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Up Date from the Factors Related to the Development of Endocrine Diseases: A perspective biological chemistry

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Abstract

The endocrine system contributes to the functioning of the body's metabolism, the increase or decrease in hormones favors the formation of disorders that develop metabolic diseases. Aim. Compile information on factors that intervene in the development of endocrinological diseases, mainly in hormonal action and alterations of certain metabolites. Methodology. A bibliographic search was carried out in Google Schoolar and PudMed using keywords, the information was analyzed in pairs. Results. Hormonal action is affected by desynchronization in hormonal production, triggering metabolic diseases. Measurement of calcium, phosphorus, PTH, 25OHD, 1.25OHD, calcitonin and osteocalcin help in the diagnosis. Conclusion. Circadian alterations favor the development of metabolic diseases, the participation of the clinical laboratory with specific tests is important.

Keywords: Hormonal Action, Desynchronization, Diagnosis, Evidence

Introduction

The endocrine system es the set of internal hormone-secreting glands that are responsible for the proper functioning of the body's internal metabolism, both intracellularly and for tissues, organs and systems. Hormones are the body's chemicals messengers transmitting signals between cells and organs, they are transported through the circulatory system to tissues and organs, regulating heart rate, metabolism, appetite, mood, sexual function, growth and more. The increase or decrease in the concentration of hormones favors the appearance of physiological and behavioral disorders that culminate in the development of metabolism diseases. The target of the present review es to compile recent and relevant information on the factors involved in the development of endocrinological diseases, emphasizing the hormonal action and the alterations that some metabolites undergo to trigger a pathology.

In this sense, a bibliographic analysis was carried out in science dissemination databases such as Google Scholar and PubMed, which were chosen by searching for keywords that each topic contains, the inclusion criteria used to choose the sources of information are: Complete articles in English and Spanish, articles with a publication date from 2018 to 2023, the main ideas of each article were analyzed in pairs and a paraphrase was carried out with said information without altering its meaning and what the author intends to communicate, establishing the following categories.

Hormonal Action

Hormonal action es understood as the regulatory activities in which hormones, receptors, organs and target tissues participate to achieve an increase or decrease in the concentration of a specific hormone. Estos concentrations are of ultimate relevance for the clinical laboratory, because there are references indices that act as indicators of possible endocrine alterations, which in turn trigger the development of metabolism diseases [1, 2, 3].

The main protagonists of Este regulation are hormones, chemicals substances that can be secreted by both glands and endocrine tissues, where production obeys rhythmic patterns influenced by environmental factors, these can have a protein structure, steroids, amines or lipid structures such as cyclic fatty acids [1]. The hormonal regulation process works under feedback mechanisms, where two variables that may be hormonal, molecular or cellular in nature, maintain a communication relationship in which, through the increase or decrease in plasma concentration, they suggest a change in the production of the variable reciprocal [1].

