Research Article

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Experimental Study of Thyroid Disturbances and Interplay with Growth Hormone on Body Development and Lipid Profile in Immature Male Rats

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Abstract

The endocrine system is an essential feature of the pituitary gland, which controls various hormonal activities. Disturbances in development-associated thyroid and growth hormones may lead to significant alterations in body weight, length, and mass index (BMI), abdomen circumference, and tail length, as well as the plasma lipid profile, thus changing body physiology. This research employed fifty immature male rats, which were separated into control, GH deficiency (GHD), GH supplementation, hypothyroidism, and hyperthyroidism groups. Over 21 days, the rats underwent hormonal treatments, observing changes in the pituitary glands' physiology through developmental and plasma lipid profile changes. Body development demonstrated similar trends and patterns. Indeed, the control group has grown in length over the past 10 weeks. The highest length growth was observed with GH addition and the lowest with GH deficiency. Hence, the Hypothyroid Group grew substantially less in length compared to the Control. In contrast, the Hyperthyroid Group showed minimal deviation or even paralleled the Control Group. In the lipid profile, in the growth hormone deficiency group the average cholesterol, triglyceride, LDL and VLDL level were decreased, while the average HDL level increased. In the growth hormone treatment group, the average cholesterol, HDL, LDL, and VLDL levels increased, whereas the average triglyceride level decreased. In the hypothyroidism group, the mean cholesterol, triglyceride, and VLDL levels decreased, as the HDL, LDL levels. The hyperthyroidism group experiences an increase in cholesterol, triglycerides, and LDL levels, while maintaining a constant HDL level and a decrease in VLDL levels. This work reveals more about the important influences of thyroid and growth hormone disruptions on pituitary gland

physiology and rat health, highlighting the organ's flexibility and essential controlling function in childhood.

Keywords: Pituitary Gland, Lipid, System, Development, Rats.

Introduction

The endocrine system's center of orchestration is the pituitary gland; this overseer secretes some key hormones that are responsible for regulating a variety of functions in the body (1). The anterior and posterior lobes separate this master gland, which releases tropic hormones to control various endocrine glands, such as the thyroid. In order to achieve homeostasis and development, among other things, the pituitary works closely with these glands (2). For instance, during puberty, thyroid hormones are very important for the normal development as well as the function of the pituitary since they aid in growth, metabolic regulation, and organ maturation for example, during puberty, thyroid hormones are critical for normal development as well as the functioning of the pituitary because they aid in growth, metabolic regulation, and organ maturation (more fluency) (3). Previous studies have shown developmental manifestations in the pituitary gland associated with varying levels of circulating growth hormone and thyroid hormones. Indeed, growth hormone plays a huge role in the normal functioning of the body and its development, which demonstrates the direct stimulating effects of growth hormone on the body (4).

All measurements of bodyweight, body length, BMI, abdomen circumference, and tail provide a clarified understanding of the physiological consequences of various experimental approaches. Thus, it was clearly stated that the growth hormone and the thyroid hormone are essential to rat growth and development (5). Growth hormone has a direct effect on plasma lipid profile; it may be leading to an increase or decrease in cholesterol, triglycerides, VHDL, HDL, and LDL by augmenting or falling in liver LDL receptor activity, and this may be on the order of magnitude of the increase or decrease induced by statins (6).

The goal is to enhance our understanding of developmental physiology by exploring this crucial developmental stage and examining the effects of varied GHRH or TRH exposures during prepubertal stages in a controlled environment.

Materials and Methods

Animal Model and Experimental Design

The research was structured as a speculative comparative research study, achieved at the physiology Department of Basic Sciences, College of Dentistry / University of Mosul / Mosul, Iraq. The study spans from February 21st, 2023, to May 2nd, 2023.Ten healthy and pregnant female albino rats were taken from the Animal House at the College of Veterinary Medicine at the University of Mosul. They were separated individually into clean, standard rat cages. Wood chips were spread as bedding until the pups were

born. Typical conditions were provided by temperature (22 ± 2 °C), humidity (50 ± 10%), a 12-h light/dark cycle, and animals being left *ad libitum* (7). All pregnant rats were monitored every morning until the time of delivery. Fifty immature male rats of one month's age, weighting about 160 ± 5g, will be used in this study, were divided into 5 equal groups (10 rats in each group) and treated for 21 days as follows:

1. Control group (G1): were given normal saline (Pioneer Company, Iraq) subcutaneously (S/C).

2. Growth hormone deficiency group (G2): were induced growth hormone deficiency by using sodium diethyldithiocarbamate (SDDTC) Sodium diethyldithiocarbamate has the formula NaS2CN (C2H5)2. It is an organosulfur compound. It is a pale-yellow, water-soluble salt), (100 mg/kg. BW) subcutaneously (S/C) (8).

