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Thyroid hormone metabolism in renal disease

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Abstract

Introduction and research aim: Hyperthyroidism or hypothyroidism may play different important and regulatory roles in multiple organs of the body. The absence or excess of the TH hormone has harmful effects on all tissues of the body, so thyroid hormone (TH) is necessary for kidney function and development. The aim of the research: to evaluate the function of the thyroid gland and its relationship to kidney disease and to know its relationship to the severity of the disease.

Background: The hormone responsible for triggering the secretion of triiodothyronine (3T) is known as thyroid stimulating hormone. It has a significant function in regulating the body's metabolism, which encompasses several mechanisms that govern the pace of activity in cells and tissues. This includes the production of 4T by the thyroid gland. Thyroid hormone (T_4) is one of the primary hormones released by the thyroid gland. Thyroid function is associated with alterations in renal function. Thyroid function indicators might differ depending on age, chronic illnesses, and genetic background. Hence, there exists a connection between thyroid function and reduced kidney function that can be attributed to both direct and indirect factors. Hormone receptors selectively bind to molecules that possess certain conformations and functional groups, hence only responding to the hormones that exhibit such characteristics. Cells from various tissues in the body may possess identical types of receptors. The levels of FT_3 steadily declined, resulting in heightened kidney injury and the presence of albumin in the urine. Despite having regular thyroid function.

Methods: A group of solid methodological studies, published between the years 2017 to 2023, collected the relationship between thyroid hormones and natural and chronic kidney disease.

Results: Chronic kidney failure may be a condition resulting from hypothyroidism. It has become clear that iodine clearance takes place in the glomerular filtration in the kidneys, which is the cause of chronic kidney failure. Iodine secretion diminishes, leading to a rise in the plasma concentration of inorganic iodide and thus its absorption. A high concentration of inorganic iodide within the body may inhibit the formation of thyroid hormones. Iodine is filtered out in the glomerular filtration in the kidneys, which is the cause of kidney failure. Also, there was a difference in the severity of the infection between males and females with diabetes, and there was no difference between those without diabetes. People with diabetes with kidney tissue damage experienced an abnormal change in TSH secretions, and there was a direct relationship with the age of those affected, genetic history, and infections of the immune system.

Discussion: FT_3 and FT_4 exhibit a negative correlation with SCr and ACR, while showing a positive correlation with eGFR. Conversely, TSH showed a negative correlation with eGFR and a positive correlation with ACR. The cause is a disparity in the iodine concentration within thyroid secretions. Conversely, hypothyroidism can be associated with constriction of the blood arteries in the kidneys and a reduction in efficient blood flow to the kidneys, leading to kidney injury and the development of microalbuminuria.

Conclusion: Increased TSH was associated with a decrease in glomerular filtration rate (eGFR), especially in the reference range. The direct effect of increasing fT_4 was a decrease in glomerular filtration rate (eGFR). There are significant differences between males and females in nephropathy due to increased TSH, while there are statistically significant differences in males, less than females, in cases of type 2 diabetes. It has also been shown that chronic nephropathy (CKD) and increased severity of hypothyroidism with gradual decline result from glomerular filtration rate (eGFR). On the other hand, patients may have hypothyroidism, which has led to a significant decrease in the number of white blood cells and hemoglobin levels, with a quarter of TSH increasing. In addition, this study demonstrated that kidney function in older individuals who had a low normal FT_3 level and a high normal TSH level was impaired. It has also been shown that the lack of iodine secretion leads to an increase in the plasma concentration of inorganic iodide, and thus FT_3 levels gradually decrease, causing an increase in the severity of kidney damage and albuminuria. Even with normal thyroid function.

Keywords: Hypothyroidism, eGFR, T_4, T_3, nephropathy, CKD

Introduction

End-stage renal disease (ESRD) is relatively common and co-occurring with thyroid disordersIt leads to high rates of morbidity and mortality in many countries of the world, including the United States, for example 230, 000 people receive dialysis annually, with an

annual increase of 8-9% in frequency over the last ten years. It has been found that people over the age of 64 years are most susceptible to kidney disease, and here thyroid hormone metabolism changes often, in addition to a change in metabolism, and endocrine disorders resulting from end-stage renal disease, which often kills these people. Patients with a large number of hypothyroidisms, diabetes, infections, and malnutrition lead to disturbances in thyroxine secretions, leading to kidney disease. Moreover, thyroid diseases may occur due to this conflict between kidney functions and the metabolism of thyroid hormones, including: These include goiter, hypothyroidism, thyroid nodules, and thyroid cancer (Beatriz *et al*., 2022) [1].

