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SPERM COUNTS AND HORMONAL PROFILES OF ADULT MALES IN ABA METROPOLIS, SOUTH EAST NIGERIA

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ABSTRACT

Aim: This study aimed at providing baseline data on the fertility status and profile of male residents in Aba metropolis, South East Nigeria. **Materials and Methods:** One hundred and eighty-six men (186) between the ages of twenty-five to fifty years (25-50), comprising 84 fertile control, and 102 men diagnosed with infertility and attending infertility clinics in Aba metropolis were recruited into the study. The infertile subjects comprised of current smokers (26), those exposed to pesticides used in the control of pests and weeds in the farms and other pollutants (46) and bicycle riders (30). Semen samples were collected from the subjects after a minimum of 5 days abstinence from sexual intercourse. The percentage of motile spermatozoa, viable spermatozoa, sperm count and morphology of the spermatozoa were determined using standard WHO procedures. Follicle stimulating hormone, luteinizing hormone, testosterone and prolactin were analysed using enzyme linked immunosorbent assay kits. Analysis of variance (ANOVA) was used to compare mean differences among subgroups whilst student t-test was used to compare the differences between two groups. Values were considered significant at $p<0.05$. **Results:** The levels of FSH, LH, testosterone and sperm counts in the infertile subjects were significantly decreased ($p<0.05$) when compared with the levels in the fertile control subjects. Of the 102 diagnosed infertile males, (22.55%) of them were oligoospermic while (2.94%) were azoospermic. The levels of the pituitary hormones were significantly ($p<0.05$) increased in the oligospermic and azoospermic infertile males whereas the values were significantly reduced in the general subgroups of the infertile population. The sperm counts and testosterone level were, however, significantly reduced in both groups of the infertile males. **Conclusion:** This study showed significant decrease in gonadotrophins hormone (FSH, LH) in 74.51% and an increase in 25.49% but testosterone and sperm counts were low in all subgroups.

KEYWORDS: Infertility, follicle stimulating hormone, luteinising hormone, testosterone, prolactin.

INTRODUCTION

Procreation is one of the main reasons for the institution of marriage.[1] Infertility has been defined as the failure to conceive after one year of regular unprotected sexual intercourse in the absence of known reproductive pathology.[2] Because some couples, who are not infertile, may not be able to conceive within the first one year of unprotected sex, the World Health Organisation, therefore recommends the epidemiological definition of infertility, which is the inability conceive within two years of exposure to pregnancy.[2] In view of the importance attached to parenthood in Africa, it is not surprising that infertility is reportedly considered a major cause for divorce and marital instability.[3,4]

In Africa, whenever infertility occurs, women are often blamed while men assumed innocence. Ignorantly, women are made to bear the brunt of infertility.[5] There is a high prevalence of infertility in sub-Saharan Africa, and prevalence rate of 20-46% has been reported.[6,7] In Nigeria, however, studies have reported incidence of 20-30 %.[8] In Western countries, 10-15% of couples experience infertility.[6] Generally, it has been reported that male partners directly or indirectly contribute about 25-30% and 25 % of infertility respectively.[6,9]

Majority of cases of male infertility are attributable to abnormal semen parameters whose causes are often not easy to identify.[6] Recently, younger men have also been reported to have lower sperm count and poorer sperm quality than in the past.[10] The rapid expansion of the chemical industry in both developed and developing countries have resulted in the release of a plethora of xenobiotics into the environment.[11] Exposure to certain chemicals in the environment, particularly persistent chlorinated lipophilic compounds, have been reported to alter normal embryonic development in ways that adversely impact subsequently on reproductive,

endocrine, nervous and immune system of a variety of fish, bird and mammalian wildlife species.[12]

Aba, the industrial nerve center of Nigeria is located in Abia State, in South Eastern Nigeria. It is one town where various commercial and industrial activities take place ranging from manufacturing and agricultural activities with resultant emission of dangerous gases and waste from industrial plants and vehicles to gases from fermented dumped refuse on major roads. These activities frequently exposed the residents to various environmental chemicals and anthropogenic substances which could have potential effects on their reproductive health. Data on the hormonal profiles and reproductive integrity of male residents in most industrial cities in Nigeria is scarce. This study is therefore aimed at providing baseline data on the fertility status and profile of male residents in Aba metropolis, in South East Nigeria.