3. Growth hormone treatment group (G3): were given human growth hormone (Norditropin novo nordisk, Swiss mad, Somatotropin 10mg/1.5 ml. (200 µg/kg BW) subcutaneously (S/C) (9).

4. Hypothyroidism group (G4): were induced Hypothyroidism by giving Propylthiouracil (PTU, by Takeda group, Turkey, each tablet contains 50 mg of Propylthiouracil), (1mg/kg Bw) as a fresh suspension prepared every day and administered orally by gavage needle (10).

5. Hyperthyroidism group (G5): were induced hyperthyroidism by being given Levothyroxine (anthrax25µg ®, Mark, Germany, each tablet contains 25µg of levothyroxine) (400 µg/kg Bw), a fresh suspension prepared every day and administered orally by gavage needle the hyperthyroidism group (G5): was given Levothyroxine (anthrax 25g®®, Mark, Germany) to induce hyperthyroidism. Each tablet contains 25g of levothyroxine (400 g/kg Bw), a fresh suspension prepared every day and administered orally by gavage needle (more fluency) (11).

Statistical Analysis

SPSS software was used in analyzing the data. In order to identify significant dissimilarities among groups, there was a one-way ANOVA (version 19), followed by post hoc tests. The values were expressed as mean ± standard deviation (SD), statistically significant at $p<0.05$. (12).

Results

1. Developmental Findings:

All measurements of body weight, body length, and BMI, as well as abdomen circumference and tail, provide a clear understanding of the physiological consequences of various experimental approaches. Thus, it was clearly stated that the growth hormone and the thyroid hormone thyroid are essential to rat growth and development.

The results recorded in table (1) showed drastic fluctuations in body weight for the experimental groups over the 21-day period. The control group continued to grow gradually throughout the period from 7 to 21 days, indicating normal increased

significantly ($p \leq 0.05$) in body growth. To treat GH deficiency, rats were given sodium diethyldithiocarbamate, and to treat hyperthyroidism, rats were given levothyroxine. The GH deficiency group's growth rate slowed down significantly ($p \leq 0.05$), which was due to a negative effect on body growth when compared to animals that were treated with growth hormone. The rats that were treated with exogenous GH had significantly ($p \leq 0.05$) high body growth with an upward trend, which was caused by the external provision of GH.

Table1: Effect of GH deficiency, GH treatment, hypo and hyperthyroidism on body weight in immature male rats (Mean ± SD), n= 10.

Groups	Body weight (BW)			
	D 0	**D7**	**D14**	**D21**
Control	68.4± 3.17 B	85.03±3.18 b	99.58±3.07 C	137.41±4.15 B
Growth hormone deficiency (400mg/kg)	69.44±3.51 B	77.59±3.04 c	102.22±4.53 C	127.29±4.19 C
Growth hormone treatment (200mg/kg)	77.14±4.07 A	114.11±4.12 a	149.70±4.57 A	163.84±5.06 A
Hypothyroidism (1mg/kg)	69.87±3.11 B	89.87±3.07 b	118.66±4.24 B	162.35±5.22 A
Hyperthyroidism (400ug/kg)	69.11±3.03 B	82.50±3.53 b	100.70±4.01 C	118.97±4.00 C

The different small letters refer to significant differences at ($p \leq 0.05$) .

Body length demonstrated similar trends and patterns table (2). Indeed, the control group, group treated with GH and hypothyroidism have increased significantly ($p \leq 0.05$) in growth in body length throughout days 7 to 21 when compared with other treated groups. GH addition led to the highest length growth, while GH deficiency resulted in the lowest growth.Hence, the Hypothyroid group grew substantially less in length compared to the control. In contrast, the hyperthyroid group showed minimal to significant ($p \leq 0.05$) growth in body length through (7 to 21) days. The BMI data provided additional insight into the rats' health and growth efficiency. The results from table (3) indicate a significant increased ($p \leq 0.05$) in the group of hypothyroidisms treated with (PTU 1mg/kg) with an advanced period of study. wWen compared with other treated groups, the GH deficiency group has a significantly ($p \leq 0.05$) lower BMI, which means that rats grew less efficiently. The GH addition group has a slightly higher ($p \leq 0.05$) BMI compared with the hyperthyroidisms and control groups which means it's an indicator of more efficient growth. The BMI of hyperthyroidism group suggests that it decreased significantly ($p \leq 0.05$) when compared with the hypothyroidism group with an advanced period of treatment.

