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Department of Biology

Some Hormonal Changes in Aggressive Behavior in Iraqi Prisoners

A Research

Submitted to Council of the College of Science / University of Babylon in Partial Fulfillment to the Requirements for the Degree of High Diploma in Science /Forensic Evidence

By

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بسم الله الرحمن الرحيم

﴿يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ﴾

صدق الله العلي العظيم

(سورة المجادلة: الآية١١)

Certification

I certify that research is prepared under my supervision at the Department of Biology, College of Sciences, University of Babylon, as partial requirements for the Degree of Higher Diploma in Forensic Evidence.

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Data: / /2021

In view of available recommendation, I forward this research for debate by the Examination Committee.

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Committee Certification

We, the examining committee, certify that we have read the research entitled **(Some Hormonal Changes in Aggressiveness Behavior in Iraqi Prisoners**) and examined the student (Waleed Adil Abbas Mohsen) in its contents at / /2021, and that in our opinion it is accepted as a research for the degree of **High Diploma in Forensic Evidence** with excellence estimation.

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Dean of Collage of Sciences –University of Babylon

Data: / /2021

Dedication

To the cleanest two hearts in my life

To those of them who have gained the power of love without limits ..... My Mother and dear father

To those who had a great impact on many obstacles and difficulties in my life ..... my dear brothers

To the homeland we are looking for, and we yearn to see it one day as we wish it safe and upright

To all those whose spring butterflies dance to those who open the anemones and yasmin ..... to the martyrs

Waleed 2021

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Waleed 2021

Summary

The aim of current study is determination relations aggressive behaviors with some hormone. Target hormones in the current study were (triiodothyronine (T3) , thyroxine (T4) , thyroid stimulating hormone TSH , testosterone and cortisol). The study comprised of the collecting of (40) blood samples, 20 of which were from inmates convicted of violent offenses and 20 samples from non-prisoners. The study was carried out in the Al-Hakem hospital from June 2021 to July 2021. The individuals ranged in age from 18 to 62 years . that measured for both groups. B group (healthy people) and A group (Iraqi Prisoners). The results showed serum level of fT3 was higher in (A) group than the control group with significant difference (P value= 0.009). The fT4 serum level in the control group was higher than in the (A) group with a significant difference (P value <0.05) . The TSH serum level in the control group was higher than the (A) group with no significant difference (P value 0.729). The cortisol serum level in (A) group higher than control group with a significant difference (P value <0.05). The control group had a testosterone serum level lower than the (A) group with no significant difference (P value >0.05) . In conclusion that group A suffers from hormonal changes that make them aggressive people, so the new treatment, rehabilitation, and researchs programs should be established.

.

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LIST OF ABBREVIATION

1.	ADHD	Attention Deficit Hyperactivity Disorder
2.	APD	Antisocial Personality Disorder
3.	BMR	Basal Metabolic Rate
4.	CHF	Congestive Heart Failure
5.	CNS	Central Nervous System
6.	CRT	Cortisol
7.	CVE	Cerebrovascular Events
8.	fT3	Free Triiodothyronine
9.	fT4	Free Thyroxine
10.	HPA	Hypothalamic - Pituitary - Adrenal (axis)
11.	L-T3	L-triiodothyronine
12.	MI	Myocardial Infarction
13.	SVT	SupraVentricular Tachycardia
14.	T	Testosterone
15.	T3	Triiodothyronine
16.	T4	Thyroxine
17.	TRH	Thyrotropin Releasing Hormone
18.	TSH	Thyroid Stimulating Hormone

CHAPTER ONE

INTRODUCTION

1 Introduction

Different causes induce aggressiveness, which may be linked to hormonal imbalances and the resulting neurotransmitters, T3 and T4, which function as neurotransmitters and are associated with aggression. This implies significant shifts in the hypothalamic-pituitary-thyroid axis, which lead to alterations in long-term aggressive behavior, particularly among criminals. In addition, it is possible that people with thyroid hormone imbalance develop mental and behavioral problems (Acar and Ulgen 2020).

It is possible that genetics, hormones, the environment, and other illnesses all contribute to aggressiveness in people. Gene-based analyses were used to examine the genetic structure underpinning violent behavior in children in a study group that consisted of 18,988 people who were part of a larger research collaboration with 12 different studies funded by the European Union. Arginine vasopressin receptors were shown to be associated with greater levels of aggressive behavior in this research. A separate study found that the oxytocin and oxytocin receptor gene variations were implicated with early childhood aggressiveness, according to research by Malik *et al*. Girls with the OXTR SNPs rs6770632 variations and boys with the OXTR SNPs rs1042778 variations were at an increased risk of excessively and chronically hostile behavior (Pappa *et al*. 2016; Malik *et al*. 2012) .

The aim of current study was relationship between aggressive behavior and some hormones

The objective were :

1. Determination aggressive behaviors relations with the concentration of

(triiodothyronine T3 ,thyroxine T4 hormone hormone and TSH hormone).

2. Determination aggressive behaviors relations with the concentration of (testosterone and cortisol).

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CHAPTER TWO

REVIEW OF LITERATURE

2 Review of Literatures

2.1 Hormone—Behavior Relationship

The study of hormone and behavior interactions is known as behavioral endocrinology. This is an asymmetrical interaction: both behavior and hormone concentrations may change as a result. The body's endocrine glands, such as the hypothalamus, release hormones into the bloodstream that then affect the neurological system to control a person's physiology and behavior. hormones that regulate body functions have throughout time been co-opted to affect other behaviors associated with those functions (Nelson 2010).

For a better understanding of the hormone-behavior connection, it is necessary to provide a short overview of hormones. Hormones are organic chemical messengers secreted by endocrine glands, which are distributed to all of the cells in the body . These glands secrete hormones into the circulation, where they may then travel to various locations to operate on target tissues. Both hormones and neurotransmitters work as messengers in the neurological system. But hormones have the ability to send signals over a larger distance and for a longer time period than neurotransmitters. such as a sample testosterone (Trainor *et al* 2006).

2.1.1 The Relationship between Aggression and Thyroid Hormone

The aggression is a prominent feature of antisocial personality disorder (APD). Studies have linked elevated levels of thyroid hormones to an increased propensity for aggressiveness. One of the manifestations of hyperthyroidism is high levels of energy. While a few of research show a connection between serum thyroid hormone levels and aggressiveness and a propensity to commit crime, this relationship has not been studied extensively (Evrensel *et al* 2016).

Aggression is something that everyone experiences in their day-to-day lives, which takes place constantly. In addition, the term aggressiveness is defined as the physical or psychological damage or act of executing a verbal or violent action to the opposing side. Aggression is described as the focused, deliberate, targeted violence which poses a risk to people and the society, resulting in injury, property damage, or even death. The first thing we must recognize about aggressiveness is that it is self-generating (Hazar *et al* 2018).

