World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

Check for updates

Expressions of thyroid and growth hormones as risk factors for Achilles tendinopathy in a population of Nigerian football players: A preliminary study

Itunu Opeyemi Owoeye [1], Ayoola Ibifubara Aiyegbusi [1,*], Chidinma S. Umeadi [2], Tobi S. Daniels [3] and Oluyemi Akinloye [2]

[1] *Department of Physiotherapy, Faculty of Clinical Sciences, University of Lagos, Lagos.*
[2] *Department of Medical Laboratory Sciences, Faculty of Basic Medical Sciences, College of Medicine, University of Lagos, Lagos.*
[3] *North Tees and Hartlepool NHS Foundation Trust*

World Journal of Biology Pharmacy and Health Sciences, 2025, 23(01), 423-430

Publication history: Received on 14 June 2025; revised on 20 July 2025; accepted on 22 July 2025

Article DOI: https://doi.org/10.30574/wjbphs.2025.23.1.0687

Abstract

Background: Achilles Tendinopathy (AT) has become a global concern in Sports Medicine especially as it results in interruption of the athletes' career. Several intrinsic and extrinsic factors have been postulated to predispose a person to AT. The aim of this study was to investigate the role of expressions of thyroid and growth hormones and their association with Achilles Tendinopathy.

Materials and Methods: This study involved 70 football players who were recruited across football clubs in Lagos, Nigeria. Ethical approval was obtained from the institutional Health Research and Ethics Committee and informed consent was obtained from each participant. The presence of Achilles tendinopathy was determined using the Royal London Hospital Test and confirmed by ultrasonography. 5ml of blood was taken from each footballer using EDTA and plain bottles. Growth and Thyroid hormones were measured with Enzyme Linked Immunosorbent Assay (ELISA). Significance was set at $p< 0.05$.

Results: The serum level of growth hormones and triiodothyronine (fT_3) was lower in participants with AT 2.98 ± 3.3.69 and 2.98 ± 3.69 compared to controls 3.39 ± 1.18 and 6.79 ± 8.08 ($p<0.05$). There was no significant association of growth hormone with Achilles tendinopathy, $p= 0.050$. Triiodothyronine (fT_3) and thyroxine (fT_4) were significantly associated with Achilles tendinopathy $p<0.001$ and $p =0.028$ respectively, but there was no significant difference in the level of TSH of players with and without Achilles tendinopathy.

Conclusion: Reduced thyroid hormone is strongly associated with Achilles tendinopathy among Nigerian footballers.

Keywords: Growth Hormone; Thyroid Hormone; Triiodothyronine; Thyroxine; Achilles Tendinopathy; Footballers

1. Introduction

Achilles tendinopathy is a foot and ankle debilitating overuse injury which is common in running and jumping sporting activities and affects multiple domains of tendon health and physical function [1, 2.] The incidence of Achilles tendinopathy appears to be on the increase, and the average age at which rupture of the Achilles tendon occurs has also reduced over time, and it is projected to keep reducing as more people engage in athletics [3, 4]. The introduction of the U-13 (under 13) and U-15 (under 15) football tournaments by the Nigeria Football Federation in 2016, has resulted in increase in football participation in Nigeria, with consequent increased number of injuries [5,6]. The prevalence of

