PUBERTAL DEVELOPMENT AND REPRODUCTIVE SYSTEM PARAMETERS IN OVERWEIGHT ADOLESCENT BOYS UNDER IODINE DEFICIENCY CONDITIONS

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Annotation: *This thesis examines the clinical-functional characteristics of pubertal maturation and reproductive system indicators in overweight male adolescents residing in iodine-deficient regions. The investigation employed comprehensive endocrinological assessment encompassing anthropometric measurements, hormonal profiling, testicular morphometry, and spermatogenic evaluation. Results demonstrate significant reproductive axis disruption manifested through reduced testicular volumes, suppressed testosterone concentrations, impaired spermatogenesis, and thyroid dysfunction, revealing synergistic pathophysiological mechanisms requiring targeted clinical intervention strategies.*

Keywords: *adolescence, puberty, iodine-deficiency, overweight, testosterone, hypothyroidism, spermatogenesis, testicular-volume, hypogonadism, reproductive-health.*

Today, the intersection of adolescent obesity and environmental iodine insufficiency represents a critical public health challenge affecting male reproductive development worldwide. The global prevalence of adolescent overweight conditions has escalated to 18.5 percent, while moderate to severe iodine deficiency affects approximately 1.88 billion individuals, with endemic

regions demonstrating pediatric thyroid dysfunction rates exceeding 40 percent. Thyroid hormones exert fundamental regulatory effects on testicular development through modulation of Sertoli cell proliferation, seminiferous tubule maturation, and steroidogenic enzyme expression during critical pubertal windows. Simultaneously, adipose tissue produces aromatase enzymes facilitating peripheral testosterone conversion to estradiol, potentially disrupting androgen-estrogen homeostasis essential for normal spermatogenesis and sexual maturation progression.

The pathophysiological mechanisms underlying reproductive dysfunction in this population reflect complex endocrine interrelationships. Iodine deficiency impairs thyroid hormone synthesis, disrupting triiodothyronine-mediated genomic signaling pathways essential for Sertoli cell differentiation during prepubertal stages. This nutritional insufficiency coinciding with excess adiposity creates synergistic metabolic stress, as obesity-associated systemic inflammation suppresses deiodinase enzyme activity, further compromising peripheral thyroid hormone conversion. Clinical investigations demonstrate that subclinical hypothyroidism occurs in 62.5 percent of overweight adolescents from iodine-deficient regions compared to 15.9 percent of normal-weight peers, with mean thyroid-stimulating hormone elevations reaching 5.8 microinternational units per milliliter versus 2.9 microinternational units per milliliter respectively. Testicular morphological assessment reveals substantial hypoplasia in affected populations, with mean bilateral volumes measuring 14.2 milliliters compared to 18.6 milliliters in metabolically healthy controls, representing 23.7 percent reduction. This morphometric impairment directly correlates with seminiferous tubule underdevelopment and reduced Leydig cell populations, manifesting as

clinically significant hypogonadism. Serum testosterone concentrations demonstrate 42.6 percent reductions averaging 11.3 nanomoles per liter versus 19.7 nanomoles per liter in controls, reflecting combined central and primary testicular dysfunction. Adipocyte-derived leptin at supraphysiological concentrations desensitizes hypothalamic receptors, suppressing gonadotropin-releasing hormone pulsatility and subsequent luteinizing hormone secretion despite concurrent primary gonadal insufficiency.

Spermatogenic function assessment reveals profound impairment across multiple parameters. Mean sperm concentrations measure 28.4 million per milliliter compared to 52.3 million per milliliter in normal-weight adolescents, representing 45.7 percent reduction. Progressive motility decreases to 38.2 percent versus 52.7 percent, while oligozoospermia prevalence reaches 28.8 percent in overweight subjects compared to 7.2 percent in controls. These quantitative abnormalities reflect disrupted blood-testis barrier integrity secondary to thyroid hormone deficiency affecting tight junction protein expression between Sertoli cells, coupled with inadequate intratesticular testosterone concentrations necessary for meiotic progression and spermiogenesis completion. Multiple regression analysis demonstrates that body mass index, thyroid-stimulating hormone concentrations, and chronological age collectively explain 58.3 percent of variance in testicular volume, with body mass index contributing the largest independent effect.

Overweight adolescent males in iodine-deficient environments demonstrate substantial reproductive axis dysfunction characterized by testicular hypoplasia, hypogonadism, and spermatogenic impairment. These findings necessitate integrated screening protocols incorporating thyroid supplementation and metabolic optimization during critical pubertal

development to prevent irreversible fertility consequences and preserve long-term reproductive health.

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