iJRASET
International Journal For Research in Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 8 **Issue:** VI **Month of publication:** June 2020

DOI: http://doi.org/10.22214/ijraset.2020.6354

www.ijraset.com

Call: 08813907089 | E-mail ID: ijraset@gmail.com

International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.429
Volume 8 Issue VI June 2020- Available at www.ijraset.com

Usage of Plant Extracts in Treating Obesity - A Mini Review

Jyothi Kanagaraj[1], S. Sheik Asraf[2]
[1, 2]*Department of Biotechnology, School of Bio and Chemical Engineering, Kalasalingam Academy of Research and Education, Krishnankoil.*

Abstract: ***Obesity represents one of the major health emergencies in developed countries. India is now behind US and China in this international list of top 10 countries with elevated amount of obese people. Obesity has been attributed in the causation of a number of metabolic disorders together with diabetes, hypertension, dyslipidaemia, cardiovascular disease and cancers that enhance the mortality and morbidity hazard in adulthood. Alterations in thyroid functions were reported roughly in each obese patient. There is substantiation that an augmented Thyroid Stimulating Hormone can be an end result of obesity, since weight loss will lead to TSH level. Herbal medicines can be defined as unprocessed or extracted products isolated from plants, and used for impediment and healing of numerous chronic diseases, together with obesity, since they have smaller number of side effects compared with pharmacological drugs .In the recent decades, these herbal medicine have been used due to the presence of several components with different anti-obesity and anti-oxidant effects on body metabolism and fat oxidation. Medicinal plants have been investigated and reported to be useful in treatment of obesity, diabetes and other chronic diseases.***
Withania somnifera (Ashwagandha), family Solanaceae, has been used in copious ayurvedic formulations outstanding to its extensive assortment of health-promoting properties. The roots of W. somnifera , endorse natural weight loss devoid of any negative side effects and are very competent in the improvement of good health. In human case studies, treatment with W. somnifera caused noteworthy reductions in serum total cholesterol, TAG, LDL levels. Although there was no report for side effect of this plant, we believe that safety of this plant still remains to be elucidated by further long-term studies.
Keywords: Obesity, Thyroid, Withania somnifera.

I. INTRODUCTION

Obesity is becoming one of the most prevalent health concerns among all populations worldwide, resulting into a significant increase in mortality and morbidity related to diabetes type 2, metabolic syndrome, stroke and cancers. Prevention and treatment of this problem are an important deal for health systems, whose aim is to reduce the obesity and overweight prevalence, and related complications over the world. Obesity and hypothyroidism are the medical conditions that have been very much related. The relation becomes more appropriate in the perspective of increase in the predominance of obesity worldwide. Obesity is regarded by patients as being less important to thyroid dysfunction (1). Reported studies indicate that alteration in TSH could well be less important to obesity.

Recent information have also disclosed a corelation between obesity and thyroid autoimmunity with leptin seems to be the key feature connecting these two conditions (2). Leptin, a hormone released by white adipose tissue, for the most part in the subcutaneous fat, persuades TSH discharge.. Leptin signal seems to be a shows potential relationship linking obesity and alterations of thyroid hormones.

Leptin regulates energy homeostasis, and its non existence in rodents and humans effects relentless obesity (3). Lipin1 is a constituent of the lipin protein family that is synchronized by pituitary hormones (4). It is also testimony to be involved in the directive of lipid metabolism as its term is modified by thyroid status.

The naturopathic treatment for obesity has been explored extensively since ancient times and gaining momentum in the present scenario. Herbal medicinal plants and their active metabolites have been used as anti-obesity medicine. Many herbal plants and their bioactive compounds were also tested by clinical trials and are effective in treatment of obesity. *Withania somnifera* (Ashwagandha) has been used in copious ayurvedic formulations outstanding to its extensive assortment of health-promoting properties (5). There are reports showing the chemical and pharmacological properties of *W. somnifera* in regulating thyroid utility but there is an obvious need of detailed reports about its effectiveness on treating hypothyroidism at the molecular marker and its contact on lipid metabolism. This review focus on the usage of *W. somnifera* extracts with their mechanism of action in treating obesity.

