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Measuring the level of the happiness hormone serotonin, dehydroepiandrosterone, and some biochemical variables in patients with enlarged prostate

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Abstract
An enlarged prostate indicates that the gland has become bigger. As men age, virtually all of them develop prostate enlargement, which is commonly referred to as benign prostatic hyperplasia (BPH). The study found that greater levels of the hormone DHEA in prostate serum may predict the existence of individuals with benign prostatic hyperplasia or prostatitis as compared to healthy people. 5-hydroxytreptamine, which is related with prostatitis and enlargement, was also investigated, and we found that patients had lower levels of 5-hydroxytreptamine than controls. Prostatitis and hypertrophy were far more prevalent compared to the control group. (p<0.05). Their impact on a variety of markers, including blood pressure, diabetes, age, smoking, the effectiveness of therapy, the types of therapies employed, body mass, the environment.

Keywords: DHEA, prostate enlargement, prostatitis, benign prostatic hypertrophy

Introduction
Normal progression mild prostatic hypertrophy. The prostate gland is tiny at birth (1.5 g) and remains so until early adolescence when it grows completely. An androgen-dependent adolescent growth phase [1]. For adolescents and young adults, the median weight is 20 g (±6g) [2, 3] Following the first. The expansion and rejuvenation stage, encompasses the complete gland of the prostate (outside, essential and transitional zones); the area that transitions experiences a further selective development phase, which occurs in roughly Half of males by the tender age of fifty, and 90% of men over the age of 80. This development is pathologically known as BPH, which is and medically known as benign prostatic enlargement (BPE) or simple prostatic obstructions. It is believed that appropriate connections between the cellular and fibromuscular tissue constituents.

The area where the transitional prostate tissue has been modified resulting in a lower epithelial/stromal percentage, leading to micronodular remodeling, which defines BPH. Clinically, BPH develops by cells growth of epithelium and mesenchymal components inside the Prostate gland. These alterations, which begin by histology in the 3rd century of age and medically in their fifth period, are principally caused by elevated levels of dihydrotestosterone (DHT) in the prostate and proceed throughout life. When the prostate swells, greater resistance in the proximal urethra may limit urine flow during urination, causing pathophysiologic alterations to the bladder wall. As a result, the symptoms of bladder detrusor dysfunction are fundamentally linked to LUTS due to prostate blockage [4, 5]. Medically, BPH is defined by the growing development of LUTS, with symptoms include nocturia, partial closing, urinary delay, weak the road, frequency, and urgency to severe urinary retention [6-7].

The classic cause of BPH is hormones and relatives have a history of it, although modifiable risk variables include weight gain, high fasting blood sugar, cardiac illness, diabetes, dyslipidemia, and the metabolic syndrome. The condition is a common condition, with an incidence ranging from 4.5% to 9% [8].

Given its prevalence, the condition presents a challenge in medical treatment. This is partly because of the many meanings of Prostatitis is easily available. The National Institutes of Health (NIH) classifies four illnesses as prostatitis. Prostatitis pelvic pain syndrome (CP/CPPS) is classified in the second category and III by the national Institutes of and it is notoriously difficult to treat, with a recurrence rate of up to 50% [9, 10]. While serotonin (5-hydroxytryptamine, or 5-HT) was discovered 60 years ago [11], investigation into serotonin and its receptors continues to yield novel biological discoveries with medicinal implications in almost all major organ systems, including the cardiovascular, pulmonary, gastrointestinal (GI), and genitourinary systems, as well as the central nervous system (CNS) [12].

Neuroendocrine prostatic glands generate a range of neuroendocrine chemicals, the least common among them being 5-HT. The unusual The architecture of certain neurons, with dendritic canals reaching into the interior with projecting encircling the epithelial-stroma The user interface gives support to the notion that neuroendocrine factors, such as 5-HT, may influence prostate growth [13]. Notably, hormonal prostate lymphocytes are mostly found in the transition zone of the healthy human prostate [14]. This is when BPH occurs [15] meanwhile evaluating BPH tissues to a healthy transition area (Without BPH), the number of neurons with hormones is far decreased [16-18]. Also, 5-HT was discovered to be significantly decreased in BPH cells [19]. Furthermore, a recent investigation in a large cohort of Swedish males revealed that LUTS is related to benign prostate development with decreased plasmatic 5-HT amounts [20].

