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Association of Anti-Mullerian Hormone with some physiological and immunological parameters of infertile males

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ABSTRACT

The measurement of anti-Müllerian hormone (AMH), which is released in the serum and seminal fluid is a specific indicator of Sertoli cell function. This study aimed to evaluate AMH as a potential clinical indicator of infertility in males with normal and low sperm concentrations, as well as those with primary and secondary infertility. It was a retrospective analysis of 140 males chosen from sole infertility and IVF center in Erbil. without a known cause of infertility, 40 men with normal sperm counts, 100 men with primary, and 40 men with secondary infertility were assessed. An in-house ELISA was used to assess the serum AMH. Semen parameters, Semen and sera cytokines, and certain sex hormone mean levels were compared and correlated with AMH as the primary outcome measures. Seminal and serum AMH levels in infertile males were significantly lower. While an insignificant correlation was detected between AMH and LH, prolactin, or testosterone in azoospermic men, there was a significant adverse association between seminal AMH and FSH. A substantial positive association between seminal AMH and testosterone was found in oligospermia men, although no significant correlations were observed with FSH, LH, or prolactin. In conclusion, AMH in seminal plasma is a reliable marker of male infertility with a role in sperm production.

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Introduction

Anti-Müllerian hormone (AMH) is a homodimeric glycoprotein connected by disulfide bonds with a molecular weight of 140kDa that belongs to the transforming growth factor-beta (TGF-β) superfamily. It mediates male sexual differentiation and, in collaboration with testosterone, sexual maturation from childhood until puberty (1). It is extensively expressed in male fetal testis and is responsible for Mullerian duct regression. In men, this hormone remains increased until puberty and then quickly diminishes during the adult transition (2). While the role of AMH persistence in adulthood is unknown, the presence of AMH-specific receptor (AMHRII) in various organs (e.g., the adrenal gland, liver, lung, skeletal muscle, and spleen) implies that AMH might have a role in adult life other than reproductive system development (3). Serum AMH levels are inversely associated with cardiovascular events (in older males), infrarenal aortic diameter, and all-cause mortality in men. The processes underlying such relationships, however, are unknown (4).

Additionally, several socioeconomic factors impacting both men's and women's fertility may also affect how well in vitro fertilization (IVF) works (5,6). Serum T and AMH have a negative correlation during pubertal development. If androgen is abnormally high (e.g., due to a mutation that activates the LH receptor) but gonadotropin is low, this association still exists (7). Serum AMH levels are abnormally high in cases with androgen sensitivity or decreased androgen production, demonstrating that follicle-stimulating hormone (FSH), along with luteinising hormone (LH) and testosterone are used as markers for spermatogenesis and the testis activity in males (8–11), increases AMH production when androgen has no inhibitory effects (12). In an investigation, serum levels of the inflammatory cytokine interleukin-6 in men were adversely correlated with serum levels of the hormone AMH. The researchers compared the levels of AMH in serum and seminal plasma in healthy males and those with pathology in sperm count (SC), anti-spermatozoal antibodies (ASA) positivity, and normal or reduced sperm concentration to see if serum AMH determination would offer any diagnostic advantage over existing endocrine diagnostics (13).

This study aimed to investigate the AMH impact on anthropometric and demographic factors, a few laboratory markers of male fertility, serum and Semen cytokines, and serum hormones in both primary (without using assisted reproductive technique (14)) and secondary infertile and fertile males in the Kurdish population.

Materials and Methods

Study individuals

Men made up the population for this retrospective analysis, and 140 suspected patients were found to have infertility. The patients were adult males ranging in age from 19 to 63 years old. In addition to 40 fertile males as a control group aged 19 to 61 years, the recruited male patients divided into primary and secondary infertility were referred to the *in Vitro* Fallopian (IVF) center and other private labo-

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ratories.

Semen and sera analyses

Individuals who visited IVF clinics and private laboratories in Erbil, Iraq, provided their sera and semen for collection. Seminal fluid was given after 3 days of abstinence, liquefied, and examined for counting, motility, and morphology before being centrifuged. The seminal plasma was kept at -20 C for testing. Five ml of the blood should be collected in a vacuum tube and stored for a serological test after centrifuging.

