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Role of Testosterone levels on the Polycystic Ovary Syndrome Women

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Abstract

Polycystic ovary syndrome (PCOS) is a critical endocrine medical condition that affects a major number of women at the age group (18-45) worldwide. The present article searches the effect of testosterone (TET) levels on the women at their reproductive ages based on a real data set of 1000 women, and the data set is available. The testosterone levels analysis results are obtained in the article using statistical joint generalized linear models. It is obtained herein that mean testosterone (TET) level is negatively associated with the joint interaction effect (JIE) of the subject's menstrual irregularity (MIT) and body mass index (BMI) i.e., MIT* BMI ($P<0.0001$), while it is positively associated with both MIT ($P<0.0001$) and BMI ($P<0.0001$). Mean TET level is negatively associated with the JIE of BMI and antral follicle count (AFC) i.e., BMI*AFC ($P=0.0005$), while it is positively associated with both BMI ($P<0.0001$) and AFC ($P=0.0006$). Further, mean TET level is negatively associated with the JIE of AFC and MIT i.e., AFC*MIT ($P<0.0001$), while both AFC ($P=0.0006$) and MIT ($P<0.0001$) are positively associated with TET level. Mean TET level is positively associated with the JIE of BMI and the subject's polycystic ovary syndrome (PCOS) diagnostic status i.e., BMI*PCOS ($P<0.0001$), while it is positively associated with BMI ($P<0.0001$) and negatively with PCOS ($P<0.0001$). Mean TET level is positively associated with the JIE of AFC and PCOS i.e., AFC*PCOS ($P<0.0001$), while it is positively associated with AFC ($P=0.0006$) and negatively with PCOS ($P<0.0001$). TET level's variance is negatively associated with PCOS ($P<0.0001$). This report concludes that the marker TET level has several joint significant effects on PCOS women. These above findings regarding the marker TET levels may be helpful for the researchers, practitioners and PCOS women. Care should be taken on menstrual irregularity, BMI, antral follicle count and testosterone levels for PCOS women.

Keywords: Antral follicle count; Body mass index; Joint mean-variance model; Testosterone levels; Polycystic ovary syndrome

Abbreviations: AFC: Antral Follicle Count; BMI: Body Mass Index; JGLMs: Joint Generalized Linear Models; JIE: Joint Interaction Effect; MIT: Menstrual Irregularity; TET: Testosterone levels; PCOS: Polycystic Ovary Syndrome

Introduction

Polycystic ovary syndrome (PCOS) is the most normal endocrine disorder that assails 10-15% of women in their reproductive age group [18-45] [1,2]. Generally, PCOS is discriminated against by a heterogeneous set of calamities, including body mass index, irregular menstrual cycles, antral follicle count, testosterone level, and polycystic ovarian morphology [3,4]. Biochemical (or clinical) hyperandrogenism is a diagnostic criteria for PCOS. Commonly adopted criteria for PCOS diagnosing is the Rotterdam criterion suggested by The American Society for Reproductive Medicine in 2003 [5], that comprises mainly three criteria such as (a) oligomenorrhoea or chronic anovulation; (b) polycystic ovaries on ultrasonography and (c) the peculiarities of biochemical (or clinical) signs of hyperandrogenism. Additionally, congenital adrenal hyperplasia, androgen secreting tumor, Cushing's syndrome and other diseases should be omitted from these suggestions. Hyperandrogenemia is the most leading clinical PCOS manifestation, which might result in cutaneous

