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THYROID AS A TARGET FOR NUTRITIONAL INTERVENTIONS - PLENARY LECTURE

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Thyroid activity is influenced by both food amount and composition. It is affected by protein to carbohydrate ratio, fat composition and level of dietary cholesterol. The role of thyroid hormone in the regulation of metabolism implies the necessity of taking into consideration the relationship between nutrition and thyroid activity in the design for nutritional interventions

HYPOTHALAMUS – PITUITARY – THYROID AXIS

Hypothalamus – pituitary – thyroid (HPT) axis is composed of hypothalamic peptide thyrotropin releasing hormone (TRH) stimulating the secretion of pituitary thyrotropin (TSH) which in turn acts on thyroid to enhance thyroid hormone synthesis by inducing iodide and aminoacid uptake, as well as thyroglobulin (Tg) and thyroid peroxidase (TPO) synthesis. Thyroid peroxidase is a key enzyme in thyroid hormone biosynthesis catalysing iodide oxidation, iodination of thyrosine residues of thyroglobulin and coupling of iodothyronines [Dunn & Dunn, 2001]. The main hormone secreted by thyroid is 3,3',5,5'-tetraiodothyronine (T4). In target tissues T4 undergoes 5' deiodination of outer ring to metabolically active 3,3',5-triiodothyronine (T3) or inner ring 5-deiodination to inactive 3,3',5'- triiodothyronine (reverse-T3). Three types of deiodinases differing in specificity catalyse 5 and 5' deiodination (type I), 5' deiodination (type II) and 5 deiodination (type III). Hepatic iodothyronine deiodinase type I (DI) catalyses both outer and inner ring iodothyronine deiodination yielding metabolically active triiodothyronine and inactive reverse triiodothyronine. It is considered to be responsible for the circulating T3 level [Bianco *et al.,* 2002]. Thyroid hormones fasten the feed back loop in HPT by inhibiting synthesis of TRH and TSH.

THYROID ACTIVITY IN BODY WEIGHT REDUCTION

Thyroid hormones regulate multiple points of metabolism stimulating such diverse processes as energy expenditure and deposition, growth and differentiation. The influence of thyroid hormones on metabolic rate due to their effect on adenosine triphosphatase activity, mitochondrial biogenesis and uncoupling protein expression subordinate HPT axis activity to reciprocal regulation by the amount of food consumed. Fasting or shortage of energy supply elicit adaptation in physiology of hormones of HPT leading to diminution of energy expenditure. This adaptive process contributes to major obstacles in successful weight loss. Changes in HPT axis comprise decrease in TRH and TSH synthesis [Blake *et al.,* 1991], decline in TSH, as well as total and free T4 and T3 plasma concentrations [van Haasteren *et al.,* 1996]. Our study conducted in obese and overweight women revealed the dependence of thyroidal response during weight reducing treatment on the magnitude of energy deficit produced either by energy intake equal to 80% or 50% of calculated total energy expenditure [Kozłowska & Rosołowska-Huszcz, 2004]. Twenty percent energy deficit caused a decrease in T3, T4 and TSH serum concentrations, an increase in T4/TSH, but did not affect fT4 and fT4/T4. Concomitantly decline in BMI, percent body fat (fm%), serum leptin concentration and Lep/fm ratio were observed. Increase in energy deficit from 20% to 50% provoked greater decline in thyroid hormone serum concentration, however significant changes were not observed with regards to fT4 level and fT4/T4 ratio. This effect could be due to both considerable interpersonal variability in fT4 serum concentration and to influence plasma free fatty acids, usually elevated during energy restriction because of enhanced lipolysis. Triiodothyronine serum concentration and T3/T4 ratio diminished in stepwise manner dependent on the magnitude of energy deficit. The relation between HPT axis and leptin was suggested by positive correlation between TSH and leptin level, however, observed only after 50% energy deficit treatment. Predictive significance of TSH serum concentration and T4/TSH ratio for success in weight reducing therapy could be concluded from direct relations of these starting values with changes in fm% evoked by

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treatment. Our study indicating direct relation between decline in thyroid activity and the magnitude of energy restriction implies that smaller energy deficit should be recommended because it causes minimal decrease in metabolic rate during weight loss.