These results imply a possible relationship involving prostatic 5-HT deficiency and BPH pathogenesis; however, the role of 5-HT in limiting benign prostate development has not been extensively investigated. The adrenal cortex produces the most abundant hormones in humans: DHEA and its sulphated version, DHEA-S. Primates, including humans, are unique among animal species, their adrenal glands release significant quantities [21]. DHEA and DHEAS have also been recognized as potent neuroactive substances [22]. Fernand Labrie coined the phrase "inside crinology," which describes the production and degradation of corticosteroids in distal targets [23, 24]. BPH tissues use enzymes 17β-hydroxysteroid phosphatase (17β-HSD), 3β-HSD, and 5α-reductase to regulate effective amounts of sex hormone [25]. The synthetic adrenal steroid DHEA is assumed to be an essential supplier of testosterone, and once processed the prostate tissues, it can produce as much as a sixteenth of the DHT present in the prostate [26].

Materials and Methods

Sample collection: From February 2022 to October 2023, sera were collected from 50 patients with benign prostatic hyperplasia and 50 patients with prostatitis, as well as 45 healthy controls. From a hospital. The patients' ages varied from 32 to 85, with an average of 73.09±8.38 years, and a medical history spanning 1 to 11 years. Five individuals experienced acute urine retention. The sample was transported to the laboratory to be separated into serum and plasma using a separator. It was then placed in a deep freeze to investigate the impact of the parameters on the samples.

Determination of biochemical per blood serum: Serum hormone serotonin activity has been determined by using Kit assayed according to the manufactured Procedure (Bioassay technology Laboratory, Cat.NO EA0028Hu, China). Serum dehydroepiandrosterone activity was tested using a kit (Fine Test Laboratory, Cat. No. EH4005, China) according to the manufacturer's instructions.

Statistical method: The biochemical data were statistically evaluated using the statistical software tool SPSS17.0. The mean standard deviation (SD) was estimated using ANOVA, and statistical significance was regarded whenever the P value was equal to or less than $p<0.05$.

Results

Biochemical Investigation: This study showed in table (1) concentration dehydroepiandrosterone in table (14) prostate enlargement patients decrease a significant (236.2±50) while increased in prostatitis patients (313±54.1) compared with control (160.44±52.46). And this study showed in table (1) concentration hormone serotonin a significant decrease (27.23±5.84) patient prostate enlargement and prostatitis patient (25.33±5.02) compared with control (44.72±6.32).

Table 1: Serum concentration of, dehydroepiandrosterone, in the studied groups

Parameters	Prostate enlargement	Prostatitis	Control	p value
Dehydroepiandrosterone (ng/ml)	236.2±50	313±54.1	160.44±52.46	0.0005
hormone serotonin (µg/dL)	27.23±5.84	25.33±5.02	44.72±6.32	0.0008

Data expressed as mean ± SD, *^ indicate significant differences at $p<0.05$, *as compared to either control or prostate enlargement, ^ as compared to control group.

In table (2) a significant increase (277.80±41.90) dehydroepiandrosterone prostatitis patients in age (30-45) compared with prostatitis patients in age (44.07±10.60), While in prostate enlargement patients in age (30-45) increase a significant (64.63±12.44) compared the age (46-85) (69.95±14.39) prostate enlargement patients.

in enlarged prostate patients. There is a significant increase (a) in patients group a obese bodies compared to prostate enlargement who are overweight (282.9±42.7a) and patients normal (261.9±24.1a), while prostatitis patients group b a significant decrease (237.8±70.5b) in over wait while increase the obese body (265.5±52b), and normal body (257.2±77.6b). No significant (254.2±53.2) in patients prostate enlargement smoking compared to not smoking (250±53.0). But decrease a significant (255±57.7) in patients prostatitis smoking compared to patients prostatitis not smoking (387.2±43.1). In patients prostate enlargement with diseases increase a significant (259.1±65.4) compared with diseases patients (282.7±51.8) while a significant decrease (300.4±50) in patients prostatitis with diseases compared without diseases (304.6±77.5). In prostate enlargement, increase -significant in rural areas compared in locations rural (318.9±81.2) compared with patients in urban (321.8±76.7) and increase in prostatitis patients significant (232.4±86.7) urban compared with prostatitis patients (232.4±86.7) rural. In prostate enlargement and

prostatitis, patients without genetic link no a significant (207.7±58.1) (209±57.3). Compared without genetic factor increase significant (302±63.1), (405.2±64.7) respectively. Hypertension has non-significantly changed the level of dehydroepiandrosterone in either group of prostate enlargement or prostatitis, compered no hypertension decreased a significant (185.9±43.1) prostatitis patients. Hyperglycemia has significantly elevated serum of dehydroepiandrosterone levels in prostate enlargement increase a significant (261.6±83.0) compared with no hyperglycemia (241.3±47.90) while in patients prostatitis hyperglycemia decrease a significant (105.8±61.96) compered no hyperglycemia (105.8±61.96).