manifestations, like acne, hirsutism and alopecia.The article by Munzker [6] examined the effects of salivary testosterone (TET) levels and salivary androstenedione levels for the PCOS diagnosis in a cross-sectional study of 110 PCOS women and 65 without PCOS. The authors of the article [6] have tested testosterone to dihydrotestosterone ratio as a new biomarker for the PCOS diagnosis. The testosterone (TET) level in the patient's blood, an important hormonal biomarker (or indicator) of PCOS, ranges from 20 to 100 ng/dL [7,8]. TET levels are typically measured in nanograms per deciliter (ng/dL) via blood tests. Generally, the normal reference range of TET levels for biological females is approximately 15 to 70 ng/dL, depending on some factors like age, health status, BMI and the lab's reference standards. TET levels above 70 ng/dL are prescribed to as hyperandrogenism, which are often associated with medical conditions like PCOS, a hormonal disorder that affects ovulation and leads to many symptoms such as irregular menstrual cycles, male-pattern hair growth (hirsutism), acne, hair thinning, and infertility [9,10]. Present day, different advanced research techniques such as statistical modelling, machine learning, data mining etc. are adopted in the analysis of PCOS data sets [11,12]. In machine learning approach, many algorithms such as multilayer perceptron, locally weighted learning, decision table, random forest, random tree, etc. are adopted in the PCOS data analysis [12-15]. Classical statistical techniques such as correlation, linear/ multiple regression, analysis of variance, testing of hypotheses etc. are used in PCOS data analysis[8, 11, 16], which are not appropriate for non-normal and heterogeneous PCOS data sets. The considered PCOS data set is a non-normal, heteroscedastic and physiological data set. The earlier testosterone (TET) levels marker of PCOS reports do not consider that the response marker TET levels are of a heteroscedastic nature. Therefore, most of the earlier reports of TET marker invite many debates and doubts. Moreover, the previous TET marker reports do not adopt any suitable model fitting diagnostic tools for accepting the final models. Thus, the research approaches may not have a strong faith on the previous TET marker analysis outcomes. The TET marker effective roles on PCOS women are very little examined based on advanced statistical probabilistic modeling. The current report of TET marker examines the following research hypotheses associated with PCOS women.

- Is there any association of marker TET levels with age, irregular menstrual cycles, BMI and polycystic ovarian morphology of PCOS women?
- If it is affirmative, how can we develop the most probable marker TET levels association model?
- What is the most probable marker TET levels statistical model?
- What are the effects of marker TET levels on PCOS women?

The present report investigates the above research hypotheses about the marker TET levels considering the following paragraphs such as materials & methods, statistical analysis & results, discussions, and conclusions. Joint statistical model of the marker TET levels is presented in Table 1, using the PCOS data set that is reported in the materials section. The joint statistical model of the marker TET levels is developed by joint generalized linear models (JGLMs) that is shortly expressed in the methods section. Analysis outcomes of the marker TET levels are reported in the result section, while the results are clearly illustrated in the discussion section. Finally, the basic TET levels analysis information is reported in the conclusions section.

Materials and Methods

Materials

The present study dataset is related with PCOS women, while PCOS is a general hormonal endocrine disorder affecting women of their reproductive age groups. The study dataset consists of 1000 subjects, each representing a woman sample unit, and along with six key characters that are typically correlated with the PCOS diagnosis risk factors. These characters take measures of valuable insights into the subjects' health conditions, and they can be adopted for exploratory data analysis such as statistical model development, machine learning and feature engineering for predicting PCOS diagnoses status. The present PCOS data set contains six characters such as the subject's age, menstrual irregularity (MIT) (0=No, 1= Yes), antral follicle count (AFC), testosterone level (TET), body mass index (BMI), and polycystic ovary syndrome (PCOS) (0=No, 1=Yes) diagnosis status. A total of 1000 random sample women units' records are obtained in the current study. The study women subjects are selected from their reproductive age group 18 to 45. A binary indicator representing whether the study subject has irregular menstrual cycles (0 = No, 1 = Yes). The menstrual cycle is the regular natural system that makes the female body for pregnancy. The body mass index (BMI) represents a measure of body fat that is measured using height (in meter) and weight (in Kg) and is given by BMI= Weight (kg) / Height (m2), which is ranging from 18 to 35.

The number of antral follicle counts (AFC) identified during an ultrasound, ranging from 5 to 30 that helps in measuring ovarian reserve and PCOS presence. AFC is a test adopted to assess a woman's ovarian reserve that refers to the number and quality of eggs she has available for reproduction. It's typically done during the early menstrual cycle follicular phase, usually on day 2 or 3, by transvaginal ultrasound. Note that AFC indicates the number of small, fluid-filled follicles (2-10 mm in diameter) in both ovaries. The testosterone (TET) level in the study subject women's blood is a significant hormonal marker of identifying the PCOS status of women that is ranging from 20 to 100 ng/dL.