In order to suppress the decline in metabolic rate during the weight reducing treatment increase in physical activity is recommended. The influence of enhanced energy expenditure on thyroid activity was examined in the studies performed both in humans and animals. Thyroid hormone metabolism has been suggested to be stimulated by physical exercise [Balsam & Leppo, 1975; Katzeff *et al.,* 1988; Katzeff & Selgrad, 1991]. However, the effects of acute exercise and chronic enhancement of physical activity usually were not separated. Exercise trained male Wistar rats have been shown to reveal energy economizing adaptations such as suppression of cold induced thermogenesis [Richard & Arnold, 1987] and brown adipose tissue (BAT) activity [Larue-Achagiotis *et al.,* 1995]. They were also stated to reduce their total energy expenditure [Richard *et al.,* 1989].

We studied the influence of exercise training intensity on thyroid activity at rest in male Wistar rats trained on treadmill at different intensities [Rosołowska-Huszcz, 1998b]. The five schedules of training were applied: exercise at the speed of: 20 m/min was performed over a 5-week period with different frequency: 2x60 min, 3 – 4x60 min, 4 – 6x20 min, 5 – 6x40 min and 6 – 6x60 min weekly. Thyroid peroxidase activity was decreased in groups trained 240 min and 360 min weekly, T_4 plasma concentration in all trained groups and T_3 only in groups exercising 120 min weekly. Hepatic 5'DI activity and rT_3 plasma concentration were not affected by training. Thus, exercise training in rats has been shown to elicit diminishing in TPO activity and T_4 plasma concentration at rest without changing hepatic 5'DI activity and T_3 and rT_3 plasma concentrations. Fall in T_3 plasma concentration, observed in rats trained with the lowest intensities, could be treated as a transitional effect in adaptation to chronic exercise. Diminishing in TPO activity at rest could contribute to the energy conservation trends in trained rats observed by others [Richard & Arnold, 1987; Richard *et al.,* 1989; Larue-Achagiotis *et al.,* 1995].

Similar conclusions to ours could be drawn from the studies regarding the influence of exercise on thyroidal iodine uptake. They revealed a decrease in iodine uptake in trained rats and humans [Rhodes, 1967; Hooper *et al.,* 1980]. Rats exercising spontaneously were found to store only half as much iodine in the thyroid as did the nonexercising controls with the amount of exercise and amount of iodine being negatively correlated [Rhodes, 1967]. This effect has been suggested to be connected with depression of thyroid activity or increase in thyroid hormone production and release.

The adaptative response of thyroid to increase in energy demand and/or energy deficit clearly indicates that dietary strategies aiming at body weight reduction ought to be combined with enhancement of physical activity, however, the energy deficit produced by both should not exceed certain limits.

THYROID AND DIETARY PROTEIN TO CARBOHYDRATE RATIO

Thyroid activity responds not only to starvation and amount of food consumed but also to changes in macronutrient and several micronutrient supply. Realimentation with diets of various macronutrient proportions has been shown to restore thyroid activity after period of starvation with different efficacy. In humans only diets containing carbohydrates, pure or mixed with protein, were able to increase T_3 and decrease rT_3 plasma level [Azizi, 1978]. However, in rats similar effects gave refeeding with protein and carbohydrates [Burger *et al.,* 1980]. Triiodothyronine plasma level has been shown to increase with carbohydrate to protein ratio in diet [Smallridge *et al.,* 1982; Rosołowska, 1998a]. whereas the opposite was found for T_4 concentration [Smallridge *et al.,* 1982; Rosołowska, 1998a]. In our study performed on male Wistar rats fed different protein and carbohydrate levels direct relation of TPO activity with protein intake and inverse relation with carbohydrate intake was demonstrated [Rosołowska, 1998a]. It suggests a decrease in thyroid activity on low protein diets. This is consistent with data showing a decline in the uptake of iodide [Florsheim *et al.,* 1970], a fall in the thyroid hormone secretion rate [Singh *et al.,* 1971] and the existence of ultrastructural changes in thyroid revealing hypothyroidism [Worthington *et al.,* 1975] under conditions of protein malnutrition. Hepatic DI activity was found to be directly related to carbohydrate intake [Rosołowska, 1998a]. This could confirm the stimulating effect of carbohydrate on hepatic 5'deiodination found by others [Gavin *et al.,* 1988].

