**Hormonal Profiles in Primary Infertility: Evaluating AMH, LH, and Progesterone Levels in Women Attending a Tertiary Hospital in Abuja, Nigeria**

**Abstract**

**Background:** This analytical cross-sectional study investigated serum levels of anti-Müllerian hormone (AMH), luteinizing hormone (LH), and progesterone (PRG) among women diagnosed with primary infertility in Abuja, Nigeria.

**Methods:** Conducted at the University of Abuja Teaching Hospital, the study recruited 142 women comprising 71 with primary infertility and 71 age-matched fertile controls. Blood samples were collected during specific phases of the menstrual cycle: AMH and LH between days 2–5, and PRG between days 21–23. Laboratory analysis utilized enzyme-linked immunosorbent assay (ELISA) for AMH and chemiluminescence immunoassay (CLIA) for LH and PRG. Independent sample t-tests compared hormonal levels, and Pearson correlation explored relationships between age and hormones.

**Results:**Mean AMH levels were significantly lower in the primary infertility group compared to controls (1.85 ± 1.40 ng/ml vs. 3.52 ± 2.41 ng/ml; p<0.001), indicating diminished ovarian reserve among affected women. Similarly, mean progesterone levels were modestly but significantly reduced in women with primary infertility (11.44 ± 7.91 ng/ml) compared to fertile controls (13.65 ± 6.86 ng/ml; p=0.034), suggesting possible luteal phase insufficiency. LH levels showed a slight but non-significant difference between groups (8.65 ± 3.58 mIU/ml vs. 8.48 ± 3.43 mIU/ml; p=0.739). A moderate negative correlation was observed between age and AMH (r= –0.305, p<0.001), while a weak yet statistically significant positive correlation existed between AMH and progesterone (r= +0.180, p=0.036).

**Conclusion**: These results highlight the important role of AMH and progesterone in the reproductive profile of women with primary infertility in Abuja. The study recommends routine infertility evaluation should include assessment of AMH and PRG to improve diagnostic precision and guide personalized treatment strategies, ultimately supporting better reproductive outcomes.

**Keywords**: Primary infertility, AMH, LH, progesterone, ovarian reserve

**Introduction**: Infertility is a significant reproductive health challenge worldwide, affecting millions of couples and carrying substantial medical, psychological, and socioeconomic burdens. In Nigeria, infertility remains a common reason for gynecological consultations, with profound sociocultural consequences for affected women, including stigmatization, marital tension, and psychological distress (Ademola *et al.*, 2023). The World Health Organization defines infertility as the inability to achieve pregnancy despite at least 12 months of regular, unprotected sexual intercourse (WHO, 2020). Clinically, it is classified into primary infertility—where a woman has never conceived—and secondary infertility, where a woman who has previously conceived cannot do so again (Zegers-Hochschild *et al*., 2017).

Primary infertility is of particular concern in sub-Saharan Africa, including Nigeria, where cultural expectations strongly emphasize childbearing as a measure of a woman’s social status (Agarwal *et al*., 2021). This societal pressure can exacerbate emotional distress and delay timely medical evaluation. Limited access to advanced reproductive technologies and the high cost of treatment further constrain effective infertility management in Nigeria (Hassan *et al*., 2022).

Multiple factors contribute to female infertility, including anatomical abnormalities, infections, genetic predisposition, environmental exposures, lifestyle factors, and hormonal imbalances (Sauer, 2021). Among hormonal factors, anti-Müllerian hormone (AMH), luteinizing hormone (LH), and progesterone play central roles in regulating ovarian function and fertility (Hagen et al., 2018). AMH, secreted by granulosa cells of growing ovarian follicles, serves as a sensitive biomarker of ovarian reserve and reproductive lifespan (Thompson and Roberts, 2023). Abnormal AMH levels can signal diminished ovarian reserve or disorders such as polycystic ovary syndrome (PCOS).

Luteinizing hormone, produced by the anterior pituitary gland, triggers ovulation and supports luteal function (Williams *et al.*, 2022). Disruptions in LH secretion patterns can lead to anovulation or luteal phase defects. Progesterone, synthesized by the corpus luteum post-ovulation, prepares the endometrium for implantation and supports early pregnancy maintenance (Tehrani and Noroozzadeh, 2019). Inadequate luteal progesterone production may compromise implantation and increase miscarriage risk.

An imbalance among these hormones may result in ovulatory dysfunction, inadequate luteal support, and ultimately, infertility (Chen and Zhang, 2023). Accurate evaluation of AMH, LH, and progesterone is thus crucial in the diagnostic workup of women with infertility, especially where invasive tests or assisted reproductive techniques are limited. While previous studies have examined these hormones in various populations, limited data exist on Nigerian women, highlighting the need for context-specific research (Oladimeji *et al*., 2023).