* Corresponding author: Ayoola Ibifubara Aiyegbusi

Achilles tendinopathy among recreational sports participants was reported as 21% and 15.9% among football players in Nigeria [7,8]. A recent systematic review on running related musculoskeletal injuries found that Achilles tendinopathy presented with a prevalence of 10.3% and incidence of 6.6% while it accounted for 13.7% of the pathology with the greatest [9,10]. Achilles tendinopathy clinically presents with symptoms of pain, swelling, and impaired function during sporting activities and activities of daily living [11]. Excessive mechanical loading because of high intensity exercises such as long distance running and jumping leads to tendinopathy [12]. The cause of tendinopathy is multifactorial and inherited genes or gene variants have been found to predispose some individuals to this injury more than the others. However, the increasing prevalence of metabolic disorders in recent times has prompted investigations into the possible connection between metabolic disorders and musculoskeletal diseases [13, 14]. Mechanical loading during exercise modulates the availability of growth factors, including growth hormone (GH) and insulin-like growth factor-I (IGF-I), which are important in maintaining muscle-tendon homeostasis and regulating collagen synthesis [15, 16]. The growth hormone has been reported to stimulate collagen synthesis and connective tissue maturation while the thyroid hormone improves the collagen structure in tendons [15, 17]. Thyroid hormones (THs), T3 and T4, play essential roles in the development and metabolism of many tissues and organs, and exert profound metabolic effects on changes in oxygen consumption, protein, carbohydrate, lipid, and vitamin metabolism [15,17]. The effects of THs are mediated mainly through T3, which regulates gene expression by binding to the TH receptors (TR)-α and –β [6, 18]. These hormones have protective action against apoptosis induced by serum deprivation, which is considered an important factor for failed healing response observed in human tendinopathies [19].

While young individuals are increasingly interested in sporting activities, there is a knowledge gap concerning the risk of development of Achilles tendinopathy and the role of thyroid and growth hormones in its development. Investigations on the expression of growth and thyroid hormones may show specific link that could contribute to the understanding of the pathogenesis of this disorder. The overall aim of this study was to determine the association between Achilles tendinopathy and changes in thyroid and growth hormones.

2. Methods

This was a descriptive case-control study that involved seventy (70) amateur football players who were recruited by convenience from 13 football clubs under the Lagos junior league, Nigeria. Twenty-three of the participants tested positive to the Royal London Hospital Test while 47 tested negative. Participants that were recruited had played football actively in the last six months and were currently engaged in full training and match responsibilities at the time of the study. Players who had undergone surgical repair of the Achilles tendon, beach footballers and footballers who were physically challenged or with disability in the lower limbs were excluded. Ethical approval was sought and obtained from the institutional Health Research and Ethics Committee with protocol number CMUL/HREC/09/19/601. Informed consent was sought and obtained from all the participants after duly explaining the study procedure to them. The consent of the coach was obtained for participants who were under the age of eighteen (18) years. 5ml of blood was taken from each participant into EDTA bottles for hormonal assay. Anthropometric and physical measurements were taken at the beginning of the study: age, gender, height and weight of each participant were recorded using a data form. Participants' body mass index (BMI) was calculated from the ratio of their weight to height.

2.1. Sample size calculation

The sample size was calculated using Cohen effect size [20]

$$n = \frac{z^2 \times P \times (1 - P)}{d^2}$$

Were

- n = Sample size
- Z = Statistics corresponding to confidence level=1.96
- P = Expected prevalence = 15.9% ≈0.159 [6]
- d = Margin of error

For a descriptive survey of this nature, the following values were set

- Z = 1.96 for a 0.05 level of confidence
- P = 0.159 (15.9%) Prevalence of Achilles tendinopathy among Nigerian footballers [6].
- d = 0.1 (10%)

- Hence, n = 51.4 ≈ 52

Royal London Hospital test was performed on each participant while lying prone on a plinth/flat surface as previously described [21]. Participants were positioned prone on a plinth with their ankles hanging relaxed just over the edge of the plinth, the portion of the Achilles tendon which was maximally tender to palpation was identified. Then the participant was asked to actively dorsiflex the ankle while the tender part of the tendon was palpated again. However, this time in maximal dorsiflexion, participants with Achilles tendinopathy reported a substantial decrease or absence of pain when the palpation technique was repeated in dorsiflexion.

2.1.1. Ultrasonography

Diagnostic Ultrasound Machine XARIO 200 (China) was used for imaging in gray scale with a 5-12 MHz 50mm linear array transducer as previously described [18]. The Achilles tendon of all participants who were positive to the Royal London Hospital test were assessed in prone position with the feet hanging over a plinth. A musculoskeletal sonographer performed all imaging in the sagittal and axial planes (supero-inferiorly and medio-laterally) along the length of the Achilles tendon. The hypoechoic region and/or focal thickening in both planes were noted. Color Doppler was used to note areas of neovascularization.