Hormonal secretion es not carried out continuously and uniformly, rather Item presents a pulsatile behavior where there are periods of secretion and rest. Secretion can vary throughout the day, causing several cycles in a single day or up to 1 cycle in 28 days. Three sets of factors intervene in hormonal action: The synthesis and secretion of the hormone in its producing cells, hormonal metabolism. And the response of the target tissues with their specific receptors [4].
In the target tissues we find groups of membrane receptors and steroid hormones that perform signaling functions that will allow the team work of the endocrine, immune and nervous systems to culminate in the increase or decrease in hormonal production, the total daily secretion of a hormone is called secretion rate [1].
Free hormones are those that establish physiological effects in the human body, they play an important role in multiple laboratory tests, designed to quantify hormone concentrations *in vitro* to determine whether they are within or outside the referencerange [2].
Currently, as a consequence of modernization and industrialization, hormonal regulation can be altered or blocked by the action of polluting chemicals compounds found in the environment, which are known as “endocrine disruptors.” estos belong a variety of chemicals substance. Natural or artificial 6 such as herbicides, insecticides, fungicides, raw materials in the plastic, cosmetics and food industries, which are sometimes pollutants of the effluents of various industries; long- term exposure to these endocrine disruptors could cause permanent negative effects [3].
The concentration of endocrine disruptors in the bodysuit es look and low [2], many times below the limit of laboratory analysis capacity. The measurement of concentrations requires sophisticated equipment, something that makes Item difficult to maintain effective control of the levels in places of exposure [4], however substances cannot be transported by proteins since they have the characteristics of being biologically active components that alters the balance. Hormonal compromising the hormonal axes [3], interrupting hormonal action at a peripheral level, modifying its biosynthesis, transport or metabolism of steroids and at the same time induction enzymes of the CYP2B and CYP3A family. At the level of target organs, they activate or inhibit hormonal receptors, affecting their transcriptional activity [3].
A study carried out by Guo in 1996.5 demonstrated that in newborn males contaminated with polychlorinated biphenyl compounds (PCBs) and DDT, upon binding with the estrogen receptor, micropenis was observed; however, no effects on the size were described. Testicular [5]. There es direct evidence on clinical and epidemiological data that coincides with implicate endocrine disruptors as causes of various pathologies such as alterations of the reproductive and thyroid axes in men and women, including the pathogenesis of obesity and diabetes [3, 7].
The endocrinology laboratory maintains a crucial role in assisting in the diagnosis of hormonal disorders, providing objective elements that allow quantification and comparison of established values with the data obtained from the patient, which favor clinical assessment [2] and confirm or rule out the existence of a pathology.

Biorhythms

The endocrinology laboratory maintains a crucial role in assisting in the diagnosis of hormonal disorders, providing target elements that allow quantification and comparison of established values with the data obtained desde the patient, which please clinical assessment and confirmation or rule out the existence of a pathology [2].
The circadian system es responsible for the temporary organization of there the physiological processes that occur in the organism. Item es structured in inputs that perceive temporal information. A central pacemaker receives and processes the signals desde the inputs and transmits Item to the rest of the organism, generating a multitude of rhythms dependent on the circadian system. In mammals, the central circadian clock es the suprachiasmatic nucleus of the hypothalamus, t hese nuclei receive information desde the retina and send humoral and neuronal signals to the rest of the bodysuit to control the Cardiac rhtyms [5, 8, 9, 10].

Desynchronization

Desynchronization are alterations in the body's biorhythms. Circadian alteration es detected by a loss of rhythmicity due to advances, delays or phase instability; item may be the result of internal desynchronize ion of the individual or desynchronization of the individual in relation to its environment [9]. Desynchronization can be caused by factors such as constant lighting, constant darkness, and even temporary alterations in darkness during nighttime sleep [11].

Risk of metabolism diseases

The alteration of biorhythms is a risk factor that favors the appearance of physiological and behavioral disorders, which culminate in the development of metabolism diseases (weight gain, obesity, metabolism deregulation), oncological diseases and even neuroinflammation induced by sleep deprivation [11]. The imbalance of the internal circadian systems induces glucose intolerance and insulin resistance; it has been proven that mechanisms can be restored through the administration of melatonin [11]. Likewise, absorbed foods are metabolized, following a rhythmicity that es the product of the release of endocrine regulators; these digestive biorhythms are regulated by food intake [4].
Melatonin receivers located in the pancreas can change the rhythms of insulin secretion by introducing melatonin. Light influences pancreatic insulin secretion through the suppression of melatonin produced during the night Este suggests an influence of shedding light on the mechanisms of glucose homeostasis [12].
Metabolic homeostasis associated with shift work indicates that night shift workers are more likely to develop cardiometabolic syndrome, possibly due to increased postprandial levels of insulin, glucose, and triacylglycerol during the night shift, as well as increased energy intake and circulation triglycerides, as well as reducing insulin sensitivity. Workers also have a higher risk of developing cancer, gastrointestinal disorders and cardiovascular diseases such as ischemic heart disease [13].
Circadian misalignment has adverse effects on metabolic and hormonal factors such as glucose and insulin [4]; including glucocorticoids, melatonin and pituitary hormone [14]. Disruption of circadian rhythms es known as a disturbance of the endocrine system and the progression of the cellular cycle [14]. Item es important to consider that disruption of circadian rhythms can cause significant damage to brain function [11].

