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Comparative Effects of Ginger and Ashwagandha Extracts, Alone and Combined, Versus Levothyroxine on Thyroid Hormone Recovery in PTU-Induced Hypothyroid Female Rats Treated During Pregnancy

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ABSTRACT:

Introduction: Maternal hypothyroidism reduces thyroid hormone availability during a period of increased endocrine demand. Objective: To compare 70% ethanolic extracts of Zingiber officinale (ginger) and Withania somnifera (ashwagandha), alone or combined, with levothyroxine in PTU-induced hypothyroid female rats treated during pregnancy.

Methods: Sixty adult female albino rats were allocated to six groups (10/group). G1 received water, whereas G2-G6 received propylthiouracil (PTU; 50 mg/kg/day) orally for 28 days. Hormonal analyses were based on n=3 animals/group in each phase. After confirmation of hypothyroidism and pregnancy, G1 remained the healthy control and G2 the untreated hypothyroid control. During the 21-day gestational treatment period, G3 received levothyroxine (20 µg/kg/day), G4 ginger (200 mg/kg/day), G5 ashwagandha (200 mg/kg/day), and G6 both extracts (200 mg/kg/day each). Serum T3, T4 and TSH were measured by ELISA.

Results: PTU decreased T3 and T4 and increased TSH before treatment. One day after parturition, G2 retained marked dysfunction (T3 0.893 ± 0.05 ng/mL; T4 1.94 ± 0.20 µg/dL; TSH 11.00 ± 1.10 µIU/mL). All treatments improved the three indices versus G2. In G3, T3 and TSH were not significantly different from G1, while T4 was higher. In G4, T3 and TSH were not significantly different from G1, while T4 remained lower. In G5 and G6, T3, T4 and TSH were not significantly different from G1. G6 was not significantly different from G5 for any thyroid index.

Conclusions: Ginger and ashwagandha improved the PTU-induced hormonal disturbance. Ashwagandha alone and the combination produced the broadest control-comparable profiles, but the data did not demonstrate pharmacological synergy.

1. INTRODUCTION

Thyroid hormones are central regulators of metabolism, growth and tissue development, and their physiological importance is particularly evident during pregnancy. Maternal thyroxine (T4) and triiodothyronine (T3) contribute to fetal development before fetal thyroid function is fully established, while pregnancy alters maternal thyroid physiology and increases endocrine

demand [1,2]. Inadequate maternal thyroid hormone availability may affect maternal homeostasis and fetal development, making recognition and treatment of hypothyroidism during pregnancy important [3].

Primary hypothyroidism is characterized by reduced thyroid hormone production and a compensatory increase in thyroid-stimulating hormone (TSH). Propylthiouracil (PTU) is widely used experimentally because it inhibits thyroid peroxidase-dependent reactions involved in thyroid hormone synthesis and also reduces peripheral conversion of T4 to the more biologically active T3 [4]. The fall in circulating T3 and T4 weakens hypothalamic-pituitary negative feedback and consequently increases TSH [5].

Levothyroxine, a synthetic form of T4, is the standard replacement treatment for hypothyroidism [6]. Its effectiveness is based on replenishment of the circulating T4 pool and subsequent peripheral conversion to T3. Experimental comparisons with plant extracts remain useful for examining whether non-hormonal interventions can modify thyroid hormone recovery in induced hypothyroidism.

Ginger *(Zingiber officinale*) and ashwagandha (*Withania somnifera*) are medicinal plants with chemically diverse extracts. In the present experiment, HPLC characterization of the 70% ethanolic extracts demonstrated distinct phytochemical profiles. Ginger contained gallic, ferulic, caffeic and p-coumaric acids as well as rutin, chlorogenic acid and quercetin, whereas ashwagandha shared several of these constituents and additionally contained sinapic and vanillic acids. These analytical findings characterize the whole extracts used experimentally; they do not, by themselves, prove the mechanism responsible for any endocrine effect.

Previous experimental studies have reported thyroid-related responses to both plants. Ginger improved thyroid hormone disturbances in different experimental models [7,8], and a PTU-based study in female rats reported increases in T3 and T4 after ginger administration [9]. Ashwagandha improved T3, T4 and TSH in PTU-induced hypothyroid rats [10], while recent work also reported improvement of thyroid hormone profiles after *Withania somnifera* root extract [11]. Evidence directly comparing ginger, ashwagandha, their combination and levothyroxine within the same pregnancy-associated PTU model remains limited.

Therefore, this study aimed to compare the effects of 70% ethanolic ginger and ashwagandha extracts, administered individually or together, with conventional levothyroxine treatment on serum T3, T4 and TSH in pregnant female rats after PTU-induced hypothyroidism. The study also examined whether the combined extract produced a more complete hormonal recovery than either extract alone.

2. MATERIALS AND METHODS

2.1 Preparation and characterization of plant extracts

Ginger and ashwagandha were obtained from a local herbal market. Homogenized plant material (20 g) was defatted with 100 mL chloroform on an electric vibrator for 3 h. After removal of the chloroform layer and drying at 50 °C, 10 g of dried sample was extracted with ethanol/water (70/30, v/v) in an ultrasonic bath at room temperature for 1 h. After filtration, solvent was removed under vacuum using a rotary evaporator and the extract was dried at 40 °C to constant mass and stored at 4 °C. Individual phenolic compounds were quantified by reversed-phase HPLC using a SYKAM system with UV detection and a C18-OSD column (25 cm × 4.6 mm), following the published reversed-phase HPLC procedure [12]. Total phenolic content was determined by the Folin-Ciocalteu method with absorbance at 765 nm and gallic acid calibration [13]. Total flavonoid content was determined by the aluminium-chloride colorimetric method with absorbance at 510 nm [14].

2.2 Experimental animals and housing

Sixty adult female albino rats, 8-10 weeks old and weighing approximately 250-270 g, were obtained from the animal house of the Biotechnology Research Center, Al-Nahrain University. Animals were housed in plastic cages with metal mesh lids under controlled laboratory conditions (22-25 °C; 40-60% relative humidity; continuous access to feed and water). The animals were randomly divided into six groups, with 10 rats initially allocated to each group. The hormonal endpoints reported in this manuscript were based on n=3 animals per group in each experimental phase, and the two phases were analysed separately.

2.3 Induction of hypothyroidism

During the first experimental phase, G1 served as the negative control and received water only. Hypothyroidism was induced in G2-G6 by oral gavage of PTU at 50 mg/kg/day for 28 consecutive days. At the end of this period, serum thyroid

hormones were evaluated to confirm induction. Importantly, G2-G6 were induction groups only during this phase; no levothyroxine, ginger, ashwagandha or combined treatment had yet been administered. PTU administration was limited to the 28-day induction period.

2.4 Mating and treatment during pregnancy

After confirmation of hypothyroidism, females were mated with healthy males at a 1:1 ratio. Early pregnancy was confirmed by the presence of a vaginal plug and/or detection of sperm in a vaginal swab. Once pregnancy was confirmed, the second experimental phase began. G1 remained the healthy negative control and continued to receive water. G2 served as the untreated hypothyroid positive control. G3 received levothyroxine at 20 µg/kg/day, G4 received ginger extract at 200 mg/kg/day, G5 received ashwagandha extract at 200 mg/kg/day, and G6 received both ginger and ashwagandha extracts at 200 mg/kg/day each. Treatments were administered orally by gavage for 21 days throughout pregnancy. The second blood collection was performed one day after parturition; therefore, the second-phase hormone concentrations represent the one-day postpartum sampling point after treatment throughout gestation rather than hormone concentrations measured during pregnancy itself.

2.5 Blood collection and thyroid hormone assays

At each experimental phase, the animals selected for hormonal examination were anesthetized by chloroform inhalation until cessation of movement. Blood was then collected by cardiac puncture, transferred to serum-separator gel tubes and centrifuged at 2000 rpm for 10 min to obtain serum. Serum T3, T4 and TSH concentrations were measured using pre-prepared assay kits according to the manufacturer's instructions and the ELISA technique.

2.6 Statistical analysis

Statistical analysis was performed using SAS (Statistical Analysis System). The least significant difference (LSD) test was used for comparison between group means. Results are presented as mean ± standard error (SE). Differences were considered significant at $P \leq 0.05$ and highly significant at $P \leq 0.01$. Hormonal analyses in each experimental phase were based on n=3 animals/group.

3. RESULTS

3.1 Extract characterization

The two 70% ethanolic extracts showed distinct descriptive phenolic and flavonoid profiles. Ginger recorded higher numerical total phenolic and flavonoid contents than ashwagandha (137.58 vs 112.6 mg Gallic/100 gm and 47.9 vs 21.6 mg Rutin/100 gm, respectively). Gallic acid, ferulic acid, caffeic acid, rutin, quercetin and p-coumaric acid were quantified in both extracts. Sinapic and vanillic acids were recorded in ashwagandha, whereas pyrogallol, Hydrobenzoic acid, chlorogenic acid and ellagic acid were recorded in ginger under the applied analytical conditions. These were single analytical values without measures of dispersion or statistical testing; therefore, differences are descriptive rather than inferential.

Table 1. Phenolic and flavonoid characterization of the 70% ethanolic ginger and ashwagandha extracts.

Phytochemical/compound	Ginger	Ashwagandha
Total phenolic content (mg Gallic/100 gm)	137.58	112.6
Total flavonoid content (mg Rutin/100 gm)	47.9	21.6
Gallic acid (µg/gm)	55.9	41.5
Pyrogallol (µg/gm)	18.9	–
Hydrobenzoic acid (µg/gm)	20.6	–
Ferulic acid (µg/gm)	32.6	27.9
Caffeic acid (µg/gm)	12.9	11.5
Sinapic acid (µg/gm)	–	13.6

Phytochemical/compound	Ginger	Ashwagandha
Vanillic acid (µg/gm)	–	9.5
Rutin (µg/gm)	33.2	21.6
Chlorogenic acid (µg/gm)	17.9	–
Quercetin (µg/gm)	10.6	6.9
Ellagic acid (µg/gm)	10.3	–
p-coumaric acid (µg/gm)	12.6	11.6

Values are descriptive analytical measurements. A dash indicates that no corresponding value was reported under the applied analytical conditions and should not be interpreted as definitive proof of complete absence.

3.2 Thyroid hormone profile after PTU induction

Oral PTU administration for 28 days established a consistent hypothyroid hormonal pattern before mating and before therapeutic intervention. Compared with G1, all PTU-exposed groups (G2-G6) had significantly lower serum T3 and T4 and significantly higher TSH. T3 differed among groups at P=0.0001, T4 at P=0.0001 and TSH at P=0.0155. All PTU-exposed groups carried the same statistical letter for each hormone, indicating no significant differences among the induction groups before treatment. Hormonal analyses in this phase were based on n=3 animals/group.

Table 2. Serum T3, T4 and TSH after 28 days of PTU induction.

Group	T3 (ng/mL)	T4 (µg/dL)	TSH (µIU/mL)
G1	3.63 ± 0.17 a	5.86 ± 0.20 a	1.70 ± 0.21 b
G2	1.90 ± 0.26 b	1.40 ± 0.11 b	3.30 ± 0.45 a
G3	1.88 ± 0.14 b	1.43 ± 0.11 b	3.38 ± 0.31 a
G4	1.60 ± 0.21 b	1.33 ± 0.22 b	3.46 ± 0.28 a
G5	1.90 ± 0.06 b	1.33 ± 0.08 b	3.50 ± 0.32 a
G6	1.73 ± 0.14 b	1.67 ± 0.17 b	3.40 ± 0.32 a
LSD	0.532 **	0.485 **	1.017 *
P value	0.0001	0.0001	0.0155

Values are mean ± SE (n=3/group). Means with different letters within the same column differed significantly. *P ≤ 0.05; **P ≤ 0.01. During this phase G2-G6 were PTU induction groups only; therapeutic treatments had not yet begun.

3.3 Thyroid hormone profile after gestational treatment

After the 21-day gestational treatment period, the second blood sample was collected one day after parturition. Group differences were highly significant for T3 (P=0.0006), T4 (P<0.0001) and TSH (P<0.0001), with LSD values of 0.929, 1.596 and 1.559, respectively. G1 recorded T3 of 3.50 ± 0.20 ng/mL, T4 of 6.77 ± 0.17 µg/dL and TSH of 2.72 ± 0.25 µIU/mL. G2 showed the most severe residual disturbance (T3 0.893 ± 0.05 ng/mL; T4 1.94 ± 0.20 µg/dL; TSH 11.00 ± 1.10 µIU/mL). All treated groups differed significantly from G2 for the three thyroid indices.

In G3, T3 (3.38 ± 0.65 ng/mL) and TSH (3.99 ± 0.28 µIU/mL) were not significantly different from G1, whereas T4 (9.36 ± 1.01 µg/dL) was significantly higher. In G4, T3 (2.91 ± 0.09 ng/mL) and TSH (1.83 ± 0.28 µIU/mL) were not significantly different from G1, whereas T4 (4.96 ± 0.38 µg/dL) remained significantly lower. In G5, T3 (3.01 ± 0.07 ng/mL), T4 (7.63 ± 0.27 µg/dL) and TSH (2.23 ± 0.20 µIU/mL) were not significantly different from G1. G6 showed the same pattern: T3 3.01 ± 0.26 ng/mL, T4 7.36 ± 0.55 µg/dL and TSH 2.26 ± 0.26 µIU/mL were not significantly different from G1. Compared with G4, G6 had significantly higher T4, while T3 and TSH did not differ significantly. G6 did not differ significantly from G5 for T3, T4 or TSH.

Table 3. Serum T3, T4 and TSH one day after parturition following treatment throughout the 21-day gestational period.

Group	T3 (ng/mL)	T4 (µg/dL)	TSH (µIU/mL)
G1	3.50 ± 0.20 a	6.77 ± 0.17 b	2.72 ± 0.25 bc
G2	0.893 ± 0.05 b	1.94 ± 0.20 d	11.00 ± 1.10 a
G3	3.38 ± 0.65 a	9.36 ± 1.01 a	3.99 ± 0.28 b
G4	2.91 ± 0.09 a	4.96 ± 0.38 c	1.83 ± 0.28 c
G5	3.01 ± 0.07 a	7.63 ± 0.27 b	2.23 ± 0.20 c
G6	3.01 ± 0.26 a	7.36 ± 0.55 b	2.26 ± 0.26 c
LSD	0.929 **	1.596 **	1.559 **
P value	0.0006	<0.0001	<0.0001

Values are mean ± SE (n=3/group). Means with different letters within the same column differed significantly. **P ≤ 0.01. G1, negative control; G2, untreated hypothyroid control; G3, levothyroxine 20 µg/kg/day; G4, ginger 200 mg/kg/day; G5, ashwagandha 200 mg/kg/day; G6, ginger 200 mg/kg/day + ashwagandha 200 mg/kg/day.

4. DISCUSSION

The present study demonstrated two major findings. First, oral PTU at 50 mg/kg/day for 28 days produced a clear hypothyroid hormonal phenotype in adult female rats before mating, characterized by reductions in T3 and T4 and a compensatory elevation in TSH. Second, levothyroxine, ginger, ashwagandha and the ginger-ashwagandha combination all improved thyroid hormone status compared with the untreated hypothyroid group, although each treatment produced a distinct pattern of recovery.

The induction phase provides the baseline for interpreting treatment responses. All PTU-exposed groups showed the same direction of change and the same significance grouping for T3, T4 and TSH before treatment, indicating no significant group-level differences in the severity of hormonal disturbance at that stage. The pattern agrees with Rongala et al. [15], who reported decreased T3 and T4 with increased TSH after PTU in female Wistar rats, and with Singh et al. [16], who observed the same hormonal direction in PTU-induced thyroid toxicity.

The physiology of this response is consistent with the known actions of PTU. Thyroid peroxidase is required for iodide oxidation, organification and coupling during thyroid hormone synthesis. Inhibition of these reactions reduces new hormone production, while PTU also limits peripheral conversion of T4 to T3 [4]. The fall in circulating thyroid hormones weakens hypothalamic-pituitary negative feedback and promotes TSH secretion [5]. The elevated TSH therefore represents a compensatory response that cannot fully overcome the pharmacological inhibition of hormone synthesis.

One day after parturition, G2 retained pronounced hormonal dysfunction even though PTU had been limited to the induction phase. The low T4 suggests incomplete recovery of thyroid synthetic and secretory capacity, while the low T3 is consistent with limited thyroidal contribution and reduced T4 substrate for peripheral T3 formation. Persistently high TSH indicates continued inadequate negative feedback. A comparable direction of thyroid suppression during reproductive states was reported by Hapon et al. [17].

Levothyroxine produced a strong hormonal response. T3 and TSH were not significantly different from the healthy control, whereas T4 rose significantly above G1. Exogenous levothyroxine directly expands the circulating T4 pool and provides substrate for peripheral conversion to T3, explaining the recovery of T3. The fall in TSH relative to G2 is consistent with re-establishment of thyroid-hormone-mediated pituitary feedback, although the high T4 indicates that the overall profile did not reproduce the healthy control pattern. The increases in T3 and T4 are consistent with Dizay et al. [9], who reported significant increases in both hormones after levothyroxine treatment in hypothyroid rats.

Ginger significantly improved all three thyroid indices compared with G2. T3 and TSH were not significantly different from G1, whereas T4 remained significantly lower. This pattern suggests substantial recovery of biologically active thyroid

hormone availability and pituitary-thyroid feedback, with less complete recovery of the circulating T4 pool at the one-day postpartum sampling point.

The ginger response is supported by experimental studies, although the models are not identical. Dizay et al. [9] reported increases in T3 and T4 after ginger in PTU-induced hypothyroid female rats. Mohammed et al. [8] reported improvement in T3, T4 and TSH in a bisphenol-A model of thyroid disruption, while Al-Amoudi [7] reported improvement of the same indices in lambda-cyhalothrin-induced thyroid injury. Differences in inducing agent, extraction procedure, dose and physiological state limit direct numerical comparison.

The ginger extract contained gallic acid, ferulic acid, caffeic acid, rutin, chlorogenic acid, quercetin and p-coumaric acid, and it had higher numerical total phenolic and flavonoid contents than the ashwagandha extract. These findings provide chemical context for the whole extract and support the plausibility of cellular-protective and redox-related contributions discussed in previous ginger studies [7,8]. However, oxidative stress, deiodinase activity and thyroid peroxidase activity were not measured in the present experiment; therefore, the hormonal response should be attributed to the administered extract as a whole rather than to a specific compound or unmeasured mechanism.

Ashwagandha produced substantial recovery of the measured thyroid profile. T3, T4 and TSH were all significantly improved versus G2 and were not significantly different from G1. The T3 and T4 responses are consistent with improved circulating thyroid hormone availability, while the TSH response suggests recovery of negative-feedback regulation. The study design cannot determine whether these changes resulted from altered synthesis, secretion, peripheral metabolism or a combination of these processes.

This finding agrees with Abdel-Wahhab et al. [10], who reported that methanolic ashwagandha extract improved T3 and T4 and reduced TSH in PTU-induced hypothyroid rats. Hossain et al. [11] likewise reported improvement of PTU-induced thyroid hormone disturbances after Withania somnifera root extract. A partially different response was reported by Panda and Kar [18], who found increased serum T4 without a significant increase in T3 in female mice. Species, dose, initial thyroid status and experimental design may account for part of this variation.

The combination also significantly improved T3, T4 and TSH versus G2, and none of the three values differed significantly from G1. Compared with ginger alone, the combination produced significantly higher T4, whereas T3 and TSH did not differ significantly. Compared with levothyroxine, the combination showed no significant difference in T3 but had significantly lower T4 and TSH. Thus, the combination produced a broad hormonal recovery without the marked T4 elevation observed in G3.

Importantly, G6 did not differ significantly from ashwagandha alone for T3, T4 or TSH. The data therefore support an effective combined response but do not demonstrate pharmacological synergy or superiority over ashwagandha alone. No directly comparable study was identified in the literature reviewed for this study in which ginger and ashwagandha were administered together throughout pregnancy in PTU-induced hypothyroid rats with simultaneous measurement of T3, T4 and TSH.

The phytochemical profiles provide a plausible context for the combined response. Ginger had higher numerical total phenolic and flavonoid contents, whereas ashwagandha shared several phenolic constituents and also contained sinapic and vanillic acids. Co-administration therefore exposed the animals to overlapping but non-identical chemical profiles. Complementary effects are biologically plausible, but the present study did not measure pharmacokinetic interactions, oxidative stress, thyroid enzyme activity or gene expression; a molecular mechanism for the combination therefore cannot be established from these data.

The timing of the second sample is important for interpretation. Treatments were given throughout the 21-day gestational period, but blood was collected one day after parturition. The second-phase values therefore represent the one-day postpartum sampling point rather than hormone concentrations measured during gestation itself.

Taken together, the treatment patterns were distinct. Levothyroxine yielded T3 and TSH values not significantly different from G1 but increased T4 above control. Ginger yielded T3 and TSH values not significantly different from G1 while T4 remained lower. Ashwagandha and the combination each produced T3, T4 and TSH values that were not significantly different from G1. Because the combination did not differ significantly from ashwagandha alone, the results do not confirm synergy.

5. STUDY LIMITATIONS

This study evaluated circulating T3, T4 and TSH but did not directly measure thyroid peroxidase, deiodinase activity, oxidative stress markers, inflammatory mediators, thyroid-related gene expression or pharmacokinetics. The second blood sample was obtained one day after parturition, so treatment-phase values represent the one-day postpartum sampling point. Finally, the combination did not differ significantly from ashwagandha alone for the three thyroid indices and therefore does not provide statistical evidence of synergy.

6. CONCLUSION

PTU administered orally at 50 mg/kg/day for 28 days produced a consistent hypothyroid hormonal pattern in adult female rats. Treatment during pregnancy with levothyroxine, ginger, ashwagandha or the ginger-ashwagandha combination significantly improved T3, T4 and TSH compared with untreated hypothyroid animals. Levothyroxine yielded T3 and TSH values not significantly different from control but increased T4 above control. Ginger yielded T3 and TSH values not significantly different from control while T4 remained lower. Ashwagandha and the combination each produced T3, T4 and TSH values not significantly different from the healthy control, but the combination was not significantly superior to ashwagandha alone. Further studies with direct mechanistic measurements are required before clinical interpretation.

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