Hyperthyroidism and hypothyroidism have distinct and significant regulatory functions in several organs of the body. Both the deficiency and overabundance of the TH hormone have detrimental impacts on all bodily tissues. Thyroid hormone (TH) is essential for the functioning and growth of the kidneys. It is well-established that hormone levels in the bloodstream regulate the equilibrium of water and sodium. As for kidney tissue, hyperthyroidism leads to an increase in the rate of glomerular filtration and tubular reabsorption, but at the same time it reduces the concentration of creatinine in the serum (Zhongyu *et al*., 2023) [17], and the effect is opposite when hypothyroidism occurs as well, as disorders affect the Kidney function also results in an increase in secretions of thyroid hormones and metabolism. Patients with nephrotic syndrome experience significant protein loss, primarily of thyroxine binding globulin (TBG) and albumin. Albumin directly binds to TH, resulting in a reduction in the overall concentration of thyroid hormone (T_4) in the bloodstream (Zhao *et al*., 2018) [16].

The thyroid gland regulates most of the body's physiological functions. The kidneys and thyroid are physiologically and functionally related to each other. It is now known that chronic kidney disease (CKD) alters the hypothalamic-pituitary-thyroid axis along with the metabolism of peripheral thyroid hormones. Many researchers have noticed an imbalance in the thyroid gland, such as hypothyroidism, hyperthyroidism, and the state of the thyroid gland, in patients with kidney failure, specifically, more than others. This is because thyroid hormones have a very crucial role in regulating the metabolism process, and the synthesis or regulation of proteins, salts, and growth. And influencing the functions of various other hormones in the human body in general. At the same time, the kidneys play a fundamental and major role in thyroid hormone metabolism by converting thyroid hormone (T_4) into triiodothyronine (T_3). In patients with chronic renal failure, abnormal thyroid function is frequently observed (Chen *et al*., 2020) [3].

Background

Regulation of thyroid hormone synthesis

The thyroid stimulating hormone that causes the release of 3T is triiodothyronine. It plays an important role in the body's control of the metabolism process (The many processes that control the rate of activity in cells and tissues), as for T_4, which is produced by the thyroid gland. Thyroid hormone (T_4) is one of the two main hormones secreted by the thyroid gland. The T_4 hormone contains four molecules of iodine and is produced by the thyroid gland in much larger quantities than T_3. T_4 plays a role in vital body functions and therefore must be monitored. T_4 analysis test is used alone or in combination with T_3 and TSH to identify thyroid disorders. The total T_4 test measures free and bound T_4 levels in the blood, which helps diagnose thyroid disorders. (Yoko *et al*., 2021) [14].

The pituitary gland, sometimes referred to as the hypophysis, exerts significant influence on physiological processes by modulating the responses of target cells to stimulus via the secretion of hormones. The pituitary gland releases thyroid-stimulating hormone, which is contingent upon the iodine level in the body. There exist two categories of hormones: those that are derived from amino acids and those that are formed from fatty acids. Modified amino acids refer to hormones that are derived from amino acids. The amino acid's initial structure is altered by removing the COOH−, NH+, or carboxyl group. The compounds mentioned are carboxyl and the amino acids tryptophan or tyrosine. Melatonin, a hormone derived from tryptophan, is secreted by the pineal gland and plays a crucial role in regulating the circadian rhythm. Thyroid hormones, which are derived from tyrosine, serve as an illustration of tyrosine derivatives and play a crucial role in regulating metabolism. These responses play a role in the reproduction, growth, and development of bodily tissues, as well as in metabolism, fluid balance, and homeostasis (You *et al*., 2019) [15].

The hormone receptor, a protein situated either intracellularly or on the cell membrane, receives the signal transmitted by the hormone. The receptor will facilitate the processing of the message by initiating subsequent signaling events or cellular mechanisms that elicit a response in the target cell. Hormone receptors selectively identify molecules based on their unique conformations and functional groups, and exclusively react to hormones that are identified by them. Cells from various body tissues may possess identical receptor types (Kalantar *et al*., 2019) [6], resulting in somewhat varied reactions. Consequently, the reaction triggered by hormones is contingent not only upon the hormone itself, but also upon the specific target cell. Upon receiving the hormone signal (Figure 1), a target cell has the ability to respond through many mechanisms. The reaction may involve the stimulation of protein synthesis, inhibition or activation of deactivation, modification of cell membrane permeability, and alteration of enzyme activity.