Hormonal axes

To maintain the homeostasis of the organism, the human

body regulates the variations of the endocrinological axes28 with processes of regulation, production, release and hormonal action that are governed by positive and negative feedback mechanisms where hormones and efferent neurofibers desde brain regions intervene [29]. Likewise, there es participation of the immune system since Item integrates a physiological network where cytokines, neuropeptides, peptide and steroid hormones intervene that regulate and modify the immune response, complementing the homeostasis of the organism [30]. In the clinical laboratory, the analysis of hormonal values in peripheral blood, as well as in different biological fluids and tissues, provides a better knowledge of the physiology of the different endocrinological axes and in turn the understanding and timely diagnosis of numerous pathologies of the themselves [31].

Hormonal determinations and specifics functional tests for each axis are of great help in defining the origin of the pathology, [31] the interactions between the immune system and the hormonal axes such as hypothalamus - pituitary - adrenal (HPA) and hypothalamus-pituitary-gonads (HPG) are activated in stress situations and affect the adaptation and maintenance of homeostasis during pathological processes caused by viruses, bacteria, parasites or autoimmune diseases [30]. On the other hand, the axes that involve the influence of the environment, the central nervous system (CNS) and the pituitary are the adrenal, thyroid, gonadal, somatotrophic axis and also the secretion of Prolactin (PRL) [30].

The indicative diagnosis of pathologies of the endocrine axes es fundamentally biochemical, hormonal determinations must be carried out with highly sensitive and specific techniques. The clinical definition es finished with imaging techniques, such as nuclear magnetic resonance, computed tomography with or without contrast or ultrasound [31]. This corroborates the presumptive medical diagnosis of the different diseases and with the different functional tests helps to elucidate yourself the etiology of the diseases. Themselves, a fundamental aspect to adapt appropriate treatment [31].

There are specific tests aimed at the assessment of a single hormonal axis in order to obtain better results oriented according to to the physiology of the axis, for example, the Trier social stress test [32] induces significant changes in the hypothalamic - pituitary -adrenal axis to identify the generation of pathologies associated with stress [30]. Certain health habits such as smoking, coffee consumption and alcohol, have been discussed as powerful modulators of the response of the HPA axis to acute stress [33], smoking habitually changes the responses of the HPA axis towards the stress [33-34].

Parathyroid hormone. PTH

Parathyroid hormone (PTH) is synthesized and secreted by the main cells of the parathyroid glands, it es stored in vesicles and secretory granules inside the main parathyroid cells. The main regulator of PTH secretion es the concentration of ionic calcium in the blood [35].

The actions at the renal level carried out by the PTH hormone are to stimulate calcium reabsorption, inhibit phosphate reabsorption and stimulation the synthesis of calcitriol. At the bones level, PTH acts on the osteoblast, stimulating the conversion of the osteoclast precursor into osteoclasts. At the intestinal level, it acts by marketing the absorption of calcium and phosphorus desde the diet, acting on its target organs through the PTH/ PTHrP receptor [35-55].

The regulation of PTH levels es controlled by a feedback mechanism, where the levels of ionic calcium, calcitriol or its derivatives, or, where appropriate, low levels of phosphorus inhibit the secretion of PTH, this es mediated by receivers, such as the calcium-sensing receptor, the vitamin D receptor, and the fibroblast growth factor receptor [56]. Its regulation is a complex process; if low calcium concentration persist for periods of more than seconds or minutes, transcription of the PTH gene is regulated and parathyroid cells proliferation increases. In contrast, calcitriol is a known inhibitor of parathyroid cell proliferation [56].