The low protein – high carbohydrate diet is commonly used in conservative therapy of chronic kidney disease in attempt to retard the rate of renal function decline. Hypothalamo–pituitary-thyroid axis activity is affected in many ways in CKD, including TSH secretion and clearance [Lim *et al.,* 1993], decline in both total and free T_3 and T_4 plasma concentrations [Docter *et al.,* 1993], decreased hepatic uptake of T_4 and diminished peripheral T_4 to T_3 conversion [Lim *et al*., 1993]. Thyroxine binding by thyroxine binding globulin is usually reduced, which is attributed to the presence of inhibitors [Oppenheimer *et al*., 1982], although their role has never been proven. We investigated the influence of 8 weeks of a low protein diet (0.6 g/kg body weight daily) treatment on pituitary–thyroid axis activity in patients with estimated creatinine clearance 39.5±11.1 mL/min [Rosołowska-Huszcz *et al.,* 2005]. Unlike the effects of low protein – high carbohydrate found in humans and animals with normal renal function, treatment evoked an increase in T_3 and total and free T_4 serum concentrations. Changes in hormonal levels induced by therapy were related to starting values of hormonal and renal function indices. Changes in T_3, T_4 and fT_4 serum concentrations as well as calculated peripheral deiodinase activity correlated negatively with their baseline values. Subjects with lowest initial T_3, T_4, fT_4 and deiodinase activity had the best response to the diet. Lower initial TSH was associated with a greater increase in T_3 concentration. Moreover, the effect of a low protein diet on thyroid hormone level was not seen in patients with normal initial hormone concentration. Triiodothyronine level after treatment correlated negatively

with baseline urea level. Changes in T_4 and T_4/TSH were inversely related to vegetal protein intake. This could indicate the influence of protein nutrition in CKD on thyroid sensitivity for TSH stimulation.

The effects of low protein diet on HPT axis in CKD could reflect diminishing in inflammation and uremic toxin level. Inflammatory cytokines, like TNF-alpha, have been shown to inhibit thyroid activity and deiodinase gene expression [Davies *et al.,* 1997; Tang *et al.,* 1995]. Uremic toxins impair thyroid hormone metabolism influencing their cellular uptake [Lim, 2001].

THYROID ACTIVITY AND DIETARY LIPIDS

Thyroid hormones regulate lipid metabolism, influencing fatty acid synthesis [Blenneman *et al.,* 1992] and oxidation [Germack *et al.,* 1996], lipolysis [Jackson-Hayes *et al.,* 2003], cellular uptake of cholesterol and transformation into bile acids [Ness & Lopez, 1995]. A reciprocal influence of fatty acids on thyroid activity has been demonstrated at several points of the thyroidal axis and thyroid hormone metabolism. The first effect identified was the displacement of thyroid hormones by fatty acids from plasma binding proteins [Hollander, 1964]. Further studies corroborated and expanded data concerning competition between free fatty acids and thyroid hormones in binding plasma proteins [Lim *et al.,* 1995]. Fatty acids have also been shown to interfere with binding of T_3 to nuclear receptors [Thurmond *et al.,* 1998; Yamamoto *et al.,* 2001] as well as influencing T_3 receptor number and activity [Noel-Suberville *et al.,* 1998]. Effects of dietary fat on plasma thyroid hormone level [Takeuchi *et al.,* 1995], TSH secretion [Kennedy *et al.,* 1994], TPO activity [Rosołowska-Huszcz *et al.,* 2001] and T_4 to T_3 conversion [Chopra *et al.,* 1985] have been also found.