Sex hormone analyses

The serological tests performed on the study group's sera included anti-Mullerian hormone, FSH, testosterone (TES), prolactin (PRL), thyroid stimulating hormone (TSH), interleukin-1 beta (IL1β), interleukin-6 (IL-6), interleukin-17 (IL17), anti-sperm antibody (ASA), LH, and tumor necrosis factor-alpha (TNFα) concentration. An in-house ELISA test was used to evaluate the anti-Mullerian hormone levels (15).

The samples were categorized into:

1-Group 1: including 100 patients with primary infertility

2-Group 2: including 40 patients with secondary infertility

3-Group 3: including 40 fertile males as a control group

The Infertility and IVF Public Center is the only referral infertility center in Erbil province. Being a man infertile for at least one year, having regular intercourse, and seeking infertility treatment at IVF Infertility Center during the study period were the inclusion criteria. Diabetes, azoospermia, and smoking, as well as receiving drugs impacting sperm parameters or sex hormone levels, were all exclusion criteria. Over the course of four months, 161 participants were screened via consecutive sampling, 21 of whom were eliminated due to the exclusion criteria (primarily azoospermia and smoking). The other 140 male cases were assessed. The measurements were taken with standard and calibrated tools. Their height was measured using a standard metal ruler while standing without shoes. Weight was assessed while wearing light clothing. Body mass index (BMI) was computed by dividing weight in kg by squared height in m (kg/m^2). The BMI was characterized as below in order to analyze the data:

< 20.0 kg/m^2 characterized as underweight, 20.0–24.9 kg/m^2 characterized as normal weight, 25–29.9 kg/m^2 characterized as overweight, and >30.0 kg/m^2 characterized as obese.

A morning blood sample of 5 mL was collected and sent to an authorized laboratory to determine serum levels of TES, PRL, TSH, LH, and FSH. Semen samples were also collected from the hospital subjects. In the few cases with their samples moved out of the hospital, care was taken to ensure that the samples were securely transferred in less than 30 minutes. The sample was repeated after one month if there were problematic semen-analysis parameters, including sperm motility < 50%, sperm concentration < 20×10^6/mL, or normal sperm morphology < 30%, before the subject could be included. In such cases, the second sample was analyzed. The semen-analysis categorization was performed on the basis of Fritz and Speroff (16).

Semen and serum cytokine concentrations were performed by Omnikine Assay Biotech kit-USA/ ELIZA, while serum hormone concentrations were done by Accu-bind kit-USA/ ELISA.

Statistical analysis

Comparisons between groups were made using the nonparametric Mann-Whitney test (for 2 groups) or the Kruskal-Wallis test (for 3 groups). If there were significant differences, Dunn's multiple-comparison post hoc test was applied. For correlation analysis, data were log-transformed to simulate a normal distribution, and the Pearson correlation coefficient (r) was computed to determine the relationship between normally distributed numeric scales. GraphPad Prism was used for all calculations (version 8.00 for Windows; GraphPad Software, San Diego, CA) (FSH ((MIU/ml)), LH ((MIU/ml)), PRL (Pg/mL), TSH ((MIU/ml)), and TES (Ng/mL) mean serum sex-hormone levels). The parameters of sperm analysis were also compared in the same way. All statistical tests were performed on a two-tailed basis, with a P-value < 0.05 deemed statistically significant. The research was authorized by Koya University's medical ethics committee. All participants in the research provided informed consent.

Results

Anthropometric characteristics and semen analysis results

The current research revealed that higher BMI values enhanced the incidence of oligospermia because of the related impact of AMH on demographic and seminal fluid features. Overweight males were shown to be more likely to experience oligospermia than those with normal BMI. According to Table 1, the mean total sperm count was lower in the seminal fluid of primary and secondary infertile patients (17.08±1.060 and 31.15±2.773), respectively, compared with the control group (151.6±4.864) with highly significant differences (P <0.001). Moreover, there was a highly significant (P<0.0001) decrement in the mean levels of progressive sperm motility and normal sperm shapes in seminal fluid in both primary and secondary infertile men. However, a highly significant (P<0.0001) increment was observed in the mean levels of non-progressive sperm motility and abnormal sperm shapes in seminal fluid compared with control groups. Regarding the age and duration of the marriage, the effects were highly significant only in the primary infertile men.