2.1.2. Blood sample Collection

5mls of blood sample was collected by venipuncture in an EDTA (Ethylenediaminetetraacetic Acid) vacutainer bottle and 5mls of blood sample was collected in a plain bottle from each study participant. This was taken between 8 and 11 in the morning before activity commenced for all participants.

2.1.3. Laboratory Analysis

Enzyme Linked Immunosorbent Assay (ELISA) was done for both thyroid and growth hormones from the blood samples collected in plain bottles. The Calibiotech fT_3 test kit solid phase ELISA was used to determine the level of free triiodothyronine (fT_3) and Calibiotech fT_4 test kit solid phase competitive ELISA was used to determine free thyroxine (fT_4) level. Thyroid Stimulating Hormone (TSH) level was determined with Calbiotech TSH solid phase sandwich ELISA and Human Growth Hormone (hGH) assay was done with Calibiotech solid phase sandwich hGH ELISA. All protocol followed producer's manual. Absorbance was read at 450nm within 15 minutes after adding the stop solution for all assay.

2.2. Data analysis

Data analysis was done using Statistical Package for Social Sciences (IBM, SPSS) version 26.0 software. Data was analyzed using descriptive statistics of frequency, mean, and standard deviation and T-test. Spearman correlation, logistic regression and odd ratio was used to describe the association of growth and thyroid hormone with Achilles tendinopathy. Significance was set at $p< 0.05$.

3. Results

A total of 70 amateur footballers participated in this study and consisted of 23 participants with Achilles tendinopathy and 47 control footballers without AT. Demographic characteristics is as described (Figures 1 and 2.) Fifty-one (51) were males (72.9%) and nineteen (19) were females (27.1%). Their mean age (years) was 23.17 ± 5.96, mean height (cm) was 172.44 ± 9.83, mean weight (kg) was 66.19 ± 11.54 and mean body mass index (BMI) (kg/cm^2) was 22.27 ± 5.22.

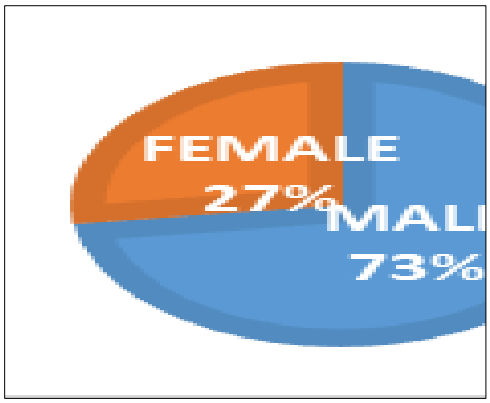

Figure 1 Gender distribution of Participants

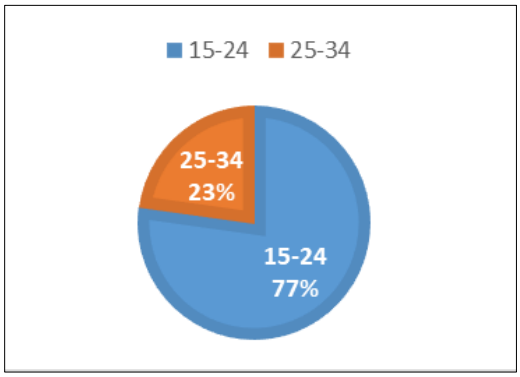

Figure 2 Distribution of Age Group

The difference in the growth hormone and thyroid hormone of cases of AT and controls are described in Table 1. The human growth hormone (hGH) for cases was significantly lower at 2.98±3.69 compared to control at 6.79±8.08 (p=0.008), free triiodothyronine (fT3) was 1.95±0.81 and control 3.39±1.18 (p =<0.001) while free thyroxine (fT4) was significantly higher in cases at 2.25±0.54, compared to control at 1.89±0.63 (p=0.016)

Table 2 shows the association between Achilles tendinopathy and growth and thyroid hormones using the spearman's rank correlation. Both fT3 and fT4 were significantly associated with p = <0.001 and 0.023 respectively while the thyroid stimulating hormone (TSH) was not significant with p = 0.809. Logistic regression result shows fT3 and fT4 were still positively correlated (r=0.611, 0.272) and significant with p values of <0.001 and 0.028 respectively while there was no significant association of TSH with AT (p = 0.321) (Table 3, figure 3).