This study was conducted to assess serum levels of AMH, LH, and progesterone among women with primary infertility attending the University of Abuja Teaching Hospital in Abuja, Nigeria, and compare them to those of fertile controls. By providing localized hormonal data, the findings aim to contribute to improved diagnostic accuracy and individualized treatment strategies, ultimately enhancing reproductive outcomes for Nigerian women facing infertility.

**Materials and Method**

**STUDY DESIGN:** The study was a Cross-sectional study of confirmed pregnant women who consented to be part of the study, age and gender- was matched monitored visiting the antenatal clinic at university of Abuja teaching Hospital

**STUDY AREA:** This study was conducted in university of Abuja Teaching Hospital gwagwalada, Federal Capital Territory (FCT) Abuja, Gwagwalada is about 62 km away from the FCT. It is one of the settler’s towns of the FCT. The town is close to the Nnamdi Azikwe international air port along the Abuja –Lokoja Express way, it is located between latitude 8°55’ and 9°00’N and longitudinal 7°00’ and 7°05’E.

The centrality of this town in relation to other area councils’ headquarters makes it influential and important in various socio‑economic activities. The climate condition of this town is not far-fetched from that of the tropics having several climatic elements in common; most especially the wet and dry season characteristic. The temperature of the area ranges from 30°C to 38°C yearly, with the highest temperature experienced in the month of March and mean total rainfall of approximately 1650 mm/annum.

About 60% of this rain falls between the months of May to August. The area council is an industrial zone of FCT that stands out as the second most cosmopolitan city of the FCT, after the capital city with 10 political wards. These have brought about the inflow of people into the council. 75% of the residents live in close proximity with poor drainage system, several pot-holes on their streets and indiscriminate environmental dumpsites.

**Study Population:** Women of reproductive age (20–40 years) diagnosed with primary infertility (inability to conceive after 12 months of regular unprotected intercourse) attending the infertility clinic at the University of Abuja Teaching Hospital, Gwagwalada, were recruited between December 2024 and Febuary 2025.

**Sample Size Determination:** The minimum sample size for this analytical cross-sectional study was determined using Fisher’s formula, as described by Ogbeibu (2014):

N = (Z² × P × q) / d²

Where:

N = the desired sample size

Z = the standard normal deviation (1.96 at 95% confidence level)

P = the estimated prevalence of primary infertility in women

q = 1 − P

d = the margin of error (typically 0.05)

The estimated prevalence of infertility among women in Nigeria is 3.9% (Mascarenhas *et al*., 2012). Substituting into the formula:

Z = 1.96

P = 0.039

q = 1 − 0.039 = 0.961

d = 0.05

N = (1.96)² × 0.039 × 0.961 / (0.05)²

= 3.8416 × 0.037479/ 0.0025

= 0.1434/ 0.0025

= 57.592

 ≈ 58

Na = n/1-r

Nadjustment (Na) = sample adjustment

n = initial sample size (58)

r = non response rate (18%)

Na = 58/1-0.18

= 58/0.82 = 70.7 ≈ 71

Thus, the final sample size for this study was rounded up to 71 participants.

**Selection Criteria**

**Inclusion Criteria:**Women aged 20–40 years,Diagnosed with primary infertility (for cases).Fertile women with at least one live birth and no history of infertility (for controls).

**Exclusion Criteria:** Known endocrine disorders (e.g., polycystic ovary syndrome, thyroid disorders).Chronic medical conditions (e.g., diabetes, hypertension).History of ovarian surgery or pelvic radiation.Use of hormonal contraceptives or fertility medications within the last three months.

**Ethical clearance and informed consent:** Ethical clearance was obtained from the ethical committee of university of Abuja teaching Hospital. Informed consent will also be obtained from all participating subjects in accordance with the standards of human experimentation and with the Helsinki Declaration of 1975, as revised in 70. This will be done via informed consent from study participants

**Blood Sample Collection and Processing:** All samples were collected between 8:00–10:00 AM using standard venipuncture technique into serum separator tubes (BD Vacutainer).

Blood samples were collected on two different occasions based on the hormones being evaluated.

A 5 mL blood sample was collected on days 2–5 (early follicular phase) of the menstrual cycle for the measurement of anti-Müllerian hormone (AMH) and luteinizing hormone (LH). Collecting samples during the early follicular phase provides a stable baseline for these hormones.

Blood samples were collected on days 21–23 of the menstrual cycle for the measurement of progesterone. Progesterone levels peak during the luteal phase (Williams and Richards, 2017).

**Sample processing:** Samples were allowed to clot at room temperature (22-25°C) for 30 minutes, followed by centrifugation at 3500 rpm for 15 minutes. Serum was immediately aliquoted into pre-labeled Cryovials tubes (3000 µL per aliquot) and stored at -80°C until analysis within three months (Rahman *et al*., 2023)

**Analytical methods**

 **Measurement of anti-Müllerian hormone**

Serum AMH concentrations were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Calbiotech Inc., USA), with an analytical sensitivity of 0.018 ng/mL. The assay was conducted according to the manufacturer's instructions, and all samples were analyzed in duplicate to ensure accuracy and precision (Kumar *et al.*, 2023).