Calcitonin

The peptide calcitonin was discovered in 1962 as a new hypocalcemic hormone [16], it es made up of a structure of 32 amino acids, produced mainly by the thyroid17 Calcitonin es known for its ability to inhibit bones resorption, as well as the activity of osteoclasts, also Calcitonin has been shown to affect renal calcium regulation by marketing its excretion [16], by directly inhibiting osteoclast-mediated bones resorption and by increasing calcium excretion by the kidneys, decreasing blood levels, therefore calcitonin maintains calcium homeostasis acting as a key modulator in bone resorption [16-18].

The physiological actions of calcitonin are mediated by the ability of its receptor to couple at least two signals transduction pathways [18]. Intracellular calcitonin binds to the CTR receiver, located on the plasma membrane of effector cells; Este binding triggers the activation of multiple G protein-mediated signaling pathways, leading to activation of the cyclic adenosine monophosphate (cAMP)/ protein kinase A pathway or protein kinase C pathways [16].

The functions of calcitonin in the kidney es to stimulate diuresis and increase the rate of fractional excretion of sodium and chloride; in addition, in the urine Item increases the excretion of calcium and phosphate [18]. At the level of the digestive system, it increases the secretion of gastric acid and pepsin and decreases pancreatic amylase and pancreatic polypeptide, in high concentrations, increases the net secretion of water and electrolytes desde the human jejunum and ileum. Calcitonin es found within lung neuroendocrine cells, located near the basement membrane, and these often extend into the lumen of the airways. Calcitonin affects the transcellular and intracellular movements of calcium, therefore, it can be exerted intrapulmonary paracrine signaling [18]. CT produced in other tissues has paracrine signaling that modulates functions such as proton transport, acid -base balance, secretion of prolactin and gastrointestinal motility [19]. Controlled trials demonstrate that calcitonin stabilizes and, in some cases, produces a short- term increase in bone density at the level of the lumbar spine [20].

there es literature that states that calcitonin in mammals es not involved in calcium homeostasis or in any other important physiological function, except, as a complement to the protection of the skeleton in conditions of calcium stress, there es even the idea that calcitonin seems to be in the process of becoming vestigial [19].

Analytical methods

The measurement of serum calcitonin functions as a

sensitive and accurate marker of medullary thyroid carcinoma [21]. Patients whose diagnosis includes calcitonin concentrations in basal and/ or stimulated plasma greater than 100 pg / mL should undergo surgery because they run a substantial risk of suffer desde medullary thyroid carcinoma or C- cell hyperplasia, a potentially precancerous condition [22]. Measurement of serum calcitonin in nodular thyroid diseases es expected to reveal sporadic medullary thyroid carcinoma at an early stage [21], this will increase the possibility of having a timely diagnosis of medullary thyroid carcinoma and provide the possibility of curative surgery [22].
Calcitonin has also demonstrated its clinical value as a tumor marker. Basal and stimulated levels of serum calcitonin have been shown to be particularly valuable in families with multiple endocrine neoplasia type 2 (MEN2) and familial medullary carcinoma (MTCF) a high risk for family medullary thyroid carcinoma [24].
The first methods to determine calcitonin were bioassays carried out in young rats, this method had a relatively low sensitivity and failed to detect the levels of calcification present in human peripheral plasma [24]. Radioimmunoassays proven to be a more reliable and sensitive method for the assay of calcitonin. The first radioimmunoassays detected calcitonin in human plasma at pathological levels. Estos assays offered the opportunity to study pathophysiology and normocalcitonemia. Today, radioimmunoassays are capable of detecting concentrations from 10 pg to 25 pg of calcitonin /ml in plasma [24].
Other techniques for the detection of calcitonin that have been developed include specific calcitonin receptor assays, plasma membrane preparations of bones or kidney cells in a competitive displacement assay with radiolabeled calcitonin or as a modification of the calcitonin receptor can be employed with a measurement of adenyl cyclase activity generated *in vitro*. Unfortunately, the radio receiver assay and the adenyl cyclase assay for calcitonin have not been shown to be as effective and reliable as the radioimmunoassay technique [24].
The basal calcitonin test, as well as the combined basal and stimulated tests, have high sensitivity and specificity that guides the primary diagnosis and, in the same way, the post-surgical follow-up [26], of medullary thyroid cancer in patients with thyroid nodules [25]. Autoimmune thyroid diseases, chronic renal failure, and elevated baseline serum calcitonin give indeterminate results with respect to the diagnosis of medullary thyroid cancer [27].
Serum calcitonin levels are highest in newborns and remain elevated during the first years of life. Estos levels of calcitonin are associated with lengthening of long bones, which grow through endochondral bones formation calcitonin stimulates the formation and maturation of cartilage [23].
Recently, the determination of calcitonin in lavage fluids desde thyroid nodules suspected of medullary thyroid carcinoma has been described. This trial demonstrated that serum calcitonin has greater precision with respect to cytology. Adopting the use of calcitonin in thyroid nodule lavage fluids complemented by cytology could reduce false negative and inconclusive results, with a sensitivity close to 100% [27].