The relationship matrix between AMH and baseline characteristics of the whole study sample and each group is presented in Table 2. Although a significant positive correlation of AMH with BMI and slow progressive sperm motility ($r = 0.3342$, $p < 0.0403$, $r = 0.3859$, $p < 0.0167$), respectively, was approved, there was a non-significant correlation between AMH and age, duration of the marriage, non-progressive and progressive sperm motility, normal and abnormal sperm shapes in seminal fluid, and BMI specific to secondary infertility. In primary infertility, there was a significant correlation between AMH with normal ($r = 0.3473$, $p < 0.0011$) and an inversely but significant relationship with abnormal sperm shapes in seminal fluid ($r = -0.3313$, $p < 0.0020$).

Results of serum and semen analysis for cytokines

The present research revealed a highly significant elevation (P≤0.0001) in serum and semen cytokines (IL1β,

Table 1. Association between AMH as a dependent variable with socio-demographic and anthropometric parameters as independent variables in control, primary, and secondary infertility male subjects (Mean ±MSE).

Stat. Variables	AUC		95%CI		P value		Mean ± MSE		
Demographical and Semen Parameters	Primary	Secondary	Primary	Secondary	Primary	Secondary	Control	Primary	Secondary
Age (Years)	0.7893	0.6134	0.7023 to 0.8762	0.4889 to 0.7379	**<0.0001**	ns	40.80±1.58	31.28±0.58↓	36.60±1.16↓
BMI (Kg/m^2)	0.8918	0.8594	0.8322 to 0.9513	0.7764 to 0.9423	**<0.0001**	**<0.0001**	23.73±0.33	28.04±0.28↑	27.66±0.45↑
Duration of Marriage (Years)	0.7330	0.5119	0.6336 to 0.8324	0.3834 to 0.6404	**<0.0001**	ns	10.20±1.40	4.463±0.27↓	8.550±0.90↓
Non-progressive Sperm Motility (%)	0.7728	0.7094	0.6948 to 0.8507	0.5947 to 0.8240	**<0.0001**	**0.0013**	32.13±1.87	47.70±1.65↑	43.13±2.53↑
Slow Progressive Sperm Motility (%)	0.5924	0.5150	0.4881 to 0.6967	0.3863 to 0.6437	ns	ns	29.00±2.15	33.70±1.32↑	30.25±2.61↑
Progressive Sperm Motility (a + b) (%)	0.9620	0.9016	0.9254 to 0.9986	0.8230 to 0.9802	**<0.0001**	**<0.0001**	38.88±1.24	18.60±0.71↓	26.63±0.63↓
Total Sperm Count(x10^6)	1.000	1.000	1.000 to 1.000	1.000 to 1.000	**<0.0001**	**<0.0001**	151.6±4.86	17.08±1.06↓	31.15±2.77↓
Normal Sperm Shapes in Seminal Fluid (%)	0.9879	0.8325	0.9753 to 1.000	0.7458 to 0.9192	**<0.0001**	**<0.0001**	71.38±1.72	33.30±0.95↓	54.00±2.12↓
Abnormal Sperm Shapes in Seminal Fluid (%)	0.9915	0.8313	0.9815 to 1.000	0.7439 to 0.9186	**<0.0001**	**<0.0001**	28.63±1.65	66.95±0.91↑	46.00±2.12↑

Note: AUC= Area under the ROC curve, CI= Confidence Interval; Data were expressed as Mean ± MSE; Total P-values were obtained by the Kruskal-Wallis test and the Sum of the volume (overall sperm count) of the right and left testis.

Table 2. Correlation (r) between AMH as a dependent variable with socio-demographic and anthropometric parameters as independent variables in control, primary, and secondary infertility in male subjects.

