

Antithyroid Screening of Polyherbal Formulation containing hydro-alcoholic extract of *Drimiopsis kirkii* Baker and *Momordica charantia* Linn.

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Abstract

The use of medicinal plants to treat endocrine diseases is a promising and holistic approach. Herbal treatments have long been used to treat illnesses such as diabetes, thyroid dysfunction, adrenal problems, and polycystic ovarian syndrome (PCOS). Several plant-derived bioactive chemicals, including alkaloids, flavonoids, and polyphenols, have medicinal actions that promote hormonal homeostasis. However, despite their benefits, herbal medicines do have certain hazards. Potential adverse effects, contraindications, and combinations with conventional drugs underscore the significance of exercising caution and seeking professional advice. As researchers continue to investigate and evaluate the usefulness of medicinal plants, combining herbal therapies with modern medication may provide more effective and safer treatment alternatives. In the present study polyherbal formulation (tablet) was prepared from the hydro-alcoholic extract of bulb of *Drimiopsis kirkii* and fruits of *Momordica charantia*. These hyperthyroid treatments related histopathological changes of thyroid gland was dramatically inhibited by treatment of PHF-T 500 mg/kg or PTU 10mg/kg.

Key-words: Thyroid, Polyherbal Tablet, Screening

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Introduction

The two most common thyroid disorders are hyperthyroidism and hypothyroidism. Hyperthyroidism is characterised by an increased level of thyroid hormone in the blood, whereas hypothyroidism is described as thyroid hormone underproduction¹. Grave's disease, thyroid adenoma, multinodular goitre, and an excess of exogenous thyroid hormones are the most common causes of hyperthyroidism². Hypothyroidism can be caused by genetics or acquired through a lifestyle. Hashimoto's thyroiditis is the leading cause of hypothyroidism³. Thyroid diseases are among the most common and are generally dictated by the availability of an iodine-rich diet, which is required for the production of thyroxine and triiodothyronine hormones⁴. Several laboratory techniques have been developed to examine thyroid function in the body. The most common tests recommended by clinicians are TSH, T4, T3, and thyroid antibody tests⁵. Hyperthyroidism is mostly treated with antithyroid medications such as methimazole, which prevents the conversion of T4 to T3, as well as radioactive iodine, which accumulates specifically in the thyroid gland and destroys thyroid tissue. The final option for hyperthyroidism is subtotal thyroidectomy, which lowers thyroid mass (Reid and Wheeler,

2005). The primary treatment for hypothyroidism is thyroid hormone replacement therapy and continuous monitoring of thyroid levels in the bloodstream. Full thyroid hormone replacement with levothyroxine can be followed by the addition of T3, which helps improve the patient's mood and memory⁶. *Drimiopsis kirkii* remains a valued ornamental plant, its potential as a medicinal resource should not be overlooked, and further scientific investigation is essential to uncover its full therapeutic potential. Also, the diverse pharmacological activities of *Momordica charantia* support its traditional use in medicine and highlight its potential for future therapeutic applications⁷. Hence, these two plants were taken for the study to prepared a polyherbal tablet and screen the Antithyroid activity.

Material and Methods

Selection and Collection of Plants with Endocrine Potential

Two plants, *Drimiopsis kirkii* Baker's bulb and *Momordica charantia* Linn.'s fruits, were chosen for the current study because of their endocrine potential in diabetes treatment and thyroid level maintenance. These plants were taken from the Mhow region of Indore, Madhya Pradesh, India.

Authentication of Selected Plant

Botanist Dr. S. N. Dwivedi, Retd. Professor & HOD, Janata PG College & Visiting Professor, APS University, Rewa, Madhya Pradesh, India, verified the *Drimiopsis kirkii* Baker bulb and *Momordica charantia* Linn fruits. Voucher specimen numbers J/Bot/DKB-12 and J/Bot/MCF-13, dated 14/08/2023 were assigned.

Extraction of Plant Materials

The extraction was carried out using shade-dried coarsely powdered plant material, specifically *Drimiopsis kirkii* Baker's bulb and *Momordica charantia* Linn. fruits. 250 gm of powdered crude medication was extracted with ethanol:water (70:30) using a soxhlet device. After 48 hours, the filtrate was evaporated in a water bath until dry and stored in desiccators.

Acute Toxicity Studies of Extract

Five healthy male wistar rats were fasted for 3-4 hours before being subjected to acute toxicity experiments in accordance with OECD guidelines no. 425 (Up and Down Procedure)⁸.

Antithyroid screening of a polyherbal formulation (tablets)

Four groups of animals, six in each, were given the following treatment schedule.