Table 2: Effect of GH deficiency, GH treatment, hypo and hyperthyroidism on body length in immature male rats (Mean ± SD) n= 10.

Groups	Body Length			
	D 0	**D7**	**D14**	**D21**
Control	11.05± 0.17	13.15± 1.02	14.24± 2.06	15.37± 2.00
	A	a	A	B
Growth hormone deficiency (400mg/kg)	10.95± 1.11	11.68± 1.18	12.54± 1.64	13.4± 1.98
	A	b	C	C
Growth hormone treatment (200mg/kg)	11.06± 1.02	13.8± 1.04	16.77± 2.92	18.41± 3.06
	A	a	A	A
Hypothyroidism	10.96± 1.67	11.43± 1.28	13.22± 1.11	14.36± 2.72
(1mg/kg)	a	b	B	B
Hyperthyroidism (400ug/kg)	10.89± 1.10	11.26± 1.14	12.63± 2.05	13.65± 2.22
	a	b	C	C

The different small letters refer to significant differences at (p≤0.05).

In Table (4), there is a noticeable difference in abdomen circumference measurements between GH addition and other groups with an advanced period of study. The table showed an increase significantly (p≤0.05) in abdomen circumference in the group of animals treated with GH from days 0 to 21 when compared with GH deficiency, the group of hypo and hyperthyroidisms, and the control group. The GH deficiency appeared to have decreased significantly (p≤0.05) than other groups. When comparing the groups of animals treated with GH from days 0 to 21, the table showed that their abdomen circumference went up significantly (p≤0.05) compared to those with GH deficiency, hypothyroidism, hyperthyroidism, and the control group. The group with GH deficiency seemed to have gone down significantly (p≤0.05) more than the other groups.

Table 3: Effect of GH deficiency, GH treatment, hypo and hyperthyroidism on BMI in immature male rats (Mean ± SD) n= 10.

Groups	Body Mass Index (BMI)			
	D 0	D7	D14	D21
Control	6.14± 0.30 a	6.49± 1.24 c	7.77± 1.04 B	8.94± 1.00 C
Growth hormone deficiency (400mg/kg)	6.37± 1.20 A	6.64± 1.01 c	7.01± 1.67 B	7.89± 1.74 D
Growth hormone treatment (200mg/kg)	6.40± 1.34 A	8.28± 1.36 a	8.92± 1.09 A	9.83± 1.94 B
Hypothyroidism (1mg/kg)	6.64± 1.20 A	7.86± 1.04 b	8.97± 1.45 A	11.30± 1.84 A
Hyperthyroidism (400ug/kg)	6.86± 1.09 A	7.32± 1.22 b	7.97± 1.11 B	8.72± 1.06 C

The different small letters refer to significant differences at (p≤0.05).

The GH treatments showed a significant increase (p≤0.05) in the development of the tail from 0 to 21 days of experiments when compared with other treated groups table (5). The same table showed a significant decrease (p≤0.05) in tail GH deficiency compared to hypothyroidism and control groups.

2. Lipid Profile Analysis:

This section explores the effects of growth hormone and thyroid axis manipulations on the lipid profiles of immature male rats across different experimental groups (Table-6).The study found out what each group's cholesterol levels were. The results showed that the table went down significantly (p≤0.05) in the group of animals that didn't have enough GH compared to other treated groups and controls. On the other hand, it went up significantly (p≤0.05) in the group of animals that were given extra GH compared to other groups. There was no significant difference between the hypothyroidism and hyperthyroidism groups. When compared with other groups, triglyceride showed a significant increase (p≤0.05) in the group of hyperthyroidism animals, while hypothyroidism recorded a significant decrease (p≤0.05) when compared with GH deficiency and GH

addition groups. The same table showed that HDL levels went up significantly ($p \leq 0.05$) in the group that received GH addition compared to other treated groups. On the other hand, LDL levels went up significantly ($p \leq 0.05$) only in the group of animals with hypothyroidism compared to other treated groups. The same table also showed that VLDL levels went up significantly ($p \leq 0.05$) in the group that received GH addition compared to other treated groups and decreased significantly ($p \leq 0.05$) in the group of animals with hyperthyroidism

Table 4: Effect of GH deficiency, GH treatment. hypo and hyperthyroidism on abdomen circumference in immature male rats (Mean ± SD) n= 10.