TET is an important androgen hormone produced primarily in the testes in males and in smaller amounts by the ovaries and adrenal glands in females. TET is commonly labelled as a "male hormone" but it also plays an important role in female health. TET is considered as an individual with ovaries, but it plays several vital functional roles such as muscle strength, including bone density, energy levels, libido, and the regulation of reproductive health. A binary indicator of identifying the subject woman has been diagnosed with PCOS (0 = No, 1 = Yes), using several combinations of risk factors such as high BMI, menstrual irregularity, testosterone levels, and antral follicle count.

Statistical Methods

The present article takes into account the testosterone (TET) levels is the aimed response random variable that is to be modeled with the left five variables such as BMI, age, MIT, AFC and PCOS. It is identified that the aimed response TET level is non-normally and heteroscedastic distributed random variable. The variation of TET levels can't be stabilized by any suitable transformation, so TET level is modeled in the article using joint generalized linear models (JGLMs) under both the Log-normal and Gamma distributions that is explicitly described in [17-20]. JGLMs is illustrated in the book by Lee, Nelder and Pawitan [17] and in the book by Das [18]. For ready reference, a short form of JGLMs for TET levels under both the Log-normal and Gamma distribution is reported as follows.

JGLMs for Log-normal distribution: For the positive response Yi (=TET) with E(Yi=TET) = μi (mean) and Var (Yi=TET) = μi2 = say, where 's are dispersion parameters and V ( ) reveals the variance function. Generally, log transformation Zi = log (Yi=TET) is adopted to stabilize the variance Var (Zi) ≈, but the variance may not always be stabilized [21]. For developing a TET improved model, JGLMs for the mean and dispersion are considered. For the response TET, assuming log-normal distribution, JGL mean and dispersion models (with Zi = log (Yi=TET)) are as follows:

$E(Z_i) = \mu_{zi}$ and $Var(Z_i) = \sigma_{zi}^2$, μzi=xit β and log (σzi2) = git γ, where xit and git are the explanatory factors/variables vectors of TET level associated with the mean regression coefficients β and dispersion regression coefficients γ, respectively.

JGLMs for Gamma distribution: In the above stated Yi's (=TET), the variance has two portions such as (based on the mean parameters μi's) and (free of μi's). The variance function V ( ) displays the GLM family distributions. For instance, if V()= 1, it is normal, Poisson if V( ) = , and gamma if V( ) = etc. Gamma JGLMs mean and dispersion models of GLU are as follows:

$$\eta_i = g(\mu_i) = x_i^t \beta \text{ and } \varepsilon_i = h(\sigma_i^2) = w_i^t \gamma,$$

where and are the GLM link functions attached with the mean and dispersion linear predictors respectively, and , are the explanatory factors/variables vectors of TET level attached with the mean and dispersion parameters respectively. Maximum likelihood (ML) method is used for estimating the mean parameters, while the restricted ML (REML) method is applied for estimating the dispersion parameters, which are explicitly stated in the book by Lee, Nelder and Pawitan [17].