Changing cell growth and mitosis of altered rates, and stimulating secretion, stimulation of the secretion of products, thyroid hormones Thyroid hormones, which contain benzene rings studded with iodine, are soluble-lipid and therefore can enter the cell. From this we conclude that protein synthesis in the body’s tissues in general depends, in one way or another, on the presence of thyroid hormones in the blood, as a high level of this hormone will certainly affect the functioning of the kidneys, negatively or positively. (Cystatin, 2021) [4].

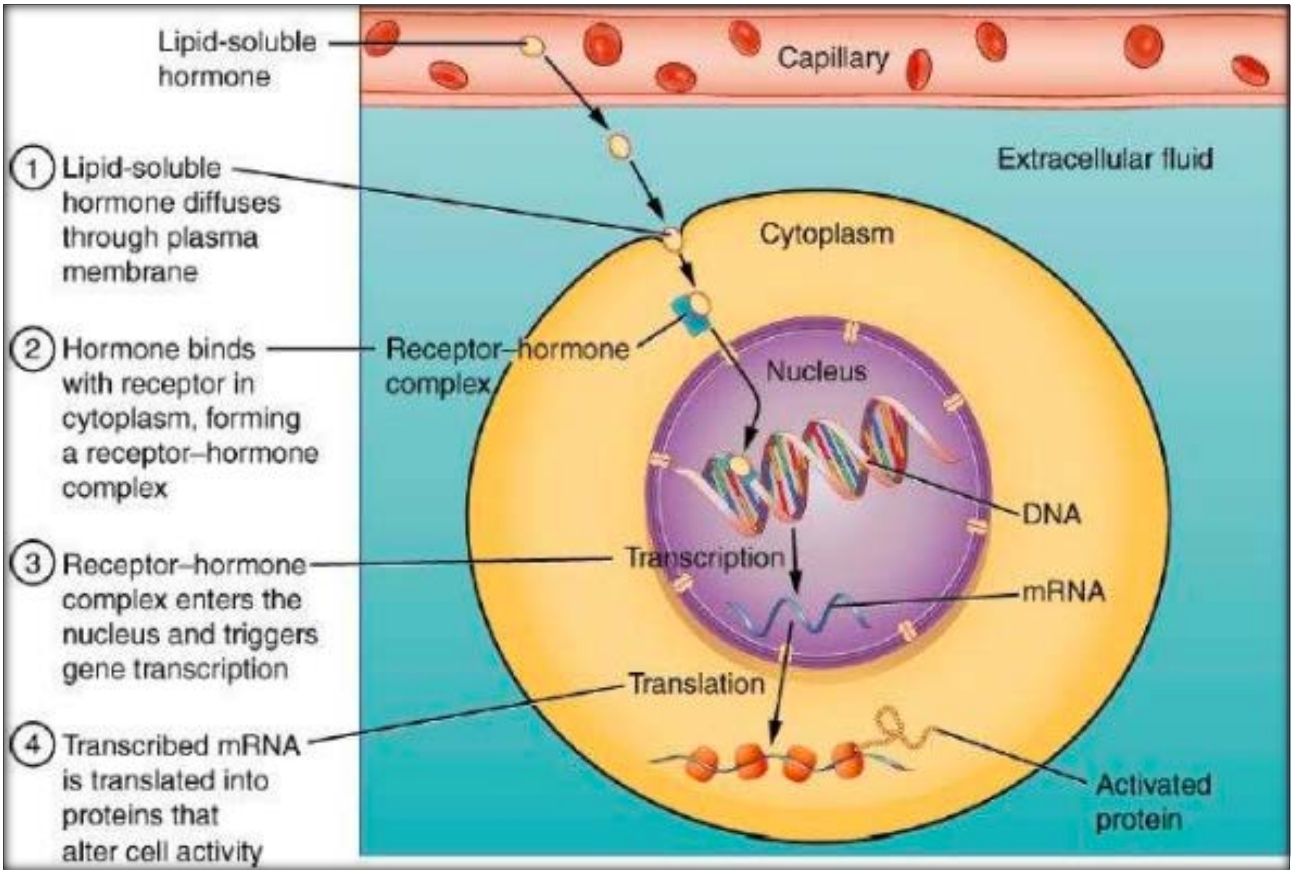


Fig 1: Thyroid hormones bind to receptors. All hormones derived from amino acids bind to cell membrane receptors with the exception of thyroid hormones, which are fat-soluble., (You *et al*., 2019) [15].