In the studies conducted on male Wistar rats we examined the effects of feeding diets differing in level (5, 10, 20% w/w) and composition of fats as well as the level of cholesterol. Thyroid peroxidase activity was found to be directly related to docosahexanoic and eicosapentanoic acid intakes [Rosołowska-Huszcz & Lachowicz, 2004], but also higher in rats fed palm oil than lard, rapeseed and sunflower oils [Rosołowska-Huszcz *et al.,* 2001]. This is in agreement with the results obtained by others [Makino *et al.,* 2001] who showed protective effects of methyl ester of eicosapenthanoic acid before destruction of thyroid tissue provoked by treatment with goitrogen. Docosahexanoic acid has also been found to stimulate TSH release in rats [Clandinin *et al.,* 1998]. However, other results also suggest the involvement of n-6 polyunsaturated fatty acids in the stimulation of thyroid activity. The endogenous ligand of peroxisome proliferator activated receptor γ (PPARγ) – 15-deoxy-$\Delta^{12,14}$-PGJ_2 (15d PGJ_2) – a product of arachidonic acid metabolism was shown to facilitate the synthesis of thyroglobulin in a functional rat epithelial cell line [Kasai *et al.,* 2000]. On the other hand, PPARγ has recently been found to promote differentiation of thyrocytes and demonstrate anticancer activity in the thyroid [Frohlich *et al.,* 2005]. Taking into consideration the similar mode of regulation of thyroglobulin and TPO gene transcription [Espinoza *et al.,* 2001], 15dPGJ_2 might represent an intermediate in the signaling pathway by which polyunsaturated fatty acids affect TPO level. Rather astonishing stimulating influence of palm oil on TPO activity could be attributed to the high content of palmitic acid present in this oil and the role of palmitic acid in TSH receptor functioning which is dependent on the palmitoilation [Tanaka *et al.,* 1998].

Hepatic DI activity in our studies was directly related to docosahexanoic and archidonic acid intakes [Rosołowska-Huszcz & Lachowicz, 2004]. This could be due to stimulating effect of *n*-3 fatty acids on liver T_3 receptor expression [Noel-Suberville *et al.,* 1998] since T_3 was found to upregulate DI gene expression [Bianco *et al.,* 2002].

Effect of higher level of cholesterol on thyroid activity in our studies depended on the kind of dietary fat. It increased TPO activity in rats fed butter, fish oil or standard diet, whereas it did not influence TPO activity in rats receiving rapeseed, sunflower and grape seed oils or lard [Sotowska & Rosołowska-Huszcz, 2004].

CONCLUSIONS

Results of our studies prompted us to conclude that the amount of fat consumed and its composition influence thyroid activity, thus affecting various points of thyroid hormone metabolism including synthesis, deiodination, binding to plasma proteins, and presumably cellular uptake.

