FEEDBACK REGULATION CHART

HORMONE	ORIGIN	CLASS/ TYPE	TARGET(S)	MAJOR ACTIONS	FEEDBACK REGULATION	
					POSITIVE	NEGATIVE
GH	**ANT. PIT.**	**PROT.**	LIVER → IGFs	SOMATIC, GROWTH METABOLISM **Acute** **Prolonged** + glucose uptake – glucose uptake + prot. synthesis – prot. synthesis – fat breakdown + fat breakdown	GHRH FASTING	SOMATOSTATIN
PRL	ANT. PIT.	PROT.	MAMMARY GL.	MILK PRODUCTION	PRH(?), STRESS	DA
ADH	HYPOTH. (Synthesis); POST.PIT (Secretion)	PEPTIDE 9aa	KIDNEY BLOOD VESSELS	WATER CONSERVATION; VASOCONSTRICTION	OSMOTIC PRESSURE, VOLUME DEPLETION, HYPOGLYCEMIA, HYPOXIA, STRESS, PAIN, EXERCISE, Pgs, BETA BLOCKERS,	ALCOHOL, ALPHA BLOCKERS, GLUCOCORT.

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OXYTOC.	HYPOTH (Synthesis) POST. PIT. (Secretion)	PEPT. 9aa	MAMMARY MYOEPITHEL. CELLS; UTERINE SMOOTH MUSCLE	MILK EJECTION (LET DOWN); UTERINE CONTRACTIONS (PARTURITION)	(INFANT SUCKLING, SIGHT, SOUND); (UTERINE STRETCH)	----
FSH, LH [MALE]	ANT. PIT. GONADO-TROPHS	GLYCO-PROT.; GONADO-TROPINS	FSH → TESTES (SERTOLI C.); LH → TESTES (LEYDIG C.)	FSH: SPERMATOGENESIS; LH: TESTOSTERONE SYNTHESIS	GnRH	↑ ANDROGENS, INHIBIN
FSH, LH [FEMALE]	ANT. PIT. GONADO-TROPHS	GLYCO-PROT.; GONADO-TROPINS	FSH → OVARIES (GRANULOSA C.); LH → OVARIES (THECAL C.)	OVARIAN FOLLICLE DEVELOPMENT AND OVULATION, OVARIAN STEROID SYNTHESIS: ESTROGEN + PROGESTERONE	GnRH	↑ ESTROGENS

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HORMONE	ORIGIN	CLASS TYPE	TARGET(S)	MAJOR ACTIONS	FEEDBACK REGULATION	
					POSITIVE	NEGATIVE
ACTH [POMC]	HYPOTHAL. and ANT. PIT. CORTICO-TROPHS	PEPTIDE 39aa	ADRENAL CORTEX	STIMULATES ADRENAL GLUCO-CORTICOID (CORTISOL) SYNTHESIS	CRH	↑ GLUCOCORTI-COIDS (NATURAL and SYNTHETIC)
TSH	ANT. PIT. THYRO-TROPHS	PEPTIDE 9aa	THYROID FOLLICULAR CELLS	STIMULATES THYROID GLAND SYNTHESIS OF T_3 and T_4	TRH	↑ **FREE T_3,** ↑ **FREE T_4**

