Hormones and their Regulation of Body Processes in Animals

Parwe S.S[1] & Shinde S.Y[2]
Department of
Zoology& Botany
Late Shankarrao Gutte Gramin ACS college,
Dharmapuri.Tq. Parli (V.) Dist. Beed

Abstract

Unlike plant hormones, animal hormones are often produced in specialized hormone-synthesizing glands. The hormones are then secreted from the glands into the blood stream, where they are transported throughout the body. There are many glands and hormones in different animal species. Hormones have a wide range of effects and modulate many different body processes. The key regulatory processes that will be examined here are those affecting blood glucose, hunger, metamorphosis, stress, and sex. The recent paper focused on the hormones and their regulation of body processes in animals.

Keywords: Hormones, animal hormones, synthesizing glands, Blood etc.

Introduction:

Hormones are chemicals secreted by various endocrine glands directly into the blood and transported to the respective organs, where they act by regulating various metabolic processes. The organ affected by the hormone is called the target organ. The target cells have specific receptors to recognize the respective hormones. There are two types of glands present in our body.

- Endocrine Glands– These glands that do not have ducts and transport their secretions directly to the site of action through the blood, e.g. adrenal glands, pituitary glands, etc.

- Exocrine Glands– These glands have ducts to pass their secretions, e.g., sweat, liver, etc.

Hormones are secreted by endocrine glands. Hormones control the functions of all the organs. They affect the diverse processes of growth and development, reproduction and sexual characteristics. Very small amounts of hormones can induce very prominent responses in the body. Most of the hormones are proteins or steroids.

Hormone Regulation of Body Processes in Animals:

Blood Glucose:

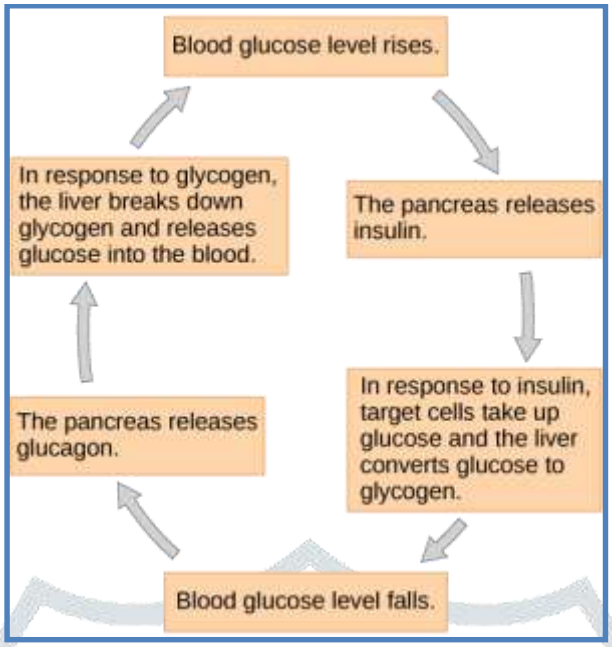
Glucose is the primary energy source for most animal cells, and it is distributed throughout the body via the blood stream. The ideal, or target, blood glucose concentration is about 90 mg/100 mL of blood, which equates to about 1 tsp of glucose per 6 quarts of blood. After a meal, carbohydrates are broken down during digestion and absorbed into the blood stream. The amount present following a meal is typically more than whatÂ the body needs at that moment, and so the extra glucose must be removed and stored for later use. The opposite phenomenon occurs following a period of fasting. **Insulin** and **glucagon** are the two hormones primarily responsible for maintaining appropriate blood glucose levels.

Insulin is produced by the beta cells of the pancreas, which are stimulated to release insulin as blood glucose levels rise (for example, after a meal is consumed). Insulin lowers blood glucose levels through several processes:

- enhances the rate of glucose uptake and utilization by target cells, which use glucose for ATP production
- stimulates the liver to convert glucose to glycogen, which is then stored by cells for later use
- increases glucose transport into certain cells, such as muscle cells and the liver
- stimulates the conversion of glucose to fat in adipocytes and the synthesis of proteins.

These actions together cause cause blood glucose concentrations to fall, called a hypoglycemic ‘low sugar’ effect, which inhibits further insulin release from beta cells through a **negative feedback loop**.The hormone **glucagon** is released from the alpha cells of the pancreas.

Glucose can then be utilized as energy by muscle cells and released into circulation by the liver cells. These actions mediated by glucagon result in an increase in blood glucose levels to normal homeostatic levels. Rising blood glucose levels inhibit further glucagon release by the pancreas via a **negative feedback mechanism**. In this way, insulin and glucagon work together to maintain homeostatic glucose levels, as shown in below.