**Test principle:** The Calbiotech AMH ELISA was based on a solid-phase sandwich enzyme immunoassay technique. The microplate wells were pre-coated with streptavidin, which bound biotinylated anti-AMH antibodies. Patient serum was added along with two conjugate reagents: a biotin-conjugated anti-AMH antibody and a horseradish peroxidase (HRP)-labeled anti-AMH antibody.

The present of AMH in the sample bound to both antibodies forming a sandwich complex, which was immobilized on the microplate via streptavidin-biotin interaction. After incubation, unbound components were removed through a washing step. A chromogenic substrate (TMB) was then added, and the enzyme catalyzed a color change. The intensity of the color was directly proportional to the AMH concentration in the sample. The reaction was stopped with a stop solution, and absorbance was read at 450 nm using the BALIO ELISA plate reader (BALIO Diagnostics, USA). A standard curve was generated from the optical densities of the standards, and sample concentrations were extrapolated from the curve (Calbiotech Inc., 2023).

**Procedure :**Prior to to assay, Reagent and samples were brought to room temperature.

Reagent were mix gently bofore used.

The required number of coated microplate strips was placed in a strip holder.

50 µL each of AMH standards, controls, and patient samples were pipetted into designated wells.

50 µL of biotin-conjugated anti-AMH reagent was added to each well.

50 µL of HRP-conjugated anti-AMH reagent was added to the same wells.

The plate was covered and incubated for 90 minutes at room temperature (20–25°C) on an ELISA plate shaker at 650 rpm.

Wells were washed five times with 300 µL of 1X wash buffer using the Stat Fax 2600 ELISA washer. Remaining liquid was blotted with absorbent paper.

100 µL of TMB substrate was added to each well and incubated for 15 minutes at room temperature on the shaker.

50 µL of stop solution was added to each well, and the plate was gently shaken to mix.

Absorbance was measured using the BALIO ELISA plate reader (BALIO Diagnostics, USA) set at 450 nm within 15 minutes of adding the stop solution.

 **Result presentation** At 450 nm the absorbance values were used to generate a standard curve by plotting the optical densities of the AMH standards against their known concentrations. The levels of AMH in patient samples were extrapolated from this curve, and all results were expressed in nanograms per milliliter (ng/mL) and were validated against control values.

 **Measurement of LH (Luteinizing Hormone):** Luteinizing hormone (LH) levels in serum samples were measured using the MAGLUMI 1000 plus automated chemiluminescence immunoassay analyzer (Snibe Diagnostic, Shenzhen, China). The assay has an analytical sensitivity of 0.1 mIU/mL and a measurement range of 0.2–200 mIU/mL, and the intra-assay and inter-assay coefficients of variation were 3.2% and 4.1%, respectively (Zhang *et al*., 2022).

**Principle:**The LH assay was based on a two-site sandwich chemiluminescence immunoassay. Patient samples, calibrators, or controls were mixed with magnetic microbeads coated with anti-LH monoclonal antibodies and ABEI-labeled anti-LH monoclonal antibodies.

Upon incubation, LH in the sample binds to the antibodies forming a sandwich complex. The magnetic field separates bound from free components, and a washing cycle was performed to removes unbound substances. Chemiluminescent substrates (Starter 1 and Starter 2) were added to trigger the luminescent reaction. The light signal was measured by a photomultiplier as relative light unit (RLU) which is directly proportional to the concentration of LH presentent in the sample.

**Procedure:**The reagent kit was scanned and loaded into the MAGLUMI 1000 plus analyzer.

Magnetic microbeads were resuspended by mixing for 30 minutes.

Calibration was performed using provided calibrators.

Quality control samples were analyzed.

Patient samples were entered with unique identification numbers.

The start command was initiated to begin the assay.

Results were automatically calculated and validated by the system.

**Result Presentation:** The results were automatically generated by the analyzer using the standard calibration curve. LH concentrations were displayed quantitatively in milli-international units per milliliter (mIU/mL).

**Measurement of Progesterone:** Serum progesterone levels were measured using the MAGLUMI 1000 plus automated chemiluminescence immunoassay analyzer (Snibe Diagnostic, Shenzhen, China). The assay is designed for quantitative determination of progesterone concentrations in human serum.

**Principle:** The progesterone assay is a competitive chemiluminescence immunoassay. The sample (calibrator/controls), buffer, magnetitc microbeads coated with progesterone antigen, ABEI labeled with anti-PRG monoclonal antibody are mixed thoroughly and incubated, PRG present in the serum sample (calibrator/controls) compete with PRG antigen immobilized on the magnetic microbeads a limited number of binding sites on the ABEI-label anti-PRG antibody. After precipitation in the magnetic field, the supernatant is decanted and then perform a wash cycle. Subsequently, the starter 1 and 2 are added to initiate chemiluminescent reaction. The light signal is measured by a photomultiplier as relative light unite (RLU), which is inversely proportional to the concentration of PRG present in the sample (calibrator/controls).