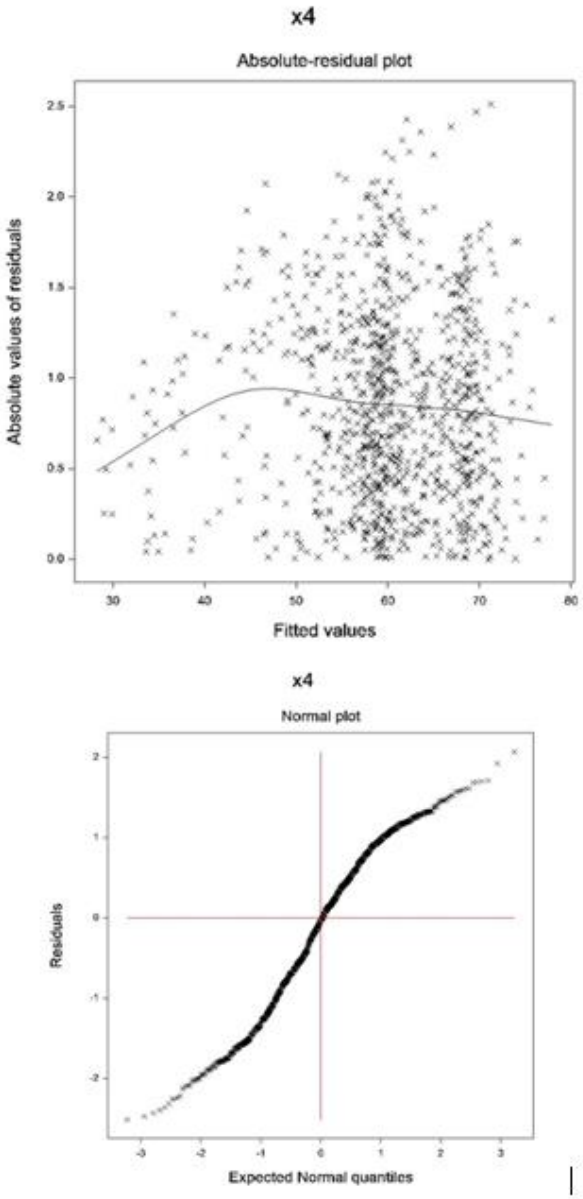

Figure 1: *For the joint Gamma fitted models of Testosterone Levels (Table 1), the (a) absolute residual plot with the fitted values, and (b) the normal probability plot for mean model.*

Statistical Analysis & Results

Statistical Analysis

The article aims to derive the effects of TET levels on the PCOS women. Joint statistical TET levels model has been developed

based on the remaining five independent variables such as BMI, age, AFC, MIT and PCOS diagnostic status. Final TET levels model has been accepted on the basis of smallest Akaike information criterion (AIC) value (within each class) that minimizes both the squared error loss and predicted additive errors [22, p. 203-204]. According to the AIC rules, JGLMs Gamma fit (AIC= 8950.762) is better than Log-normal fit (AIC=8993). In the mean model, all the marginal and joint interaction effects are significant. Due to the marginality rule by Nelder [23], if any interaction effect is significant, then its all lower order interaction effects and marginal effects will be incorporated in the model. Two partial marginal effects such as AFC (P=0.1471) and AGE (P=0.1263) are included in the in the dispersion model, for improving the model [22]. It is noted that in Epidemiology, partial significant effects are known as confounders, which may have some effects with the risk factor or marker.

The obtained TET levels Gamma fitted statistical JGLM (Table 1) is a data evolved model that is to be verified by model checking plots. All the valid interpretations about TET levels are obtained from the data exhibited Gamma fitted TET levels probabilistic model (Table 1) that is to be accepted based on suitable graphical diagnostic plots, which is displayed in Figure 1. Figure 1(a) reveals the absolute residuals plot for the Gamma fitted TET levels model (Table 1) with respect to the fitted values, which is almost flat linear, indicating that variance is constant with the running means. Figure 1(b) shows the normal probability plot for the Gamma fitted TET levels mean model (Table 1) that does not indicate any lack of fit. So, both the (figures 1(a) and (1b)) do not present any discrepancy in the Gamma fitted TET levels models (Table 1). The above Figure 1(a) and Figure 1(b) confirm that the Gamma fitted TET levels model is an approximate form of the unknown true TET levels model.

Results

Table 1 shows the TET levels analysis summarized outcomes. According to the AIC rule, Gamma fitted (AIC= 8950.762) JGLM shows better results for TET levels analysis than Log-normal fitted model (AIC=8993). So, the final accepted TET levels model is Gamma fitted JGLM. These two fitted models for TET levels (Table 1) have very similar conclusions, but there are some discrepancies between these two fitted models. Some critical common discrepancies between the fitted Gamma and Log-normal models are reported in the articles [24, 25]. Here TET level is considered as the dependent (or response) variable, and the left five others are used as the independent (or explanatory) variables. It is obtained herein that mean testosterone (TET) level is negatively associated with the joint interaction effect (JIE) of the subject's menstrual irregularity (MIT) and body mass index (BMI) i.e., MIT* BMI (P<0.0001), while it is positively associated with both MIT (P<0.0001) and BMI (P<0.0001). Mean TET level is negatively associated with the JIE of BMI and antral follicle count (AFC) i.e., BMI*AFC (P=0.0005), while it is positively associated with both BMI (P<0.0001) and AFC (P=0.0006). Further, mean TET level is negatively associated with the JIE of AFC and MIT i.e., AFC*MIT (P<0.0001), while both AFC (P=0.0006) and MIT (P<0.0001) are positively associated with TET level. Mean TET level is positively associated with the JIE of BMI and the subject's polycystic ovary syndrome (PCOS) diagnostic status i.e., BMI*PCOS (P<0.0001), while it is positively associated with BMI (P<0.0001) and negatively with PCOS (P<0.0001). Mean TET level is positively associated with the JIE of AFC and PCOS i.e., AFC*PCOS (P<0.0001), while it is positively associated with AFC (P=0.0006) and negatively with PCOS (P<0.0001).