Table 2: Serum concentration of dehydroepiandrosterone relative to their demographic parameters in patient groups

Demographic Parameters		Prostatitis	Prostate enlargement
Age (years)	30 – 45	440.1±83.3	383.9±68.3
	46-85	185.2±42.5	277.4±94.7
BMI	Normal	257.2±77.6 b	261.9±24.1a
	Over Weight	237.8±70.5 b	282.9±42.7a
	Obese	265.5±52 b	237.8±65.1a
Smoking status	Positive	254.2±53.2	255±57.7
	Negative	250.0±53	387.2±43.1
Diseases status	Positive	300.4±50	282.7±51.8
	Negative	304.6±77.5	259.1±65.4
Geography	Rural	260.7±70.4	318.9±81.2
	Urban	232.4±86.7	321.8±76.7
Genetic status	Positive	209.0±57.3	207.7±58.1
	Negative	405.2±64.7	302±63.1
Hypertension	Positive	254.7±68.9	204.2±63
	Negative	185.9±43.1	198.7±64.65
Hyperglycemia	Positive	105.8±61.96	261.6±83
	Negative	389.5±77.8	241.3±47.9
Therapy Current	Urimax capsule	--------	244.3±78.3
	Xradal tablet	----------	253.3±79.9
	Prostacalm capsule	289.4±87.5	----------
	Prostanil tablets	205±79.23	----------

Data expressed as mean ± SD, different letter indicate significant differences at $p<0.05$ within the same group, * Significant differences ($p<0.05$) between the two groups, prostanil=Finasteride, Prostcalm=a combination of natural plant extracts with organic and mineral antioxidants, Xradal =alfuzosin, Urimax= tamsulosin

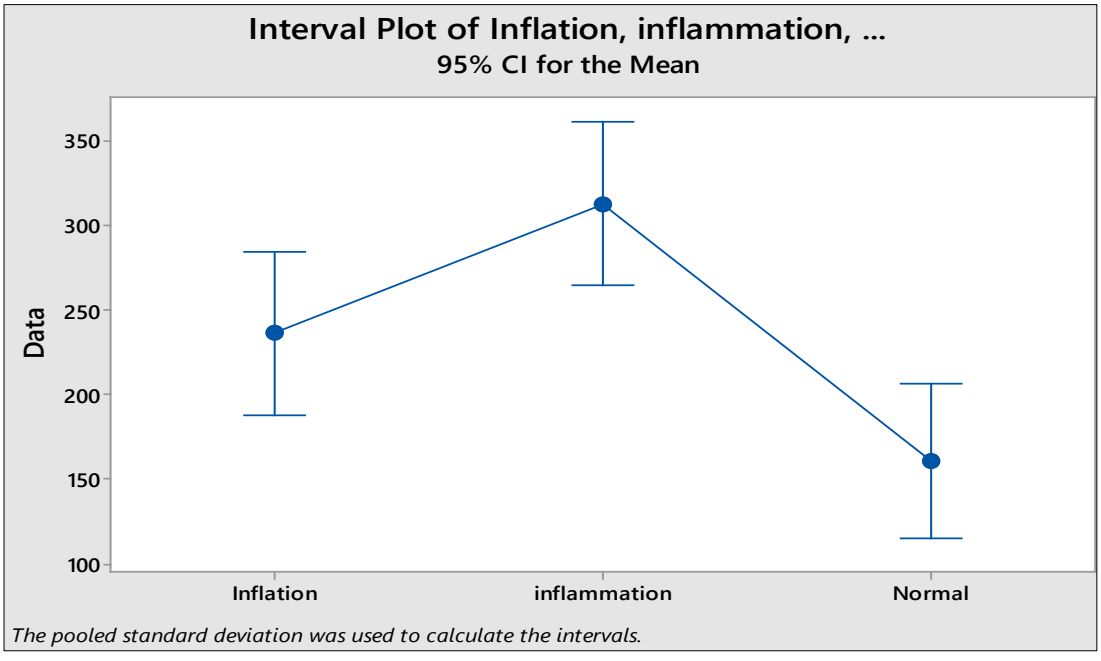

Fig 1: Concentration of dehydroepiandrosterone in patient compered normal

Table 3: Serum concentration of hormone serotonin relative to their demographic parameters in patient groups