REFERENCES

1. Azizi F., Effect of dietary composition on fasting–induced changes in serum thyroid hormones and thyrotropin. Metabolism, 1978, 27, 935-942.
2. Balsam A., Leppo L.L., Effect of physical training on the metabolism of thyroid hormones in man. J. Appl. Physiol., 1975, 39, 212-215.
3. Bianco A.C., Salvatore D., Gereben B., Berry M.J., Larsen P.R., Biochemistry, cellular and molecular biology and physiological roles of the iodothyronine selenodeiodinases. Endocrine Rev., 2002, 23, 38-89.
4. Blake, N.G., Eckland D.J.A., Foster O.J.F., Lightman, S.L., Inhibition of hypothalamic thyrotropin–releasing hormone messenger ribonucleic acid during food deprivation. Endocrinology, 1991, 129, 2714-2718.
5. Blennemann B., Monn Y.K., Freake H.C., Tissue-specific regulation of fatty acid synthesis by thyroid hormone. Endocrinology, 1992, 130, 637-643.
6. Burger A.G., Berger M., Wipfheimer K., Danforth E., Interrelationship between energy metabolism and thyroid hormone metabolism during starvation in the rat. Acta Endocrinol., 1980, 93, 322-331.
7. Chopra I.J., Huang T.S., Beredo A., Solomon D.H., Chua Teco C.N., Mead J.F., Evidence for an inhibitor of extrathyroidal conversion of thyroxine to 3,5,3'-triiodothyronine in sera of patients with nonthyroidal illness. J. Clin. Endocrinol. Metab., 1985, 60, 666-672.
8. Clandinin M.T., Claerhout D.L., Lien E.L., Docosahexaenoic acid increases thyroid-stimulating hormone concentration in male and adrenal corticotrophic hormone con-