The variance of TET levels is negatively associated with PCOS (P<0.0001). There are two partial marginal effects such as AFC (P=0.1471) and AGE (P=0.1263) that are included in the dispersion model, for improving the model [22]. These two partially significant effects are known as confounders.

From Table1, Gamma fitted TET levels mean ($\hat{\mu}$) model i $\hat{\mu}$ = exp(3.2542 + 0.0310 BMI + 1.4093 MIT – 0.0488 MIT* BMI + 0.0376 AFC - 0.0014 BMI*AFC – 0.0181AFC*MIT – 1.1881 PCOS + 0.0411 BMI*PCOS + 0.0259 AFC*PCOS), and from Table 1, the Gamma fittedTET levels variance ($\hat{\sigma}^2$) model is $\hat{\sigma}^2$ = exp.(- 1.6704 + 0.0096 AFC – 0.9344 PCOS – 0.0081 AGE).

From the above, TET level mean ($\hat{\mu}$) model is explained by many marginal and interaction effects such as BMI, MIT, MIT* BMI, AFC, BMI*AFC, PCOS, BMI*PCOS, AFC*PCOS while the variance ($\hat{\sigma}^2$) model is explained by AFC, PCOS and AGE.

Discussions

The summarized TET levels analysis findings are displayed in Table 1. The most selected fitted mean and variance models of TET levels are displayed above from Table 1. These above two mean-variance TET level models show the associations of TET levels with the remaining explanatory factors such as BMI, age, MIT, AFC and PCOS. These different associations of TET levels are illustrated in the following paragraphs.

From the TET level's mean model (Table 1), it is observed that mean TET level is negatively associated with the joint interaction effect (JIE) of the subject's menstrual irregularity (MIT) and body mass index (BMI) i.e., MIT* BMI (P<0.0001), while it is positively associated with both MIT (P<0.0001) and BMI (P<0.0001). This indicates that TET level increases as the joint

interaction effect MIT*BMI decreases. Note that both the marginal effects MIT (0=No, 1=Yes) and BMI are positively associated with TET levels, which implies that TET level increases for the women with higher BMI, or abnormal MIT, or both. But it is not always possible as their joint interaction effect MIT*BMI is negatively associated with TET level. Therefore, it should not always be concluded that obese women, or women with menstrual irregularity, or both may have higher TET levels. Note that if the joint interaction effect is significant, the marginal effects are not important.

Table 1: *Results for mean and dispersion models for Testosterone Levels (X4) from Log-normal & Gamma fit.*

Model	Covariate	GAMMA FIT				LOG-NORMAL FIT			
		estimate	s.e.	t(990)	P-value	estimate	s.e.	t(990)	P-value
Mean	Constant	3.2542	0.2068	15.73	<0.0001	3.2233	0.2141	15.05	<0.0001
	BMI	0.0310	0.0077	4.02	<0.0001	0.0300	0.0079	3.75	0.0002
	MIT	1.4093	0.1806	7.80	<0.0001	1.3925	0.1879	7.41	<0.0001
	MIT*BMI	-0.0488	0.0062	-7.76	<0.0001	-0.0492	0.0065	-7.53	<0.0001
	AFC	0.0376	0.0108	3.46	0.0006	0.0329	0.0112	2.92	0.0035
	BMI*AFC	-0.0014	0.0004	-3.48	0.0005	-0.0013	0.0004	-3.08	0.0021
	AFC*MIT	-0.0181	0.0042	-4.29	<0.0001	-0.0164	0.0043	-3.75	0.0002
	PCOS	-1.1881	0.2627	-4.52	<0.0001	-1.0586	0.2675	-3.95	<0.0001
	BMI*PCOS	0.0411	0.0083	4.95	<0.0001	0.0388	0.0084	4.59	<0.0001
	AFC*PCOS	0.0259	0.0052	4.980	<0.0001	0.0253	0.0053	4.76	<0.0001
Dispersion	Constant	-1.6704	0.2088	-8.00	<0.0001	-1.5683	0.2075	-7.55	<0.0001
	AFC	0.0096	0.0066	1.45	0.1471	0.0110	0.0065	1.67	0.0950
	PCOS	-0.9344	0.1151	-8.11	<0.0001	-1.0109	0.1151	-8.77	<0.0001
	AGE	-0.0081	0.0053	-1.53	0.1263	-0.0087	0.0053	-1.64	0.1013
	AIC	8950.762				8993			