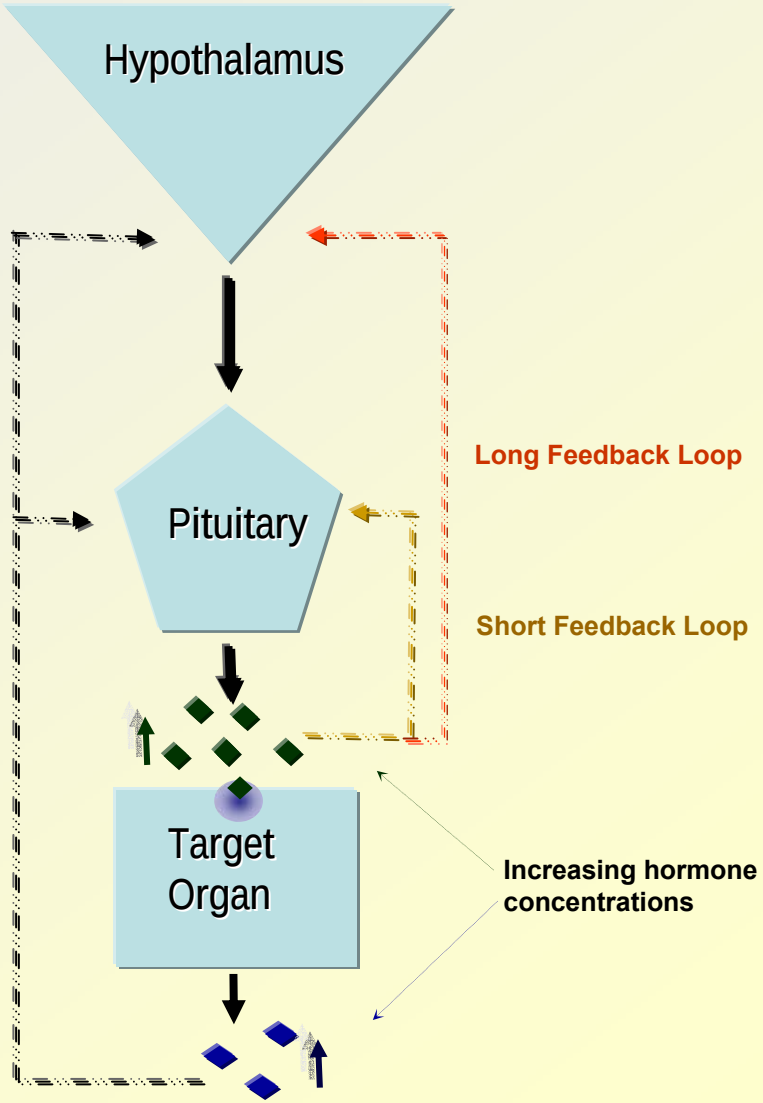
FEEDBACK REGULATION CHART ≟ Practice Sheet

HORMONE	ORIGIN	CLASS/ TYPE	TARGET(S)	MAJOR ACTIONS	FEEDBACK REGULATION	
					POSITIVE	NEGATIVE

General Scheme for Feedback Regulation in the Endocrine System

Hypothalamic-Hypophyseal-Target Organ Axis

- The hypothalamus influences pituitary hormonal secretions by *releasing* and *inhibiting factors;*
- The pituitary responds by secreting *hormones that stimulate target organs;*
- Target organs secrete hormones that may exert positive or negative feedback at the level of the pituitary and/or hypothalamus
- Note that they can increase the secretion of inhibitory (as well as releasing) factors



Different types of feedback regulation occur in the endocrine system. Feedback can be positive, negative, endocrine, paracrine, autocrine, and/or neurocrine

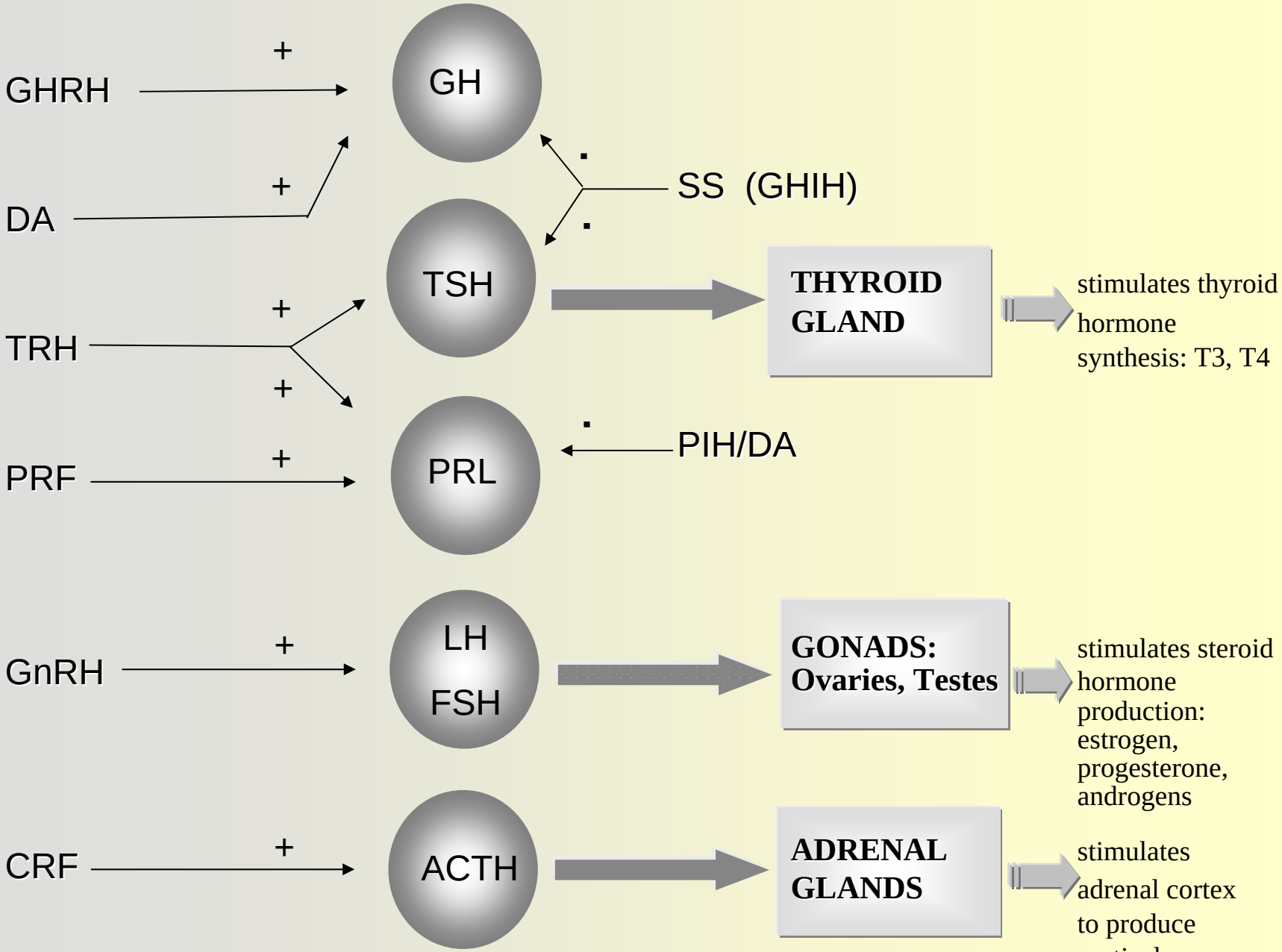
In ***endocrine regulation*** hormones regulate 'distant' targets, usually by traveling through the bloodstream.