Table 1 Differences in the Expression of Thyroid and Growth Hormones of participants with and without Achilles tendinopathy

Hormones	Achilles Tendinopathy (Mean ± SD)	Non-Achilles Tendinopathy (Mean ± SD)	t-test	p-value
hGH (ng/ml)	2.98±3.69	6.79±8.08	2.712	0.008*
TSH (µIU/ml)	0.88±0.36	1.08±0.89	1.345	0.183
fT3 (pg/ml)	1.95±0.81	3.39±1.18	5.924	<0.001*
fT4 (ng/dl)	2.25±0.54	1.89±0.63	2.502	0.016*

*Significant at *p<0.05*; Key: SD – Standard Deviation, hGH –Human Growth Hormone, TSH – Thyroid Stimulating Hormone, fT3 – free triiodothyronine, fT4 – free thyroxine

Table 2 Relationship between Achilles Tendinopathy and Thyroid and Growth hormones

Parameters	rs	p-value
TSH	0.029	0.809
fT3	0.611	<0.001*
fT4	0.272	0.023*
hGH	0.227	0.059

*Significant at *p<0.05*; Key: rs – Spearman's rank correlation coefficient, fT3 – free Triiodothyronine, fT3 – free Triiodothyronine, hGH – Human Growth Hormone, TSH – Thyroid Stimulating Hormone

Table 3 Association between Growth and Thyroid Hormones and Achilles Tendinopathy

Parameters	Odds Ratio (OR)	p-value
hGH	0.893	0.050
fT3	0.131	<0.001*
fT4	2.891	0.028*
TSH	0.615	0.321

*Significant at *p<0.05;* Key: fT3 – free Triiodothyronine, fT3 – free Triiodothyronine, hGH – Human Growth Hormone, TSH – Thyroid Stimulating Hormone.

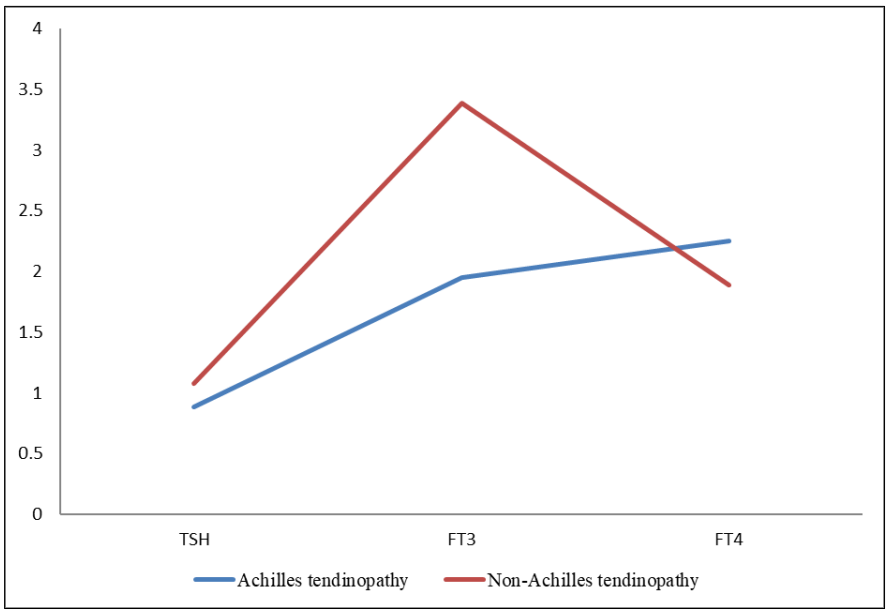

Figure 3 Graph showing Relationship between Thyroid hormones and Achilles tendinopathy

4. Discussion

Achilles tendinopathy (AT) is a time loss injury that results from loss of functional strength and endurance, thereby impacting negatively on an athletes' activity of daily living (ADL) and career. This was a preliminary study carried out as part of a continuous attempt to investigate intrinsic factors that may predispose an athlete to AT in order to devise preventative measures against its development. It was a cross-sectional study to investigate the association between tendinopathy and the expressions of growth and thyroid hormones among a population of Nigerian footballers. The knowledge of this is very important as previous studies have reported the link between expression of hormones and

soft tissue injuries [17, 18, 19 22]. It is therefore necessary to investigate the expression of the selected hormones in Achilles tendinopathy and determine their association among Nigerian footballers.