Like other endocrine glands in the human body, the thyroid has significant effects on the brain and neurological system. These early accounts of thyroid illness, especially mood disorders and cognitive impairment, recognized a connection to neuropsychiatric conditions such as depression and cognitive failure. The thyroid gland makes T4 and T3, also known as L-triiodothyronine (L-T3), under the stimulus of the pituitary gland, which generates thyroid-stimulating hormone (TSH). An increase in free T4, together with a decrease in TSH, confirms the diagnosis of hyperthyroidism. In contrast, low thyroid function is often

associated with high TSH levels. When thyroid hormone concentrations are in the normal range, but TSH is either lower than normal (subclinical hyperthyoidism) or higher than normal (subclinical hypothyroidism), subclinical thyroid dysfunction is present (Ritchie and Yeap, 2015)

Research into the sources of aggressiveness points to many genetic, hormonal, environmental, and illness variables as causal factors. Thirty individuals with attention-deficit hyperactivity disorder had their blood thyroid hormone, thyroid-stimulating hormone (TSH), and thyroid hormone levels tested (ADHD). Two-thirds of the patients had high T3 levels, whereas the other third had low T3 levels (Acar and Ulgen 2020).

Alterations in mood and cognition are associated with changes in cognitive processes. Since some of the first reports of thyroid illness, a connection between thyroid dysfunction and changes in mood and cognition has been noted. Researchers have sought to more clearly characterize the whole spectrum of thyroid diseases, especially in terms of their underlying condition, in order to get a better understanding of the illness and to improve therapy recommendations. Studies conducted lately have shifted their focus to determining the connection between thyroid hormones and cognitive decline, including newer insights into the relationship between thyroid hormones and cognitive decline (Ritchie and Yeap, 2015).

2.1.1.1 Normal Histology of Thyroid Gland

The normal thyroid is comprised of numerous follicles surrounded by a fibrous capsule, which forms septae that divide the parenchyma into multiple lobules . The septae also contain the nerves and blood vessels supplying each lobule . Each lobule contains 20-40 round follicles, 200 μm in average diameter and lined by simple, flat to low columnar epithelium, depending on the state of functional activity; the more active the follicle, the taller the follicular epithelium (Kumar, *et al* 2014; Mescher 2015).

Follicular cells have uniform dark, small nuclei that are centrally located, and some have abundant granular cytoplasm, a variant known as Hürthle cells . Sanderson polsters, which are small follicles extending into the central spaces of larger follicles, can be seen scattered throughout the thyroid, and should not be mistaken for papillary structures . Follicles contain colloid, a viscous material composed predominantly of the thyroid hormone precursor protein thyroglobulin (Rosai 2011).

The normal thyroid gland contains up to three months' worth of thyroglobulin stored within colloid ((Kumar, Abbas, and Aster 2014; Mescher 2015)). The final cell type of the thyroid is the parafollicular, or C cell, a derivative of the neural crest by way of the ultimobranchial body . Parafollicular cells form clusters – as the name suggests – within and in between follicles, and are found in highest concentration within the mid- and upper portions of the lobes . These cells form and secrete calcitonin, thereby participating in calcium homeostasis (Beynon and Pinneri 2016).

2.1.1.2 Hyper- and Hypothyroidism

Hyper- and hypothyroidism are two of the most common disorders of the endocrine system worldwide . Approximately 4-5% of the population of the United States are affected, and the number is even higher in iodine-deficient countries . Most importantly, both symptomatic and silent versions of these two conditions are associated with increased mortality – in particular, due to cardiovascular disease (Wei *et al*, 2013).

2.1.1.3 Normal Physiology

In order to discuss hyper- and hypothyroidism and their sequelae, one must first recall the normal physiology of the thyroid gland. The primary function of thyroid follicular cells is the synthesis of thyroid hormones, of which there are predominantly two: tetraiodothyronine (T4), more commonly known as thyroxine, and triiodothyronine (T3) . These hormones are extremely important for a significant variety of functions throughout the body, including development, growth, and basal metabolic rate (BMR) control. Thyroid hormone production and release is stimulated through the hypothalamic-pituitary axis. Thyrotropin-releasing hormone (TRH) from the hypothalamus causes the anterior pituitary to release thyrotropin, also called thyroid-stimulating hormone (TSH) . In response to TSH, thyroid follicular cells produce thyroglobulin, an inactive protein, which is then released from the apical surface into the follicle as colloid (Kumar, Abbas, and Aster 2014).

The mechanism of iodide transport by sodium-iodide cotransporters on the basal surface of follicular cells take up iodide from the bloodstream, which is then released via transport protein, pendrin, into the follicle and oxidized by thyroid peroxidase into iodine . Next, tyrosine

residues on thyroglobulin are iodinated and then conjugated via oxidative coupling, forming T3 and T4. Iodinated thyroglobulin is taken back into the follicular cell, where lysosomal protease degradation releases the T3 and T4 to exit into capillaries. Thyroid hormones travel in the bloodstream bound predominantly to thyroxine-binding protein . T4 is significantly more abundant, making up 90% of the total thyroid hormone; however, T3 is two to ten times more bioactive . To combat this problem, the target tissues contain 5′-iodinase, which can convert T4 into T3 (Kumar, *et al* 2014; Buttaro *et al*. 2019)

The activity of thyroid hormone is very broad, as it can act by essentially three main mechanisms: 1) directly at the cellular level, 2) via the sympathetic nervous system, and 3) through changing metabolism and affecting the circulation . Thyroid hormone increases BMR, body temperature, gluconeogenesis, lipolysis, proteolysis, and glucose absorption. It increases stroke volume and heart rate, leading to increased cardiac output. In the young, it promotes growth and leads to bone maturation and fusion of growth plates. It is essential for central nervous system (CNS) maturation during fetal development (Klein 2002).

This series of biochemical events leading to thyroid hormone formation is controlled by a negative feedback loop whereby increased levels of thyroid hormone, especially T3, inhibit release of TSH from the anterior pituitary. When released into the circulation, it forms T3 through the process of de-iodination. T4 and T3 can then exert negative feedback on the anterior pituitary with high levels of T3/T4 decreasing TSH secretion and low levels of T3/T4 increasing TSH release. The opposing forces of TRH and T3 allow for maintenance of a relatively steady thyroid

state in the normal individual . However, when derangements occur within this delicate system, serious and potentially fatal conditions may result (Zhang *et al*. 2014).

2.1.1.4 Hyperthyroidism

Increased concentration of thyroid hormone has many causes, the most common of which is Graves disease, which will be discussed in detail below . Less common causes of hyperthyroidism include hyperfunctional (or toxic) adenoma, toxic multinodular goiter, thyroid malignancy, increased TSH from pituitary adenoma (secondary hyperparathyroidism), increased TRH (tertiary hyperparathyroidism), exogenous thyroid hormone ingestion, or thyroid damage from amiodarone toxicity, radiation, or trauma . No matter the inciting factor, for the most part, the signs and symptoms of hyperthyroidism are the same, related to the previously described activity of thyroid hormone. These include weight loss despite adequate or increased caloric intake, heat intolerance, tremor, hyperreflexia, sweating, skin flushing, weakness, irritability, and nervousness (Hampton 2013).