Group I: Normal Diet (Control)

Group II: Animals were given 600µg/kg/ml of thyroxine for 14 days to induce hyperthyroidism.

Group III: Animals with hyperthyroidism were treated with the conventional medication (PTU propyl thio uracil 10mg/kg) for 21 days.

Group IV: Animals with hyperthyroidism were treated for 21 days with a polyherbal formulation (tablet) (500mg/kg).

The study included twenty-four Wistar male adult rats divided into four groups of six. The serum of the experimental rats was tested for thyroid hormone levels (ELISA METHOD) and lipid profile (KIT METHOD) before and during the experiment. Blood was collected via retro-orbital puncture while under light diethyl ether anaesthesia. Serum was separated by centrifugation at 2000 rpm for 15 minutes in a standard centrifuge and used for analysis. To generate hyperthyroidism in rats, Thyroxine (600µg/kg/ml) was administered orally for 14 days. Serum thyroid hormone levels and liver complications were analysed to validate the induction⁹.

Body and Organ Weights

Each rat's body weight was measured from the first day of the experiment to the last day. The rats are to be sacrificed at regular intervals using an automatic electronic balance. At sacrifice, the weight of the liver and left thyroid gland was measured at g levels (absolute weights). To decrease variances from individual body weights, the relative weight (% of g or mg/g body

weight) was calculated as [(absolute organ weight (g or mg)/body weight at sacrifice (g)) ×100].

Measurement of Food and Water Intake

All animals were monitored daily for clinical symptoms for 5 weeks after the first injection. Each rat's body weight and food consumption were measured at the start of therapy and once a week during the treatment period. Throughout the treatment period, the amounts of food and drink consumed were averaged weekly.

Estimation of total cholesterol, HDL, LDL, and VLDL

Lipid abnormalities may be attributed to a reduced thyroid capability. Prior to the availability of blood thyroid hormone estimates, serum cholesterol levels were used to aid in the diagnosis of thyroid issues. Total cholesterol, HDL, LDL, VLDL, and triglycerides were identified. Following twenty-one days of therapy with regular medication and polyherbal formulation (tablet), total cholesterol, HDL, and LDL levels increased, while VLDL and triglycerides decreased significantly and returned to normal levels.

Estimating Hormone Levels in Blood Serum

Blood samples for hormonal testing were taken from the deceased animal using a cardiac punch, and the samples were centrifuged at 2000 rpm for 15 minutes. Following this the serum was separated and deposited in micro centrifuge tubes and maintained in ice cold condition till analysis. The serum concentrations of triiodothyronine (T3), thyroxine (T4), and thyroid stimulating hormone (TSH) were then assessed.

Statistical analysis

All results were expressed as mean ± standard error of mean (SEM). The data was analysed using one-way ANOVA, followed by Dunnett's test. P-values < 0.05 were considered statistically significant.