II. OBESITY AND HYPOTHYROIDISM

Obesity and hypothyroidism are significant public health problem in India (6) Obesity leads to countless health consequences and in India reported occurrence of obesity in range of 2.9%
to14.3% (7), and of overweight ranges 1.5%–24.0% (8). In India these studies were conducted at different areas at different time period. The diverse standards used in these studies included WORLD HEALTH ORGANIZATION, CDC growth charts, IOTF references, and IAP 2007 reference.

Hypothyroidism is linked with dwindle metabolic rate, elevated BMI, which leads to obesity. (7). Reported scientific proof suggests that hypothyroidism is associated to considerable changes in body weight and correspond to a hazard factor for obesity (6,8). Biochemical assays additionally noted that little variations in serum TSH caused by negligible changes in L-T4 dosage during substitution.

However, there is a scarcity of data concerning the definite scope of weight gain and weight loss with leptin management in hypothyroidism. Weight loss induces a noteworthy decrease in serum TSH levels (9). Alteration in body composition reduces the condition of inflammation, decreases the discharge of cytokines, and consequential deterioration of thyroid function. This observable fact further entail that autoimmune annihilation of thyrocytes is not accountable for the raised serum TSH and the alteration in thyroid utility tests are largely functional (10). There is no hint for leptin administration to control body weight with the exception of in obese hypothyroid subjects. Additional research is essential to decide whether subclinical hypothyroidism is causally involved in the progress of obesity. The association between leptin, and progress of obesity associated with hypothyroidism needs to be studied. (11)

III.ROLE OF LEPTIN IN OBESITY

Leptin, a hormone released by white adipose tissue, for the most part in the subcutaneous fat, persuades TSH discharge. Leptin signal seems to be a shows potential relationship linking obesity and alterations of thyroid hormones. Leptin regulates energy homeostasis, and its non existence in rodents and humans effects relentless obesity (12). Lipin1 is a constituent of the lipin protein family that is synchronized by pituitary hormones (4).

It is also testimony to be involved in the directive of lipid metabolism as its term is modified by thyroid status. It is also reported to be implicated in the directive of lipid metabolism since its expression is modified by thyroid status. Leptin and Lipin regulations might be interrelated.

Leptin regulate energy homeostasis, involve in glucose and lipid metabolism, immune function, and other systems. Leptin binds to its specific receptor and activates many pathways like Janus kinase 2 ,signal transducer and activator of transcription 3, insulin receptor substrate phosphatidylinositol 3 kinase, SH2-containing protein tyrosine phosphatase 2 /mitogen-activated protein kinase, and 5' adenosine monophosphate-activated protein kinase / acetyl-CoA carboxylase. Leptin is a
16kD bioactive protein secreted from adipocytes and acts as a regulator (13). Normal intensity of leptin in the blood is 5–10ng/mL in healthy patients and in obese patients it is of 50ng/mL(14). Leptin stimulates class I cytokine-receptor family, consisting of six isoforms that dimerize with each other, but not have intrinsic kinase activity (14). Leptin receptor isoforms show a discrepancy with tissue and cell type in addition to ligand stimulation. Auto-regulation of receptor levels as well as ligand-dependent activity may perhaps furthermore lead to leptin resistance (15,16).

Modern medical treatments for obesity have many disadvantages, affecting the monoamine neurotransmitters and the potential for drug abuse and dependency.

The safety of these medications requires improvement. Ayurvedics treatment has been used for curing many diseases for more than 2000 years, and it has proven worth. Reported scientific studies have also confirmed the effective role of plant metabolites in treatment of obesity, but the mechanisms are not clear. *Withania somnifera* has a wide array of health-promoting properties and has been used in numerous Ayurvedic formulations.