Most important to the discussion of thyroid-related mortality, however, are the substantial cardiac manifestations of hyperthyroidism. Indeed, symptoms related to effects on the heart are a consistent feature, and often the presenting complaint, of a hyperthyroid patient . These include tachycardia, palpitations, and arrhythmias, notably atrial fibrillation, which is not only the most common cardiac arrhythmia in this population, but also a substantial contributor to cardiovascular disease-related morbidity and mortality, particularly cerebrovascular events (CVE) or stroke (Chaker *et al*. 2015)

Thyroid hormones have a central role in cardiovascular homeostasis. In myocardium, these hormones stimulate both diastolic myocardial relaxation and systolic myocardial contraction, have a pro-angiogenic effect and an important role in extracellular matrix maintenance. Thyroid hormones modulate cardiac mitochondrial function. Dysfunction of thyroid axis impairs myocardial bioenergetic status. Both overt and subclinical hypothyroidism are associated with a higher incidence of coronary events and an increased risk of heart failure progression. Endothelial function is also impaired in hypothyroid state, with decreased nitric oxide-mediated vascular relaxation. In heart disease, particularly in ischemic heart disease, abnormalities in thyroid hormone levels are common and are an important factor to be considered. In fact, low thyroid hormone levels should be interpreted as a cardiovascular risk factor. Regarding ischemic heart disease, during the late post-myocardial infarction period, thyroid hormones modulate left ventricular structure, function and geometry (Mori *et al.*, 2019)

Congestive heart failure may be the sole symptom of so-called "apathetic" hyperthyroidism in a subset of elderly patients . Almost 6% of patients with hyperthyroidism present with CHF and atrial fibrillation . Thyrotoxic cardiomyopathy often presents as acute heart failure in young patients without cardiovascular risk factors, and may be fatal if not treated properly. Stabilization of thyroid function status may reverse the process; however, chronic dilated cardiomyopathy can also result (Soh and Croxson 2008).

2.1.2 Testosterone and Aggressive Behavior

Aggressiveness is exhibited in various forms and intensities from; thoughts, body arousal and anger to verbal, dominant, competitive traits and serious acts of violence. The manifestation of this behavioral spectrum is associated with and served by the mobilization of the muscular system. Studies of testosterone's relationship with aggressive and violent behavior have been performed in parallel with those on the mediators of aggressiveness ("Testosterone and Aggressive Behavior" 2004; Negri-Cesi *et al*. 2004)(Batrinos 2012).

Clinical data from the non-prisoner population necessary to confirm the above findings in normal free men is limited. Most studies have been based on self-report questionnaires, which record actual aggression and its intensity with questionable likelihood. In a series of such studies, which gave conflicting results, the majority of these confirmed the relationship of testosterone with aggressiveness reported in prisoners . An investigation of testosterone, cortisol and thyroxin in a sample of 4179 veterans, which has increased credibility because of its size, has shown that basal testosterone levels were positively related to antisocial and aggressive behavior . It is of interest, however, that supraphysiological doses of testosterone in the order of 200 mg weekly , or even 600 mg weekly (O'Connor *et al*. 2002) .

In particular, correlational and experimental work suggests that trait dominance may play a role in moderating relationships between T and human dominance behavior. People with dominant personality styles tend to behave in assertive, forceful, and self-assured ways to achieve or maintain high social status. In one study, a rise in T after winning a

competition predicted increased aggressive behavior in a subsequent task, but only among men scoring high in trait dominance (Carré, Putnam, and McCormick 2009).

Also, baseline T concentrations were positively correlated with men's dominance behavior during a mate competition, but only for men scoring high in trait dominance . Finally, a single administration of T to women increased their competitive motivation after a victory, but only for those scoring high on trait dominance . An individual's ability to exert self-control under affectively charged situations might also mitigate the effect of T on aggression. Some research indicates that individuals scoring high on trait-based measures of self-control are more efficient at inhibiting aggressive impulses during social provocation (Bettencourt *et al*. 2006; Mehta *et al*. 2015).

Other research indicates that tasks designed to bolster self-control decreased participants' subsequent aggression, whereas those designed to disrupt or temporarily reduce selfcontrol increased participants' subsequent aggression . According to one theoretical model of aggression , instigating triggers, such as provocation, and impelling forces, such as T, may promote aggressive impulses, but these impulses may not manifest behaviorally among individuals high in trait self-control because these individuals are better equipped to override such impulses. Thus, T's effects on aggression may be reduced among those high in self-control but pronounced among those low in self-control (Denson 2015).

2.1.3 Cortisol and Aggressive Behavior

It is well established that mood disorders are often associated with alterations in hypothalamic–pituitary–adrenal (HPA) axis function . Although some studies have found no alterations in HPA axis activity in major depression, evidence of hypercortisolism is one of the most consistent biological findings among psychiatric patients . Findings of HPA axis dysregulation in patients with primary anxiety disorders are generally less robust than in those with major depression (Varghese and Brown, 2001).

However, HPA axis hyperactivity has been documented in studies of social phobia , panic disorder , obsessive–compulsive disorder, and mixed anxiety–depressive disorder . In contrast, patients with post-traumatic stress disorder show evidence of decreased basal cortisol levels and increased negative feedback regulation of the HPA axis as compared to normal controls. Although dysregulation of stress hormones was originally thought to be an epiphenomenon of mood disorders, a growing database suggests that excess cortisol secretion may contribute to the mood symptoms, in part, by increasing CRH expression in the amygdala and other key brain regions (Faravelli *et al.*, 2012).

There is evidence that neuroendocrine dysregulation often normalizes with antidepressant treatment and that a lack of normalization may be associated with early relapse In contrast, patients with post-traumatic stress disorder show evidence of decreased basal cortisol levels and increased negative feedback regulation of the HPA axis as compared to normal controls.. Furthermore, nonaffected family members of depressed probands often have abnormal Hypothalamic - Pituitary -

Adrenal (axis) HPA axis function, suggesting that glucocorticoid abnormalities may represent genetic vulnerability factors that predispose to and/or exacerbate the course of mood disorders (Oswald *et al*. 2006).

The steroids cortisol (CRT) and testosterone (T) have become well-established targets in the search of hormonal modulators of social aggression. In the literature, aggression is commonly divided into an impulsive and instrumental subtype (Trainor *et al* 2006).

Impulsive aggression, also named reactive aggression, is unplanned and driven by affect. Instrumental aggression, also named proactive aggression, is premeditated and is characterized by a lack of emotions . Recently, it has been suggested that the balance between T and CRT levels, that is the testosterone/cortisol ratio (T/CRT), might be predictive for both types of aggression (Terburg, Morgan, and van Honk 2009).

Indeed, CRT is strongly linked to social withdrawal, which is shown by many studies that will be discussed in this review. Recent studies have found similar relationships between T, CRT and aggressive behavior in clinical populations (children with conduct disorder and adults with psychopathy) as well as healthy humans (Glenn and Raine 2011).

Researchers found that low levels of cortisol, a hormone normally secreted by the brain in response to stressful or threatening situations, is associated with persistent and early aggression. Boys with low cortisol concentrations exhibited three times the number of aggressive symptoms and were named most aggressive by peers three times more often when

compared with boys who had higher cortisol levels at either sampling time (Ramirez, 2003)

.