Chronic Kidney Failure: Research has substantiated that chronic kidney disease is characterized by an estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73 m2 and/or indications of renal impairment persisting for a minimum of three months. In clinical practice, testing commonly encompass the most prevalent conditions. In the case of chronic kidney disease, the glomerular filtration rate (eGFR) was determined by estimating the serum creatinine concentration using the CKD-EPI equation (CKD Epidemiology Collaboration). The albuminuria was assessed by measuring the urinary albumin-to-creatinine ratio (ACR) according to Kramer *et al*. (2018) [7].

The study conducted by Whelton *et al*. (2017) [13] found that the therapy of chronic kidney disease involves mitigating the patient's susceptibility to developing CKD and the associated consequences, including cardiovascular disease, acute kidney injury (AKI), anemia resulting from CKD, and metabolic acidosis induced by CKD. Chronic kidney disease, initially caused by excessive thyroxine hormone secretion, as well as mineral and bone abnormalities, originating from a thyroid secretion abnormality.

Estimated glomerular filtration rate in the kidney

According to a study conducted by Bullock *et al*. in 2017 [2], it is established that in order to prevent the onset of chronic kidney disease, it is necessary to set an individual blood pressure target of less than 130/90 mm Hg or higher, and to consider the use of angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB). Indicated for those with albuminuria, hypertension, hemoglobin A1c levels greater than or equal to 7%, diabetes, and a need for medical nutrition therapy. Figure (2) illustrates the connection between metabolic processes in the body and the protein levels in the blood serum, which is influenced by the secretion of hormones from the pituitary gland. This, in turn, causes stress on the kidney and accelerates the deterioration of renal tissue in individuals with chronic nephropathy. In order to mitigate the potential hazards associated with drugs, When prescribing medication, it is important to take into account the predicted glomerular filtration rate. In general, it is advisable to avoid nephrotoxic substances, such as prolonged use of non-steroidal anti-inflammatory medicines (NSAIDs) (Shobhit *et al*., 2023) [1].

The relationship of the thyroid gland to kidney functions

Prior research, conducted both domestically and globally, has demonstrated a correlation between thyroid function and alterations in kidney function. Thyroid function indicators might differ depending on age, chronic illnesses, and genetic background. Hence, the association between thyroid function and reduced kidney function may manifest as both a direct and an indirect connection. For instance, kidney function may vary between aged individuals and younger persons. The reference is from Sequist *et al*. in 2018 [10]. Multiple studies have demonstrated a correlation between thyroid dysfunction and estimated glomerular filtration rate (eGFR), as well as the risk of developing chronic kidney disease in individuals with chronic illnesses and the elderly. However, it remains unclear which specific indicator of thyroid function is more strongly linked to kidney function and this association. There is a dispute or disagreement between them. For instance,. According to a study, it was found that free thyroid hormone (FT_4) has a beneficial impact on the glomerular filtration rate in individuals who are affected. This effect is directly linked to the age of the

affected person. However, another study revealed that high levels of FT_4 are connected to a higher risk of developing chronic kidney disease in affected individuals. Interestingly, this risk is not influenced by age. It is important to note that there is currently no available data investigating the relationship between normal thyroid function and age-related kidney function in older adults in general (Huang *et al*., 2020) [5].