Experimental Groups / Demographical and Semen Parameters	Control (n=40)		Primary Infertility(n=100)		Secondary Infertility (n=40)	
	r	P	r	P	r	P
Age	0.3880	**0.0160 ***	-0.1134	ns	0.1378	ns
BMI	-0.4061	**0.0114 ***	0.0380	ns	0.3342	**0.0403 ***
Duration of Marriage	0.1824	ns	0.0194	ns	0.1904	ns
Non-progressive Sperm Motility	-0.5329	**0.0006 *****	-0.0471	ns	-0.2181	ns
Slow Progressive Sperm Motility	0.1836	ns	0.03909	ns	0.3859	**0.0167 ***
Progressive Sperm Motility	0.4615	**0.0035 ****	-0.0399	ns	-0.2209	ns
Total Sperm Count	-0.0501	ns	0.0977	ns	0.02592	ns
Normal Sperm Shapes in Seminal Fluid	0.3773	**0.0195 ***	0.3473	**0.0011 ****	0.1026	ns
Abnormal Sperm Shapes in Seminal Fluid	-0.4441	**0.0052 ****	-0.3313	**0.0020 ****	-0.1026	ns

IL17 in serum, TNF α, and ASA) for males with primary and secondary infertility compared with controls. Meanwhile, a significant ($p \leq 0.001$) increment in IL6 in serum and Semen and IL17 in Semen was observed in association of AMH with seminal and serum parameters of the studied cytokines compared to control groups (Table3). According to Table 4, there was a correlation between AMH and cytokines in the entire samples and each group. Also, IL1β in semen showed a significant correlation ($r = -0.2107$, $p < 0.0373$, $r = 0.3472$, $p < 0.0327$), respectively, in primary and secondary infertile men, while IL1β in serum showed a significant ($p<0.0038$) correlation ($r = 0.2872$) in primary infertility only. Although a significant positive correlation was observed between AMH and IL6 in serum ($r = 0.4480$, $p < 0.0048$), specific to secondary infertility, there was no significant correlation concerning IL6 in Semen, IL17, TNFα, and ASA in both serum and Semen.

The serum hormonal concentration analysis

The association between AMH and serum hormonal concentrations (FSH, LH, TSH, testosterone, and prolactin) is presented3 in Table 5. When compared to the control group, the current research found significant ($P<0.0004$ and $P<0.0001$) increases in the mean levels of hormones (LH and prolactin) in primary and secondary infertile men. Meanwhile, the mean levels of testosterone decreased significantly($P<0.0001$) in primary and secondary infertility, while TSH concentration increased significantly ($P<0.0035$); however, FSH showed no significant differences in primary and secondary groups compared with the control group.

The correlations between AMH and sera hormones are represented in Table 6. According to the current study results, there were negative correlation ($r = -0.2463$, $p < 0.0222$ and $r = -0.3032$, $p < 0.0045$) between AMH and FSH and testosterone, respectively, in primary infertility, although a positive significant correlation ($r = 0.4069$, $p < 0.0169$) was observed between AMH and LH in secondary infertility, as well as AMH and TSH($r = 0.2584$, $p < 0.0163$) in primary infertility.

Discussion

Anthropometric characteristics and semen analysis results

The total number of semen samples examined in this study was 140. Men with primary infertility made up 71.43% of the population, while those with secondary infertility made up 28.57%. This was consistent with earlier research results conducted in Iraq (17–19). Other comparable studies have been accomplished as well. Compared to the current data, primary infertility has been more prevalent in Nigeria (20). In contrast, primary infertility has affected 70.7% of Egyptian couples, while secondary infertility has affected 29.3% (21).

To the best of our knowledge, this is the first study on Kurdish infertile males to evaluate the assumed association between AMH and sperm quality, the fertility of semen samples, and sperm functioning characteristics. The fact that AMH is made by Sertoli cells present in seminiferous tubules (the sperm production location) clarifies the correlation between AMH and semen characteristics (22).

A higher rate of sperm aneuploidy and sperm DNA damage in males with poor semen parameters may impact the outcome for infertile men, according to the relationship between serum AMH and semen parameters [non-progressive sperm motility (%) and aberrant sperm morphologies in seminal fluid (%)] (23). The serum AMH level is moderately adversely associated with semen quality. Since defective Sertoli cell function and progressive motility are adversely connected with serum AMH concentration, spermatogenesis may be affected by abnormal AMH production (24).

Results of serum and semen analysis for cytokine

Seminal plasma cytokine levels in the current study were considerably greater in infertile patients compared to healthy controls, shedding light on its function in the pathophysiology of male infertility. According to recent research (25), IL-17 concentration rises in infertile men and may be a useful biomarker of chronic urogenital tract inflammation. The current study found a negative correlation between the seminal plasma IL-17 level and AMH; similar findings were also found by Qian et al. (26). It is believed that the mechanism by which IL-17 may reduce sperm motility is oxidative stress increasing in the seminal plasma caused by various cytokines (27).

