones: signaling between organisms
2) local regulation: direct signaling between cells
3) hormonal: indirect signaling through blood or interstitial fluid

[See Fig. 11.3]

Examples of local regulators

NO (nitric oxide) is a gas

- **neurons: acts as neurotransmitter**
- **white blood cells: used to kill invaders and damaged cells**
- **endothelial cells: relaxes smooth muscle**

***Viagra (sildenafil) inhibits phosphdiesterase type V (PDE-V) and prolongs effect of NO. Used to treat disorders of blood flow like angina and impotence*. NO ⇒ guanylate cyclase ⇒ cGMP ⇒ PKG ⇒ phosphorylation; cGMP + PDE ⇒ GMP**

Growth factors are generally peptides (proteins)

- **nerve growth factor (NGF)**
- **epithelial growth factor (EGF)**
- **insulin-like growth factor (IGF)**
- **transforming growth factor (TGF)**

Prostaglandins (PGs) are modified fatty acids

- **discovered in semen (prostate secretion)**
- **released from most cells into interstitial fluid**
- **PGE and PGF relax and constrict blood vessels of lung to regulate oxygenation**
- **PGs also regulate fever and pain (*aspirin and ibuprofen* inhibit PG synthesis)**

Vertebrate endocrine system

(don’t forget organs of the digestive system, excretory system, and circulatory system)

[See Fig. 45.6]

Antagonistic hormones insure accurate regulation

[See Fig. 45.1]

the posterior pituitary (neurohypophysis) is an extension of hypothalamus

[See Fig. 45.7a]

the anterior pituitary (adenohypophysis) develops from the roof of the mouth (adenoids)

[See Fig. 45.7b]

[See Fig. 45.7b]

- **GH is a 200 amino acid protein**
  <u>stimulates growth directly</u>
  <u>stimulates release of other factors</u>: tropic action (e.g. IGF from liver)

  too much ⇒ gigantism (childhood) or acromegaly (middle age)
  too little ⇒ dwarfism

Gigantism in identical twins

Acromegaly: Before and after

Dwarfism

The anterior pituitary also secretes gonadotropins (FSH, LH) to regulate gonadal function

[See Fig. 46.14]

***mineralocorts*.**
(e.g.
<u>aldosterone</u>)
***glucocorts*.**
(e.g. <u>cortisol</u>)

[See Fig. 45.14a]

[See Fig. 45.15]

Thyroid gland and thyroid hormones

[See Fig. 45.8 & 45.9]

Thyroid gland and thyroid hormones

⇑ = <u>hyperthyroidism</u>: ⇑ body temp, sweating, weight loss, blood pressure, irritability

⇓ = <u>hypothyroidism</u>: opposite symptoms in adults, <u>cretinism</u> in infants (decreased brain and bone growth)

<u>goiter</u> (enlarged thyroid) caused by lack of iodine in diet (reason salt is <u>iodized</u> now).

[See Fig. 45.10]

[See Fig. 45.11]

Islets of Langerhans contain α & β cells (1-2% of pancreas)

[glucose] = 90 mg/dL

Diabetes mellitus

<u>Diabetes</u> is from *Greek* for ⇑ urination (diuresis)
<u>mellitus</u> is *Greek* for honey (glucose in urine)

⇓ beta cells ⇒ ⇓ insulin ⇒ ⇑ glucose in blood ⇒ ⇑ glucose secretion ⇒ ⇑ urination ⇒ ⇑ thirst

⇓ glucose in cells ⇒ ⇑ fat metabolism ⇒ ⇓ blood pH (acidosis)

<u>Type I</u> (insulin dependent)
- **usually occurs in childhood**
- **may be caused by autoimmune disorder**
- **β cells are destroyed**

<u>Type II</u> (non-insulin dependent)
- **usually occurs after age 40**
- **>90% of diabetics are Type II**
- **may be caused by change in <u>insulin receptors</u>**
- **heredity and weight are important**

[See Fig. 45.14b]

[See Fig. 46.8]