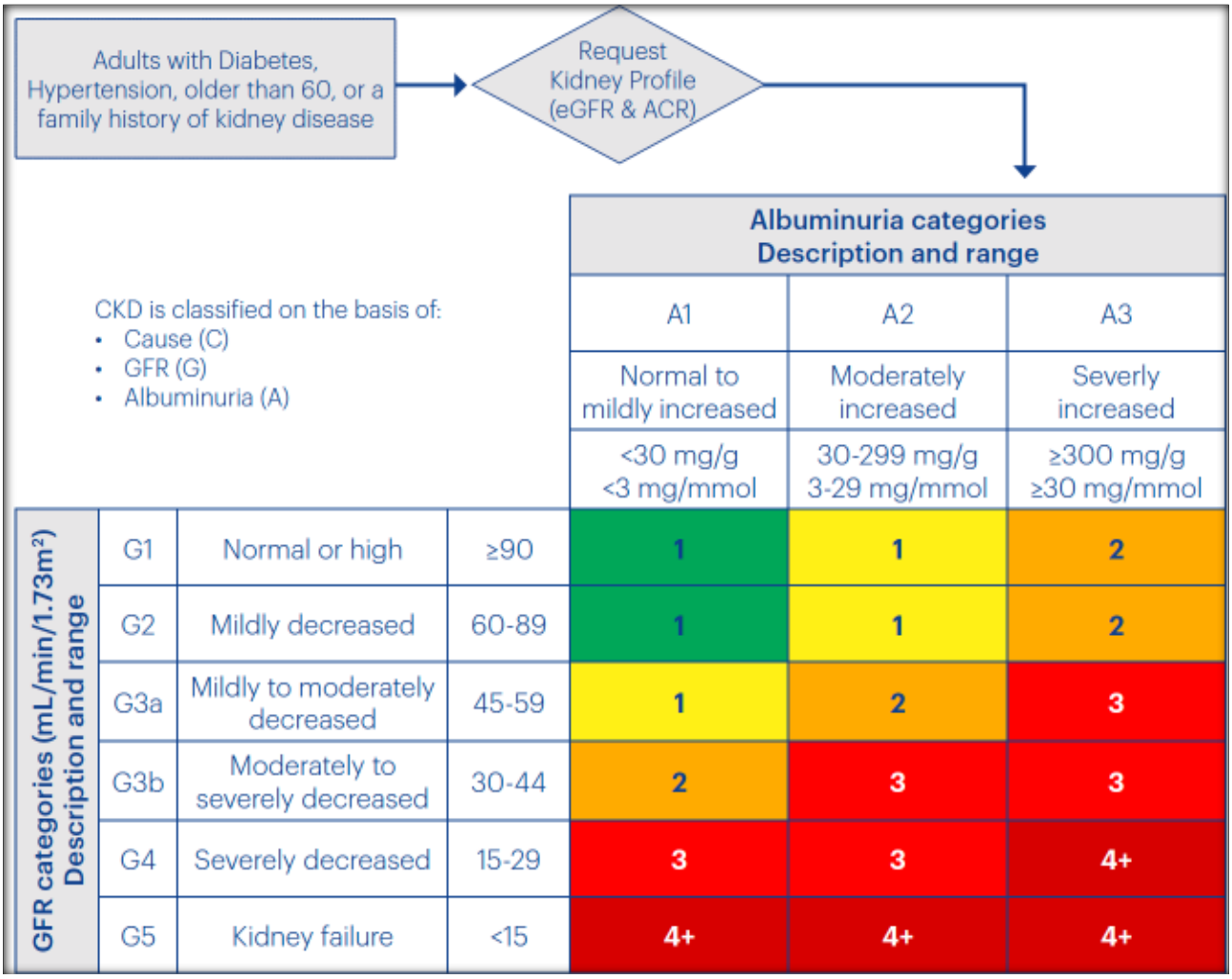


Fig 2: The relationship of kidney failure to thyroid activity and glomerular filtration, and the indirect link between the kidney and thyroxine secretions. (Beatriz *et al*., 2022) [1]

Methods: We relied on a group of rigorous methodological studies and research papers from the Google Surge and Resage Kit platform, published between the years 2017 to 2023, which collected the relationship between thyroid hormones (FT_4) and (FT_3) and normal and chronic nephropathy (CKD), and includes a study Three axes: the first: regulation of thyroid hormone synthesis: the second axis: estimated glomerular filtration rate in the kidney (eGFR): and kidney failure; and the third axis: the relationship of the thyroid gland to kidney functions.

Results

Chronic kidney failure can arise as a consequence of hypothyroidism. There is a significant occurrence of subclinical hypothyroidism (SCH) and clinical hypothyroidism in patients with chronic kidney disease (CKD) in this study. The seriousness of hypothyroidism escalates when the glomerular filtration rate (eGFR) gradually decreases. The occurrence of hypothyroidism in patients with chronic kidney disease (CKD) might be attributed to various factors apart than the presence of thyroid antibodies. The study conducted by (Shobhit *et al*. in 2023) [1] several researchers have reported the prevalence of hypothyroidism to range from 13% in early stages of kidney failure to 70% in patients with end-stage renal disease (ESRD). Uremia is one important link between thyroid dysfunction and chronic kidney disease. Thyroid disease, and treated cases have a lower chance of developing renal dysfunction. Kidney dysfunction with variable values is specific to thyroid hormones. According to the latest studies, it has been found that there are important links between kidney disease and thyroid function. This knowledge is important because it anticipates a connection between two separate entities. (Cystatin, 2021) [4].