Calcium

Calcium is a cation with the ability to act as a second messenger in different cells groups of the immune system (T and B lymphocytes, macrophages, mast cells, and others hand) [34]. For its distribution in the intra- and extracellular spaces, specialized pumps and channels are required, and the influence of a state of cellular repolarization [34]. Of the total amount of bodysuit calcium, 99% is found in bone tissue, it es crystallized in the form of hydroxyapatite crystals and only 1% is available to exchange with extracellular calcium. Around 50% of total plasma calcium circulates bound to proteins, mainly albumin, free calcium es the biologically active fraction and the one that es under hormonal control [35].
Tubular calcium reabsorption is a major factor determining serum calcium concentration as well as its urinary excretion [36]. Calcium in the bodysuit participates in many essential physiological processes, such as coagulation, muscle contraction, glycogenolysis and also controls cells adhesions [36]. Item es estimated that the serum calcium level remains within a range of 8.5 mg/ dL and 10.5 mg/ dL; Item es important to emphasize that estos values are modified by factors such as gender, age and pathological conditions, Calcium levels influence the coordinated actions of the skeleton, intestine and.

Hypocalcemic

They are low concentrations of calcium in the body, which can be caused by factors such as hypersensitivity of calcium receivers that trigger autosomal dominant hypocalcemia that es the result of activation mutations in the receptors [42].

Hypercalcemic

There are multiple manifestations associated with the increase in calcium concentration in the body, some of they are; Family hypocalciuric hypercalcemia is a pathology caused by mutations in the loss of functions of the calcium receptor that triggers resistance to the calcium ion [42].

Analytical methods

The analytical determination of total calcium can be done using flame atomic absorption spectrometry (FAAS), this technique determines the total calcium, lithium and magnesium in blood serum samples [38]. Quantification of calcium in serum is normally performed with ultraviolet-visible spectrophotometry, where ocresolphthalein is used complexone at alkaline pH (2-4) as a color developing reagent [39].
The concentration of ionic calcium regulates the parathyroid [40], so that when it decreases in serum, an inactivation of the calcium receptor occurs, which results in promoting the synthesis of PTH and an increase in the production of PTH from the granules and vesicles, so on the contrary, when the concentration of ionic calcium in serum increases, the activation of the calcium receptor occurs, leading to a decrease in the production of PTH [35]. The effects of calcium on PTH are mediated by the action of calcium receptor 15, a G protein-coupled receptor located in the membrane of gland cells [40]. In general, significant mutations in the calcium receptor have been shown to induce diseases that modify calcium homeostasis due to changes in the set point that regulate PTH release [41].