MATERIALS AND METHODS

Subjects' selection and study area: One hundred and eighty-six men (186) between the ages of twenty-five to fifty years (25-50), attending infertility clinics in Aba metropolis were recruited into the study. They included patients attending infertility clinics at Abia State Teaching Hospital, Aba, Rosevine Hospital, Best Care Hospital, Living Word Mission Hospital, Hariket Medical Diagnostics, Excellence Medical Diagnostics and Solution Medical Diagnostics. The men were all married but were unable to impregnate their wives after a period of 2-5 years of regular unprotected sexual intercourse.

The major inclusion criteria into the study was men who were accompanied by their wives to the clinics and diagnostic laboratories and did accept the methods of seminal fluid collection. Men who refused to allow the methods of seminal fluid collection and were not accompanied by their wives were excluded from the study. Withdrawal method was discouraged since semen may be lost with withdrawal method or get contaminated with vaginal secretions. Effort was made to procure non-spermicidal condoms (Milex limited) for the participants following the reports of Zarvos and Goodpasture,[13] that ejaculates produced and recovered into non-spermicidal condoms used during intercourse at home are of a higher quality. Some of the study subjects were involved in series of activities and occupations such as agriculture which exposed them to pesticides used in the control of pests and weeds in the farms, local manufacturing while others were bicycle riders. Some of the subjects were current smokers of various forms of cigarettes. The semen samples were collected from the subjects after about a minimum of 5 days of abstinence from sexual intercourse.

Ethical approval

Ethical approval was obtained from the Ethics Committee of the Abia State Health Management Board and Abia State University Teaching Hospital, Aba, Nigeria and informed consent was obtained from all participants that were involved in this study.

Sample size calculation

The number of samples used in this research was determined using the formula below:

$$N = \frac{Z\alpha^2 pq}{d^2}$$

Where N = desired sample size
$Z\alpha$ = the α level of the coefficient interval at 95% (1.96)
p = proportion of occurrence
q = (1-p) proportion of non-occurrence
d = precision

Substituting the expected occurrence of p= 9% i.e. 0.09 from WHO[14] we have

$$N = \frac{1.96^2 \times 0.09\ (1 - 0.09)}{(0.05)^2}$$

$$= 125$$

Sample collection

Blood: Blood samples were collected from the subjects by venous puncture according to the method of Sood.[15] The blood samples were allowed to clot, retract and serum was separated within two hours of collection and used for the hormonal assay.

Semen: Modified 'masturbation' was the main method of collecting semen from the subjects in the hospitals and diagnostic laboratories where the study was conducted. Here the wife did the 'masturbation' in a dedicated room with dedicated bed and other facilities that made them relax. Some men who lived very close to the laboratory where the samples were analysed were allowed to collect at home and brought to laboratory within 15-20 minutes. In that case, the laboratory scientists were ready for them to analyse the semen immediately within 30 minutes to one hour of collection.

Laboratory determinations

Semen analysis

The percentage of motile spermatozoa was determined by putting a drop (10-15µl) of well mixed liquefied on a clean, grease-free dry slide and covered with 22 x 22 mm glass cover slip. It was then focused using 10X objective and 40X objective was used to examine several fields to assess motility. A total of 100 spermatozoa were counted and the percentage of motile and non-motile spermatozoa were estimated.[17]

Estimation of the percentage of viable spermatozoa was done by mixing one drop (10-15µl) of 0.5% eosin solution on a slide and examining the preparation microscopically after 2 minutes by using 10X objective to focus and 40X objective to count the percentage of viable and non-viable spermatozoa. Viable spermatozoa are motile while non-viable spermatozoa are not motile after 2 minutes in 0.5%eosin solution.[17]

The sperm count was performed by diluting semen 1:20 with sodium bicarbonate formalin solution and mixed properly. An improved Neubaeur ruled counting chamber was filled with the well mixed diluted semen and allowed to settle for 3-5 minutes. The 10X objective was then used to count the number of spermatozoa in 1 ml of area of 2sq mm (i.e. 2 large squares). The number of spermatozoa in 1 ml of fluid was calculated by multiplying the number counted by 100,000.[17]