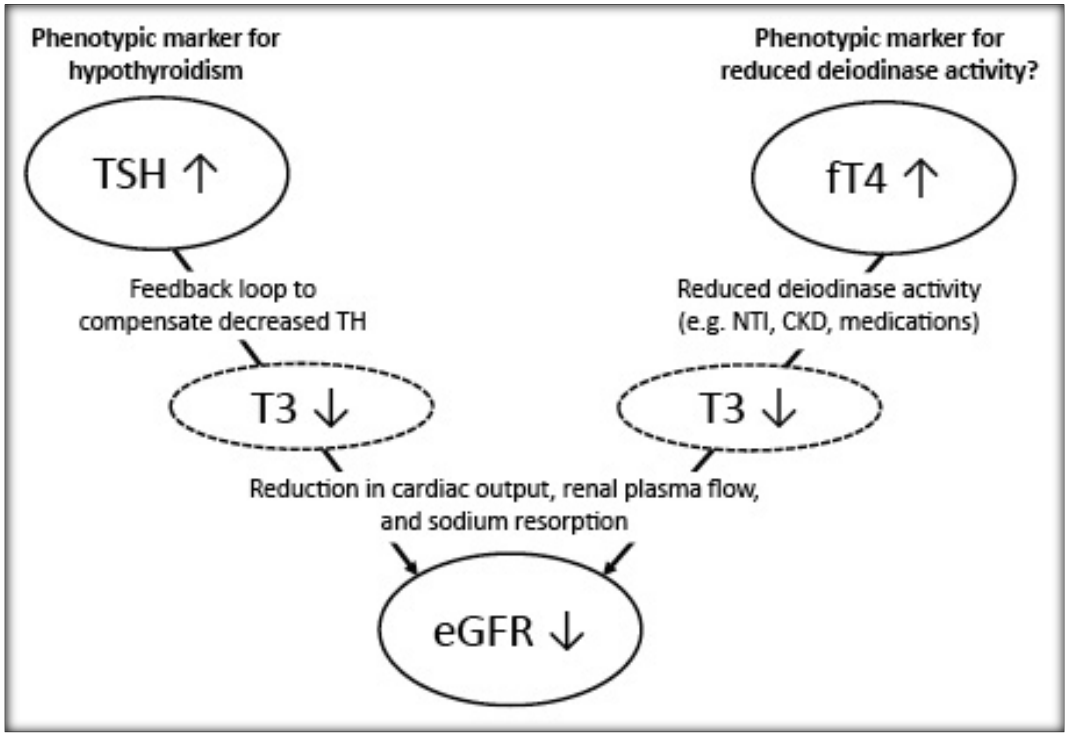


Fig 3: The hypothesis of the link between thyroid function on the one hand and kidney function on the other hand. Where TSH, thyroid hormone. fT_4, free thyroid hormone. Also T_3, triiodothyronine. Th, as the thyroid hormone. NTI, non-thyroid disease. Chronic kidney disease as well. eGFR represents the estimated glomerular filtration rate. (Chen *et al*., 2020) [3].