Mean TET level is negatively associated with the JIE of BMI and antral follicle count (AFC) i.e., BMI*AFC (P=0.0005), while it is positively associated with both BMI (P<0.0001) and AFC (P=0.0006). This implies that TET level increases as the joint interaction effect BMI*AFC decreases. Here also, both the marginal effects BMI and AFC are positively associated with TET levels, which implies that TET level increases for the women with higher BMI, or AFC values, or both. But it is not always possible as their joint interaction effect BMI*AFC is negatively associated with TET level.

Mean TET level is negatively associated with the JIE of AFC and MIT i.e., AFC*MIT (P<0.0001), while both AFC (P=0.0006) and MIT (P<0.0001) are positively associated with TET level. This reveals that TET level increases as the joint interaction effect AFC*MIT decreases. Here also, both the marginal effects AFC and MIT (0=No, 1=Yes) are positively associated with TET levels, which implies that TET level increases for the women with higher AFC values, or menstrual irregularity, or both. But it is not always possible as their joint interaction effect AFC*MIT is negatively associated with TET level.

Mean TET level is negatively associated with the JIE of AFC and MIT i.e., AFC*MIT (P<0.0001), while both AFC (P=0.0006) and MIT (P<0.0001) are positively associated with TET level. This reveals that TET level increases as the joint interaction effect AFC*MIT decreases. Here also, both the marginal effects AFC and MIT (0=No, 1=Yes) are positively associated with TET levels, which implies that TET level increases for the women with higher AFC values, or menstrual irregularity, or both. But it is not always possible as their joint interaction effect AFC*MIT is negatively associated with TET level.

Mean TET level is positively associated with the JIE of BMI and the subject's polycystic ovary syndrome (PCOS) diagnostic status i.e., BMI*PCOS (P<0.0001), while it is positively associated with BMI (P<0.0001) and negatively with PCOS (P<0.0001). This indicates that TET level increases as the joint interaction effect BMI*PCOS increases. Here one marginal effect BMI is positive and the other PCOS (0=No, 1=Yes) status is negatively associated with TET levels, so the joint effect BMI*PCOS may not always increase. In other words, this interpretation can be restated as TET level may be higher for the obese women with no PCOS status.

Mean TET level is positively associated with the JIE of AFC and PCOS i.e., AFC*PCOS (P<0.0001), while it is positively associated with AFC (P=0.0006) and negatively with PCOS (P<0.0001). This denotes that TET level increases as the joint interaction effect AFC*PCOS increases. Here one marginal effect of AFC is positive, and the other PCOS (0=No, 1=Yes) status is negatively associated with TET levels, so the joint effect AFC*PCOS may not always increase. In other words, it can be restated as TET level may be higher for the women with higher AFC values and no PCOS status.

The variance of TET levels is negatively associated with PCOS (P<0.0001). It shows that TET levels are highly scattered for the women without PCOS status. TET level's variance is positively partially associated with AFC (P=0.1471). It indicates that TET

levels are highly scattered for the women with higher AFC values. In addition, TET level's variance is negatively partially associated with AGE (P=0.1263). This implies that TET levels are highly scattered for the women with lower age groups.