The morphology of the spermatozoa was also determined. A thin film of liquefied well-mixed semen was made on a dry grease free slide, fixed with 95% v/v ethanol while still wet for 5-10 minutes and allowed to air dry. The slide was then washed with bicarbonate formalin solution to remove any mucus which may be present and the smear was rinsed with several changes of water. The washed smear was covered with dilute (1 in 20) 0.2% carbon fuschin and allowed to stain for 3 minutes. The stain was then washed off with water and the smear was counterstained with dilute (1 in 20) 1.0% Loeffler's methylene blue for 2 minutes and later washed off. The smear was air dried and examined using 40X objective and 100X was used to confirm the abnormlities.100 spermatozoa were counted and percentage estimate of normal and abnormal form was done. Normal spermatozoa have long tail and oval head while abnormal spermatozoa have double or pointed head with forked or curled tail.[17]

Determination of the serum concentration of Hormones

Follicle stimulating hormone, luteinizing hormone, testosterone and prolactin were analyzed using Enzyme Linked Immunosorbent Assay kits obtained from DRG International Incorporated, East Mountain Side, US.A. The tests were carried out according to the manufacturer's instruction.

Statistical analysis

The results were statistically analysed using the Statistical Package for Social Sciences (SPSS) version 21. Data were expressed as mean ± SD. Analysis of variance (ANOVA) was used to compare mean differences among groups whilst student t-test was used to compare the differences between two groups. Values were considered significant at $p<0.05$.

RESULTS

The general motility and morphology of spermatozoa in the subject groups studied is shown in table 1. The table showed that actively motile spermatozoa were observed in 34.92% of the subjects while moderately active spermatozoa were seen in 24.87% of the subjects studied. The percentage of the subjects with sluggishly motile and non-motile and dead spermatozoa were 20.43% and 18.83% respectively). The observed morphology of the spermatozoa revealed that 59.79% of the population studied exhibited spermatozoa whose forms could be described as normal forms while 40.21% showed abnormalities (abnormal forms include spermatozoa with forked tail, coiled tail, tapered and large heads) according to criteria described in the WHO.[16]

The percentage of the fertility profile of subjects in the infertile subgroups and fertile control is shown in table 2. Assessment of the percentage fertility of the subgroups based on normal FSH, LH, testosterone and sperm count with low or normal prolactin levels showed that 100% of the control subjects were fertile while only 76.92% of smokers were fertile. The proportion of fertile male populations exposed to environmental pollutants and pesticides and bicycling were 67.39% and 76.67% respectively. Also, the percentage of the infertile male population assessed based on high FSH, LH, prolactin with low testosterone and sperm count were 23.08%, 32.61% and 23.33% for smokers, male populations exposed to environmental pollutants and pesticides and bicycling respectively.

The concentration of follicle stimulating hormone (FSH), luteinising hormone (LH), testosterone, prolactin and sperm count in the various categories of subjects who participated in the study is shown in table 3. The table shows that the mean ±SD of FSH in the control subjects, smokers, subjects exposed to environmental pollutants and bicyclists were 10.08± 5.28 mlU/ml, 5.67±2.48mlU/ml, 7.97.±3.28mlU/ml and 12.48±4.11mlU/ml respectively. Comparison of the means of FSH between the sub-groups of subjects showed significant difference ($p<0.05$, $F=3.99$). The means ±SD of the luteinising hormones (LH) in the control subjects, smokers, those exposed to environmental pollutants/pesticides and bicyclists were 9.20±5.24 ng/ml, 7.89±4.14ng/ml, 7.01±1.50ng/ml, and 6.28±2.44 respectively. Significant difference ($p<0.05$, $F=4.21$) in means between the sub-groups was also observed. Testosterone levels in the subgroups were 6.15±2.00 ng/ml, 3.38±1.68 ng/ml, 5.30±3.11 ng/ml and 3.98±2.55 ng/ml respectively. No significant difference ($p>0.05$, $F=1.99$) in means was observed between the subgroups. Also, the means ±SD of prolactin in the control subjects, smokers, subjects exposed to environmental pollutants and bicyclists were 8.20±4.11ng/ml, 9.40±3.93ng/ml, 8.47±5.30ng/ml and 6.54±2.83 ng/ml respectively with no obvious significant variation ($p>0.05$, $F=1.89$) in mean between the subgroups. The sperm count values for the control subjects, smokers, subjects exposed to environmental pollutants and bicyclists were 40.49±13.42 (x 10^6/ml), 13.94±6.98 (x 10^6/ml), 10.51±5.47 (x 10^6/ml) and 10.05±4.49 (x 10^6/ml) respectively. Comparison of the means of the sperm count between the sub-groups of subjects showed significant difference ($p<0.05$, $F=3.56$).