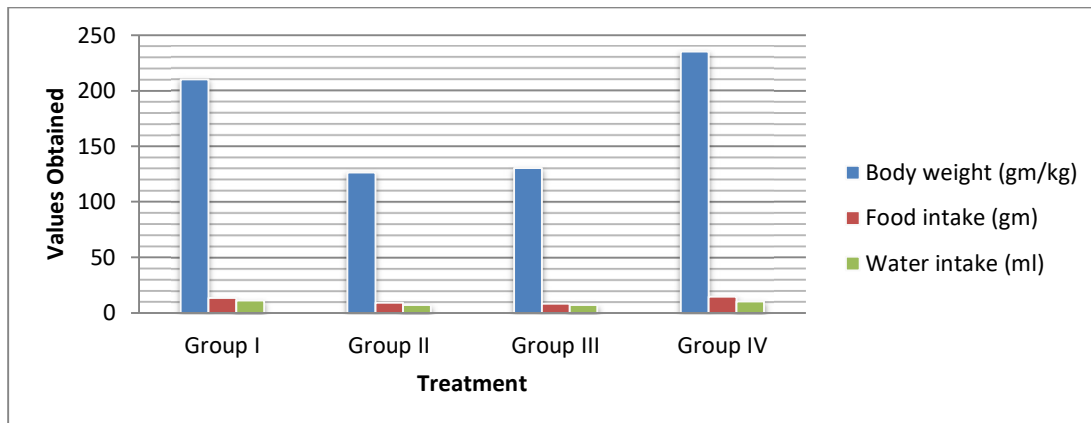
Results and Discussion

In the present experiment anti-thyroid screening of the polyherbal formulation tablet [PHF-T] containing hydro-alcoholic extract of bulb of *Drimiopsis kirkii* Baker and fruits of *Momordica charantia* Linn were reported. In this investigation, the animals were separated into four groups. Group I functioned as the control group, Group II was the thyroxine-induced group, Group III was the standard group, and Group IV was the test group. The various parameters were analysed and reported. Significant ($P < 0.01$) decreases in body weight, food and drink intake were observed. Treatment with PTU 10mg/kg and PFH-T 500mg/kg over 21 days significantly reduced body weight loss ($P < 0.01$ or $P < 0.05$) (Table 1). Lipid abnormalities may indicate decreased thyroid function. Prior to the availability of blood thyroid hormone assays, serum cholesterol levels were utilised to help diagnose thyroid disorders. Table 2 depicts the lipid profile of various experimental groups of rats. Total cholesterol, HDL, LDL, VLDL, and triglycerides were measured. In hyperthyroid rats, total cholesterol, HDL, and LDL levels fell while VLDL and triglycerides increased. After 21 days of therapy with conventional medication plus PFH-T 500mg/kg, total cholesterol, HDL, and LDL levels climbed, but VLDL and triglycerides reduced dramatically at the 5% level and returned to normal range. The control animals' T3, T4, and TSH levels were 1.03 ng/ml, 23.19 ng/ml, and 1.85 µg/ml, respectively. In hyperthyroid induced rats, T3 and T4 levels increased to 2.73 and 74.86 ng/ml, respectively, whereas TSH reduced considerably to 0.72 µg/ml. T3 and T4 levels declined to 0.93 and 32.06 ng/ml, respectively, whereas TSH increased to 1.46 µg/ml in rats treated with the usual medication. Rats treated with PHF-T at 500mg/kg showed a drop in T3 and T4 levels and an increase in TSH levels (Table 3). The group treated with 500mg/kg of PHF-T demonstrated highly significant results and was determined to be as effective as the standard treatment (PTU). The weight of the thyroid gland was considerably lower in the experimental group over the control group. Control rats had significantly lower relative thyroid gland weights ($P < 0.01$) than thyroxine-induced rats. Treatment with PTU and PHF-T resulted in much higher

declines in organ weights ($P<0.01$) compared to thyroxine-induced results (Table 4). Histopathological examinations were conducted to examine tissue sections of the thyroid gland from various experimental groups of rats. Histomorphometric analysis revealed significant ($P<0.01$) decreases in mean thicknesses of cross thyroid glands and follicular lining epithelium, together with significant ($P<0.01$) increases in hepatocyte counts compared to control rats. Treatment with PHF-T 500 mg/kg or PTU 10mg/kg significantly reduced the histological alterations associated with hyperthyroidism. (Figure 1).

Table 1: Body weight and intake in treatment groups

Group	Days	Body weight (gm/kg)	Food intake (gm)	Water intake (ml)
Group I	35	210.39±0.32	13.26±0.18	11.22±0.04
Group II	14 induced	152.85±0.18	11.92±0.04	8.23±0.10
	21 induced	126.29±1.10	9.10±0.06	7.19±0.18
Group III	14 induced	173.35±2.18	12.02±0.61	9.20±0.11
	21 treatment	130.29±1.18	8.26±0.52	7.22±0.67
Group IV	14 induced	275.18±3.14	21.02±0.36	15.17±0.02
	21 treatment	235.22±2.26	14.68±0.11	10.29±0.17



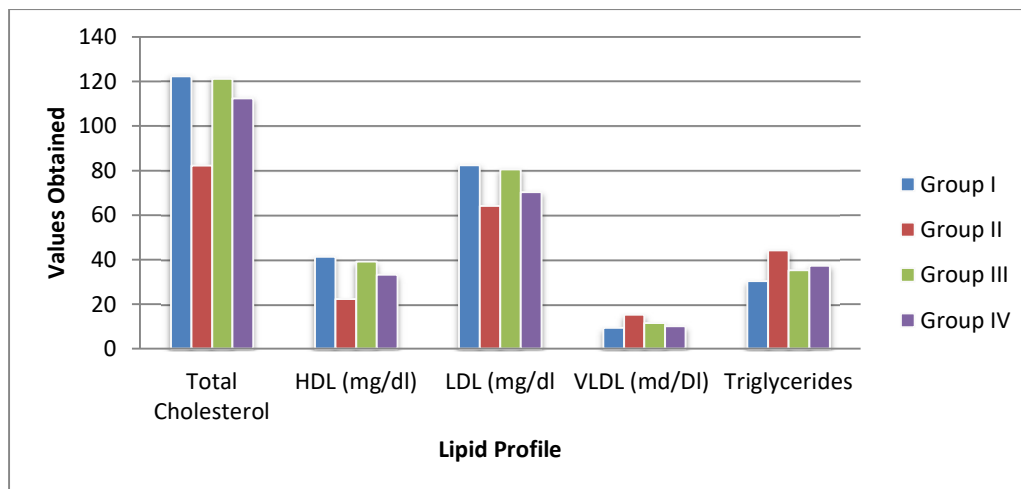
Graph 1: Body weight and intake in treatment group