centration in female weanling rats. J. Nutr., 1998, 128, 1257-1261.
9. Davies P.H., Sheppard M.C., Franklyn J.A., Inflammatory cytokines and type I 5'-deiodinase expression in phi1 rat liver cells. Mol. Cell. Endocrinol., 1997, 16, 191-198.
10. Docter R., Krenning E.P., de Jong M., Hennemann G., The sick euthyroid syndrome: changes in thyroid hormone serum parameters and hormone metabolism. Clin. Endocrinol. (Oxf.), 1993, 39, 499-518.
11. Dunn J.T., Dunn A.D., Update on intrathyroidal iodine metabolism. Thyroid, 2001, 11, 407-414.
12. Espinoza C.R, Schmitt T.L., Loos U., Thyroid transcription factor 1 and Pax8 synergistically activate the promoter of human thyroglobulin gene. J. Mol. Endocrinol., 2001, 27, 59-67.
13. Florsheim W.H., Suhr B.Z., Mirise R.T., Thyroid function in protein depleted rats. J. Endocrinol., 1970, 46, 93-99.
14. Frohlich E., Machicao F., Wahl R., Action of thiazolidinediones on differentiation, proliferation and apoptosis of normal and transformed thyrocytes in culture. Endocr. Relat. Cancer, 2005, 12, 291-303.
15. Gavin L.A., Moeller M., Mc Mahon F.A., Castle J.N., Gulli R.R., Cavalieri R.R., Carbohydrate feeding increases total body and specific tissue 3,5,3'-triiodothyronine neogenesis in the rat. Endocrinology, 1988, 123, 1075-1081.
16. Germack R., Adli H., Vassay R., Perret G.Y., Triiodothyronine and amiodarone effects on β_3-adrenoreceptor density and lipolytic response to the β_3-adrenergic agonist BRL 37344 in rat white adipocytes. Fundam. Clin. Pharmacol., 1996, 10, 289-297.
17. Hollander C., Free fatty acids: a possible regulator of free thyroid hormone levels in man. Endocrinol. Metab., 1964, 27, 1219-1223.
18. Hooper P.L., Rhodes B.A., Conway M.J., Exercise lowers thyroid radio iodine uptake. J. Nucl. Med., 1980, 21, 835-837.
19. Jackson-Hayes L., Song S., Lavrentyev S.N., Jansen M.S,. Hilgartner F.B., Tian L., Wood P.A., A thyroid hormone response unit formed between the promoter and first intron of the carnitine palmitoylotransferase-Iα gene mediates the liver-specific induction by thyroid hormones. J. Biol. Chem., 2003, 278, 7972-7972.
20. Kasai K., Banba N., Hishinuma A., Matsumura M., Kakishita H., Matsumara M., Motohashi S., Sato N., Hattori Y., 15-deoksy-delta(12,14)-prostaglandin J(2) facilitates thyroglobulin production by cultured human thyrocytes. Am. J. Physiol. Cell Physiol., 2000, 276, C1859-1869.
21. Katzeff H.L., Bovbjerg D., Mark D.A., Exercise regulation of triiodothyronine metabolism. Am. J. Physiol., 1988, 255, E824-E828.
22. Katzeff H.L., Selgrad C., Maintenance of thyroid hormone production during exercise – induced weight loss. Am.J. Physiol., 1991, 261, E382-E388.
23. Kennedy J.A., Nicolson R., Wellby M.L., The effect of oleic acid on the secretion of thyrotrophin and growth hormone by cultured rat anterior pituitary cells. J. Endocrinol., 1994, 143, 557-564.
24. Kozłowska L., Rosołowska-Huszcz D., Leptin, thyrotropin and thyroid hormone response to two level of energy deficit in overweight – obese women. Endocrine, 2004, 24, 147-154.
25. Larue-Achagiotis C., Rieth N., Goubern M., Laury M.-C., Louis – Sylvestre J., Exercise – training reduces BAT thermogenesis in rats. Physiol. Behav., 1995, 57, 1013-1017.
26. Lim C.F., Munro S., Wynne K., Topliss D., Stockigt J., Influence of nonesterified fatty acids and lysolecithins on thyroxine binding to thyroxine-binding globulin and transthyretin. Thyroid, 1995, 5, 319-324.
27. Lim C.F., Bernard B.F., de Jong M., Docter R., Krenning E.P., Hennemann G., A furan fatty acid and indoxyl sulfate are the putative inhibitors of thyroxine hepatocyte transport in uremia. J. Clin. Endocrinol. Metab., 1993, 76, 318-324.
28. Lim V.S., Thyroid function in patients with chronic renal failure. Am. J. Kidney Dis., 2001, 38, S80-84.
29. Makino N., Oda N., Miura N., Imamura S., Yamamoto K., Kato T., Fujiwara K., Sawai Y., Iwase K., Nagasaka A., Itoh M., Effect of eicosapentaenoic acid ethyl ester on hypothyroid function. J. Endocrinol., 2001, 171, 259-265.
30. Ness G.C., Lopez D., Transcriptional regulation of rat hepatic low – density lipoprotein receptor and cholesterol 7α hydroxylase by thyroid hormone. Arch. Biochem. Biophys., 1995, 323, 404-408.
31. Noel-Suberville C., Pallet V., Audouin-Chevallier I., Higueret P., Bonilla S., Martinez A., Zulet M., Portillo M., Garcin H., Expression of retinoic acid, triiodothyronine and glucocorticoid hormone nuclear receptors in decreased in the liver of rats fed a hypercholesterolemia-inducing diet. Metabolism, 1998, 47, 301-308.
32. Oppenheimer J.H., Schwartz H.L., Mariash C.N., Kaiser F.E., Evidence for a factor in the sera of patients with nonthyroidal disease which inhibits iodothyronine binding by solid matrices, serum proteins, and rat hepatocytes. J. Clin. Endocrinol. Metab., 1982, 54, 757-766.
33. Richard D., Arnold J., Influence of exercise training in the regulation of energy balance. J. Obes. Weight Regul., 1987, 6, 212-224.
34. Richard D., Lachance P., Deshaies Y., Effects of exercise – rest cycles on energy balance in rats. Am. J. Physiol., 1989, 256, R886-R891.
35. Rhodes B.A., Effect of exercise on the thyroid gland. Nature, 1967, 216, 917-918.
36. Rosołowska-Huszcz D., Wpływ niektórych czynników żywieniowych i wysiłku fizycznego na metabolizm hormonów tarczycy. 1998a, Wyd. SGGW (in Polish; English abstract).
37. Rosołowska-Huszcz D., The effect of exercise training intensity on thyroid activity at rest. J. Physiol. Pharmacol., 1998b, 49, 457-66.
38. Rosołowska-Huszcz D, Gromadzka-Ostrowska J, Wilczak J, Romanowicz K, Borysiak M, Dębska M, Mazurek B., Thyroid peroxidase activity, hepatic glucose-6-phosphate dehydrogenase activity and corticosterone level in plasma and tissues of rats fed different dietary fats. J. Animal Feed Sci., 2001,10 , 185-200.
39. Rosołowska-Huszcz D., Lachowicz K., Udział kwasów tłuszczowych w regulacji syntezy białek zaangażowanych