The comparison of the mean of FSH, LH, testosterone prolactin and sperm count between the smokers and control subjects is shown in table 4. Significant difference ($p<0.05$, $t=1.99$) in means was observed in the

mean of FSH, LH, testosterone and sperm count while no significant variation (p>0.05, t=1.99) was observed in the means of prolactin. Similarly, the comparison of the mean of FSH, LH, testosterone prolactin and sperm count between the subjects exposed to environmental pollutants/pesticides and control is shown in table 5. Significant difference (p<0.05, t=1.99) in means was observed in the mean of FSH, LH and sperm count while no significant variation (p>0.05, t=1.99) was observed in the means of testosterone and prolactin. Further comparison of the mean of FSH, LH, testosterone prolactin and sperm count between the bicyclists and control subjects is shown in table 6 and significant difference (p<0.05, t=1.99) in means was observed in the mean of FSH, LH, testosterone and sperm count while no significant variation (p>0.05, t=1.99) was observed in the mean prolactin.

Of the 102 diagnosed infertile males, 23 (22.55%) of them were oligoospermic while 3 (2.94%) were azoospermic. The result of serum FSH, LH, prolactin, testosterone levels and sperm counts in oligoospermic and azoospermic subjects and fertile control are presented in table 7. The FSH, LH, prolactin, testosterone and sperm count in the oligoospermic infertile subjects were 14.97±8.55 mIU/ml, 11.70±6.07 ng/ml, 11.83±5.83ng/ml, 2.86±1.53 ng/ml and 12.05±8.48 x 10^6/ml respectively while the levels of the hormones and sperm count in the azoospermic infertile subjects were 32.43±16.38 mIU/ml, 21.90±6.89 ng/ml, 31.70±11.98 ng/ml and 0.00±00 respectively. Comparison of the means of these parameters using ANOVA showed that significant differences (p<0.05, F=5.46) in means exist in the means of FSH between the oligoospermic infertile, azoospermic infertile and control fertile subjects, LH (p<0.05, F=6.70), prolactin (p<0.05, F=6.89), testosterone (p<0.05, F=3.67) and sperm count (p<0.05, F=11.67) respectively.

Table 1: General motility and morphology of the spermatozoa in the subject groups (combined).

Motility	**Percentage (%)**
Actively motile	35.48%
Moderately motile	25.26%
Sluggishly motile	20.43%
Non-motile	18.83%
Morphology	**Percentage (%)**
Normal forms	59.70%
*Abnormal forms	40.30%

*abnormal forms include spermatozoa with forked tail, coiled tail, tapered and large heads

Table 2: Percentage assessment of the fertility profile of the subjects.

Subject group	**% Fertile population (i.e. normal FSH, LH, testosterone and sperm count) and low or normal prolactin level**	**% Infertile population (i.e. high FSH, LH, prolactin with low testosterone) and low sperm count.**
Control (n=84)	100	0
Smokers (n=26)	76.92 (n=20)	23.08 (n=6)
Men exposed to pesticides/ pollutants (n=46)	67.39 (n=31)	32.61 (n=15)
Bicyclists (n=30)	76.67 (n=23)	23.33 (n=7)

Table 3: Mean ± SD of FSH, LH, Testosterone, Prolactin and Sperm Counts of the subjects.