It is derived herein that the lower joint effects MIT* BMI, BMI*AFC and AFC*MIT are highly risk factors for TET levels or equivalently for PCOS women. Also, the higher joint effects BMI*PCOS and AFC*PCOS are highly risk factors for TET levels. It is well-known that TET level is a biomarker of PCOS women. The report has derived the associations of mean TET level with five different joint interaction effects, and along with their marginal effects. Marginal associations of TET levels are easily understandable but the joint interaction effects are a little complex. Note that the joint interaction effects on TET levels can be located using only statistical modeling. Best of our knowledge, no earlier article identifies any joint interaction association of TET levels. The current report has focused on the associations of TET levels with the remaining five factors such as BMI, AGE, MIT, AFC and PCOS status. But recently many articles focus on different problems of PCOS women. Very lately, an article [26] has focused on PCOS women, and it is concluded that PCOS is a complex endocrine chaos that affects 6–21% women at reproductive age groups (18-45), which is discriminated by chronic anovulation, hyper-androgenism, and polycystic ovarian morphology. Modern clinical management relies on lifestyle modifications and symptom-targeted therapies due to the absence of curative interventions. The report [27] has illustrated that hormonal imbalances and glucose-lipid metabolism have minimal impact on embryo development in PCOS women. However, obese PCOS women, and along with their hormonal factors may influence on pregnancy findings such as risk of miscarriage due to androgen levels and high BMI. Also a recent report [28] has focused on that reduced miR-338-3p levels have potential predictive value in distinguishing between individuals with PCOS women from the normal population. It is reported in the article [29] that Anti-Müllerian Hormone (AMH) may be entangled in regulating impaired ovarian granulosa cells development in PCOS rats via SMAD family member 4 (SMAD4). Interested readers and researchers may go through many new ideas or concepts for studying PCOS diagnosis and management systems [11,27, 28,30].

Conclusions

The current report has examined the impacts of TET levels on PCOS women. The associations of TET levels on five explanatory factors such as BMI, AGE, MIT, AFC and PCOS status have been derived. The fitted TET levels model has been accepted herein based on graphical diagnostic checking plots (Figure 1), on the smallest AIC rule (Table 1), on comparison of joint Gamma and Log-normal models (Table 1), and standard error of the estimates. Table 1 shows both the fitted Gamma and Log-normal models have similar interpretations, which are reported in the discussion section. All these outcomes (Table 1) related to TET levels in the report focus on the real practical situations. The extracted findings regarding TET levels on PCOS women though not completely decisive but are expressive. Present day scientific research techniques should have complete belief on these TET levels obtained outcomes, as the TET levels fitted models have been taken with graphical diagnostic tests and comparison of two different models.

The obtained TET level models (Table 1) are developed from the data set as noted in the material section. For any parallel PCOS data sets, almost similar findings (Table 1) regarding the TET level will be obtained by any researcher, which is not verified herein as parallel data sets are not available. The current TET level results show many real facts, which are not pointed out in the previous articles. These TET levels results herein are completely new in the clinical endocrine literature. These results of TET levels may be helpful for PCOS women, researchers and medical practitioners. It is extracted herein that TET levels have very complex associations (Table 1) with the different factors for the PCOS women that should be known to the practitioners for appropriate treatment processes. All women at their reproductive age group (18-45) should care about testosterone level, menstrual irregularity, BMI and antral follicle count values.

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Conflict of interest

The authors confirm that this article content has no conflict of interest.

Informed consent statement

Not applicable

Sample availability

The authors declare no physical samples were used in the study.

References

1. The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group, Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Human Reproduction, 2004; 19: 41-47.
2. Azziz, R, Carmina E, Chen Z, Dunaif A, Laven JS et al. Polycystic ovary syndrome. Nature Reviews Disease Primers, 2016; 2: 16057.