The progesterone assay is a two-site sandwich chemiluminescence immunoassay. In this method, (patient serum, calibrator, or control), magnetic microbeads coated with anti-progesterone monoclonal antibody, and an ABEI-labeled anti-progesterone monoclonal antibody were mixed thoroughly and incubated to form antigen-antibody sandwich complexes.

After incubation, a magnetic field was applied to separate the bound from the free components. The unbound substances were removed by washing. Chemiluminescent reagents (Starter 1 and Starter 2) were then added to initiate a luminescent reaction. The resulting light signal was measured as relative light units (RLU) using a photomultiplier tube, which is directly proportional to the concentration of progesterone in the sample.

**Procedure:** The reagent kit was scanned and loaded into the MAGLUMI 1000 plus analyzer.

Magnetic microbeads were mixed and resuspended for 30 minutes.

Calibration was performed according to the manufacturer's instructions.

Internal quality control samples were analyzed.

Patient samples were entered into the system using unique patient IDs.

The start command was initiated to begin the assay.

After the assay run, the results were validated and stored.

**Result Presentation:** The results were generated automatically by the analyzer and displayed as quantitative values. Progesterone concentrations were expressed in nanograms per milliliter (ng/mL). All results were validated against internal quality control limits.

**Statistical Analysis:** Data analysis was conducted using IBM SPSS Statistics, version 29. Descriptive statistics (means, standard deviations, and frequencies/percentages) were used to summarize demographic and clinical characteristics. Independent sample t-tests compared continuous hormone levels between the primary infertility group and the control group. Pearson’s correlation coefficient was used to evaluate the relationships between continuous variables, such as age and hormone levels. Chi-square (χ²) tests were used to evaluate associations between categorical variables (e.g., distribution of participant characteristics across groups). For cells with expected counts < 5, Fisher’s exact test was applied as appropriate. A p-value < 0.05 was considered statistically significant (IBM Corp., 2023)

**Result:** A total of 142 women participated in the study, comprising 71 women diagnosed with primary infertility and 71 age-matched fertile control women. All participants in both groups were female, representing 100% of the study population.

Table1. Participant

| Group |  Total Participants |  All Female (%) |
| --- | --- | --- |
| Primary Infertility Women |  71 |  100% |
| Fertile Control women |  71 |  100% |
| Total |  142 |  100% |

**Table 2**: The age distribution of participants was categorized into four ranges: ≤25, 26–30, 31–35, and 36–40 years. In the primary infertility group, 8 participants (11.3%) were aged ≤25 years, 17 (23.9%) were 26–30 years, 25 (35.2%) were 31–35 years, and 21 (29.6%) were 36–40 years.
Among the fertile control women, 8 (11.3%) were also aged ≤25 years, 21 (29.6%) were 26–30 years, 20 (28.2%) were 31–35 years, and 22 (31.0%) were 36–40 years.
The overall age distribution was relatively balanced between the two groups, with the highest proportion of participants falling within the 31–35 year range (31.7%), followed by 36–40 years (30.3%).

**Table2. Age Distribution of the study participants**

| Age Range (Year) | Primary Infertility Women (n=71) |  Fertile Control Women (n=71) | Total (n=142) |
| --- | --- | --- | --- |
|  20-25 |  8 (11.3%) |  8 (11.3%) |  16 (11.3%) |
|  26–30 |  17 (23.9%) |  21 (29.6%) |  38 (26.8%) |
|  31–35 |  25 (35.2%) |  20 (28.2%) |  45 (31.7%) |
|  36–40 |  21 (29.6%) |  22 (31.0%) |  43 (30.3%) |

**Table 3:** The mean age of women in the primary infertility group was 32.51 ± 4.86 years, while that of the control group was 33.27 ± 5.66 years. Statistical comparison using the independent t-test showed no significant difference in mean age between the groups (p = 0.38).
However, when age was dichotomized into >35 years and ≤35 years, a Chi-square test revealed a statistically significant difference in distribution between the groups (p = 0.038), with an odds ratio (OR) of 0.59. This indicates that women with primary infertility were less likely to be over 35 years of age compared to the fertile controls.

**Table 3 Age Comparison Between Primary Infertility Women and Control Groups**

| Group | Sample Size (n) | Age (Mean ± SD) | p-value | Odds Ratio (Age >35) |
| --- | --- | --- | --- | --- |
| Primary Infertility | 71 | 32.51 ± 4.86 years | 0.38 | 0.59 |
| Control | 71 | 33.27 ± 5.66 years |  |  |

**Table 4:** This table presents the descriptive statistics (mean ± standard deviation) for age, luteinizing hormone (LH), progesterone (PRG), and anti-Müllerian hormone (AMH) levels in primary infertile women compared with fertile controls. Statistical comparisons between the groups were conducted using an independent samples t-test, with significance set at p < 0.05. AMH and PRG levels were significantly higher in the fertile control group (p < 0.001 and p = 0.034, respectively), while no statistically significant differences were observed in age and LH levels (p = 0.974 and p = 0.739, respectively).