Calcium-diabetes relationship

Glucose is responsible for controlling Ca^{2+} concentrations in many cellular compartments, as well as in their organelles [36]. Ca^{2+} at the intracellular level is a crucial factor for the

survival, proliferation and function of β cells; it participates together with β cells to achieve adequate insulin secretion. However, when Ca^{2+} signaling is altered it is associated with the development of insulin resistance, a key factor in diabetes mellitus 2. Ca^{2+} activity related to type 2 diabetes mellitus has a fundamental role in established physiological signaling for the calcium ion, where vitamin D deficiency and irregularities in glucose-induced insulin release generate a Ca^{2+} -dependent process [36].

Match

Phosphorus is part of the mineral component of bones and teeth, as well as hydroxyapatite crystals, in the muscle it is involved in contraction and at the intracellular level it is part of the DNA structure, its metabolism is associated with the use of energy created through mitochondrial level [45]. 85 % of the phosphate is found in the mineral phase of bone and the rest in inorganic or organic form in the extracellular and intracellular spaces; only 12% of serum phosphate is bound to proteins [44].

Extracellular phosphorus is known as inorganic because it constitutes ionic compounds circulating in blood plasma and extracellular fluids that are released and metabolized by different tissues and organs [45]. It has an important relationship with calcium, to the point that quantitative changes in one influence the other, the relationship between phosphorus and calcium is so close that no study can separate the influence of one on the other [45].

Hyperphosphatemia

Hyperphosphatemia is considered as an increase in plasma phosphorus, this increase is associated with the lack of formation of parathyroid hormone that stimulates the excretion of phosphates in the kidney, uncontrolled parenteral administration or acute absorption of high doses of potassium phosphorus or sodium phosphorus. The high presence in plasma ends up triggering a chemical reaction with calcium, forming precipitations of calcium phosphates in soft tissues that could obstruct structures such as skin, cornea, kidneys and periarticular areas [45-49].

The increase in phosphorus concentration in the body has been related to the progression of kidney disease and left ventricular hypertrophy. This indicates that a slight increase in phosphorus is correlated with a poor cardiovascular prognosis [50]. However, high serum phosphorus levels that are within the normal range are associated with a higher risk of carotid atherosclerosis and coronary calcification, and even with higher cardiovascular mortality in people without kidney failure [50]. Metastatic calcification is characterized by abnormal serum calcium and phosphorus levels that lead to the precipitation of calcium salts in a normal, structurally undamaged tissue; the degree of hyperphosphatemia determines the number and size of these calcium deposits [51]. Hyperphosphatemia is a highly prevalent condition in the population undergoing dialysis treatment [52].

Hypophosphatemia

Hypophosphatemia is the decrease in the concentration of serum phosphates. This phenomenon is classified into two categories according to the amount of phosphate deficient, moderate and severe. Values between 1.0 - 2.5 mg% are moderate deficits, while values <1 mg% or 0.3 mmol/L are part of the so-called severe hypophosphatemia. There are 3 main mechanisms that could be involved in the development of this pathology: The decrease in intestinal absorption of phosphate, the redistribution of phosphate from the extracellular fluid into the cells and the increase in renal excretion [44, 36].

Analytical methods

The alteration of phosphorus metabolism is a consequence of chronic kidney disease [46], its blood concentrations of phosphorus vary according to age, in the first years of life there is a higher concentration, and subsequently it decreases due to the stimulation and maturation in parathyroid hormone (PTH) response [44]. The phosphaturia test and fraction excreted of phosphorus in urine allows us to quantify the amount of phosphorus in the body [44].

Vitamin D

Vitamin D has a fundamental role in the regulation of mineral and bone metabolism and acts on the cardiovascular and immune systems through the so-called pleiotropic effects [57]. Serum vitamin D levels are highest in the newborn and decrease exponentially throughout life [58].