In this study, the expression of human growth hormone (hGH) was significantly (p=0.008) reduced among football players with Achilles tendinopathy compared with players without Achilles tendinopathy. The growth hormone is closely linked with collagen synthesis and connective tissue maturation and its normal expression is important for collagen synthesis in skeletal muscles and tendons [23]. The growth hormone (GH) was previously reported to have a matrix-stabilizing effect during inactivity and rehabilitation by stimulating collagen synthesis in the musculo-tendinous tissue and increasing the cross-sectional area of tendons [18, 23, 24], its decreased expression would therefore mean an alteration in collagen synthesis which explains the development of tendinopathy among the football players in this study. Unlike our study which observed reduced expression of hGH among football players with Achilles tendinopathy the study of Han *et al* [24] reported that there was no significant alteration in tendon size or collagen synthesis during hGH variations. Most previous studies on humans however corroborates our findings that increased circulation of hGH increases the mRNA expression of collagen type 1 and this increase inhibits cell death induced by anoxia in human tendon [18, 23].

The thyroid Stimulating Hormone (TSH) level was reduced in players with Achilles tendinopathy in this study although there was no significant difference (p= 0.183) in the level of TSH in players with and without Achilles tendinopathy. This finding agrees with the report of Olivia *et al* [23] who demonstrated by Western Blot analysis that thyroid hormone α or β nuclear receptor isoforms are expressed at high levels in healthy and pathological rotator cuff tendons, with no apparent difference in thyroid hormones (THs) expression between tendon from normal subjects and patients with thyroid disease. Even though the thyroid hormone enhances the proliferation of tenocytes and protects against apoptosis, this study observed no difference in the thyroid stimulating hormone level of cases versus control. Exercise causes physical stress and exertion and this is accompanied by some endocrine modifications meant to neutralize its effects on thermogenesis and substrate metabolism [25]. Regulation of the metabolic adaptation caused by prolonged physical activities by the thyroid hormones is important but remains controversial. Some authors reported that no major effect of exercise training is observed on the serum TSH levels in professional athletes and sedentary individuals, suggesting a minimal influence of long-term chronic exercises [26]. In contrast, another study concluded that long -term exercise training might lead to down regulation of thyroid hormone concentrations [26]. Exercise is quite stressful and it challenges the body's homeostasis such that the body reestablishes a new dynamic equilibrium in order to minimize cell damage. One of the body's systems usually affected is the hypothalamic-pituitary-thyroid axis [27]. Ronaldo *et al* [27] in their study on men that underwent six months of endurance training reported that T_4 and free T_4 concentrations were slightly decreased, with no change in TSH.

In this study, triiodothyronine (fT_3) level was significantly reduced (p=0.001) in footballers with Achilles tendinopathy compared to controls. This may be due to the presence of inflammation as the Achilles tendon is in an injured state. Persistent low-grade inflammation, as seen in a multitude of chronic diseases, is obviously detrimental. The findings of Han *et al* [24] on animal studies reported that immunocytochemical techniques and microscopic analysis on animal tenocytes and fibrochondrocytes from fibrocartilaginous entheses showed that injured tendons that progress toward healing show a gradual decrease in the number of triiodothyronine (T3) receptors of collagen-forming fibroblasts, and their ability to synthetize collagen reduces.