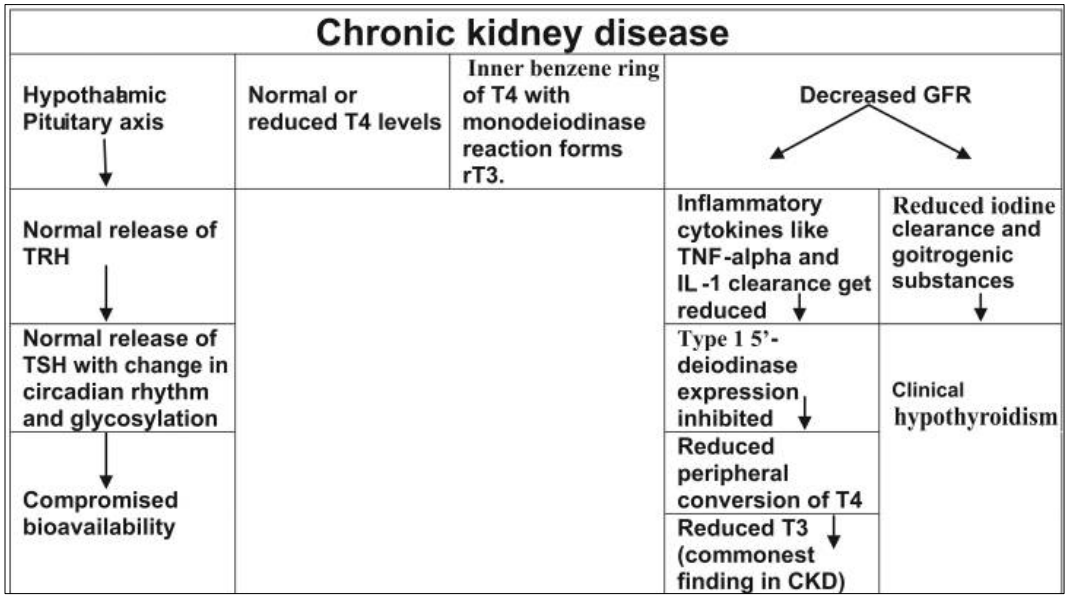
It has become clear that iodine clearance takes place in the glomerular filtration in the kidneys, which is the cause of chronic renal failure. Iodine secretion diminishes, leading to a high plasma concentration of inorganic iodide and thus its absorption. A high concentration of inorganic iodide within the body may lead to inhibition of the formation of glandular hormones. It significantly thyroids by disturbing the hypothalamic-pituitary-thyroid axis and peripheral metabolism of thyroid hormones (Wolf-Chaikov effect). Figure (3) This mechanism is responsible for hypothyroidism in patients with chronic kidney failure. Table (1) Thyroid disorders are also associated with glomerulonephritis most frequently via a common autoimmune cause (Wang *et al*., 2018) [12] and associations with thyroid and kidney malignancies have also been reported. Therefore, there is a common relationship between kidney failure and thyroid disorders. Low estimated glomerular filtration rate (eGFR) in these patients increases the likelihood of developing hypothyroidism.

Table 1: This prominent inverse relationship was also found in the subgroup analysis, which showed that statistical significance between TSH and eGFR was only present in the non-hypothyroid groups. (Sequist *et al*., 2018) [10].

Subgroup	No. of people Adjusted	Beta (95%CI, *P*)	Outcome: eGFR mL/min/1.73²
Age	<43(*n* = 2,677)	−1.2 (−2.0 to −0.4, 0.005)	
	≥ 43 (*n* = 2,901)	−1.9 (−2.7 to −1.1, <0.001)	
Sex	Male (*n* = 2,761)	−1.8 (−2.6to −1.0, <0.001)	
	Female (*n* = 2,817)	−1.7 (−2.3 to −1.0, <0.001)	
TSH	< 4mmol/L (*n* = 4,744)	−2.1 (−2.8 to −1.4, <0.001)	
	≥ 4mmol/L (*n* = 834)	−1.9 (−4.9 to1.1, 0.222)	
eGFR	< 99mL/min/1.73² (*n* = 2,793)	−1.4 (−2.1 to −0.7, <0.001)	
	≥ 99mL/min/1.73² (*n* = 2,785)	−0.8 (−1.2 to −0.4, <0.001)	
Anti-TPO Abs	Negative (*n* = 5,211)	−1.8 (−2.3 to −1.2, <0.001)	
	Positive (*n* = 367)	−1.9 (−3.6 to −0.2, 0.029)	
Urine iodine	< 281μg/L (*n* = 2,607)	−1.8 (−2.6 to −1.0, <0.001)	
	≥ 281μg/L (*n* = 2,613)	−1.8 (−2.5to −1.1, <0.001)	
WBC	< 6.1 ×10³/μL (*n* = 2,720)	−2.1 (−2.8 to −1.4, <0.001)	
	≥ 6.1 ×10³/μL (*n* = 2,856)	−1.5 (−2.2 to −0.7, <0.001)	

−5 −4 −3 −2 −1 0 1
Per 1 unit increase of log−TSH, mIU/L

Table 2: The relationship between thyroid dysfunction and kidney damage. (Sato *et al*., 2019) [9].



Analysis of the correlation between thyroid hormones and indicators of kidney function: It is noted that it was found (Table 3) that there was an inverse correlation between the levels of FT_3 and FT_4 with both SCr and ACR (r = −0.168, p<0.001 and r = −0.107, P = 0.023; r = −0.267, p<0.001 and r = −0.109, P = 0.021, respectively). Meanwhile, FT_3 and FT_4 levels were positively associated with eGFR (r = 0.325, p<0.001 and r = 0.165, p<0.001, respectively). In contrast, there was a negative correlation between TSH levels and eGFR (r = −0.128, P = 0.006), and a positive correlation between TSH and ACR levels (r = 0.104, P = 0.027).