**Table.4: Comparison of Mean Age, LH, Progesterone, and AMH Levels Between Primary Infertility Women and Fertile Controls**

| Parameters | Primary Infertility Women (n = 71) |  Fertile Control Women (n = 71) |  p-value (t-test) |
| --- | --- | --- | --- |
| Age (years) | 32.63 ± 4.78 |  32.66 ± 5.36 |  0.974 (NS) |
| LH (mIU/ml) | 8.65 ± 3.58 |  8.48 ± 3.43 |  0.739 (NS) |
| PRG (ng/ml) | 11.44 ± 7.91 |  13.65 ± 6.86 |  0.034 (Significant) |
| AMH (ng/ml) | 1.85 ± 1.40 |  3.52 ± 2.41 |  <0.001 (Highly Significant) |

NS = Not Significant at p<0.05
Note:
Age is expressed in years. LH in mIU/ml. PRG = Progesterone, expressed in ng/ml. AMH = AntiMullerian Hormone, expressed in ng/ml.

**Table 5:** This table summarizes the Pearson correlation coefficients among key hormonal and age-related variables for the combined population of primary infertile and fertile control women. At a significance level of p < 0.05, a moderate and statistically significant negative correlation was observed between age and AMH levels. A weak but statistically significant positive correlation was also found between PRG and AMH. All other correlations were weak and not statistically significant at the p < 0.05 threshold.

**Table 5: Pearson Correlation Coefficients Between Age, LH, PRG, and AMH Across All Participants**

| Correlated Variables | Pearson r | p-value | Interpretation |
| --- | --- | --- | --- |
| Age vs AMH | -0.305 | <0.001 | Moderate negative correlation |
| LH vs AMH | +0.152 | 0.078 | Weak, NS |
| PRG vs AMH | +0.180 | 0.036 | Weak positive, significant |
| Age vs PRG | +0.010 | 0.912 | No correlation |
| LH vs PRG | +0.126 | 0.137 | Weak, NS |

NS = Not Significant at p<0.05

**Discussion:** This study assessed the hormonal profiles, specifically anti-Müllerian hormone (AMH), luteinizing hormone (LH), and progesterone (PRG) of women with primary infertility in Abuja and compared them with fertile control women.

**Table1** presents the summary of participant distribution in the study. A total of 142 women were enrolled and grouped into two categories: women diagnosed with primary infertility (n = 71) and age-matched fertile control women (n = 71). Each group represented 50% of the total study population, facilitating an even comparative analysis.

All participants were biologically female, which aligns with the study's focus on evaluating female reproductive hormones, AMH, LH, and PRG. This uniformity in biological sex among participants is consistent with the study focus on the evaluation of reproductive hormones, (AMH), (LH), and (PRG). The exclusive inclusion of female participants eliminates sex-related biological variability, thus enhancing the internal validity of the hormonal comparisons between the two groups.

The equal sample size between groups ensures statistical balance and comparability, minimizing potential bias in group-specific findings. This design is particularly valuable in cross-sectional studies, where equal representation enhances the robustness of comparative analysis between cases and controls.

The demographic distribution in Table 2 demonstrates methodological rigor by maintaining equal group sizes and a focused population, allowing for accurate assessment of hormonal variations associated with primary infertility in the study cohort.

**Table 2** presents the age distribution of women with primary infertility and fertile controls. The age ranged from 20 to 40 years, providing a representative spread across key reproductive stages. Among women with primary infertility, the highest proportion (35.2%) was within the 31–35-year age group, followed by the 36–40-year group (29.6%). This distribution reflects established literature indicating that female fertility declines notably after age 30, with a steeper reduction after age 35 (Broer *et al*., 2014; Wallace and Kelsey, 2010).

This age-related decline is primarily attributed to the progressive depletion of ovarian follicles, decreased oocyte quality, and reduced endocrine function of the ovaries, particularly a decline in AMH levels and an increase in follicle-stimulating hormone (FSH) (Broekmans *et al.*, 2009; Nelson, 2013). Consequently, women in the 31–35 and 36–40 age groups may experience more pronounced difficulties with conception.

In contrast, the fertile control group showed a relatively balanced distribution between the 36–40 (31.0%) and 26–30 (29.6%) age groups. This may suggest that a proportion of women maintain reproductive competence into their late 30s, potentially due to higher ovarian reserve, healthier lifestyle choices, or earlier parity. The 20-25 age group had the lowest representation in both groups (11.3%), possibly due to underreporting, delayed diagnosis, or lower awareness of infertility issues at earlier reproductive ages.

These findings support global data that emphasize age as a major determinant of fertility potential. According to the American Society for Reproductive Medicine (ASRM, 2017), female fertility peaks in the mid-20s and begins to decline gradually in the early 30s, with a steeper decline after 35 years. The data also reflect the growing trend of delayed childbearing in urban populations, which is often associated with increased infertility rates (Umezulike and Efetie, 2004).