The current study provides the scientific validation to the anti-inflammatory, and anti-apoptotic properties of the important medicinal plant, *W. somnifera*, which may be recommended as a suitable intervention to prevent or slow down the adverse effects of obesity and its associated co-morbid conditions.

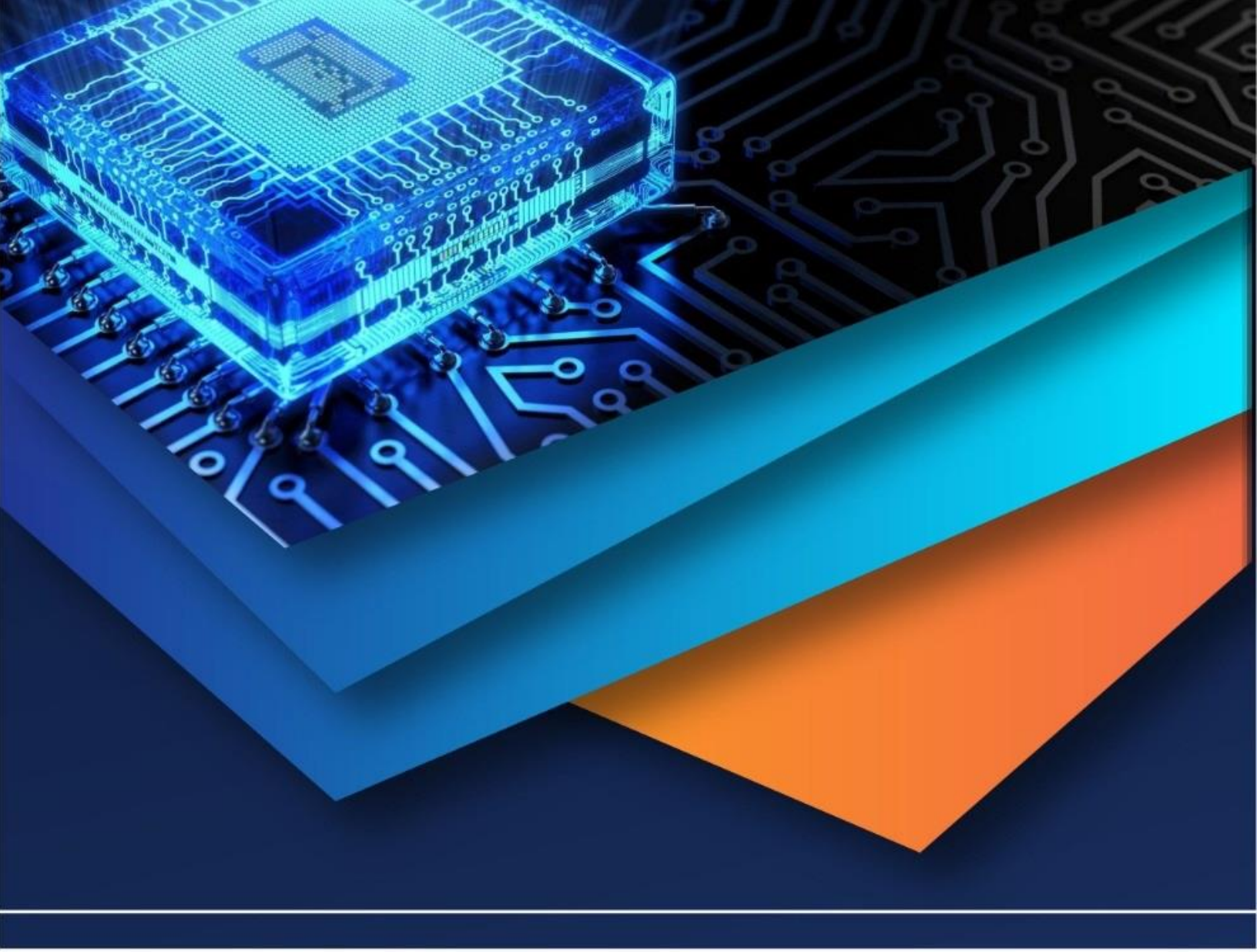
Understanding the molecular mechanism of leptin signaling will provides insights into its roles in health and disease, as leptin plays a crucial tool to fight against obesity.