w metabolizm energetyczny, 2004, *w*: Fizjologiczne uwarunkowania postępowania dietetycznego, Wyd. SGGW, pp. 58-66 (in Polish; English abstract).
40. Rosołowska–Huszcz D., Kozłowska L., Rydzewski A., Influence of low protein diet on nonthyroidal illness syndrom in chronic renal failure. Endocrine, 2005, 27, 283-288.
41. Singh D.V., Anderson R.R., Turner C.V., Effect of decreased dietary protein on the rate of thyroid hormone secretion and food consumption in rats. J. Endocrinol., 1971, 50, 445-450.
42. Smallridge R.C., Glass A.R., Wartofsky L., Latham K.R., Burman K.D., Investigations into the etiology of elevated serum T3 levels in protein-malnourished rats. Metabolism, 1982, 31, 538-542.
43. Sotowska B., Rosołowska-Huszcz D., Influence of dietary cholesterol on thyroid activity depends on dietary fat type in rats fed different fat sources. 2004, *in*: Materials of the13th International Symposium "Molecular and physiological aspects of regulatory processes of the organism", June 3-4, 2004, Cracow, Poland, pp. 439-440.
44. Tanaka K., Nagayama Y., Nishihara E., Namba H., Yamashita S., Niwa M., Palmitoylation of human thyrotropin receptor: slower intracellular trafficking of the palmitoylation-defective mutant. Endocrinology, 1998, 139, 803-806.
45. Tang K.T., Braverman L.E., DeVito W.J., Tumor necrosis factor-alpha and interferon-gamma modulate gene expression of type I 5'-deiodinase, thyroid peroxidase, and thyroglobulin in FRTL-5 rat thyroid cells. Endocrinology, 1995, 136, 881-888.
46. Thurmond D.C., Baillie R.A., Goodridge A.G., Regulation of the action of steroid/thyroid hormone receptors by medium-chain fatty acids. J. Biol. Chem., 1998, 273, 15373-15381.
47. Yamamoto N., Li Q.L., Mita S., Morisawa S., Inoue A., Inhibition of thyroid hormone binding to the nuclear receptor mobilization of free fatty acids. Horm. Metab. Res., 2001, 33, 131-137.
48. Takeuchi H., Matsuo T., Tokuyama K., Suzuki M., Serum triiodothyronine concentration and Na^+, K^+-ATPase activity in liver and skeletal muscle are influenced by dietary fat type in rats. J. Nutr., 1995, 125, 2364-2369.
49. van Haasteren G.A.C., Linkels E., van Toor H., Klootvijk W., Kaptein E., de Jong F.H., Reymond M.J., Visser T.J., Greef W.J., Effects of long–term food reduction on the hypothalamus – pituitary – thyroid axis in male and female rats. J. Endocrinol., 1996, 150, 169-178.
50. Wortinghton B.S., Enwonwu C.O., Functional variations in the ultrastructure of the thyroid gland in protein malnourished infant monkeys. Am. J. Clin. Nutr., 1975, 28, 66-78.

TARCZYCA JAKO CEL INTERWENCJI ŻYWIENIOWEJ - WYKŁAD PLENARNY

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Aktywność tarczycy zależy od ilości i składu pokarmu. Zmienia się wraz ze stosunkiem białka do węglowodanów, zależy od składu tłuszczu i poziomu cholesterolu w diecie. Z roli hormonów tarczycy w regulacji metabolizmu wynika konieczność uwzględniania zależności aktywności tarczycy od żywienia w planowaniu postępowania dietetycznego.