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# CHEMICAL FOUNDATIONS OF HORMONE ACTION AND HORMONAL RECEPTORS

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#### ABSTRACT

Hormones are biologically active chemical substances that regulate virtually all physiological processes in the human body—including growth, metabolism, reproduction, stress responses, and the maintenance of homeostasis. Their action is based on the highly selective interaction between a hormone and its specific receptor, which triggers a cascade of intracellular signaling events. This article examines the chemical nature of various classes of hormones, the mechanisms of receptor binding, the main types of hormonal receptors, and the pathways of intracellular signal transduction. Particular attention is given to the role of hormone–receptor interactions in metabolic regulation, as well as their significance for the diagnosis and treatment of endocrine disorders.

#### KEYWORDS

Hormones, hormone receptors, endocrine system, chemical structure of hormones, intracellular signal transduction, metabolism.

### Introduction

Hormones are biologically active chemical substances secreted by endocrine glands and acting on distant target organs. Their uniqueness lies in their high specificity: each hormone affects only those cells that contain its specific receptor. Understanding the chemical mechanism of hormone action is crucial for medicine, as most endocrine disorders (diabetes mellitus, hypothyroidism, Cushing's syndrome, infertility) arise from dysfunction in the hormone–receptor interaction. Modern pharmacology develops drugs that either mimic natural hormones or block their receptors.

Hormones and their receptors constitute an extremely precise chemical system that regulates all vital processes in the body. The chemical structure of a hormone determines its ability to bind to a specific receptor and initiate defined intracellular responses [1-3]. Disruption of any component-hormone, receptor, or signaling pathway-leads to endocrine pathologies. Understanding the molecular

43. December - 2025

mechanisms of hormonal action forms the foundation of contemporary endocrinology, pharmacology, and clinical medicine.

## **Main Chapters**

Hormones can be divided into several major classes according to their chemical nature. Peptide hormones (insulin, vasopressin, oxytocin) are chains of amino acids synthesized as precursors (proproteins) and processed post-translationally. These hormones are hydrophilic and cannot freely cross the lipid bilayer of the cell membrane; their receptors are located on the cell surface and belong to families such as GPCRs (G protein–coupled receptors), receptor tyrosine kinases, and ionotropic receptors. Binding of a peptide hormone to its membrane receptor rapidly activates secondary messengers (cAMP, IP<sub>3</sub>/DAG, intracellular Ca<sup>2+</sup>), which modulate the activity of enzymes and ion channels and ultimately produce a physiological response [4-6].

Steroid hormones (corticosteroids, androgens, estrogens, progesterone) are lipophilic derivatives of cholesterol. They readily diffuse across the membrane and bind to receptors in the cytoplasm or nucleus. The resulting hormone–receptor complex translocates into the nucleus, binds to regulatory DNA sequences, and modulates the transcription of target genes, thereby exerting long-lasting effects through regulation of protein synthesis and functional remodeling of tissues [7].

Amine hormones (adrenaline, noradrenaline, thyroxine) are derived from amino acids (tyrosine, tryptophan). Adrenaline and noradrenaline act predominantly through GPCRs and rapidly regulate vascular tone, heart rate, and metabolism. Thyroxine (T<sub>4</sub>) and triiodothyronine (T<sub>3</sub>) exhibit properties similar to steroid hormones: they circulate in protein-bound form and act via nuclear receptors, regulating the transcription of genes responsible for basal metabolic rate [8].

Eicosanoids (prostaglandins, thromboxanes, leukotrienes) are local lipid mediators derived from arachidonic acid. They have a short half-life and act through membrane receptors to rapidly regulate inflammatory and vascular responses.

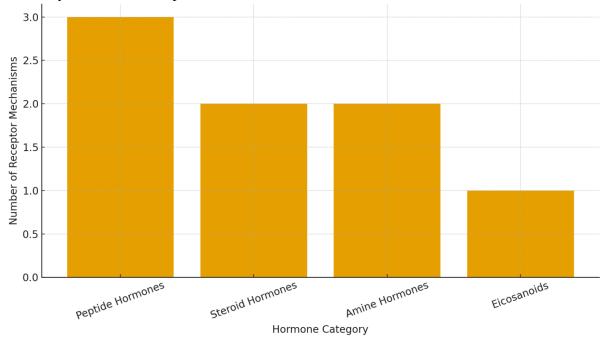


Fig.1. Hormone Types and Their Receptor Diversity

43, December - 2025

Signal transduction mechanisms include activation of adenylyl cyclase with subsequent cAMP production and activation of protein kinase A (PKA); activation of phospholipase C leading to the formation of IP<sub>3</sub> and DAG (IP<sub>3</sub> induces Ca<sup>2+</sup> release from the endoplasmic reticulum, while DAG activates PKC); as well as direct tyrosine kinase activity (phosphorylation of target proteins) and proteolysis/modification of transcription factors. The final cellular response depends on context: receptor type, availability of secondary messengers, cross-regulatory mechanisms, and the expression level of receptor isoforms.

Pathology within the hormone–receptor system may be associated with insufficient hormone secretion (primary deficiency), excessive secretion (adenomas), receptor defects (hormone resistance), or impairments in post-receptor signaling pathways (secondary messenger abnormalities). A classical example is insulin resistance in type 2 diabetes mellitus, in which normal or elevated insulin levels fail to produce adequate glucose uptake due to defects in the insulin receptor or downstream signaling pathways (PI3K/Akt) [9].

Clinical applications of knowledge about hormone–receptor interactions include replacement therapy (insulin therapy, thyroxine replacement), the use of receptor agonists and antagonists ( $\beta$ -blockers, antiestrogens, angiotensin II antagonists), as well as hormone-based therapy in oncology (aromatase inhibitors in estrogen-dependent breast cancer). Modern approaches also involve the development of small-molecule and biological agents targeting components of signaling cascades (tyrosine kinase inhibitors, monoclonal antibodies).

In conclusion, the chemical structure of hormones determines their transport pathways, receptor localization, and mechanisms of signal transduction. The distinctions between hydrophilic and lipophilic hormones underpin fundamentally different modes of action—ranging from rapid post-translational regulation to long-term transcriptional remodeling. A clinical understanding of these mechanisms forms the basis for the diagnosis and treatment of a wide range of endocrine and somatic disorders.

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43, December - 2025

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