Groups	Abdomen Circumference			
	D 0	D7	D14	D21
Control	9.92± 1.43 a	10.06± 1.90 b	11.36± 1.65 B	12.74± 1.86 b
Growth hormone deficiency (400mg/kg)	9.98± 1.08 a	9.75± 1.43 c	10.79± 1.07 C	11.42± 1.64 c
Growth hormone treatment (200mg/kg)	10.06± 1.70 a	12.77± 1.12 a	15.92± 1.00 A	31.84± 1.06 a
Hypothyroidism (1mg/kg)	10.10± 1.11 a	10.99± 1.60 b	11.7± 1.64 B	12.53± 1.70 b
Hyperthyroidism (400ug/kg)	10.01± 1.60 a	10.99± 1.05 b	11.88± 1.03 B	12.73± 1.32 b

The different small letters refer to significant differences at ($p \leq 0.05$)

Table 5: Effect of GH deficiency, GH treatment, hypo and hyperthyroidism on tail length in immature male rats (Mean ± SD) n= 10.

Groups	Tail Length			
	D 0	D7	D14	D21
Control	9.74± 1.06 a	11.69± 1.98 a	12.52± 1.13 B	13.19± 1.85 b
Growth hormone deficiency (400mg/kg)	9.91± 1.45 a	10.13± 1.64 b	10.91± 1.09 D	11.18± 1.01 d
Growth hormone treatment (200mg/kg)	9.86± 1.07 a	11.95± 1.73 a	15.06± 1.70 A	16.03± 1.43 a
Hypothyroidism (1mg/kg)	9.19± 1.29 a	10.36± 1.08 b	11.48± 1.22 C	12.85± 1.34 c
Hyperthyroidism (400ug/kg)	9.55± 1.54 a	10.42± 1.91 b	11.26± 1.51 C	12.79± 1.61 c

The different small letters refer to significant differences at (p≤0.05).

Table 6:The normal value of lipid profile in rats (Mean ± SD) n= 10

+	Cholesterol Mg/dl	Triglyceride Mg/dl	HDL Mg/dl	LDL Mg/dl	VLDL Mg/dl
Control	89.8 ± 4.39 a	75.2 ± 4.61 b	40.6 ± 3.24 c	15.5 ± 2.64 B	27.3 ± 4.50 b
Growth hormone deficiency (400mg/kg)	66.0 ± 3.30 c	60.9 ± 4.07 c	46.5 ± 4.35± b	15.1 ± 4.07 B	16.6 ± 2.17 c
Growth hormone treatment (200mg/kg)	90.8 ± 6.37 a	62.7 ± 4.65 c	58.5 ± 5.85 a	16.6 ± 2.22 B	32.8 ± 4.49 a
Hypothyroidism (1mg/kg)	85.3 ± 4.97 b	52.2 ± 6.65 d	42.2 ± 6.88 c	18.2 ± 2.44 A	17.5 ± 2.22 c
Hyperthyroidism (400ug/kg)	85.9 ± 4.89 b	84.9 ± 4.91 a	40.6 ± 3.24 c	16.9 ± 3.31 B	14.3 ± 3.33 d

The different small letters refer to significant differences at (p≤0.05)

Discussion

For this GH deficient group, the lag in growth development was noted. Indeed, growth hormone plays a huge role in the body's normal functioning and development. Sodium diethyldithiocarbamate was administered to cause the deficiency, disrupting the growth process and demonstrating the importance of growth hormones to control growth rates. The substantial growth in the GH treatment group was achieved due to the introduction of exogenous growth hormone, which demonstrates the direct stimulating effects of growth hormone on the body.

Thereby, it can be assumed that growth hormone supplementation from the outside can disrupt the deficiency in control growth and accelerate body growth beyond control growth levels. Groups with hypothyroidism and hyperthyroidism exhibit significant differences in their growth curves. Indeed, thyroid hormones play an important role in the body's normal development and functioning. Without these hormones, the growth process cannot proceed. Conversely, the latter group, which has growth rates that are between GH addition and the control group, demonstrate that excessive levels of the thyroid hormone still play a substantial role Conversely, the latter group, which has growth rates that are between GH addition and the control group, demonstrates that excessive thyroid hormone levels still play a significant role (more fluency) (13).