CHAPTER

THREE

MATERIALS AND

METHOD

3. Materials and Methods

3.1. Materials

3.1.1. Instrument and Equipment

The following instruments and equipment were used in this study are listed in (table 3-1)

Table 3- 1 Equipment and their companies

NO.	Type of equipment	Company/Origin
2.	Centrifuge	Hettich/German
3.	Disposable Syringes (5ml)	Medico/United Arab Emirates
4.	Disposable Tips	Gilson/France
5.	Gel Tubes (5 ml)	Dolphi medical/Jordan
6.	Incubator	Memmert – Germany
7.	Micropipette (0.5-10)µl, Number channel pipette (20-200) µl	Huawei – Germany
8.	Micropipette (5-50)µl	Slamed – Germany
9.	Micropipette Set (0.5-10µl)	Dragon MED/USA
10.	Micropipette Set (20- 200µl)	Dragon MED/USA

11.	Micropipette(100-1000) µl, Micropipette(10-100) µl	Dragon med. – Germany
13.	Mine vidas	Biomerieux (France)
14.	Timer	Chania

3.1.2. Kits

Kits used in this study were listed in (table 3-2)

Table 3- 2 Kit used in study

No.	Name of Kit	Company	Origin
1.	Kit of Testosterone	Biomerieux	(France)
2.	Kit of TSH	Biomerieux	(France)
3.	Kit of Cortisol	Biomerieux	(France)
4.	Kit of fT3	Biomerieux	(France)
5.	Kit of fT4	Biomerieux	(France)

3.2. Methods

3.2.1. Experimental Design of the Study

The study comprised the collecting of (40) blood samples, 20 of which were from inmates convicted of violent offenses and 20 samples from non-prisoners. The study was carried out in the Al-Hakem hospital from June 2021 to July 2021. The individuals ranged in age from 18 to 62 years . Target hormones in the current study were (T3 , T4 , TSH , testosterone and cortisol) that measured for both groups. Control group (healthy people) and APD group (Iraqi personer). The samples were put in plane tubes and left in a vertical posture for 20-30 minutes before being disposed in a centrifuge device 3000 / 5 minutes.

The serum was extracted by pipetting the top portion after centrifugation (the serum was stored in the freezer, with each sample divided into two sections to guarantee the success of hormonal testing on it), and the same procedures were done on the control samples.

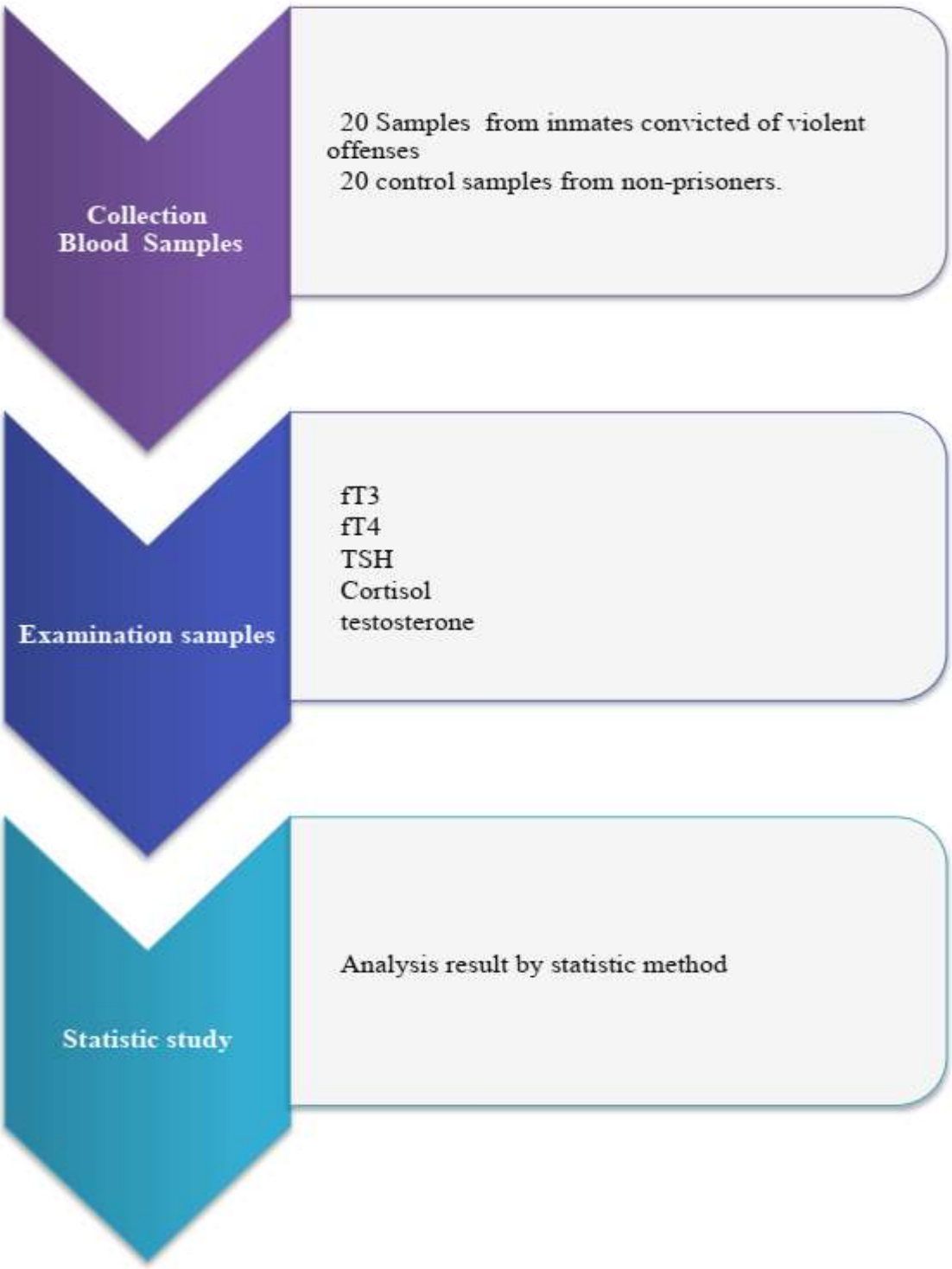

Figure 3- 1 A flow-chart representing the main stages involved of the research plane.

3.2.3. Samples Examination

All samples were tested according the kits and mini Vidas system instruction

3.2.3.1. TSH

1. Assay Procedure

2. The kit's necessary components were removed, and any unneeded components were returned to storage at 2-8°C.

3. Components Allowing for the temperature to rise to room temperature (approximately 30 minutes).

4. Each sample, control, or calibrator was examined with one "TSH" strip and one "TSH" SPR one. Confirmed because the storage bag was properly resealed once the necessary SPRs were extracted.

5. The "TSH" code on the instrument indicated the test. The calibrator is also labeled "S1" and is tested in duplicate. C1 was used to identify the control.

6. The “TSH” Reagent Strip is labeled with the sample identification numbers.

7. A vortex type mixer was used to mix the calibrator, control, and samples (for serum or plasma separated from the pellet).