According to the current study, the immune system's defense mechanisms against bacterial infections may involve the release of proinflammatory cytokines, principally IL-6 and TNF, as primary or secondary signals (28). Regarding the negative correlation between ASA and AMH, as well as the possibility that ASA's elevated levels are caused by the presence of other pathological factors other than low Sertoli cells (29).

The serum hormonal concentration analysis

The present study results were supported by Sulthan et al. (30), who found that 20%–30% of males had decreased testosterone and increased LH; the malfunctioning of basal Leydig cells is suggested as the explanation. According to a related investigation, testosterone, testosterone index, and LH were 18%, 26%, and 34% in infertile males, being lower than those of the fertile control participants (31).

The present study looked at the relationship between AMH and factors related to semen analysis and sex hormone serum levels. Results showed that serum levels of various sex hormones differed in men depending on BMI status. On the other side, some studies have found no connection between BMI and any hormonal or Semen characteristics (32).

Male infertility and serum AMH did not correlate based on the present research, consistent with Tuttelmann *et al*. (7) and El. Halawaty *et al*. (33). Seminal AMH was observed to be strongly negatively correlated with serum FSH levels in men with primary infertility, in agreement with Sabetian *et al.* (34). According to Fujisansa (35), serum levels of LH, prolactin, and testosterone did not significantly associate with the amount of AMH in seminal plasma. The production of testosterone by testicular Leydig cells, which is regulated by LH, has a significant impact on spermatogenesis, whereas FSH stimulates the production of AMH by Sertoli cells (36). Additionally, these findings show a favorable relationship between AMH and Sertoli cell activity, indicating that serum AMH may serve as an endocrinological marker in spermatogenesis (37).

In conclusion, AMH in seminal plasma is a reliable

Table 3. Correlation between AMH as a dependent variable and serum and seminal cytokines (IL1β, IL6, IL17TNFα, and ASA) as independent variables in control, primary, and secondary infertility in male subjects (Mean ± MSE).

Stat. Variables	AUC		95%CI		P-value		Mean ± MSE		
Cytokine Parameters	Primary	Secondary	Primary	Secondary	Primary	Secondary	Control	Primary	Secondary
IL1β in Semen (pg/ml)	0.8041	0.7603	0.7344 to 0.8738	0.6513 to 0.8693	<0.0001	<0.0001	5.923±0.3247	21.15±1.998↑	13.60±1.493↑
IL1β in Serum (pg/ml)	0.9006	0.9094	0.8519 to 0.9493	0.8437 to 0.9751	<0.0001	<0.0001	4.229±0.3098	19.53±1.589↑	12.36±1.057↑
IL6 in Semen (pg/ml)	0.7241	0.5719	0.6232 to 0.8251	0.4421 to 0.7016	<0.0001	ns	6.128±0.8862	10.31±0.4919↑	8.270±1.111↑
IL6 in Serum (pg/ml)	0.6818	0.6109	0.5950 to 0.7685	0.4830 to 0.7388	0.0008	ns	4.593±0.1323	6.071±0.2386↑	5.073±0.2102↑
IL17 in Semen (pg/ml)	0.7644	0.5730	0.6687 to 0.8600	0.4439 to 0.7021	<0.0001	ns	2.599±0.3010	4.293±0.1484↑	3.105±0.2532↑
IL17 in serum (pg/ml)	0.8685	0.7325	0.8104 to 0.9266	0.6103 to 0.8547	<0.0001	0.0003	2.108±0.1065	4.125±0.1781↑	3.300±0.2476↑
TNFα in Semen (pg/ml)	0.9869	0.6972	0.9730 to 1.000	0.5814 to 0.8130	<0.0001	0.0024	3.856±0.1756	6.840±0.1106↑	4.946±0.2615↑
TNFα in serum (pg/ml)	0.8868	0.6838	0.8343 to 0.9393	0.5586 to 0.8091	<0.0001	0.0044	3.519±0.1214	5.839±0.1646↑	4.594±0.2554↑
ASA in Semen	0.6957	0.7743	0.6080 to 0.7834	0.6706 to 0.8781	0.0003	<0.0001	37.13±2.570	60.98±4.094↑	61.37±4.814↑
ASA in Serum	0.7191	0.7578	0.6367 to 0.8016	0.6494 to 0.8662	<0.0001	<0.0001	34.13±2.245	66.32±5.302↑	65.75±7.239↑

Table 4. Correlation between AMH as a dependent variable and both serum and seminal cytokines (IL1β, IL6, IL17, TNFα, and ASA) as independent variables in control, primary, and secondary infertility in male subjects.