Demographic Parameters		Prostatitis	Prostate enlargement
Age (years)	30 - 45	23.47±4.7	23.14±3.08
	46-85	30.62±5	22.67±3.38
BMI	Normal	16.74±2.77 b	25.59±4.27 a
	Over Weight	24. 2±25.75	04.3.±26.35
	Obese	18±3.33 b	42.6±7.30 a

Smoking status	Positive	18.73±3.02b	30.8±4.37
	Negative	27.53±6.86 b	21.99±4.30
Diseases status	Positive	24.88±4.85	23.23±2.98
	Negative	20.97±3.8	20.53±1.73
Geography	Rural	34.87±19.09	28.55±13.08
	Urban	25.76±11.53	27.15±17.18
Genetic status	Positive	17.75±4.77	29.58±4.92
	Negative	3.32±26.38	19.99±4.59
Hypertension	Positive	26.29±3.13	21.55±2.95
	Negative	44.31±7.45	23.32±3.02
Hyperglycemia	Positive	25.83±48.41	16.27±14.46
	Negative	28±42.90	27. 93±5.54
Therapy Current	Urimax capsule	------	29.99±5.72
	Xradal tablet	-------	26.36±2.84
	Prostacalm capsule	23.93±3.53	--------
	Prostanil tablets	30.67±22.59	--------

Data expressed as mean ± SD, different letter indicate significant differences at $p<0.05$ within the same group, * Significant differences ($p<0.05$) between the two groups. prostanil=Finasteride, Prostcalm=a combination of natural plant extracts with organic and mineral antioxidants, Xradal =alfuzosin, Urimax= tamsulosin

The effect of hormone serotonin in patient prostate enlargement no a significant in age (30-45) compered in age and (46-85), (22.67±3.38) (23.14±3.08), while in prostatitis patient the result showed deceased a significant (23.47±4.7) in age (30-45) compered (30.62±0.1) in age (46-85) on the body mass of enlarged prostate patients. There is a significant increase (42.60±7.30a) in patients group a obese bodies compared to prostate enlargement who are overweight (26.35±3.04a) and patients normal (25.59±4.27a), while prostatitis patients group b a significant increase (25.75±5.14b) in over wait compered the obese body (18±3.33 b), and normal body (16.74±2.77b) in patients prostate enlargement with genetic factor no a significant (29.58±4.92) compared patients without genetic factor (19.99±4.59), while increase significant (17.75±4.77) in prostatitis patients without genetic factor compered patients without genetic factor (26.38±3.32) no significant (30.8±4.37) in patients prostate enlargement smoking compared to not smoking (21.99±4.3). But increase a significant (27.53±6.86) in patients prostatitis not smoking compared to patients prostatitis smoking (18.73±3.02) in geographical between rural and urban we noticed increase significant (27.15±17.18) in patients prostate enlargement in urban compared with patients in rural (28.55±13.08) and increase significant (34.87±19.09) of prostatitis patients in rural compared with prostatitis patients (25.76±11.53) urban patients prostate enlargement no a significant (23.23±2.98) compared without diseases patients (20.53±1.73) While a significant increase (24.88±4.85) in patients prostatitis with diseases compared without diseases (20.97±3.80). in patients prostate enlargement with high blood pressure no a significant (21.55±2.95) compered without blood pressure(23.32±3.02) in patients prostatitis without high blood pressure increased a significant (44.31±7.45) compared with high blood pressure (3.13±26.29) in patients prostate enlargement with high blood sugar increase a significant (16.27±14.46) compered without blood sugar (27. 93±5.54). in patients prostatitis with high blood sugar increase a significant (25.83±48.41) compared without blood sugar (28±42.9) in patients prostate enlargement who use kind drag urimxe increased a significant (29.99±5.72) compered who use kind drag xradal (2.84±26.36), while in table (3) in patients prostatitis who use kind drag prostalin showed increase in a significant (30.67±22.59) compered patients who use prost calm (3.53±23.93).