8. The calibrator, control, and sample test component for this test is 200 ul.

9. SPRs and “TSH” Reagent Strips were inserted into the proper positions on the instrument. Confirmed because the color labels on the SPRs and Reagent Strips match the assay code.

10. The assay processing was started according to the instructions in the Operator's Manual. The device performs all of the assay procedures automatically.

11. After pipetting, the vials were reclosed and brought to the necessary temperature.

12. The test was finished in around 40 minutes. Once the test was completed, the SPRs and strips were removed from the apparatus. The used SPRs and strips were disposed of in a suitable receiver.

3.2.3.3. T3

1. Assay Procedure

2. The kit's necessary components were removed, and any unneeded components were returned to storage at 2-8°C.

3. Components Allowing for the temperature to rise to room temperature (approximately 30 minutes).

4. Each sample, control, or calibrator was examined with one "T3" strip and one "T3" SPR one. Confirmed because the storage bag was properly resealed once the necessary SPRs were extracted.

5. The "T3" code on the instrument indicated the test. The calibrator is also labeled "S1" and is tested in duplicate. C1 was used to identify the control.

6. The “T3” Reagent Strip is labeled with the sample identification numbers.

7. A vortex type mixer was used to mix the calibrator, control, and samples (for serum or plasma separated from the pellet).

8. The calibrator, control, and sample test component for this test is 200 ul.

9. SPRs and “T3” Reagent Strips were inserted into the proper positions on the instrument. Confirmed because the color labels on the SPRs and Reagent Strips match the assay code.

10. The assay processing was started according to the instructions in the Operator's Manual. The device performs all of the assay procedures automatically.

11. After pipetting, the vials were reclosed and brought to the necessary temperature.

12. The test was finished in around 40 minutes. Once the test was completed, the SPRs and strips were removed from the apparatus. The used SPRs and strips were disposed .

3.2.3.4. T4

1. Assay Procedure

2. The kit's necessary components were removed, and any unneeded components were returned to storage at 2-8°C.

3. Components Allowing for the temperature to rise to room temperature (approximately 30 minutes).

4. Each sample, control, or calibrator was examined with one "T4" strip and one "T4" SPR one. Confirmed because the storage bag was properly resealed once the necessary SPRs were extracted.

5. The "T4" code on the instrument indicated the test. The calibrator is also labeled "S1" and is tested in duplicate. C1 was used to identify

the control.

6. The “T4” Reagent Strip is labeled with the sample identification numbers.

7. A vortex type mixer was used to mix the calibrator, control, and samples (for serum or plasma separated from the pellet).

8. The calibrator, control, and sample test component for this test is 200 ul.

9. SPRs and “T4” Reagent Strips were inserted into the proper positions on the instrument. Confirmed because the color labels on the SPRs and Reagent Strips match the assay code.

10. The assay processing was started according to the instructions in the Operator's Manual. The device performs all of the assay procedures automatically.

11. After pipetting, the vials were reclosed and brought to the necessary temperature.

12. The test was finished in around 40 minutes. Once the test was completed, the SPRs and strips were removed from the apparatus. The used SPRs and strips were disposed .

3.2.3.2. Testosterone

1. Assay Procedure

2. The kit's necessary components were removed, and any unneeded components were returned to storage at 2-8°C.

3. Components Allowing for the temperature to rise to room

temperature (approximately 30 minutes).

4. Each sample, control, or calibrator was examined with one "TES2" strip and one "TES2" SPR one. Confirmed because the storage bag was properly resealed once the necessary SPRs were extracted.

5. The "TES2" code on the instrument indicated the test. The calibrator is also labeled "S1" and is tested in duplicate. C1 was used to identify the control.

6. The “TES2” Reagent Strip is labeled with the sample identification numbers.

7. A vortex type mixer was used to mix the calibrator, control, and samples (for serum or plasma separated from the pellet).

8. The calibrator, control, and sample test component for this test is 100 ul.

9. SPRs and “TES2” Reagent Strips were inserted into the proper positions on the instrument. Confirmed because the color labels on the SPRs and Reagent Strips match the assay code.

10. The assay processing was started according to the instructions in the Operator's Manual. The device performs all of the assay procedures automatically.

11. After pipetting, the vials were reclosed and brought to the necessary temperature.

12. The test was finished in around 40 minutes. Once the test was completed, the SPRs and strips were removed from the apparatus. The used SPRs and strips were disposed .

3.2.3.4. Cortisol

1. Assay Procedure

2. The kit's necessary components were removed, and any unneeded components were returned to storage at 2-8°C.

3. Components Allowing for the temperature to rise to room temperature (approximately 30 minutes).

4. Each sample, control, or calibrator was examined with one "CORS" strip and one "CORS" SPR one. Confirmed because the storage bag was properly resealed once the necessary SPRs were extracted.

5. The "CORS" code on the instrument indicated the test. The calibrator is also labeled "S1" and is tested in duplicate. C1 was used to identify the control.

6. The “CORS” Reagent Strip is labeled with the sample identification numbers.

7. A vortex type mixer was used to mix the calibrator, control, and samples (for serum or plasma separated from the pellet).

8. The calibrator, control, and sample test component for this test is 200 ul.

9. SPRs and “CORS” Reagent Strips were inserted into the proper positions on the instrument. Confirmed because the color labels on the SPRs and Reagent Strips match the assay code.

10. The assay processing was started according to the instructions in the Operator's Manual. The device performs all of the assay procedures automatically.

11. After pipetting, the vials were reclosed and brought to the necessary temperature.

12. The test was finished in around 40 minutes. Once the test was completed, the SPRs and strips were removed from the apparatus. The used SPRs and strips were disposed .

3.2.4. Statistical Analysis

All results of experiments were recorded as M±STD for three replicates. The results significant between samples means was performed using multiple comparison between groups through SPSS software.

CHAPTER FOUR

RESULTS AND

DISCUSSION

4. Results and Discussion4

4.1. T3

The concentration of fT3 was measured in both group control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20) , all results were recorded as mean and standard division serum. Serum fT3 level of the control group was 3.15±0.84 nanograms per deciliter (ng/dL) while fT3 serum level in Iraqi Prisoners (A) was 3.58±0.70 (ng/dL), where P value= 0.009 figure 4-1.