Subjects	**FSH (mlU/ml)**	**LH (ng/ml)**	**Testosterone (ng/ml)**	**Prolactin (ng/ml)**	**Sperm count (x 10^6/ml)**
Control (n=84)	10.08±5.28	9.20±5.24	6.15±2.00	8.20±4.11	40.49±13.42
Smokers (n=26)	5.10±1.48	6.89±4.14	4.38±1.68	9.40±3.93	13.94±6.98
Men exposed to pesticides (n=46)	7.97±3.28	7.01.±1.70	3.30±3.11	8.47±5.30	10.51±5.47
Bicycling (n=30)	4.48±1.10	6.28±2.44	3.98±2.55	6.54±2.83	10.05±4.49
p- value	p<0.05, F=2.99	p<0.05, F=4.21	p>0.05, F=1.99	p>0.05, F=1.89	p<0.05, F=3.56

Table 4: The effect of smoking on FSH, LH, Testosterone, Prolactin and Sperm Counts of the subjects.

Subjects	**FSH (mlU/ml)**	**LH (ng/ml)**	**Testosterone (ng/ml)**	**Prolactin (ng/ml)**	**Sperm count (x 10^6/ml)**
Smokers (n=26)	5.10±1.48	6.89±4.14	4.38±1.68	9.40±3.93	13.94±6.98
Control (n=84)	10.08±5.28	9.20±5.24	6.15±2.00	8.20±4.11	49.49±13.42
p-value	p<0.05, t=1.99	p<0.05, t=1.99	p<0.05, t=1.99	p>0.05, t=1.99	p<0.05, t=1.99

Table 5: The effect of pesticide exposure on FSH, LH, Testosterone, Prolactin and Sperm Counts of the subjects.

Subjects	FSH (mlU/ml)	LH (ng/ml)	Testosterone (ng/ml)	Prolactin (ng/ml)	Sperm count (x 10^6/ml)
Men exposed to pesticide/ pollutants (n=46)	7.97±3.28	7.01.±1.70	3.30±3.11	8.47±5.30	10.51±5.47
Control (n=84)	10.08±5.28	5.20±1.24	6.15±2.00	8.20±4.11	49.49±13.42
p-value	p<0.05, t=1.99	p<0.05, t=1.99	p<0.05, t=1.99	p>0.05, t=1.99	p<0.05, t=1.99

Table 6: The effect of bicycling on FSH, LH, Testosterone, Prolactin and Sperm Counts of the subjects.

Subjects	FSH (mlU/ml)	LH (ng/ml)	Testosterone (ng/ml)	Prolactin (ng/ml)	Sperm count (x 10^6/ml)
Bicycling (n=30)	4.48±1.10	6.28±2.44	3.98±2.55	6.54±2.83	10.05±4.49
Control (n=84)	10.08±5.28	9.20±3.24	6.15±2.00	8.20±4.11	49.49±13.42
p-value	p<0.05, t=1.99	p<0.05, t=1.99	p<0.05, t=1.99	p>0.05, t=1.99	p<0.05, t=1.99

Table 7: The FSH, LH, Testosterone, Prolactin and Sperm Counts of infertile and control subjects.

Subjects	FSH (mlU/ml)	LH (ng/ml)	Testosterone (ng/ml)	Prolactin (ng/ml)	Sperm count (x 10^6/ml)
Oligospermia (infertile subjects) (n=23)	14.97±8.55	11.70±6.07	2.86±1.53	11.83±5.83	12.05±8 .49
Azoospermia (infertile subjects) (n=3)	32.43±16.38	21.90±6.89	1.47±0.21	31.70±11.98	0.00±0.00
Control (fertile subjects) (n=84)	10.08±5.28	9.20±5.24	6.15±2.00	8.20±4.11	49.49±13.42
p-value	p<0.05, F=5.46	p<0.05, F= 6.70	p<0.05, F= 3.67	P<0.05, F= 6.89	p<0.05, F= 11.67

DISCUSSION

Parenthood in Nigeria is of great importance and it is not surprising that when infertility is suspected among the couples, it could be very devastating and also constitute a major cause for divorce and marital instability.[3,4] There is always a tendency to blame the woman whenever conception fails to occur in a marriage.[18] Thus, infertility is a major public health problem with untold psychological disorders on the Nigerian couple. Follicle stimulating hormone (FSH), luteinising hormone (LH), prolactin and testosterone and sperm count evaluation is often employed in the management of male infertility.[19] In this study, the percentage of general population (fertile control, smokers, those exposed to environmental pollutants/pesticides and bicyclists combined) exhibiting spermatozoa that were actively motile was 35.48% while 45.69% were moderately and sluggishly motile with 18.83% of the spermatozoa being non-motile. The study further demonstrates that 59.70% of the general population were analysed with normal for normal spermatozoa morphology while 40.30% presented with abnormal spermatozoa morphology.