The main source of vitamin D is through skin synthesis after sun exposure, the incidence of UV-B solar rays on the skin transforms 7-dehydrocholesterol into provitamin D, which due to its thermolability is transformed into vitamin D. This passes into the blood circulation, binding to the vitamin D transport protein and once it reaches the liver, by the action of 25-hydroxylase, it is transformed into 25-hydroxyvitamin D (25OHD) or calcidiol, which has an approximate half-life of 2 to 3 weeks. 25(OH)D is transformed by the action of 1α-hydroxylase in the renal proximal convoluted tubule into 1,25(OH)2 D3 or Calcitriol, which has a half-life of 6 to 8 hours [36].

Vitamin D deficiency has been associated with an increased risk of secondary hyperparathyroidism (SHPT), increased cardiovascular risk, high blood pressure, diabetes, neoplastic and autoimmune diseases. Patients with chronic kidney disease have a high prevalence of vitamin D insufficiency/deficiency; deficiency has been associated with a variety of bone, metabolic, and cardiovascular disorders [57]. When cases of vitamin D toxicity occur, hypercalcemia becomes persistent and is expressed with suppressed PTH [60].

Analytical methods

Within analytical determinations in the laboratory, the quantification of 25(OH)D fulfills the function of being a biomarker that evaluates the reserve of vitamin D in the body [57]. There are currently two types of measurement that are routinely used to determine vitamin D metabolites, 25OHD3 and 25OHD2. These are competitive immunoassays and methods based on chromatographic separation followed by direct non-immunological detection [53].

Osteocalcin

Osteocalcin is a protein derived from osteoblasts, dependent on vitamin K, it exists mainly in two forms; undercarboxylated osteocalcin and carboxylated osteocalcin and each has different functions. Total osteocalcinin is the combination of circulating undercarboxylated osteocalcin and carboxylated osteocalcin [48]. It is the most abundant non-collagenous protein in bone [47], it is carboxylated intracellularly in three glutamate residues by the enzyme

gamma carboxylase. This post-translational modification gives osteocalcin a high affinity for hydroxyapatite [59], it functions as a hormone and is responsible for it regulates glucose metabolism, testosterone synthesis, muscle mass, development, brain functions and parasympathetic tone, and establishes a link between bones and many organs [47].

In the clinical laboratory, the concentration of serum osteocalcininca is a marker of bone formation [47], the level of osteocalcinin decreases with age causing the consequent decrease in cognitive functions during aging, as well as the reduction of bone health [54]. The three main forms of osteocalcinin, (carboxylated, subcarboxylated, and total osteocalcin) can be measured separately in blood by methods such as radioimmunoassay, immunoradiometric assay, enzyme-linked immunoassay, or electrochemiluminescence immunoassay [43].

It is evaluated that the investigation is not reserved for a report of results, but rather that it proposes solutions or warns of short- and/or long-term risks posed by the problem. The aim is for the results to be extended in the future through useful proposals to solve specific problems.

Conclusions

The fundamental role played by hormones is widely described, their regulation process that works under a feedback mechanism, maintains a good communication relationship between cells, tissues and organs. Long-term exposure to disruptors produces circadian alterations, caused by desynchronization in the individual's hormonal production. These factors favor the development of metabolic diseases such as overweight, obesity, glucose intolerance and insulin resistance. The participation of the clinical laboratory with tests that guide the doctor to make a safe diagnosis is important. The measurement of calcium, phosphorus, parathyroid hormone (PTH), vitamin D (25OHD) and activated vitamin D (1.25OHD) are of great importance. To correlate patterns of clinical alterations that point to pathology inside or outside the parathyroid gland, the calcitonin radioimmunoassay serves as a sensitive and accurate marker of medullary thyroid carcinoma, as well as the quantification of osteocalcin, which is a useful marker to measure bone formation.

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