Also, the thyroxine (fT_4) level of football players with Achilles tendinopathy was significantly increased (p=0.016) compared to control which implies that fT_4 is not being converted to fT_3 which is the active thyroid hormone. It has been reported that the number of participants involved in physical activities of high performance has increased annually and this has consequently increased the number of reported thyroid diseases as the body releases pro-inflammatory substances in an acute form [27]. The existence of subclinical hypothyroidism and these inflammatory markers inhibit peripheral conversion of T_4 to T_3 [27] . Free T_4 is converted to T_3 by enzyme deiodinase, and a reduction in conversion maybe related to abnormal enzymatic action usually caused by injury response or tissue repair in adults [26] which explains the high level of fT_4 in this study. It has been reported that tendon overuse with genetic predisposition and dysmetabolisms expose humans to tendinopathy [28]. Dysmetabolism can either be an increase or decrease in metabolic functions, in this study it was a decrease in metabolic function due to reduction in thyroid hormones. Hypothyroidism as commonly seen in primary care medicine is a metabolic state caused by thyroid hormone deficiency and it can be primary or secondary. Knopp *et al* [29], reported that participation in sports results in some form of injury or muscle soreness, and symptoms associated with training may be the only presenting symptoms of hypothyroidism. Also, many athletes train through minor injuries therefore, manifestations of hypothyroidism may be masked in active persons with musculoskeletal symptoms which may be perceived as the normal strain in their training routine. Prolonged physical exercise can lead to a systemic inflammatory response and a dysregulation of the glands resulting in tendinopathy as seen in this study.

Limitation of Study

This was a preliminary study so the sample size was small. A larger sample size would allow for a generalized application of the findings. Further studies with larger sample size is recommended.

5. Conclusion

Growth and thyroid hormones are contributory to the changes in the homeostasis of the Achilles tendon. Reduced thyroid hormone is an important risk factor for Achilles tendinopathy among Nigerian footballers.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Obst SJ, Barrett RS, Newsham-West R. Immediate effect of exercise on achilles tendon properties: systematic review. Medicine and Science in Sports and Exercise. 2013; 45: 1534–1544.

[2] Silbernagel KG, Hanlon S, Sprague A. Current Clinical Concepts: Conservative Management of Achilles Tendinopathy. J Athl Train. 2020;55(5):438-447. doi:10.4085/1062-6050-356-19

[3] Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. Scand J of Med and Sci in Sports.2013; 23(6): 669–686.

[4] Hagen, Mia MD; Pandya, Nirav K. MD Achilles Tendon Ruptures in Young Female Basketball Players: A Case Series, JAAOS: Global Research and Reviews: June 2019 - Volume 3 - Issue 6 - p e19.00016

[5] Ogungbe O. "NFF unveil Zenith 2017 U13, U15 future stars tournament." The Guardian. 2017. Assessed on 1 February 2019, from: https://guardian.ng/sport/nff-zenith-unveil-2017-u13-u15-future-starstournament.

[6] Owoeye OBA, Aiyegbusi AI, Fapojuwo OA, Badru OA, Babalola AR. Injuries in male and female semi-professional football (soccer) players in Nigeria: prospective study of a National Tournament. BMC Research Notes. 2017; 10: 133. doi.org/10.1186/s13104-017-2451-x.

[7] Aiyegbusi AI, Okafor UAC, Leke OP. Prevalence of Achilles tendinopathy and its association with physical characteristics in recreational sport participants in Lagos, Nigeria. Journal of Clinical Sciences. 2016; 13: 163-6.

[8] Aiyegbusi AI, Owoeye IO, Balogun OJ, Fapojuwo OO, Akinloye. Prevalence of Achilles Tendinopathy and Associated Selected Intrinsic Risk Factors among Nigerian Footballers. Muscles, Ligaments and Tendons Journal. 2021; 11 (1): 118-127.

[9] Kakouris, Nicolas et al. "A systematic review of running-related musculoskeletal injuries in runners." Journal of sport and health science vol. 10,5 (2021): 513-522. doi:10.1016/j.jshs.2021.04.001

[10] Heyward OW, Rabello LM, van der Woudea L, van den Akker-Scheek I, Gokeler A, van der Worp H, Zwerver J. The effect of load on Achilles tendon structure in novice runners. J Sci and Med in Sports. 2018; 21: 661-665.