It is believed that smoking affects some male reproductive hormones such as luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin and testosterone. However, the actual effect of smoking on these hormones is highly controversial. In this study, the FSH, LH and prolactin levels in the smokers were lower when compared with the levels in control subjects who had never smoked and the difference in the means levels were significant (p<0.05, F=1.99). Bakheet and Amarshad[20] and Mitra *et al*.[21] have reported increased serum levels of LH and FSH in the studies on smokers while Pasqualotto *et al*.[22] reported no significant differences in levels of FSH, LH and total testosterone in smokers. FSH is required for the initiation of spermatogenesis and maturation of spermatozoa while LH is important in stimulating the production of hormones by the gonads.[23] In a study assessing the effects of tobacco smoking on hormone levels, Ochedalski *et al.*[24] reported that mean levels of LH, follicle-stimulating hormone (FSH), and prolactin were lower in smokers than in non-smokers, whereas the mean levels of testosterone did not differ. Similar results were reported in other studies investigating plasma hormone levels.[25] The findings reported in this study is thus in agreement with the works of Ochedalski *et al.*[24] ,Bakheet and Amarshad[20] and Mitra *et al*.[21]

While some authors report that smoking increases serum testosterone levels;[26] others have reported that testosterone levels are unaffected;[27] while another group have reported decreased serum testosterone levels in smokers.[28] However, in this study, the mean testosterone level in the infertile smokers was lower than that observed in the fertile non-smoking population and

the variation in mean was significant ($p<0.05$). The low testosterone level observed in this study agrees with finding of Vogt *et al.*[28] In the normal functioning of the hypothalamic–pituitary–gonadal (HPG) axis, when FSH and LH levels increase, this will stimulate an increase in the level of testosterone and inhibin B, which in turn decreases FSH and LH levels by negative feedback. Since the levels of FSH and LH are low in the smokers than in non-smoking control, it could be explained possibly some elements of tobacco smoke might have interrupted the regular functioning of the HPG system resulting to the failure of the Leydig cell in accordance with the report in smokers as recorded in previous studies.[29] It has also been reported that cigarette smoking lead to increased levels of prolactin in male smokers.[30,31] This finding was observed in our study, though the increase when compared to the control was not significant ($p>0.05$). A disruption in the hormonal balance of the sex hormones may lead to a decline in the quality of semen. The quality of semen greatly affects the chances of conception as it is generally considered to be a proxy measure of male fertility.

Exposure to environmental toxicants described as endocrine disrupters could interfere with sperm production or the function of reproductive hormones or sperm and may increase the risk of male infertility.[32] Reports have suggested that exposure to environmental factors (air pollution, pesticides, phthalates, PCB and the use of mobile phones) may affect semen quality and fertility in males.[32] Some of the male participants in the study works in farms where pesticides are used regularly to control weeds and pests. Some are also involved in local manufacturing of various kinds of products whose chemical composition are unknown to them and all of them admitted daily usage of mobile cell phones. The result of the present study showed that the levels of FSH and LH in the infertile subjects exposed to various environmental pollutants/ pesticides are significantly higher ($p<0.05$) than levels in the non-exposed fertile control whereas the levels of testosterone and sperm counts were significantly ($p<0.05$) lower in the infertile exposed subjects than those in fertile control subjects. Though the level of prolactin was lower in the exposed infertile subjects when compared to the fertile control, the difference was not significant ($p<0.05$). It has been reported that declining sperm counts were observed in individuals living in regions replete with heavy environmental pollution[33,34] implying that environmental pollutants may impair male fertility.[35] Sheiner *et al.*[36] and Jensen *et al.*[37] have that variety of environmental and occupational exposures may impair male fertility. Some of these pollutants e.g. pesticides which are regarded as endocrine disrupting chemicals (EDCs) are hypothesized to cause an adverse effect by interfering in some way with the body's hormones or chemical messengers.[38,39] The results of our study indicates that the pollutants could be acting on the testes and post-testicular sites in the infertile subjects resulting the low sperm count recorded.