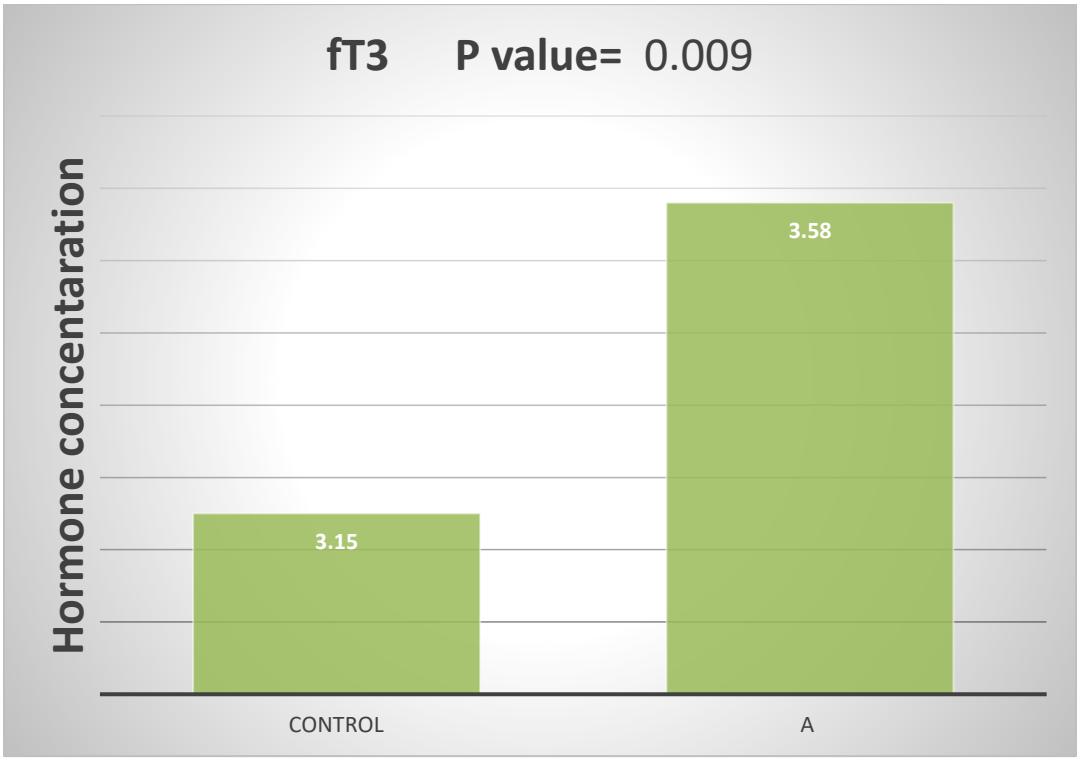

Figure 4- 1 Serum level of fT3 in control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20) .

Previous study found the mean score of free T3 level in the criminal A group was found to be significantly higher than that in the noncriminal A group. A subjects with higher free T3 levels also had higher aggression scores. In the noncriminal A group, as serum free T3

and T4 levels increased, there was also an increment in the aggression scores. However, in the criminal A group, there was no significant correlation between thyroid hormone levels and aggression (Evrensel,*et al* , 2016).

4.2. T4

All findings were reported as mean and standard division in both the control group. The serum level of fT4 in the control group was 2.24±6.1 (ng/dL), but the fT4 serum level in the Iraqi Prisoners (A) group was 0.814±0.10 (ng/dL), with a P value of 0.001 indicating a significant difference figure 4-2 .

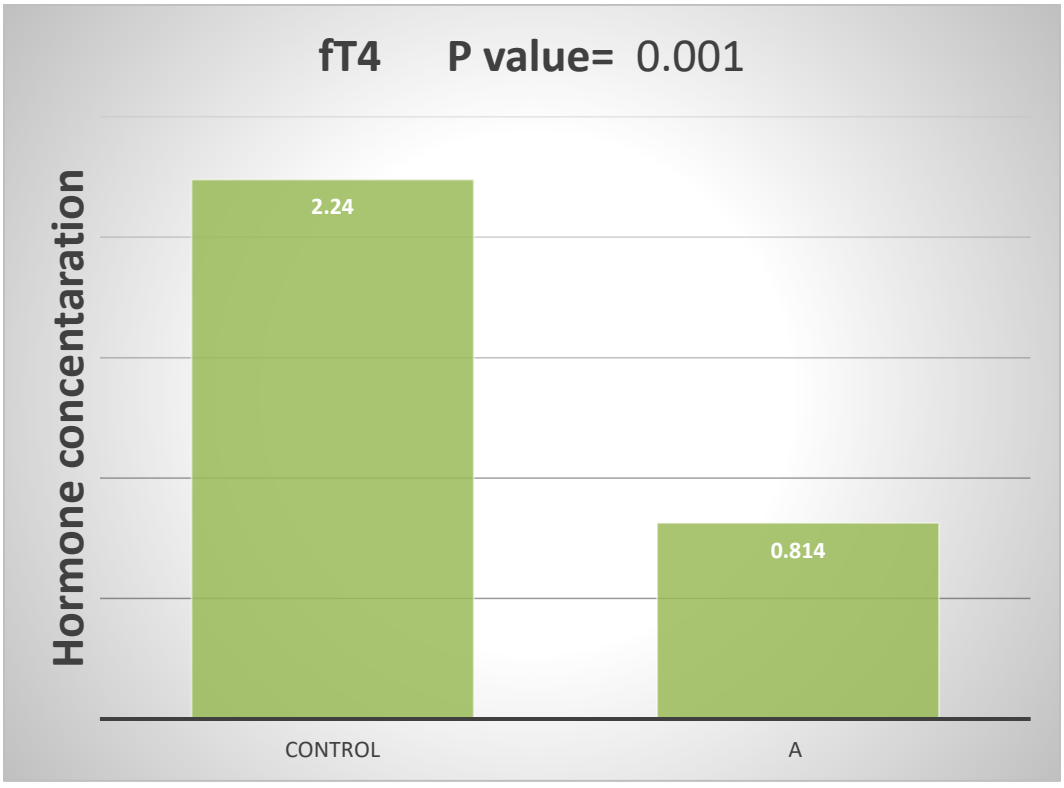

Figure 4- 2 Serum level of fT4in control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20) .

The fT4 serum level in the control group was higher than in the (A) group with a significant difference. That is agreed with previous study (Stålenheim *et al*. 1998; Stalenheim and Gunilla Stalenheim 2004) Stalenheim and Gunilla Stalenheim in their study six- to eight-year follow-up study of a forensic psychiatric subpopulation. The aim of their study was to examine the long-term validity of biological markers of psychopathy and antisocial behavior over time. As part of a follow-up, the National Council for Crime Prevention provided their with a list of criminal records. In addition to mental and psychological assessments, the forensic psychiatric examination also included results for serum triiodothyronine (T3) and free thyroxin (FT4), as well as platelet monoamine oxidase (MAO) activity. Follow-up serum T3 levels in criminal recidivists were significantly greater than those of non-recidivists and normal controls, whereas their levels of free T4 were lower. The T3 levels in criminal recidivists correlated to psychopathy- and aggression-related personality traits as measured by the Karolinska Scale of Personality. In violent recidivists, a remarkably high correlation was noted between T3 levels and irritability and detachment, traits that have previously been linked to the dopaminergic system. Psychological characteristics of criminal recidivists were associated with T3 levels, platelet MAO activity and multiple regression analyses. Over time, T3 and platelet MAO, two biological indicators of psychopathy used in forensic psychology investigations, have maintained their predictive value. Researchers found that this group of individuals had persistent changes in the hypothalamic-pituitary-thyroid axis (Stålenheim *et al*. 1998).

4.3. TSH

TSH was evaluated in both the control and A group , with all values provided as mean and standard division. The serum level of TSH in the control group was 2.33±3.2 (ng/dL), whereas the TSH serum level in the Iraqi Prisoners (A) group was 1.57±0.9 (ng/dL), with a P value of 0.729, suggesting no significant difference figure 4-3.