3. Zhao H, Qin Y, Kovacs P, Jiao, X. Genetic studies on polycystic ovary syndrome (PCOS): recent advances and perspectives. Current Molecular Medicine, 2019; 19: 218-226.
4. Dong J, Rees DA.Polycystic ovary syndrome: pathophysiology and therapeutic opportunities. BMJ Med. 2023; 2: e000548,
5. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertility and Sterility. 2004; 81: 19-25.
6. Munzker J, Hofer D, Trummer C, Ulbing M, Harger A, Pieber T, et al. Testosterone to Dihydrotestosterone Ratio as a New Biomarker for an Adverse Metabolic Phenotype in the Polycystic Ovary Syndrome. J Clinical Endocrinology Metabolism, 2015; 100: 653-660.
7. Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Lergo RS, et al. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. Endocr Rev. 2015; 36: 487-525
8. Blouin K, Veilleux A, Luu-The V, Tchernof A. Androgen metabolism in adipose tissue: recent advances. Mol Cell Endocrinol.2009; 301: 97-103.
9. Bui HN, Sluss PM, Hayes FJ, Blincko S, Knol DL, Blankenstein MA, et al. Testosterone, free testosterone, and free androgen index in women: Reference intervals, biological variation, and diagnostic value in polycystic ovary syndrome. Clinica Chimica Acta, 2015; 450:227-232.
10. Zhang J, Pan M, Zhang Q, Wang F, Qu F. Association of Tumor Markers with Total Testosterone in the Serum of Women with Polycystic Ovary Syndrome. Clin. Exp. Obstet. Gynecol. 2022; 49: 162.
11. Bairagi VK, Vanjale MS, Dolas A, Zende S, Nanthaamornphong A, Harpale S,et al. polycystic ovarian syndrome (PCOS) Detection Through Deep Learning, IJETT, 2024; 72: 159-170.
12. Kavitha K, Tangudu N, Sahu SR, Narayana GVL, Anusha V. Detection of PCOS using Machine Learning Algorithms with Grid Search CV Optimization. IJTT, 2023; 71: 201-208.
13. Al-Muqaren HM, Mansor MB, Ibrahim Z. Predictive modelling of polycystic ovary syndrome using machine learning. J Med Systems. 2020;.44: 91.
14. Modi N, Kumar Y. Detection and Classification of Polycystic Ovary Syndrome Using Machine Learning-Based Approaches, IEEE.2024; 1-6.
15. Li X, Dai J, Tang Y, Xiao T. Machine learning-based prediction models for polycystic ovary syndrome risk assessment: A review. Frontiers in Endocrinology, 2022;13: 835477.
16. Ye W, Xie T, Song Y, Zhou L. The role of androgen and its related signals in PCOS. J Cellular Molecular Medic. 2021; 25: 1825-1837.
17. Lee Y, Nelder JA, Pawitan Y. Generalized Linear Models with Random Effects :Unified Analysis via H–likelihood.2017.
18. Das RN. Robust Response Surfaces, Regression and Positive Data Analyses. London: Chapman Hall 2014.
19. Das RN, Lee Y. Log-normal versus gamma models for analyzing data from quality-improvement experiments. Quality Engineering 2009; 21: 79-87.
20. Qu Y, Tan M, Rybicki L. A unified approach to estimating association measures via a joint generalized linear model for paired binary data. Communications in Statistics -Theory and Methods 2000; 29:143-156.
21. Myers RH, Montgomery DC, Vining GG. Generalized Linear Models with Applications in Engineering and the Sciences. New York: John Wiley Sons. 2002.
22. Hastie T, Tibshirani R, Friedman J. The Elements of Statistical Learning, Springer-Verlag, 2001.
23. Nelder JA. The statistics of linear models: back to basics. Statistics and Computing. 1994; 4: 221-234.
24. Das RN. Discrepancy in fitting between log-normal and gamma models: An illustration. Model Assisted Statistics Applications 2012; 7: 23-32.
25. Das RN, Park JS. Discrepancy in regression estimates between Log-normal and Gamma: Some case studies. J Applied Statistics 2012; 39: 97-111.
26. Ding K, Wang X, Liu W, Chen Y, Xue F, Song C, et al. Engineering modification of human umbilical cord mesenchymal stem cell-derived small extracellular vesicles ameliorates polycystic ovary syndrome by enhancing the ovarian environment and regulating follicular development. Stem Cell Res Ther. 2025;16: 481.
27. Wang L, Yu X, Xiong D, Leng M, Liang M, Li R, et al. Hormonal and metabolic influences on outcomes in PCOS undergoing assisted reproduction: the role of BMI in fresh embryo transfers. BMC Pregnancy Childbirth. 2025; 25: 368.
28. Liang L, Lv J, Li W, Song C, Chen Y, Wei H, et al. Reduced miR-338-3p contributes to polycystic ovarian syndrome by inhibiting proliferation and enhancing apoptosis. Hereditas. 2025; 162: 126.
29. Dong A, Yu X, Zhang Y, Liu L, Liu F, Song W, et al. Anti-Müllerian hormone regulates ovarian granulosa cell growth in PCOS rats through SMAD4. Int J Gynaecol Obstet. 2025; 17: 209-221.
30. Tang T, Lai H, Huang X, Gu L, Shi H. Application of serum markers in diagnosis and staging of ovarian endometriosis. J Obstetrics Gynecology Res. 2021; 47: 1441-1450.