Results from previous studies show high agreement. A study conducted by (14) suggests that thyroid hormones significantly influence fat metabolism and modify cholesterol and triglyceride levels in the body. The study by (15) also discovered a negative impact on body fat levels due to GH deficiency, a finding that aligns with this study's conclusion that "GH deficiency might influence lipid metabolism. On the other hand, some studies report mixed results regarding the effects of hyperthyroidism on lipid levels. One of them suggests that hyperthyroidism may result in a reduction in cholesterol levels, a finding that contrasts with the current study's findings, which indicate an increase in cholesterol levels in the hyperthyroidism group (5).

According to the above, this study's findings are mostly consistent with previous research on growth hormones and thyroid's effects on fat metabolism, with some discrepancies due to study design or rat biology.

Conflicts of interest

The authors declare that there is no conflict of interest.

Ethical Clearance

This work is approved by The Research Ethical Committee.

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دراسة تجريبية لاضطرابات الغدة الدرقية والتفاعل مع هرمون النمو على نمو الجسم ومستوى الدهون في ذكور الجرذان الغير ناضجة

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الخلاصة

نظام الغدد الصماء هو سمة أساسية من الغدة النخامية، التي تتحكم في الأنشطة الهرمونية المختلفة. قد تؤدي الاضطرابات في هرمونات الغدة الدرقية والنمو المرتبطة بالنمو إلى تغيرات كبيرة في نمو وزن الجسم وطوله ومؤشر كتلته (BMI)، ومحيط البطن وطول الذيل، بالإضافة إلى ملف الدهون في البلازما، وبالتالي تغيير فسيولوجيا الجسم .استخدم في هذا البحث خمسين من الجرذان غير الناضجة تم فصلهم إلى مجموعات وكما يلي: مجموعة السيطرة، مجموعة نقص هرمون النمو (GHD)، مجموعة العلاج بهرمون النمو، مجموعة قصور الغدة الدرقية واخيرا مجموعة فرط نشاط الغدة الدرقية. خضعت الجرذان لعلاجات هرمونية على مدار 21 يومًا، حيث تغيرت خلالها فسيولوجيا الغدة النخامية من خلال التغيرات التنموية وتغيرات الدهون في البلازما. أظهر نمو الجسم اتجاهات وأنماط مماثلة. في الواقع، تزايد طول مجموعة المراقبة طوال الأسابيع العشرة. وقد لوحظ أعلى نمو في الطول مع إضافة هرمون النمو وأدنى نمو مع نقص هرمون النمو. ومن ثم، فقد نمت مجموعة قصور الغدة الدرقية بشكل أقل في الطول مقارنة بالمجموعة الضابطة. في المقابل، أظهرت مجموعة فرط نشاط الغدة الدرقية انحرافًا بسيطًا أو حتى موازيًا لمجموعة التحكم. في ملف الدهون، في مجموعة نقص هرمون النمو، انخفض متوسط مستوى الكوليسترول والدهون الثلاثية و LDLوVLDL، في حين ارتفع متوسط مستوى .HDL في مجموعة العلاج بهرمون النمو ارتفع متوسط مستوى الكولسترول، HDL، LDLوVLDL، في حين انخفض متوسط مستوى الدهون الثلاثية في مجموعة قصور الغدة الدرقية، انخفض متوسط مستوى الكوليسترول والدهون الثلاثية ومستوىVLDL ، وارتفاع مستوى HDLو LDLفي مجموعة فرط نشاط الغدة الدرقية في حين ارتفع مستوى الكوليسترول والدهون الثلاثية وLDL، بينما بقي مستوى HDL ثابتًا ولكن مستوى VLDL قد انخفض. يكشف هذا العمل المزيد عن التأثيرات الهامة لاضطرابات الغدة الدرقية وهرمون النمو على فسيولوجيا الغدة النخامية وصحة الجرذان، ويسلط الضوء على مرونة العضو ووظيفة التحكم الأساسية في مرحلة ما قبل النضج.

الكلمات المفتاحية: الغدة النخامية، الدهن، نظام، التطور، الجرذان.