Table 3: Analysis of the correlation between thyroid hormones and indicators of kidney function, (Cystatin, 2021) [4].

Parameters		*r* value	P value
FT_3 with	SCr	-0.168	<0.001
	eGFR	0.325	<0.001
	ACR	-0.267	<0.001
FT_4 with	SCr	-0.107	0.023
	eGFR	0. 165	<0.001
	ACR	-0.109	0.021
TSH with	SCr	0.043	0.359
	eGFR	-0.128	0.006
	ACR	0.104	0.027

Discussion

The findings of our study indicate a negative correlation between FT_3 and FT_4 levels with SCr and ACR, and a favorable correlation with eGFR. On the other hand, there was a negative correlation between TSH and eGFR, and a positive correlation between TSH and ACR. Table number two. The findings were in line with prior research (Sato *et al*., 2019) [9] and indicated a correlation between thyroid hormones and kidney function in individuals with and without diabetes. Due to the involvement of thyroid hormones in glucose metabolism, the occurrence of thyroid dysfunction is more common in diabetic patients compared to persons without diabetes. Individuals in good health. In our comprehensive investigation, we discovered that FT_3 levels exhibited a progressive decline as the severity of kidney injury and albuminuria increased. In the previous study, it was found that even when the thyroid is functioning normally, there was a positive correlation between FT_3 and eGFR, and a negative correlation between FT_3 and ACR. According to the KDIGO classification, Figure 3 shows that those at the highest risk of developing diabetic kidney disease (DKD) saw a rise in TSH levels and a drop in FT_3 levels. Prior research has shown that individuals with diabetes, particularly those with elevated TSH levels and reduced FT_3 levels as reported in Table (2), have a higher likelihood of developing diabetic kidney disease (DKD). Furthermore, there was a greater occurrence of subclinical hypothyroidism observed in both the DKD group and the non-DKD patients. A study conducted by Shobhit *et al*. (2023) [1] has identified subclinical hypothyroidism as an autonomous risk factor for DKD. Furthermore, thyroid replacement therapy has the potential to safeguard renal function.

Conversely, in other research, hypothyroidism may be associated with the narrowing of blood vessels in the kidneys and reduced efficient blood flow to the kidneys, resulting in renal injury and the development of microalbuminuria. The reference is Kramer *et al*., 2018. Furthermore, Wang *et al*. (2018) [12] demonstrated that the degree of albuminuria in individuals with chronic renal disease had the most significant impact on the levels of reverse triiodothyronine (rT_3) concentration, as indicated in Table 3, and the concentration of rT_3 in the bloodstream. It had a negative correlation with the level of albuminuria. Moreover, due to the fact that the majority of thyroid hormones are attached to proteins, the loss of proteins produced by albuminuria will result in a decrease in thyroid hormones. This decrease, in turn, triggers the hypothalamic-

pituitary-thyroid axis through negative feedback, preventing the conversion of T_4 to T_3 in tissues. The peripheral effect results in elevated levels of thyroid-stimulating hormone (TSH) and reduced levels of triiodothyronine (T_3). The citation is from Peters *et al*. (2021) [8].

Conclusion

According to this systematic analysis, it was discovered that an elevation in TSH levels is linked to a decline in glomerular filtration rate (eGFR), particularly within the normal range. An increase in fT_4 immediately led to a decrease in glomerular filtration rate (eGFR), which could potentially be influenced by age indirectly. There were no notable disparities between males and females in nephropathy caused by elevated TSH levels, however there were statistically significant disparities between males and females in the context of diabetes. Quadratic. Research has demonstrated a correlation between chronic nephropathy (CKD) and the worsening of hypothyroidism, which is accompanied by a steady decrease in glomerular filtration rate (eGFR). Alternatively, patients may experience hypothyroidism, resulting in a notable reduction in white blood cell count ($p = 0.005$) and hemoglobin levels ($p<0.001$), accompanied by a 25% increase in TSH. Furthermore, this study revealed that older persons with a slightly below average FT_3 level and a slightly above average TSH level have reduced kidney function. This study also demonstrated that insufficient iodine secretion results in elevated plasma levels of inorganic iodide, leading to a steady decline in FT_3 levels. Consequently, this contributes to the worsening of kidney injury and the occurrence of albuminuria. Despite having normal thyroid function.

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