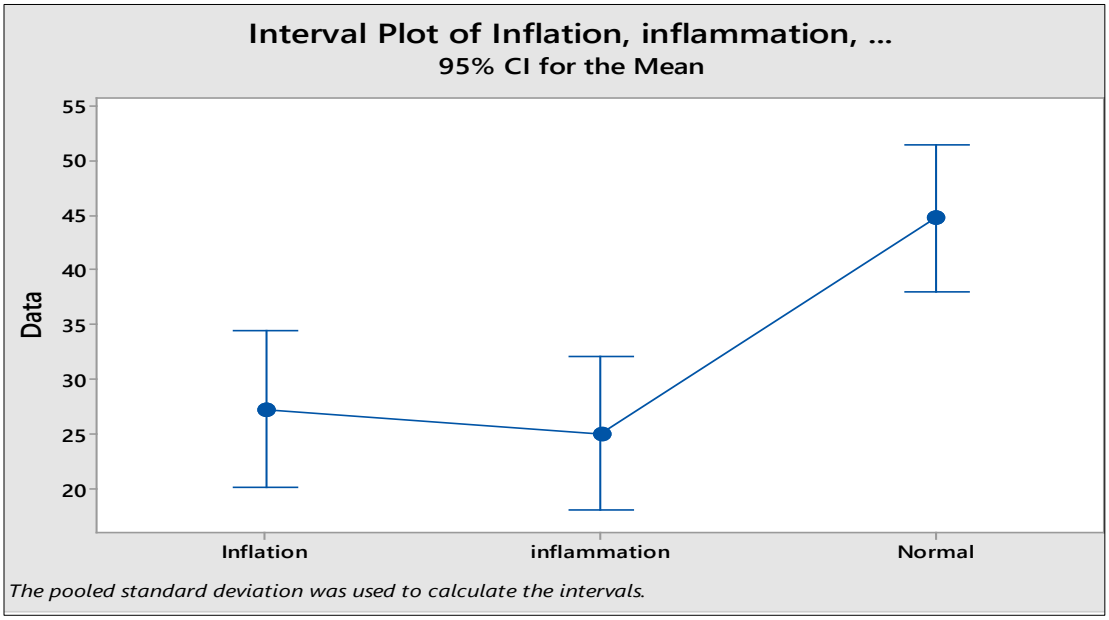

Fig 2: Concentration of hormone serotonin in patient compered normal

Discussion

Neuroendocrine prostatic cells release a variety of neuroendocrine substances, among which 5-HT is the most prevalent. The unusual architecture of certain The presence of Circular pathways reaching in the lumen with terminals around the epithelial-stroma junction in synapses support the notion that endocrine outputs such as 5-HT may influence prostate growth [27]. Hormonal prostatic neurons are mostly found in the changing zone of the natural human prostate [29], where BPH develops [28]. When comparing BPH tissue to the normal transitional region (without BPH), the quantity of neurons in the endocrine system significantly reduced [16, 30, 31]. The results revealed that the hormone serotonin no a significant in age in the age group of 46-85 and younger age group in patients with prostate enlargement, this implies that the hormone does not grow with age, but in patients prostatitis increased hormone in age 46-85 compared to the younger age group.

We discovered that patients with prostatitis and enlargement have higher levels of serotonin than healthy persons, which is consistent with the findings of researchers Emanuel Carvalho-Dias and Alice Miranda [32]. People with prostatitis and prostate enlargement had higher levels of the hormone serotonin compared to normal and obese people. Hormone concentrations were not significantly Smoking individuals who had prostate enlargement differed from non-smokers, demonstrating that prostate enlargement is connected with smoking, earlier observed. Patients with prostatitis are less prone to smoke than smokers. Some researchers demonstrate a moderate unfavorable relationship among tobacco and BPH, either diagnosed by symptoms or through clinical study [33-37].

Some studies even suggest that smoking has a preventive effect on BPH [38]. Others suggest Nicotine exposure accelerates the progression of BPH. Once more, many physicians hold that the impact of tobacco use on BPH depends on how much was consumed [39, 40]. Condition status results revealed that the hormone serotonin rose in people with prostatitis and prostate enlargement compared to those without the condition. The geographical location plays a part in the increase in the concentration of the hormone serotonin in prostate growth patients who reside in cities have higher hormone concentrations than those who live in rural areas, while the converse is true for prostate patients. The genetic factor has an influence on the hormone serotonin prostate enlargement, no a significant difference between who do not have a hereditary family history and who have a family history, but in patients prostatitis, increased in the family hereditary factor compared to do not have a hereditary family history. This suggests that the hereditary component does not impact hormone concentration in people with an enlarged prostate. Hypertension in patients with prostate enlargement, increased serotonin concentrations without blood pressure compared to those with blood pressure, but in patients prostatitis without blood pressure increased hormone serotonin, of the hormone in patients who suffer from blood pressure compared to those who do not. Individual metS components such as type 2 diabetes and hypertension were investigated [41].