The age distribution in this study highlights the importance of early fertility assessment and reproductive health education, particularly for women approaching their mid-thirties, to improve the chances of timely conception and reduce the emotional and financial burden of infertility.

**Table 3** compares the mean age and age-related infertility odds between women with primary infertility and fertile controls. The mean age for the primary infertility group was 32.51 ± 4.86 years, while that of the control group was slightly higher at 33.27 ± 5.66 years. The p-value of 0.38 indicates that the difference in mean age between the two groups was not statistically significant, suggesting that both groups were age-matched and comparable for fertility-related analysis.

Interestingly, the odds ratio (OR) of 0.59 for age >35 years implies that women with primary infertility were less likely to be older than 35 years compared to the fertile control group. Although not statistically significant, this observation may suggest that non-age-related factors, such as endocrine abnormalities, anatomical defects, or lifestyle influences, may be contributing to infertility in this population.

Numerous studies have established age as a critical factor influencing female fertility. Fertility declines with advancing age due to the depletion of ovarian follicles and a decrease in oocyte quality, particularly after age 35 (Wallace and Kelsey, 2010; Broekmans *et al*., 2009). However, the non-significant age difference in this study implies that primary infertility may be present even in younger women. Additionally, the mean age of over 32 years in both groups highlights the trend of delayed childbearing in urban Nigerian settings, often due to education, career, or late marriage (Umezulike and Efetie, 2004). This aligns with findings from international studies indicating that women are increasingly attempting conception at older ages, which may expose them to greater risk of infertility regardless of individual health status (Broer *et al*., 2014).

 While age is a recognized determinant of female fertility, the findings from this study emphasize the need to consider other reproductive and environmental factors when assessing infertility, especially in populations where women are still relatively young.

**Table 4** presents the comparison of mean age, LH, progesterone, and AMH levels between women with primary infertility and fertile controls. The mean ages of both groups were nearly identical (32.63 ± 4.78 years vs. 32.66 ± 5.36 years; p = 0.974), confirming that age was well matched and unlikely to confound hormonal comparisons.

The serum levels of luteinizing hormone (LH) did not significantly differ between the two groups (p = 0.739), indicating that LH concentrations were similar in primary infertile women and fertile controls. LH plays a critical role in ovulation by triggering follicular rupture, and its pulsatile secretion pattern is essential for normal reproductive function (Marshall and Eagleson, 2020). The absence of a significant difference in LH levels suggests that hypothalamic-pituitary-ovarian axis dysfunction related to LH may not be a predominant cause of infertility in this group.

Progesterone (PRG) levels, however, were significantly lower in women with primary infertility (11.44 ± 7.91 ng/ml) compared to fertile controls (13.65 ± 6.86 ng/ml; p = 0.034). Progesterone is pivotal for the maintenance of the luteal phase and preparation of the endometrium for implantation (Coutifaris, 2013). This finding may point to luteal phase insufficiency or suboptimal corpus luteum activity, both of which are implicated in infertility due to impaired endometrial receptivity (Güney *et al*., 2015).

Most notably, AMH levels were significantly reduced in the primary infertility group (1.85 ± 1.40 ng/ml) compared to controls (3.52 ± 2.41 ng/ml; p < 0.001). AMH is a well-established biomarker of ovarian reserve, reflecting the remaining quantity of antral and pre-antral follicles (La Marca and Sunkara, 2014). Reduced AMH in infertile women supports the hypothesis of diminished ovarian reserve contributing to primary infertility, consistent with findings in various populations (Seifer *et al*., 2011). This decline may also precede clinical manifestations of ovarian aging, emphasizing the utility of AMH in early infertility assessment.

Overall, these findings underline the multifactorial nature of infertility, where normal LH levels coexist with significant differences in progesterone and AMH, highlighting the importance of evaluating both luteal function and ovarian reserve in infertile women.

**Table 5** presents the Pearson correlation coefficients assessing the relationships between age, LH, PRG, and AMH among all study participants. A moderate negative correlation between age and AMH (r = -0.305, p < 0.001) was observed, indicating that as age increases, AMH levels tend to decrease. This inverse relationship aligns with established evidence which consistently shows that AMH levels decline with age due to follicular depletion and reduced granulosa cell activity (Nelson *et al.*, 2012; La Marca *et al*., 2009).

The correlation between LH and AMH was weak and not statistically significant (r = +0.152, p = 0.078), suggesting limited direct association between these hormones. LH secretion, governed by pulsatile GnRH release, may not have a direct regulatory effect on AMH, which is primarily secreted by pre-antral and small antral follicles independently of gonadotropins (Fanchin *et al*., 2005).

Similarly, the weak positive correlation between progesterone and AMH (r = +0.180, p = 0.036) was statistically significant but modest, indicating a slight tendency for higher AMH to be associated with higher luteal phase progesterone levels. This might reflect that better ovarian reserve (higher AMH) supports more robust follicular development and corpus luteum function, leading to increased progesterone production (McCredie *et al.*, 2006).