IV.ACKNOWLEDGMENT

The authors thanks the management of KARE.

REFERENCES

[1] Rosenbaum M, Hirsch J, Murphy E, Leibel RL. Effects of changes in body weight on carbohydrate metabolism, catecholamine excretion, and thyroid function. Am J Clin Nutr. 2000;71:1421–32.
[2] Knudsen N, Laurberg P, Rasmussen LB, Bülow I, Perrild H, Ovesen L, et al. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. J Clin Endocrinol Metab. 2005; 90:4019–24.
[3] Farooqi IS, O'Rahilly S *:* Mutations in ligands and receptors of the leptin- melanocortin pathway that lead to obesity. Nat Clin Pract Endocrinol Metab 2008;4:569–577
[4] Van Harmelen V, Rydén M, Sjölin E, Hoffstedt J: A role of lipin in human obesity and insulin resistance: relation to adipocyte glucose transport and GLUT4 expression**.** J Lipid Res.2007; 48(1):201-6
[5] Taranjeet Kaur and Gurcharan Kaur. *Withania somnifera* as a potential candidate to ameliorate high fat diet-induced anxiety and neuro inflammation. J Neuroinflammation. 2017; 14: 201.
[6] Jain S, Pant B, Chopra H, Tiwari R, Obesity among adolescents of affluent public schools in Meerut. Indian J Public Health. 2010;54:158-60
[7] Kumaravel V, Shriram V, Anitharani M, Mahadevan S, Balamurgan AN, Sathiyasekaran B.Are the current Indian growth charts really representative? Analysis of anthropometric assessment of school children in a South Indian district. Indian J Endocr Metab 2014;18:56-62.
[8] Goyal RK, Shah VN, Saboo BD, Phatak SR, Shah NN, Gohel MC, *et al.* Prevalence of overweight and obesity in Indian adolescent school going children: Its relationship with socioeconomic status and associated lifestyle factors. J Assoc Physicians India 2010;58:151-8.
[9] Danforth E Jr., Horton ES, O'Connell M, Sims EA, Burger AG, Ingbar SH, *et al.* Dietaryinduced alterations in thyroid hormone metabolism during overnutrition. J Clin Invest 1979;64:1336-47.
[10] Longhi S, Radetti G. Thyroid function and obesity. J Clin Res Pediatr Endocrinol 2013;5 Suppl 1:40-4
[11] Nannipieri M, Cecchetti F, Anselmino M, Camastra S, Niccolini P, Lamacchia M, *et al.* Expression of thyrotropin and thyroid hormone receptors in adipose tissue of patients with morbid obesity and/or type 2 diabetes: Effects of weight loss. Int J Obes (Lond) 2009;33: 1001-6.
[12] Verma A, Jayaraman M, Kumar HK, Modi KD. Hypothyroidism and obesity. Cause or effect? Saudi Med J 2008;29:1135-8
[13] Ladenson PW, Kristensen JD, Ridgway EC, Olsson AG, Carlsson B, Klein I, *et al.* Use of the thyroid hormone analogue eprotirome in statin-treated dyslipidemia. N Engl J Med 2010;362:906-16.
[14] Bjorbaek C, Kahn BB. Leptin signaling in the central nervous system and the periphery. Recent Prog Horm Res. 2004;59:305–331.
[15] Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR, et al. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. N Engl J Med.1996;334:292–295.
[16] Ceddia RB. Direct metabolic regulation in skeletal muscle and fat tissue by leptin: implications for glucose and fatty acids homeostasis. Int J Obes (Lond) 2005;29:1175–1183.



10.22214/IJRASET

45.98

IMPACT FACTOR: 7.129

IMPACT FACTOR: 7.429

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY