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Associations Between Serum Phthalates and Thyroid Hormone Levels in Long-Term Plastic Using Female Volunteers

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KEYWORDS

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ABSTRACT:

Introduction: Phthalates are a class of industrial chemicals that are often employed as softeners and plasticizers in a variety of commercial items, such as toys, furniture, medical equipment, food packaging materials, and cosmetics. Phthalates can be inhaled, consumed, or come into contact with the skin. Phthalates undergo quick metabolism to produce their corresponding monoesters and oxidative metabolites, which are then eliminated in the urine and faeces.

Objectives: This study investigates the effect of serum phthalate on Thyroid hormones in women volunteers using plastics for short and long-term.

Methods: 2500 female participants in the reproductive age range of 20–45 years were given questionnaires. Women participants are divided into two groups based on their age and level of exposure to plastic products: Group I is for short-term exposure to plastics, aged 20–32, and Group II is for long-term exposure to plastics, aged 33–45.

Under **short-term plastic usage**, 60 women were selected:

- **Group I (Control):** 30 women without hormonal disorders (thyroid dysfunctions).
- **Group I (Test):** 30 women with hormonal disorders (thyroid dysfunctions).

Under **long-term plastic usage**, 60 women were selected:

- **Group II (Control):** 30 women without hormonal disorders (thyroid dysfunctions).
- **Group II (Test):** 30 women with hormonal disorders (thyroid dysfunction)

Phthalates' existence was examined. The serum levels of Triiodothyronine (T3), Thyroxine (T4) and Thyroid stimulating hormone (TSH) were determined for the control and test groups through competitive chemiluminescent immuno assay.

Results: The mean ± standard deviation (SD) was used to express the results. The one-way analysis of variance (ANOVA) was used to determine the statistical differences between each test group and the controls. The threshold for statistical significance was set at $P<0.05$.Results on quantification of phthalate shows the significant differences in the concentrations of phthalate esters in between the group I and II. However, among the identified phthalates, DMP, DEP, DINP, DONP and DBP were below their quantification limit in the case of group I, whereas in the case of group II, all the 9 phthalates (DMP, DEP, DBP, DHP, BBP, DBEP, DEHP, DINP and DNOP) being detected. From the results we inferred that majority of phthalates residues showed a remarkable indication in group II advanced age, whereas three main low molecular weights phthalates such as Diethyl phthalate (DEP), Dimethyl phthalate (DMP) and Di butyl phthalate (DBP) showed their indication in group I when compared with group II.A significant rise in Triiodothyronine (T3), Thyroxine (T4) and Thyroid stimulating hormone (TSH) was also seen in Group II..

Conclusion: This work gives information on how exposure to phthalates affects the thyroid system and potential underlying mechanisms. While more human research is necessary to confirm our findings, more

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experimental research is also required to confirm these findings and to learn more about the underlying mechanisms and consequences of thyroid system interference on humans. Natural and herbal products should be used in place of plastics that contain phthalates.

1. Introduction

Phthalates are synthetic chemical compounds that are pseudo-persistent in nature and are commonly found in plastics and personal care products, including food packaging materials, building materials, furniture, and cosmetics [1]. These compounds are widely detected in various bodily fluids and can enter the human body through oral, respiratory, and dermal exposure routes [2].

Phthalates are diesters of phthalic acid and have been shown to disrupt the thyroid system by altering the expression of genes associated with the hypothalamic–pituitary–thyroid (HPT) axis, thyroid hormone transport, metabolism, and action, as demonstrated in experimental studies [3]. Furthermore, certain experimental findings suggest that phthalate exposure may aggravate or exacerbate thyroid autoimmunity. Given their extensive use in commercial products, the general population is continuously exposed to these chemicals. However, epidemiological evidence linking phthalate exposure to thyroid function during non-developmental stages remains limited. Therefore, the present study aims to evaluate the correlation between specific serum phthalate metabolites and markers of thyroid function [4].

Recent research indicates that early-life exposure to certain phthalates is associated with reduced thyroid function in women. Thyroid hormones are essential for the normal functioning of every cell in the body and play a critical role during developmental stages. They are particularly important for brain development, thermoregulation, and the proper functioning of the heart and other vital organs [5].

Diet has also been identified as an important contributor to elevated levels of high molecular weight (HMW) phthalates in blood [6]. Therefore, assessing both low molecular weight (LMW) and HMW phthalate esters along with thyroid hormone levels in women with short-term and long-term plastic exposure may provide valuable insights into exposure pathways and their potential associations.

Although much of the existing research has focused on children and men, women may be at increased risk of adverse health effects due to greater use of cosmetics and personal care products. Diethyl phthalate and dibutyl phthalate are particularly prevalent in such products. In vivo and observational studies by Lopez-Carillo *et al.* [7] have demonstrated an association between phthalate exposure and endocrine disruption, potentially contributing to breast cancer development. Moreover, endocrine disruptors such as phthalates may exert additive effects, whereby even low-level exposures interact with other chemicals to produce cumulative “cocktail effects.”

Population-based studies have reported moderate to significant correlations between phthalate metabolite concentrations in serum and urine, suggesting that both matrices can serve as reliable biomarkers of human exposure. Lovekamp-Swan et al. reported that urinary concentrations of MEHP, a primary metabolite of DEHP, were negatively associated with free thyroxine (fT4) and total thyroid hormone levels in 408 men. Subsequently, several epidemiological studies across diverse populations have supported an association between DEHP exposure and alterations in thyroid hormone levels. Serum concentrations of free thyroxine (T4) and total triiodothyronine (T3) in adult men were also found to be negatively associated with DEHP metabolite levels [8].

Kuo FC *et al.,* [9] reported that phthalates are well-recognized endocrine-disrupting chemicals capable of interfering with thyroid hormones through multiple mechanisms. Since 2000, numerous studies have investigated the relationship between phthalate exposure and thyroid hormone regulation, encompassing both epidemiological research and mechanistic studies in animal models. Kenji Moriyama’s article, “Thyroid hormone action is disrupted by phthalate as an antagonist,” significantly advanced understanding in this field. Recent research directions continue to explore endocrine disruption,

Journal of Chemical Health Risks
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particularly the impact of chemicals such as BPA on thyroid hormones in vulnerable populations, including pregnant women and young children.

2. Objectives

In recent decades, plastics have become ubiquitous, and plasticizers, which are utilized as crosslinking agents in plastic products, have the ability to leak and penetrate biological systems. This study attempts to determine the correlation between the duration of exposure and the blood levels of phthalate metabolites in female volunteers who have been exposed to plastic for both short-term and long-term periods of time.

The initial step was to circulate the questionnaire to categorize women participants based on their plastic exposure relating to age, then identify women participants with hormonal dysfunction. and analyze the level of hormones T3, T4, and TSH in control and test groups. From this angle, we conducted this study in women with PCOS and thyroid disorders to measure the levels of various phthalate metabolites in serum and to analyze if they differ from the healthy women and the presence of phthalate metabolites in their blood samples.

3. Methods:

Ethical clearance for the following was sought from ARC Fertility Centre's Institutional Ethical Clearance Board and received (ARCIEC/others/002/2021). Questionnaires were circulated to 2500 women volunteers of reproductive age, ranging from 20 to 45 years. Based on their age and exposure to plastic products, woman participants are categorized
in to two groups

- Short-term exposure to plastics/plasticizers in 20-32-year-old women as group I
- Long-term exposure to plastics/plasticizers in 33-45-year-old women as group II.

Under short-term plastics usage, 30 women participants without hormonal disorders (thyroid Dysfunctions) were selected for the study and they are grouped as
Group I—Control: Under short-term plastics usage, 30 women participants with hormonal disorders (thyroid dysfunctions) were selected for the study, and they are grouped as Group I (Test).

Under long-term plastics usage, 30 women participants without hormonal disorders (thyroid dysfunctions) were selected for the study, and they are grouped as Group II (Control). Under long-term plastics usage, 30 women participants with hormonal disorders (thyroid dysfunctions) were selected for the study.

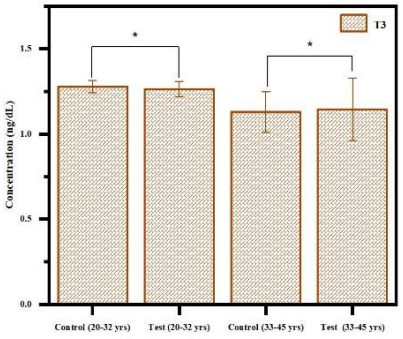
Blood Sample Collection:

5 ml of the whole blood samples were collected from each woman participant. The collected blood samples were transported in a cooling pail and centrifuged at 3000 rpm for 15 mins within 24 hours of collection. The serum was kept frozen in a glass vial at -80°C in a deep freezer until the phthalates were analyzed. The blood samples were analyzed for hormones such as T3, T4, and TSH, and phthalate ester analysis was collected from the test and control groups in the morning during the early follicular phase (second to fifth day) of a spontaneous menstrual cycle. In the present work, we conducted a study among women with hormonal disorders (thyroid abnormalities) of two different age groups (20-32 years and 33-45 years) to examine the levels of thyroid hormone imbalance.

4. Result:

Level of Triiodothyronine (T3) in Group I and Group II:

In Group I participants, the level of T3 in test participants was found to be 1.264 ng/dL when compared to control (1.279 ng/ dL).Whereas, in Group II the level of T3 was found to be 1.145 ng/ dL for test participants and 1.13 ng/ dL for the control participants. The level of T3 between control and test participants among two groups was depicted in Figure 1.



Values are Mean ± SD (n=30); * =Significance at 5% level (P<0.05)

Journal of Chemical Health Risks
www.jchr.org
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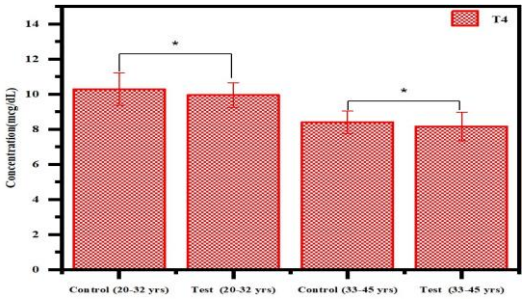
Fig 1. Levels of T3 hormone in Control (20-32 yrs 33-45 yrs), Group I (20-32 yrs) and Group II(33-45yrs).

Level of Thyroxine (T4) in Group I and Group II: The level of T4 between control and test participants among two groups was depicted in Fig 2. The level of T4 in group I, test participants was found to be mcg/dL, whereas for the control participants it was found to be 10.28 mcg/dL. In contrast to this the level of T4 in group II control participants was about 8.4 mcg/dL with that of test participants as 8.16mcg/dL.

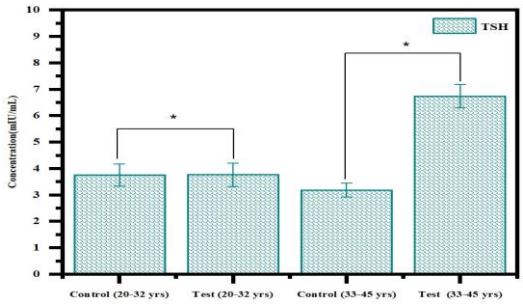


Values are Mean ± SD (n=30); * =Significance at 5% level (P<0.05)

Fig. 2. Levels of T4 hormone in control (20-32 yrs & 33-45 yrs),Group I (20-32 yrs) and Group II (33-45 yrs**).**

Level of Thyroid stimulating hormone (TSH) in Group I and Group II :

In Group I participants, TSH level for control was found to be 3.75mIU/dL and for test it was found to be 3.76mIU/dL. In Group II participants, the level of TSH was found to be 6.738mIU/dL for the test and 3.18mIU/dL for the control as depicted in Fig.3.



Values are Mean ± SD (n=30); * =Significance at 5% level (P<0.05)

Fig. 3. Levels of T4 hormone in Control (20-32 yrs & 33-45 yrs), Group I (20-32 yrs) and Group II (33-45 yrs).

5. Discussion:

Phthalate esters are widely present in cosmetic products, including lipsticks and talcum powders. Previous studies by Parlett *et al.,* [10] and Duty *et al.,* [11] have demonstrated that frequent use of personal care products by women may elevate circulating phthalate levels. Among biological matrices, blood samples are considered a more reliable indicator than urine, saliva, or breast milk for assessing systemic exposure and circulating concentrations of phthalate metabolites.

Kim MJ *et al.,*[12] reported that plasticizers exert diverse effects on thyroid function in both humans and experimental animals. Several studies have shown either positive or negative associations between plasticizer exposure and serum T3 and T4 concentrations. Plasticizers may also modify the expression of thyroid hormone (TH)-related genes and regulatory proteins, including thyroid-stimulating hormone (TSH), thyrotropin-releasing hormone (TRH), and hormone transporters. Histopathological evidence indicates thyroid follicular cell enlargement and hyperplasia following exposure to certain plasticizers. Collectively, these findings suggest that plasticizers interfere with TH homeostasis through multiple toxicological pathways.Grindler N *et al.,*[13] further demonstrated, through multivariable regression analysis, that several urinary phthalate metabolites were inversely associated with TSH levels, DNA methylation patterns, and gene expression. These findings support the hypothesis that phthalates may impair placental function by epigenetically modifying the expression of critical placental genes. However, additional investigations are necessary to elucidate the functional consequences of these molecular alterations.

Yuan N *et al.,*[14] highlighted that Diethyl hexyl phthalate (DEHP), a commonly used industrial plasticizer, may disrupt the hypothalamic–pituitary–thyroid (HPT) axis. Although epidemiological data suggest a potential association between DEHP exposure and altered thyroid function, results remain inconsistent. Their study reported an inverse association between total T4 concentrations and urinary MEHP and MEHHP levels, while thyrotropin concentrations were positively correlated with urinary mono (2-ethyl-5-oxohexyl) phthalate levels.

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(2), 01-07 | ISSN:2251-6727

Chen Y *et al.,*[15] reviewed the impact of plasticizers on thyroid function in humans and animals. The thyroid gland, one of the earliest endocrine glands to develop during embryogenesis, plays a fundamental role in metabolism, cellular proliferation, circadian regulation, and neurodevelopment. Evidence indicates that plasticizers may exert bidirectional effects on serum T3 and T4 levels. Histological analyses consistently demonstrated thyroid follicular cell enlargement and hyperplasia following exposure. These observations reinforce the concept that plasticizers may disturb thyroid hormone regulation via diverse mechanisms.

Pacyga DC *et al.,*[16] reported that higher maternal urinary MBP levels were associated with lower TSH concentrations in cord blood serum, even after adjusting for maternal thyroid status. This study is among the first to evaluate maternal phthalate exposure in relation to neonatal thyroid hormone profiles, suggesting potential transplacental endocrine effects.

Bereketoglu C *et al.,* [17] examined longitudinal associations between maternal urinary phthalate metabolites and serum thyroid hormone levels during pregnancy. Although only a few significant relationships were observed, inverse associations were identified between FT3 and MCPP, and between progesterone and MEP. These findings indicate that phthalate exposure during gestation may alter maternal thyroid and sex hormone concentrations, with the timing of exposure potentially influencing endocrine outcomes.

Phthalates are recognized environmental endocrine disruptors, and their association with thyroid hormone dysregulation has been increasingly documented.

Wang W *et al.,* [18] investigated whether thyroid autoimmunity status and metformin therapy modify the relationship between phthalate metabolites and thyroid function parameters in individuals with type
2 diabetes, highlighting the complexity of these interactions.

Experimental *in vitro* and *in vivo* studies have demonstrated that phthalates may mimic thyroid hormones and bind to thyroid receptors, thereby disturbing thyroid homeostasis and impairing normal HPT axis function [19].

In the present study, analysis of thyroid parameters revealed that TSH levels were highest in long-term plastic-using women (test group II) compared to the group II controls, short-term controls, and short-term plastic-using participants. Additionally, T3 and T4 concentrations were reduced in the long-term exposure group relative to other groups. These findings are consistent with previous reports indicating that chronic exposure to phthalates may disrupt thyroid hormone regulation [20].

The thyroid gland is particularly susceptible to chemical disruptors such as phthalate metabolites. These exogenous agents may interfere at multiple levels, including hormone synthesis, metabolism, transport, and receptor binding [21]. *In vitro* evidence further supports the detrimental impact of such chemicals on thyroid function. Therefore, minimizing prolonged plastic exposure may be crucial in reducing potential endocrine disruption and safeguarding thyroid health.

6. Conclusion

As Toxicity of phthalates has been studied extensively in recent years our quest is to know how these harmful chemicals are involved in the pathophysiology of hormonal disorders. The usage of plastic products has multiplied several folds in the past few decades. From the data, our findings clearly demonstrates that as the age advances, the level of phthalate ester and the hormone dysfunctions were elevated. This indicates the potential role of phthalate esters in the pathogenesis of hormonal imbalance in women. Hence our study suggests that phthalate esters may have an aetiological association with hormonal imbalance on a long-term exposure. As its mechanism of action is still obscure, a study at molecular level is required to understand the signalling pathways involved.

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Journal of Chemical Health Risks
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