Table 2: Effect on Lipid Profile

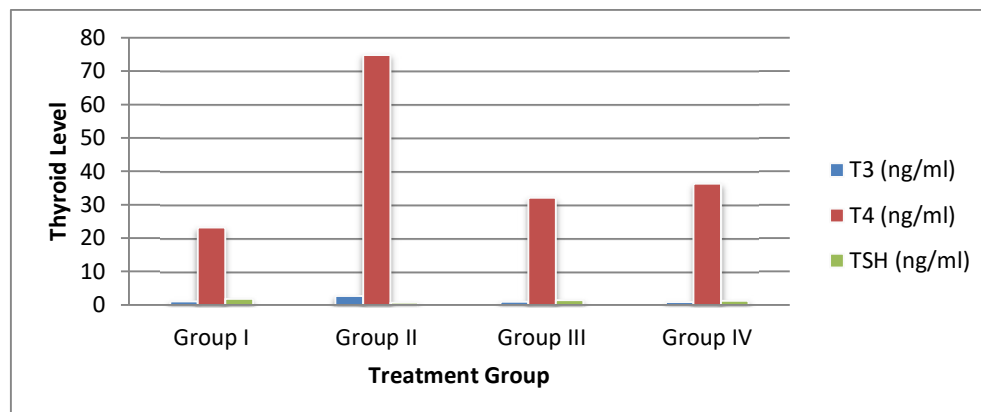
Group	Total Cholesterol	HDL (mg/dl)	LDL (mg/dl)	VLDL (md/Dl)	Triglycerides
Group I	122.29±0.19	41.26±0.11	82.48±0.28	9.39±0.82	30.48±0.22
Group II	82.26±0.17	22.32±0.18	64.10±0.37	15.36±0.03	44.19±0.04
Group III	121.22±0.11	39.06±0.18	80.58±0.72	11.47±0.39	35.25±0.11
Group IV	112.39±0.15	33.26±0.14	70.36±0.44	10.02±0.29	37.29±0.86

Table 3: Effect on T3, T4 and TSH Levels

Group	T3 (ng/ml)	T4 (ng/ml)	TSH (ng/ml)
Group I	1.03±0.39	23.19±0.83	1.85±0.87
Group II	2.73±0.67	74.86±0.21	0.72±0.29
Group III	0.93±0.48	32.06±0.20	1.46±0.53
Group IV	0.88±0.25	36.29±0.48	1.29±0.61



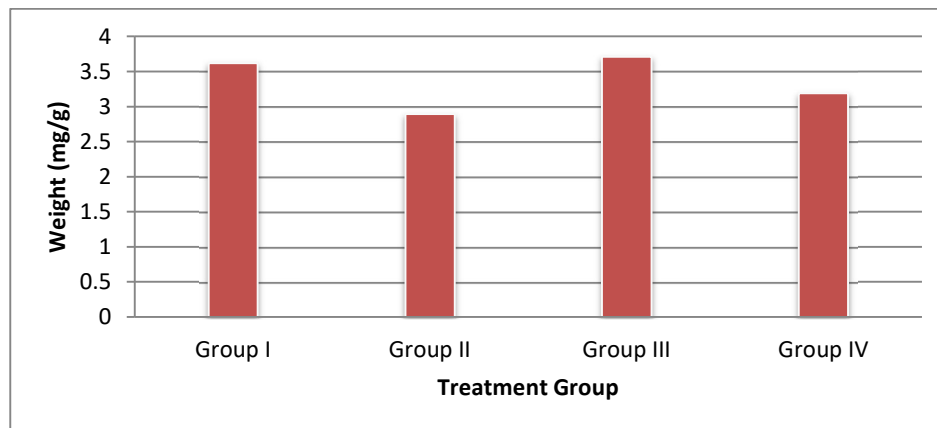
Graph 2: Lipid Profile in treatment group



Graph 3: Lipid Profile in treatment group

Table 4: Effect on Organ Weight

Group	Thyroid gland weight (mg/g)
Group I	3.62±0.17
Group II	2.89±0.42
Group III	3.71±0.63
Group IV	3.19±0.54