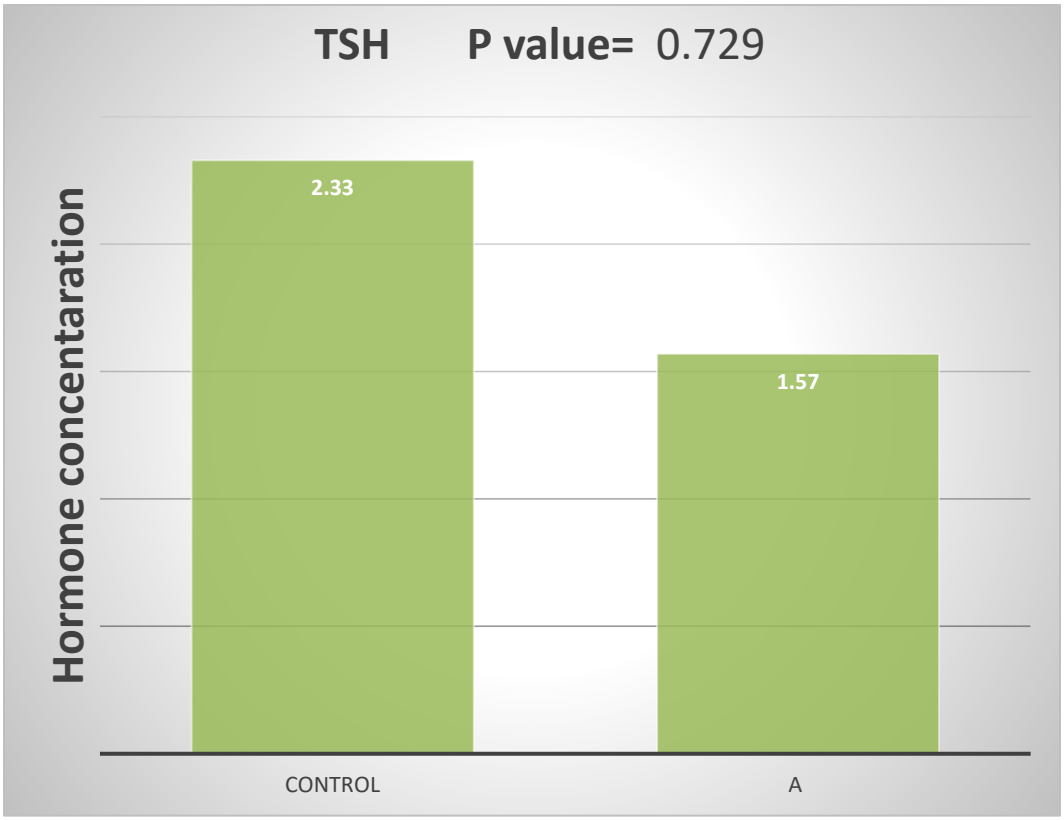

Figure 4- 3 Serum level of TSH in control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20).

The TSH serum level in the control group was higher than the (A) group was with no significant difference. That is agreed with (Stalenheim *et al* 2004) in study they found the population had a highly increased T3 level and a decreased level of free T4 compared with normal controls. The

difference in T3 level was even more pronounced when the subjects were divided into those with and those without criminal recidivism. No relationship was found between TSH level and criminal behavior.

4.4. Cortisol

Cortisol levels were measured in both the control group and the A group, and all results were given as mean and standard deviation, respectively. The cortisol serum level in the control group was 10.184.12 (ng/dL), whereas the cortisol serum level in the Iraqi Prisoners (A) group was 13.751.2 (ng/dL), with a P value of 0.006, indicating a statistically significant difference. Figure 4-4 shows the cortisol serum levels in the control and Iraqi Prisoners (A) groups.

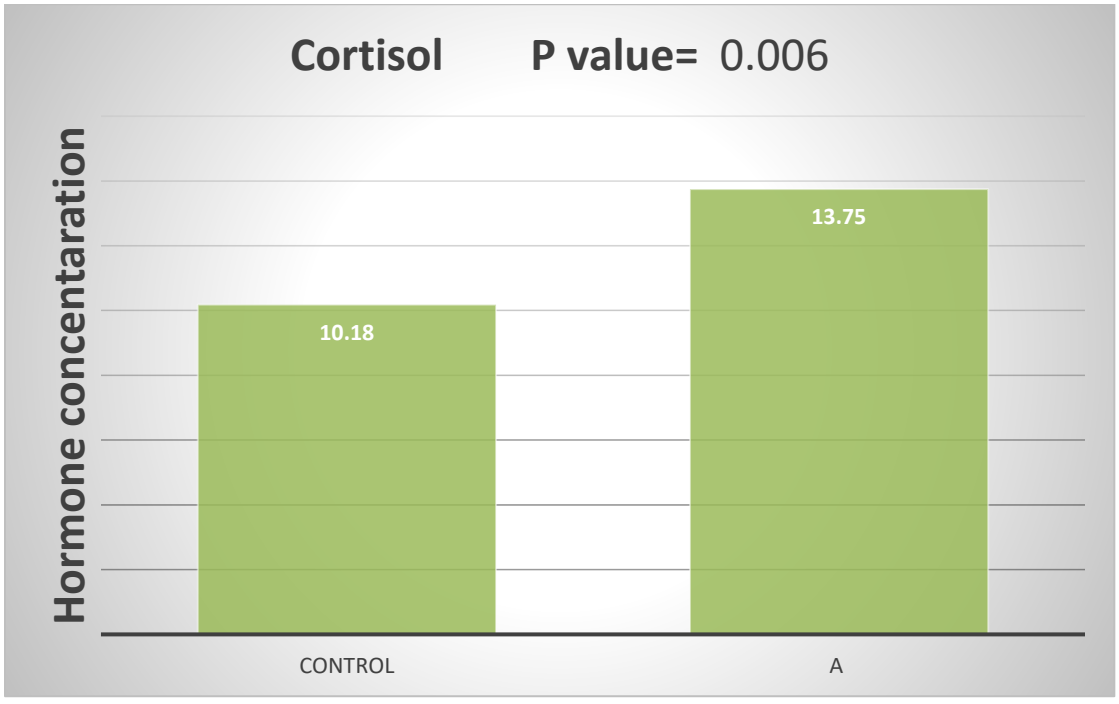

Figure 4- 4 Cortisol serum level in control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20).

The cortisol serum level in (A) group higher than control group with a significant difference. This is similar to what (Montoya *et al.*

2012) reached when as they referred in their studies to interaction does however not distinguish between the impulsive and instrumental subtypes of aggression , it has been proposed that low levels of the neurotransmitter serotonin (5-Hydroxytryptamine; 5-HT), in combination with high testosterone /cortisol ratio facilitate the impulsive subtype of aggression in particular.

4.5. Testosterone

Testing for testosterone levels was performed on participants in both the control and the A groups, and the results were provided as a mean and a standard deviation. Figure 4-5 shows that the testosterone serum level in the control group was 22.15.8.36 (ng/dL), whereas the testosterone serum level in the Iraqi Prisoners (A) group was 27.00.8.9 (ng/dL), with a P value of 0.948, which indicates that there was no statistically significant difference.