A thorough and effective evaluation of infertility in the male involves examination of seminal quality and fertilization capacity as well as measurement of the hormone profiles.[40] In this study, we analysed FSH, LH, prolactin, testosterone as well as sperm counts, motility and morphology (table 2). Significant variation ($p<0.05$) in means was observed in the means of FSH, LH, testosterone and sperm counts between the fertile control and infertile subjects involved in bicycling. Although the LH levels was lower in the infertile subjects involved in bicycling, the difference was insignificant ($p>0.05$). The present study also reported that 22.3% of the infertile smokers were actually infertile based on the observation of a low testosterone level and low sperm count while 77.78% were fertile because they had normal FSH, LH, testosterone and sperm count while the men exposed to environmental pollutants/pesticides presented fertility profile of 68.42% and those involved in bicycling 75.0%. The general assessment of motility and morphology of the spermatozoa showed that only 35.48% and 25.26% were actively and moderately motile. The percentage of the spermatozoa that were sluggishly motile and even dead were 20.43% respectively indicating that the quality of the spermatozoa especially in the population was poor. A number of studies have demonstrated that cigarette smoking is correlated with alterations in sperm quality such as semen volume, sperm concentration, motility, and morphology.[41-43]

Regular exercise and physical activity promote overall health benefits that are hard to ignore. The benefits of exercise have been known since antiquity as Marcus Cicero, around 65 BC, stated: "It is exercise alone that supports the spirits, and keeps the mind in vigor.[44] Human spermatogenesis is a sensitive process and it is well known that testicular temperature must be maintained at approximately 2.5°C below core body temperature for normal spermatogenesis. Elevation of scrotal temperature to normal core body temperature has been reported to result in complete failure of spermatogenesis in man. This increased impaired reproductive function due to heat-induced alterations has been reported extensively.[45-47] As body temperature increases with exercise it has been hypothesized by several authors that the injurious effect of exercise on spermatogenesis can be attributed to increased scrotal temperature rather than hormonal changes.[49,50] It is reasoned that this phenomenon is more prevalent in cyclists due to wearing of tight-fitting shorts and compression of the scrotum against the saddle and body resulting in decreased thermoregulation.[51] In this study, significant variation in the means of FSH, LH, testosterone and sperm count ($p<0.05$). The testosterone level is reasonably below the lower limit acceptable for anticipated spermatogenesis. This has thus reflected in the sperm counts observed. This observation therefore agrees with the report of Lucia *et al.*[49] Arce *et al.*[50] and Brant *et al.*[51]

For men who have been diagnosed as infertile men, when the concentration of FSH is higher than normal, this finding is considered to be a reliable indicator of germinal epithelial damage, and has been shown to be associated with azoospermia and severe oligospermia. [52] de Kretser et al.[53] had reported the implication of elevated levels of serum FSH with increasing severity of seminiferous epithelial destruction. In this study, the levels of the pituitary hormones were significantly increased in the oligoospermic and azoospermic infertile males but testosterone (gonadal hormone) level was significantly lower. Similar depressed value was seen in the sperm count between the subgroups. Thus, a combination of factors may be proposed to be responsible for the hypergonadotropic hypogonadism observed in this study group.

In conclusion, the overall findings made in this study showed significant decrease in gonadotrophins hormone (FSH, LH) in all subgroups. The sperm counts was, however, decreased in all subgroups. This observation indicates that 74.51% (n=76) from all subgroups of diagnosed infertile males exhibited hypogonadotropic hypogonadism while 25.49% (n=26) demonstrated hypergonadotropic hypogonadism. Effort was not made to isolate the confounding factors that could account for the observations made in this study population. However, it is possible that the levels of the gonadotropins in the infertile subgroups might have caused a disruption of spermatogenesis resulting in decline of testosterone production which subsequently led to the consistent low sperm count and the poor quality of the spermatozoa seen in the infertile males.

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