Graph 4: Thyroid gland weight in treatment group

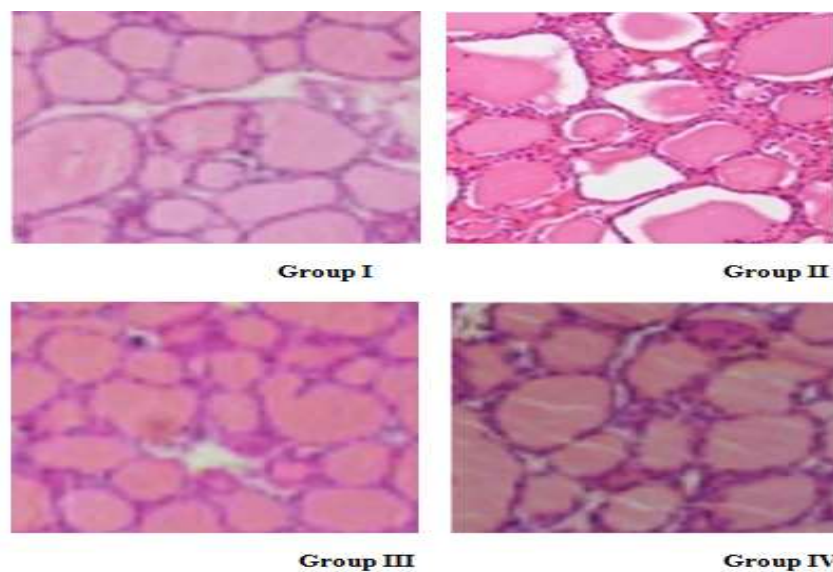


Fig. 1: Histopathology of Thyroid Gland

Conclusion

Rats treated with PHF-T at a dose of 500mg/kg had lower T3 and T4 levels and higher TSH levels. The group treated with 500mg/kg of PHF-T demonstrated significant results and was determined to be as effective as the standard treatment (PTU). Histopathological examinations were conducted on thyroid gland tissue sections from various experimental groups of rats. Treatment with PHF-T 500 mg/kg or PTU 10mg/kg significantly reduced hyperthyroid-related histological alterations in the thyroid gland.

References

1. Gessl, A., Lemmens-Gruber, R., & Kautzky-Willer, A. (2013). Thyroid Disorders. In V. Regitz-Zagrosek (Ed.), *Sex and Gender Differences in Pharmacology* (Vol. 214, pp. 361–386). Springer Berlin Heidelberg.
2. Khan, A., Khan, M. M. A., & Akhtar, S. (2002). Thyroid Disorders, Etiology and Prevalence. *Journal of Medical Sciences*, 2(2), 89–94.
3. Little, J. W. (2006). Thyroid disorders. Part II: Hypothyroidism and thyroiditis. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, 102(2), 148–153.

4. Vanderpump, M. P. J. (2019). Epidemiology of Thyroid Disorders. In M. Luster, L. H. Duntas, & L. Wartofsky (Eds.), *The Thyroid and Its Diseases* (pp. 75–85). Springer International Publishing.
5. Wellby, M. L. (1976). The Laboratory Diagnosis of Thyroid Disorders. In *Advances in Clinical Chemistry* (Vol. 18, pp. 103–172). Elsevier. [https://doi.org/10.1016/S0065-2423\(08\)60297-8](https://doi.org/10.1016/S0065-2423(08)60297-8).
6. Hueston, W. J. (2001). Treatment of Hypothyroidism. *American Family Physician*, 64(10), 1717.
7. Dwivedi D. (2009). Status survey of medicinal plants wealth of Malwa region of Madhya Pradesh with special reference to conservation of vulnerable and endangered species, *J. Econ. Taxon. Bot.*, 33(2): 443-452.
8. Sewell, F., Ragan, I., Horgan, G., Andrew, D., Holmes, T., Manou, I., ... & Corvaro, M. (2024). New supporting data to guide the use of evident toxicity in acute oral toxicity studies (OECD TG 420). *Regulatory Toxicology and Pharmacology*, 146, 105517.
9. Samy, J. V. R. A., Sayanam, R. R. A., Balasubramanian, C., Vijayakumar, N., Kim, S. J., Vijayakumar, S. & Hazazi, B. T. (2023). Effect of a polyherbal formulation on L-thyroxine induced hyperthyroidism in a rat model: In vitro and in vivo analysis and identification of bioactive phytochemicals. *International Journal of Biological Macromolecules*, 237, 124140.