In ***paracrine regulation***, a hormone produced by one cell influences the activities of another cell in the same organ. This usually occurs between 'adjacent cells'. There are many examples of this type of regulation in the endocrine pancreas and in the gonads.

In ***autocrine regulation*** the activity targeted by a hormone is in the 'same cell' that produced it. Examples of this type of regulation are demonstrated by many prostaglandins and growth factors.

In ***neurocrine regulation*** hormones reach their targets by 'dendritic or axonal' transport.

[Also note that a hormone can participate more than one kind of regulation.]


GHRH
+
GH
DA
+
SS (GHIH)
TSH
THYROID GLAND
stimulates thyroid hormone synthesis: T3, T4
TRH
+
PRF
+
PRL
PIH/DA
GnRH
+
LH
FSH
GONADS: Ovaries, Testes
stimulates steroid hormone production: estrogen, progesterone, androgens
CRF
+
ACTH
ADRENAL GLANDS
stimulates adrenal cortex to produce cortisol…

► GROWTH HORMONE

GH regulation occurs on multiple levels and includes both positive and negative feedfback loops

- **The primary metabolic action of GH is ANABOLIC;**

- **GH diverts nutrients away from fat storage and toward protein synthesis;**

- **Thus, GH ultimately causes an increase in *lean body mass***

- **GH acts on carbohydrates (CHO), proteins, and lipids.**

Major Effects of Growth Hormone

1. Promotes growth: Induces precursor cells in bone and other tissues to differentiate and secrete insulin-like growth factor I (IGF-I), which stimulates cell division. Also stimulates secretion of IGF-I by liver.
2. Stimulates protein synthesis, predominantly in muscle.
3. Anti-insulin effects:
   a. Renders adipocytes more responsive to lipolytic stimuli
   b. Stimulates gluconeogenesis
   c. Reduces the ability of insulin to stimulate glucose uptake

The Impact of GH on Metabolism and Growth

Metabolism

CHO

↑Glucose
↑Insulin
↓Peripheral
insulin sensitivity

PROTEINS

↑aa tissue uptake and protein incorporation
+ N2 balance
↑ urea production

LIPIDS

↑ lipolysis
[ketogenic]

Growth

⇧**Skeletal growth**

⇧ **Visceral growth**

⇧ **Cartilage growth**

⇧ **Long bone growth**

⇧ **rate of healing**
[mediated by IGFs]

⇩ **injury rates in athletes**
(stress fractures, torn tendons)

MAJOR HORMONES INFLUENCING GROWTH

GH ★	major stimulus for: postnatal growth, hepatic IGF-I, and other mediators of growth and protein anabolism
INSULIN ★	stimulates fetal growth; contributes to postnatal growth by enhancing IGF-I secretion; increases protein synthesis
T_3, T_4	permissive to GH's actions; permissive to development of the CNS
TESTOSTERONE	stimulates: adolescent growth spurt, GH secretion, and protein synthesis; has late effects on epiphyseal closure
E_2	stimulate GH secretion (esp. during adolescence), is mandatory for statural growth (♀ and ♂) and subsequent epiphyseal closure
CORTISOL★	has catabolic effects and is inhibitory to growth