No meaningful correlation was found between age and progesterone (r = +0.010, p = 0.912), indicating that circulating progesterone levels during the luteal phase may not be directly influenced by age in this population. This is consistent with previous findings that progesterone secretion is more closely tied to the functional capacity of the corpus luteum than to chronological age per se (Lehmann *et al*., 2013).

The weak, non-significant correlation between LH and progesterone (r = +0.126, p = 0.137) similarly suggests that these hormones vary independently within physiological ranges.

These correlation patterns highlight the complex and partly independent hormonal dynamics involved in female reproductive function. While AMH is closely linked to age and ovarian reserve, LH and progesterone appear to fluctuate based on additional regulatory mechanisms.
This study's findings reinforce the importance of hormonal assessments in infertility diagnosis. LH levels were not significantly different, suggesting LH dysfunction may not be a primary issue in this group. Progesterone’s statistically lower levels in PIW highlight its role in implantation and luteal phase sufficiency. AMH’s strong correlation with infertility supports its use in evaluating ovarian reserve in clinical settings.

Further literature comparison supports these findings. The mean LH levels in the infertility group (6.88 ± 1.95 IU/L) were not significantly different from the control group (7.13 ± 2.14 IU/L; p = 0.549; Table 5), consistent with Ibrahim et al. (2021), who noted that serial LH testing is more reliable than single-point measurements. Progesterone differences align with Okeke et al. (2023), who linked urban stress and poor diets with luteal deficiencies. Abubakar and Mohammed (2022) also noted suboptimal progesterone levels contribute to implantation failure. These reinforce the approach of combining AMH, LH, and progesterone analysis, as advocated by Yusuf et al. (2024), to assess infertility in urban populations.

**Summary of Findings:** This study found that although infertility risks generally increase with age, there was no statistically significant age difference between infertile and fertile women in the study population. This indicates that age alone may not be a determining factor for infertility among the participants.

The results also emphasize the importance of hormonal balance and ovarian reserve in female fertility. Infertile women had significantly lower levels of progesterone and anti-Müllerian hormone (AMH) compared to fertile controls, while luteinizing hormone (LH) levels did not differ significantly between the groups. These findings suggest that deficiencies in ovarian reserve and progesterone may contribute more directly to primary infertility than LH levels.

Overall, the findings highlight the value of including AMH and progesterone assessments in fertility evaluations and the need for targeted reproductive health interventions beyond age-based assumptions.

**Conclusion**: This study compared serum AMH, LH, and progesterone levels in women with primary infertility and fertile controls attending the University of Abuja Teaching Hospital. Women with primary infertility had significantly lower AMH and progesterone levels, suggesting reduced ovarian reserve and possible luteal phase insufficiency. LH levels were not significantly different, aligning with evidence that basal LH alone has limited diagnostic utility.

A moderate inverse correlation between age and AMH underscores the impact of biological aging on ovarian reserve, while the positive correlation between AMH and progesterone indicates a subtle link between ovarian reserve and luteal function. These findings emphasize that primary infertility cannot be fully explained by chronological age alone; comprehensive hormonal evaluation, especially AMH and mid-luteal progesterone, remains essential for accurate diagnosis and personalized management.

By contributing localized hormonal data, this study supports the need for tailored fertility assessment strategies and underscores the importance of including ovarian reserve and luteal function markers in routine infertility evaluation for women in Nigeria.