Hyperglycemia in patients with prostate enlargement and patients with prostatitis increases hormone in comparison to those without hyperglycemia. Diabetes is frequently associated with low PSA levels; previous research suggests a link between metS and BPH [42]. Hyperglycemia is linked to reduced parasympathetic activation through neuronal death [43]. A disparity between sympathetic and parasympathetic activity can result to urinary head obstruction with reduced uro strength. The Rho kinase system is required for prostate shrinkage [44]. Androids are essential for the prostate's correct creation, growth, and function [45]. The cortex of the adrenal gland produces Dehydroepiandrosterone (DHEA) and its dissolved counterpart, DHEA sulfate (DHEAS), are among the most common hormones found in humans [46, 47].

Previous studies have shown that DHEA could be linked to the occurrence of prostate cancer [48, 49]. Yet, the utility of blood concentrations of DHEA in prostate cancer monitoring is questionable. The results also indicated that the hormone serotonin increases in the age group of 46 to 85 in individuals with prostate enlargement compared to the young age range. In contrast to inflammatory individuals, testosterone levels rise increasing aging. Levels of hormones grow in younger age groups compared to the elderly. In obese and overweight people with prostate enlargement, the hormone levels rise while it falls for those with a typical weight. A BMI of more than 35 kg/m^2 was related with a 3.5-fold increased chance of developing an enlarged prostate capacity (>40 ml) [50]. Individuals having a BMI of less than 25 kg/m^2 were twice as many more likely to get LUTS [51].

Hormone concentrations in smokers with prostate enlargement were lower than in nonsmokers, showing that smoking caused prostate enlargement, as previously documented. In the presence of other diseases associated with prostate enlargement, there was no significant difference in the concentration of the hormone serotonin between smokers and nonsmokers; however, prostatitis patients had a higher concentration of the hormone than patients without other diseases. Individuals with a BMI < 25 kg/m^2 were 1.2 times more likely to develop LUTS [51]. Hormone concentrations in smokers with prostate enlargement were lower than in nonsmokers, indicating that smoking causes prostate enlargement, as previously reported. In the presence of other disorders linked with prostate enlargement, there was no significant difference in the concentration of the hormone serotonin between smokers and nonsmokers; however, individuals with prostatitis had a greater concentration of the hormone than those without the condition. Hypertension in individuals with prostate enlargement and prostatitis raises concentrations without blood pressure compared to those with blood pressure.

We found that patients with prostatitis had a greater concentration. Hyperglycemia in patients with prostate enlargement and patients with prostatitis increases hormone in comparison to those without hyperglycemia. People with extra visceral fat are more likely to develop diabetes, which might be a separate risk component for BPH [52-53]. As a result, obese, dyslipidemia, especially elderly men are at risk for MetS, which contains features that are strong predictors of prostate enlargement [41, 54]. Further diseases associated with belly fat involve heart failure, obstructive sleep apnea, and nonalcoholic fatty liver disease [55]. Marberger *et al*. discovered that high amounts the presence of 5α-reductase as well as Dihydro in the prostatic can lead to the development and progression of BPH, even with low systemic T values [56]. This medication has been effective in BPH people across all blood concentrations of T [57, 58].

Conversely, a year-long study of T treatment in hypogonadal males revealed no substantial rise in PV.

Conclusion

These findings link hormone serotonin and dehydroepiandrosterone to human prostate pathology, and this model provides a diverse platform for investigating the molecular underpinnings of inflammation-related prostate disorders linked with episodic or chronic hormone serotonin and dehydroepiandrosterone levels. In BPH, inflammation may be taken into consideration in the best therapy selection because if discovered, patients should have a symptomatic favorable improvement by adding anti-inflammatory drugs to routine treatments., We detected a rise in the hormone serotonin, in patients with prostatitis and enlargement compared to the control, whereas we observed a reduction in dehydroepiandrosterone. The purpose of this study was to investigate the function of dehydroepiandrosterone with the management of moderate prostatic expansion, and also to assess the pharmacological regulation by the urinary serotoninergic pathway as a potential treatment for BPH.

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