Insulin and glucagon regulate blood glucose levels. When blood glucose levels fall, the pancreas secretes the hormone glucagon. Glucagon causes the liver to break down glycogen, releasing glucose into the blood. As a result, blood glucose levels rise. In response to high glucose levels, the pancreas releases insulin. In response to insulin, target cells take up glucose, and the liver converts glucose to glycogen. As a result, blood glucose levels fall

Hunger Management:

The immediate form of energy for most animal cells is glucose, and extra glucose is stored as glycogen which is readily broken down into glucose when needed. Longer term reserves of energy are stored as fats, in cells called adipocytes. Too little fat means there may no be enough energy reserves in times when food is less available, and will cause an animal to feel hungry; however, too much fat is generally unhealthy and is likely to cause an animal to feel satisfied. The hormone **leptin** helps maintain an appropriate amount of fat reserves in the body.It is produced by adipocytes in proportion to their number and size. More and larger adiopcytes means more leptin; fewer and smaller adipocytes means less leptin. Leptin levels are detected by sensors in the **hypothalamus**. High lepin levels suppress appetite and speed up metabolism, while low levels of leptin stimulate hunger and slow down metabolism, resulting in a negative feedback loop. These activities are mediated through signaling from the **hypothalamus-pituitary axis** to the **thyroid**, which plays a major role in regulating metabolic function.

In response to high levels of leptin, the hypothalamus releases thyrotropin-releasing hormone, signals to the anterior pituitary to release thyroid-stimulating hormone. The thyroid the releases **thyroxine**, also known as **tetraiodothyronine** or **T_4**, and **triiodothyronine**, also known as **T_3**. These hormones affect nearly every cell in the body except for the adult brain, uterus, testes, blood cells, and spleen. T_3 and T_4 activate genes involved in

energy production and glucose oxidation, resulting in increased rates of metabolism and body heat production which together cause an increased rate of caloric usage. Low levels of leptin cause the opposite response, leading to a decreased metabolic rate to conserve energy.

Growth and Metamorphosis:

In vertebrate species that undergo metamorphosis, such as amphibians, surges of T_3 are responsible for initiating development of new structures, reorganization of internal organ systems, and other processes that occur during metamorphosis. In insects, metamorphosis is controlled by a set of hormones that determine whether the animal grows into the next larval stage or changes into an adult as it gets larger. The corpus allatum, an endocrine gland in the brain, secretes a hormone called **juvenile hormone** during all larval stages, which maintains the larval status of the animal. As the larvae grows, another endocrine gland in the brain releases **prothoracicotropic hormone**, which signals to the prothoracic gland to release the hormone **ecdysone**. Ecdysone promotes either molting (shedding the exoskeleton) or metamorphosis, depending on the level of juvenile hormone. Ecdysone in combination with high juvenile hormone results in molting into the next larval stage; ecdysone in combination with low juvenile hormone results in metamorphosis into an adult.

Short-term Stress Response:

When presented with a stressful situation, the body responds by calling for the release of hormones that provide a burst of energy. The hormones **epinephrine** (also known as adrenaline) and **norepinephrine** (also known as noradrenaline) are released by the adrenal medulla. These two hormones prepare the body for a burst of energy in the following ways:

- cause glycogen to be broken down into glucose and released from liver and muscle cells
- increase blood pressure
- increase breathing rate
- increase metabolic rate
- change blood flow patterns, leading to increased blood flow to skeletal muscles, heart, and brain; and decreased blood flow to digestive system, skin, and kidneys

Long-term Stress Response

Long-term stress response differs substantially from short-term stress response. The body cannot sustain the bursts of energy mediated by epinephrine and norepinephrine for long times. Instead, other hormones come into play. In a long-term stress response, the hypothalamus triggers the release of ACTH from the anterior pituitary gland. The adrenal cortex is stimulated by ACTH to release steroid hormones called **corticosteroids.**Â The two main corticosteroids are **glucocorticoids** such as cortisol, and **mineralocorticoids** such as aldosterone.

Coticosteriods are under control of a negative feedback loop (illustrated below), which can become mis-regulated in cases of chronic long-term stress.

Conclusion:

Steroid hormones have an essential role in the growth, differentiation and function of many tissues in both animals and man. It is established by animal experimentation that modification of the hormonal environment by gonadectomy, by pregnancy or by exogenous administration of steroids can greatly increase or decrease the spontaneous occurrence of tumours or the induction of tumours by applied carcinogenic agents. In man also, there is evidence that differences in endogenous hormone levels may be associated with differences in tumour incidence. It is possible, therefore, that the incidence of human tumours could be increased or decreased by a specific mode of exogenous hormone administration, but this cannot be predicted.

The intake of steroids for effective contraceptive medication has to be sufficient to disturb the hormonal environment, and in fact such a disturbance is a requisite of fertility inhibition. The possibility that a carcinogenic risk may be involved in such medication must therefore be considered. For example, the minimum effective dose of diethylstilboestrol of 6 μg/kg bw/day for mammary carcinogenesis in mice is of the same order as the doses used for therapy in women (0.5-5 mg/day). At the same time, it should be remembered in regard to both oestrogens and progestins in contraceptive medication that the steroid hormones of pregnancy have actions similar to those of the contraceptive agents.

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