Table 4- 1 The hormones serum level(fT3, fT4, TSH, Cortisol and testosterone) in tow group, control group (healthy people N= 20) and A group (N = 20)

No.	Hormones	B (Mean±SD)	A (Mean±SD)	P
1.	fT3	3.15±0.84	3.58±0.70	0.009
2.	fT4	2.24±6.1	0.814±0.10	0.001
3.	TSH	2.33±3.2	1.57±0.9	0.729
5.	Cortisol	10.18±4.12	13.75±1.2	0.006*
6.	testosterone	22.15±8.36	27.00±8.9	0.948

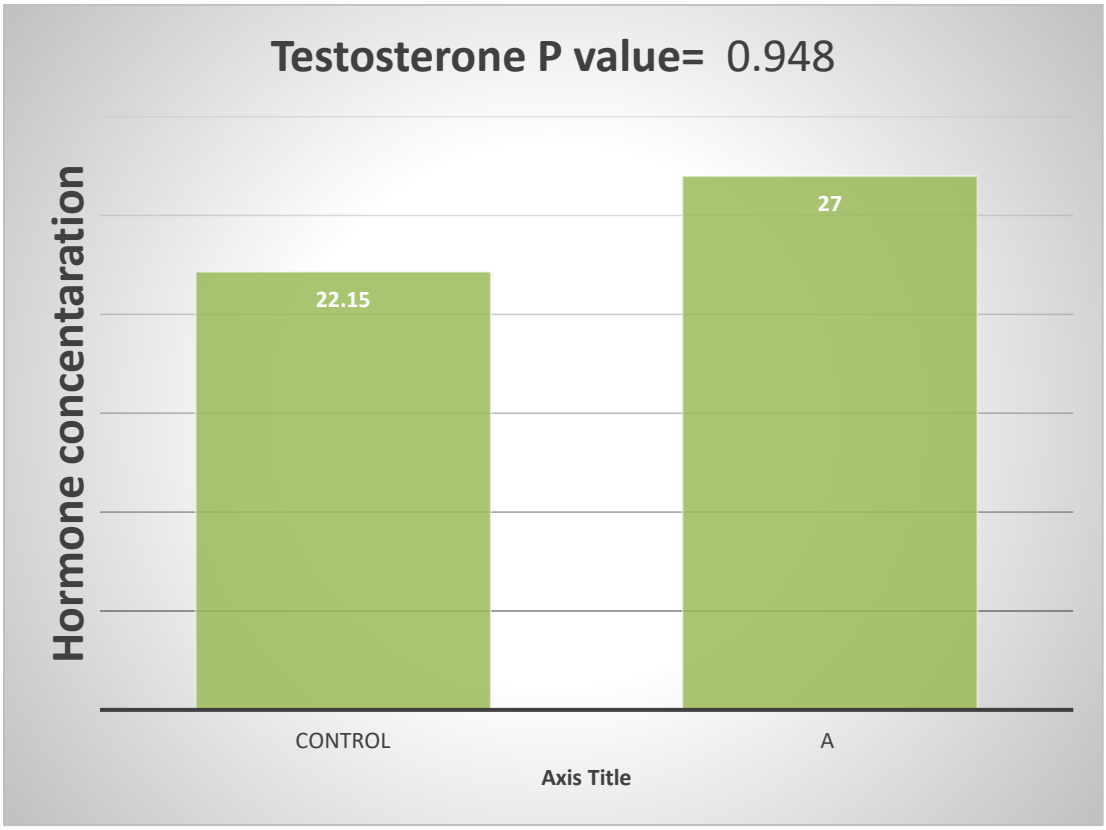

Figure 4- 5 Testosterone serum level in control group (healthy people N= 20) and A group (Iraqi Prisoners N = 20) .

The control group had a testosterone serum level lower than the (A) group with no significant difference. that is agreed with (Batrinos 2012) in his study referred that testosterone plays a significant role in the arousal of these behavioral manifestations in the brain centers involved in aggression and on the development of the muscular system that enables their realization. There is evidence that testosterone levels are higher in individuals with aggressive behavior, such as prisoners who have committed violent crimes.

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CONCLUSIONS

AND

RECOMMENDATIONS

Conclusions and Recommendations

Conclusions

1.There is a significant increase in thyroid hormones in the convicts.

2. Serum level of fT3 was higher in (A) group than the control group with significant difference.

3. The fT4 serum level in the control group was higher than in the (A) group with a significant difference.

4. The serum level of TSH in the control group was higher than the (A) group was with no significant difference.

5. The serum level of cortisol in (A) group higher than control group with a significant difference. The control group had a testosterone serum level lower than the (A) group with no significant difference.

Recommendations

Many members of society suffer from hormonal changes that negatively affect their behavior. Therefore, clinics or centers for this group must be established to reduce the effects of these changes.

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الخلاصة

اشتملت الدراسة على جمع (40) عينة دم ، 20 منها كانت من سجناء مدانين بجرائم عنف و 20 عينة من غير سجناء. أجريت الدراسة في مستشفى الحاكم في الفترة من يونيو 2021 إلى يوليو 2021. وتراوحت أعمار الأفراد بين 18 و 62 عامًا. كانت الهرمونات المستهدفة في الدراسة الحالية هي (T3 و T4 و TSH والتستوستيرون والكورتيزول) التي تم قياسها لكلا المجموعتين. المجموعة الضابطة (الأشخاص الأصحاء) ومجموعة A (سجناء عراقيين). كان الهدف من الدراسة الحالية تحديد العلاقة بين السلوك العدائية و مستوى الهرمونات في الدم . أظهرت النتائج أن مستوى مصل fT3 كان أعلى في مجموعة (A) من مجموعة التحكم مع اختلاف معنوي قيمة P = 0.009. كان مستوى مصل fT4 في المجموعة الضابطة أعلى منه في مجموعة (A) مع اختلاف معنوي بقيمة (P value <0.05). كان مستوى مصل TSH في المجموعة الضابطة أعلى من مجموعة (A) مع عدم وجود فرق معنوي بقيمة P 0.729. كان مستوى مصل الكورتيزول في مجموعة (A) أعلى من المجموعة الضابطة مع اختلاف معنوي بقيمة (P value <0.05). كان مستوى مصل التستوستيرون في المجموعة الضابطة أقل من مجموعة (A) مع عدم وجود فرق معنوي بقيمة (P value >0.05). أوصت الدراسة بأن مجموعة A يعانون من أمراض هرمونية تجعلهم عدوانيين، لذلك بالنسبة لهؤلاء الأفراد، يجب وضع برامج جديدة للعلاج وإعادة التأهيل والبحوث المتقدمة.

جمهورية العراق
وزارة التعليم العالي والبحث العلمي
جامعة بابل/ كلية العلوم
قسم علوم الحياة

بعض التغيرات الهرمونية في السلوك العدائي لدى السجناء العراقيين

بحث مقدم الى

مجلس كلية العلوم – جامعة بابل

كجزء من متطلبات نيل درجة الدبلوم العالي في العلوم/ أدلة جنائية

من قبل

وليد عادل عباس محسن

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