Experimental Groups / Cytokine Parameters		Control		Primary Infertility		Secondary Infertility	
		r	P	r	P	r	P
IL1β	In Semen	0.0540	ns	-0.2107	**0.0373** *	0.3472	**0.0327** *
	In Serum	-0.160	ns	0.2872	**< 0.0038** **	0.0896	ns
IL6	In Semen	0.1252	ns	-0.1198	ns	-0.1805	ns
	In Serum	-0.1148	ns	0.03363	ns	0.4480	**0.0048** **
IL17	In Semen	0.1700	ns	0.0304	ns	0.0930	ns
	In Serum	-0.0490	ns	0.04121	ns	0.1364	ns
TNFα	In Semen	0.1122	ns	0.0630	ns	-0.2553	ns
	In Serum	-0.0397	ns	0.0670	ns	0.0283	ns
ASA	In Semen	-0.3498	**-0.3498** *	-0.0880	ns	-0.2787	ns
	In Serum	-0.3498	ns	-0.0164	ns	-0.0550	ns

Table 5. Association between AMH as a dependent variable and sera hormones (FSH, LH, TSH, testosterone, and prolactin) as independent variables in control, primary, and secondary infertility in male subjects (Mean ± MSE).

Stat. Variables	AUC		95%CI		P-value		Mean ± MSE		
Hormonal Parameters	Primary	Secondary	Primary	Secondary	Primary	Secondary	Control	Primary	Secondary
FSH	0.5924	0.5150	0.4881 to 0.6967	0.3863 to 0.6437	ns	ns	29.00±2.15	33.70±1.32↑	30.25±2.61↑
LH	0.6924	0.6675	0.5974 to 0.7873	0.5463 to 0.7887	0.0004	0.0099	3.865±0.221	5.878±0.41↑	5.995±0.577↑
TSH	0.5660	0.6897	0.4533 to 0.6787	0.5722 to 0.8072	ns	0.0035	2.179±0.28	2.294±0.18↑	3.147±0.293↑
Testosterone	0.9948	0.8578	0.9879 to 1.000	0.7766 to 0.9391	<0.0001	<0.0001	6.176±0.14	2.737±0.081↓	4.555±0.201↓
Prolactin	0.9471	0.8134	0.9059 to 0.9883	0.7208 to 0.9060	<0.0001	<0.0001	5.886±0.58	19.02±0.79↑	11.52±0.859↑

Table 6. Correlation between AMH as a dependent variable and sera hormones (FSH, LH, TSH, testosterone, and prolactin) as independent variables in control, primary and secondary male infertile subjects.

Experimental Groups / Parameters	Control		Primary Infertility		Secondary Infertility	
	r	P	r	P	r	P
FSH	-0.0398	ns	-0.2463	**0.0222** *	0.0158	ns
LH	-0.1298	ns	-0.1181	ns	0.4069	**0.0169** *
TSH	-0.0416	ns	0.2584	**0.0163** *	0.2361	ns
Testosterone	0.1893	ns	- 0.3032	**0.0045** **	0.1518	ns
Prolactin	0.1052	ns	-0.0180	ns	-0.1261	ns

marker of male infertility with a role in sperm production. The ability to accurately predict testicular sperm extraction success in non-obstructive azoospermia is also of clinical significance. This study has established a foundation for future research by demonstrating the utility of AMH in determining Kurdish males' reproductive potential.

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None

Interest conflict
No conflicts of interest have been declared by the author. No one else was involved in the study's planning, data collection, analysis, clarification, article preparation, or decision to publish the findings except the author.

Author's contribution
Fakhir Najim K. Sabir did the work alone.

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