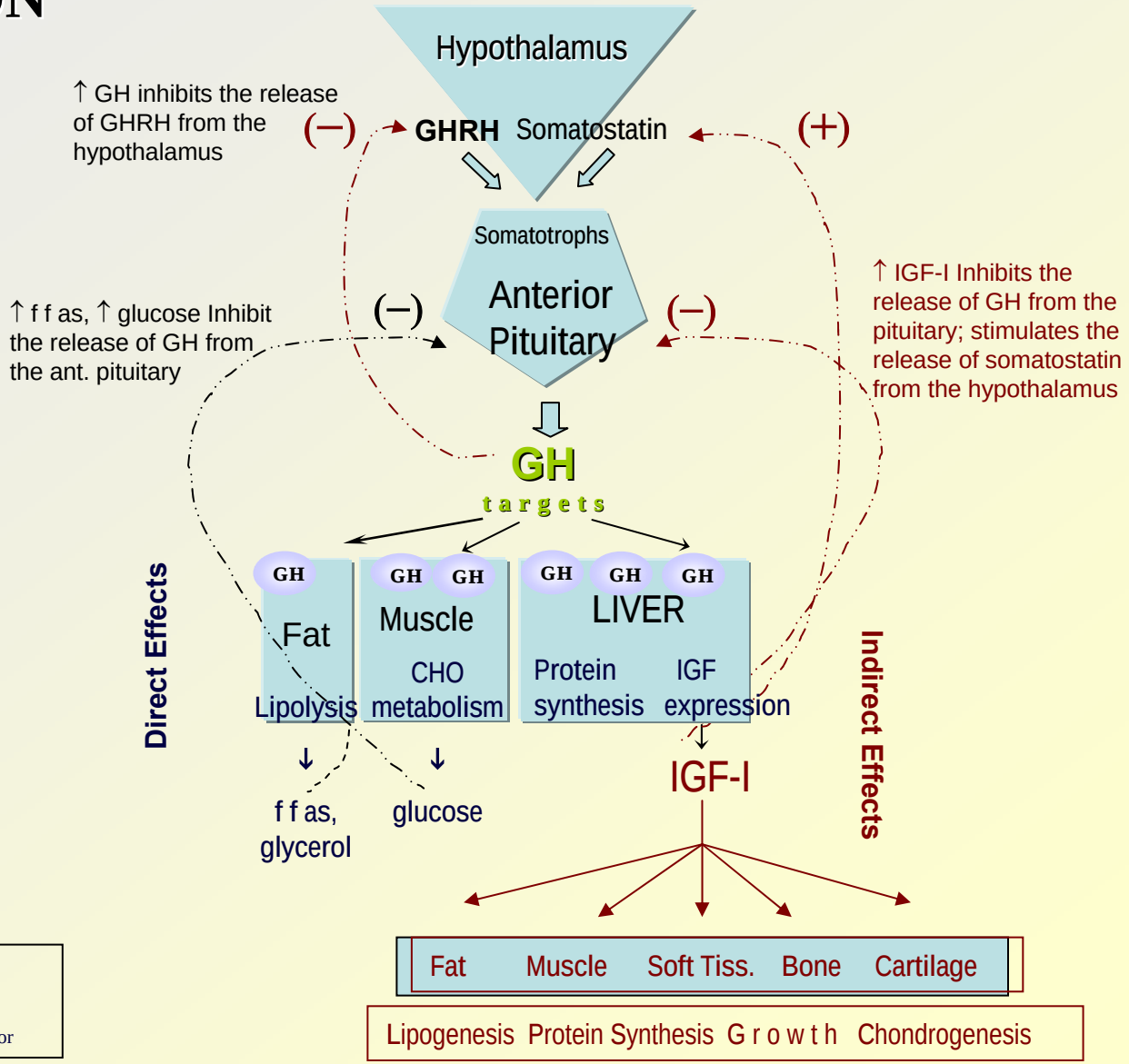
Stars denotes hormones that are considered to be ★ anabolic or ★ catabolic

- **Under normal circumstances, the primary metabolic action of GH is ANABOLIC, it increases lean body mass.**

- **However, in the *absence of adequate insulin* reserves GH is no longer anabolic, but becomes CATABOLIC, leading the breakdown of many body tissues and to ketogenesis.**

FEEDBACK REGULATION

GH Increases Post-Natal Growth and Lean Body Mass



f f a s = free fatty acids

CHO = carbohydrate

IGF = insulin-like growth factor

GH and IGF-1 Regulation and Feedback Loops