[11] Abate M, Schiavone C, Salini V, Andia I. Occurrence of tendon pathologies in metabolic disorders. Rheumatology (Oxford). 2013 Apr;52(4):599-608. doi: 10.1093/rheumatology/kes395. Epub 2013 Jan 12. PMID: 23315787.

[12] Hong-Yun L, Ying-Hui H. Achilles Tendinopathy: Current Concepts about the Basic Science and Clinical Treatments. BioMed Research International. 2016; Article ID 6492597, 9 pages.

[13] Dolphin P, Bainbridge K, Mackenney P, Dixon J. 'Functional dynamic bracing and functional rehabilitation for Achilles tendon ruptures: a case series'. Physiotherapy Practice and Research. 2016; 37(2): 119-125.

[14] Oliva, F., Berardi, A., Misiti, S. et al. Thyroid hormones enhance growth and counteract apoptosis in human tenocytes isolated from rotator cuff tendons. Cell Death Dis 4, e705 (2013).

[15] Vaughn NH, Stepanyan H, Gallo RA, Dhawan A. Genetic Factors in Tendon Injury: A Systematic Review of the Literature. The Orthopaedic Journal of Sports Medicine. 2017; 5: 8.

[16] Morimoto, K., and Inouye, K.A., Sensitive enzyme immunoassay of human thyroid–stimulating hormone (TSH) using bispecific F(ab')2 fragments recognizing polymerized alkaline phosphatase and TSH. J. Immunol. Methods, 1997;205(1):81–90.

[17] Abate M, Guelfi M, Pantalone A, Vanni D, Schiavone C, Andia I, Salini V. Therapeutic use of hormones on tendinopathies: a narrative review. Muscles, Ligaments and Tendons Journal. 2016; 6 (4): 445-452.

[18] Nevin TW, James DC, Justin CL. Imaging in the Assessment and Management of Achilles Tendinopathy and Paratendinitis. Sem in MSK Radiol. 2011; 15: 89-100.

[19] Oliva F, Piccirilli E, Berardi AC, Frizziero A, Tarantino U, Maffulli N. Hormones and tendinopathies: the current evidence. British Medical Bulletin. 2016; 117(1): 39-58.

[20] Cohen J, (1992). A power primer. Psychological bulletin, 112(1): 155.

[21] Maffulli N, Kenward MG, Testa V, Capasso G, Regine R, King JB. Clinical diagnosis of Achilles tendinopathy with tendinosis. Clin J Sport Med 2003;13(1):11-5.

[22] Perseghin G, Lattuada G, Ragogna F, Alberti G, La Torre A. Free leptin index and thyroid function in male highly trained athletes. Eur J Endocrinol 2009; 161(6): 871-876.

[23] Oliva F, Misiti S, Maffulli N. Metabolic diseases and tendinopathies: the missing link. Muscles Ligaments Tendons J 2014; 4: 273–4.

[24] Han X, Guo L, Wang F. Contribution of PTHrP to mechanical strain-induced fibrochondrogenic differentiation in entheses of Achilles tendon of miniature pigs. J Biomech. 2014; 47: 2406–14.

[25] Fortunato R, Ignacio D, Padron A, Pecanha R, Marassi M, et al. (2008). The effect of acute exercise session on thyroid hormone economy in rats. J Endocrinol 198(2): 347-353.

[26] Garras DN, Raikin SM, Bhat SB. MRI is unnecessary for diagnosing acute Achilles tendon ruptures: Clinical diagnostic criteria. Clinical Orthopaedic and Related Research. 2012; 470(8): 2268-2273.

[27] Ronaldo A, Rosa M, Moretti G. Thyroid Homeostasis and Inflammation, the Physiological Changes of Exercise of Thermogenesis. J Endocrinol Thyroid Res. 2017; 2(2): 001-005.

[28] Sharma P, Maffulli N (2006). Biology of tendon injury: healing, modeling and remodeling. J Musculoskelet Neuronal Interact. 6(2): 181-190.

[29] Knopp WD, Bohm ME, McCoy JC (1997). Hypothyroidism presenting as tendinitis. The Physician and sports medicine, 25(1): 47-55