**References**

1. Ademola, A., Smith, J., and Johnson, K. (2023). Sociocultural consequences of infertility in Nigerian women. Journal of Reproductive Health, 45(2), 123-134.
2. Agarwal, A., Mulgund, A., Hamada, A., and Chyatte, M. R. (2021). A unique view on male infertility around the globe. Reproductive Biology and Endocrinology, 19(1), 1-9.
3. American Society for Reproductive Medicine. (2017). Age and fertility: A guide for patients. Fertility and Sterility, 107(1), 52-58.
4. Broekmans, F. J., Soules, M. R., and Fauser, B. C. (2009). Ovarian aging: Mechanisms and clinical consequences. Endocrine Reviews, 30(4), 465-493.
5. Broer, S. L., Broekmans, F. J., Laven, J. S., and Fauser, B. C. (2014). Anti-Müllerian hormone: Ovarian reserve testing and its potential clinical implications. Human Reproduction Update*,* 20(5), 688-701.
6. Calbiotech Inc. (2023). AMH ELISA kit instructions for use. Calbiotech Inc.
7. Chen, L., and Zhang, M. (2023). Hormonal imbalances in female infertility: A comprehensive review*.*Reproductive Medicine International, 18(3), 245-260.
8. Coutifaris, C. (2013). Luteal phase defects and infertility. Clinical Obstetrics and Gynecology, 56(2), 340-348.
9. Fanchin, R., Schonauer, L. M., Righini, C., Guibourdenche, J., Frydman, R., and Taieb, J. (2005). Serum anti-Müllerian hormone is more strongly related to ovarian follicular status than serum inhibin B, estradiol, FSH and LH on day 3.Human Reproduction, 20(5), 1144-1150.
10. Güney, M., Oral, B., Karahan, N., and Mungan, T. (2015). Evaluation of reproductive hormones in infertile women: A clinical study. Journal of Obstetrics and Gynaecology Research, 41(9), 1420-1428.
11. Hagen, C. P., Aksglaede, L., Sørensen, K., Main, K. M., Boas, M., Cleemann, L., ... and Juul, A. (2018). Serum levels of anti-Müllerian hormone as a marker of ovarian function in 926 healthy females from birth to adulthood. Journal of Clinical Endocrinology and Metabolism, 103(4), 1365-1373.
12. Hassan, M. A., Killick, S. R., and Ahmed, B. (2022). Barriers to infertility treatment in developing countries: A systematic review. International Journal of Reproductive Medicine*,* 2022, Article ID 5647382.
13. IBM Corp. (2023). IBM SPSS Statistics for Windows, Version 29.0. IBM Corp.
14. Kumar, A., Patel, S., and Sharma, R. (2023). Analytical performance of AMH assays in clinical practice. Clinical Chemistry and Laboratory Medicine*,* 61(8), 1456-1463.
15. La Marca, A., and Sunkara, S. K. (2014). Individualization of controlled ovarian stimulation in IVF using ovarian reserve markers. Human Reproduction Update*,* 20(1), 124-140.
16. La Marca, A., Stabile, G., Artenisio, A. C., and Volpe, A. (2009). Serum anti-Müllerian hormone throughout the human menstrual cycle. Human Reproduction, 24(10), 2579-2585.
17. Lehmann, P., Vélez, M. P., Sauerbrei, A., Foster, W. G., Kornecki, A., Bartels, A., ... and Yong, P. J. (2013). Anti-Müllerian hormone (AMH) and endometriosis: A systematic review and meta-analysis. Reproductive Biology and Endocrinology, 11(1), 1-8.
18. Marshall, J. C., and Eagleson, C. A. (2020). Neuroendocrine aspects of polycystic ovary syndrome. Endocrinology and Metabolism Clinics of North America, 49(1), 135-146.
19. Mascarenhas, M. N., Flaxman, S. R., Boerma, T., Vanderpoel, S., and Stevens, G. A. (2012). National, regional, and global trends in infertility prevalence since 1990: A systematic analysis of 277 health surveys. PLoS Medicine, 9(12), e1001356.
20. McCredie, S., Paul, C., and Skegg, D. C. (2006). Infertility in New Zealand women: A population-based study. Australian and New Zealand Journal of Obstetrics and Gynaecology, 46(4), 328-332.
21. Nelson, S. M. (2013). Biomarkers of ovarian response: Current and future applications. Fertility and Sterility, 99(4), 963-969.
22. Nelson, S. M., Telfer, E. E., and Anderson, R. A. (2012). The ageing ovary and uterus: New biological insights. Human Reproduction Update, 18(5), 525-535.
23. Ogbeibu, A. E. (2014). Biostatistics: A practical approach to research and data handling. Mindex Publishing.
24. Oladimeji, O., Sanni, O., and Okeowo, T. (2023). Hormonal profiles in Nigerian women with infertility: A multicenter study.African Journal of Reproductive Health*,* 27(4), 78-89.
25. Rahman, S., Ahmed, T., and Khan, M. (2023). Sample preservation techniques for hormone analysis: A laboratory guide*.*Clinical Laboratory Science, 36(2), 89-95.
26. Sauer, M. V. (2021). Reproduction at an advanced maternal age and maternal health. Fertility and Sterility*,* 115(5), 1080-1086.
27. Seifer, D. B., MacLaughlin, D. T., Christian, B. P., Feng, B., and Shelden, R. M. (2011). Early follicular serum müllerian-inhibiting substance levels are associated with ovarian response during assisted reproductive technology cycles. Fertility and Sterility, 95(1), 474-478.
28. Tehrani, F. R., and Noroozzadeh, M. (2019). Relationship between serum AMH levels and age at menopause in women of reproductive age. Journal of Reproduction and Infertility*,* 20(2), 61-69.
29. Thompson, K., and Roberts, L. (2023). Anti-Müllerian hormone in reproductive medicine: Clinical applications and interpretations. Reproductive Endocrinology Review, 15(2), 112-125.
30. Umezulike, A. C., and Efetie, E. R. (2004). The problem of infertility in developing countries. Tropical Doctor, 34(2), 79-81.
31. Wallace, W. H. B., and Kelsey, T. W. (2010). Human ovarian reserve from conception to menopause. PLoS ONE, 5(1), e8772.
32. Williams, T., Johnson, A., and Smith, P. (2022). Luteinizing hormone dynamics in reproductive health. Endocrine Reviews, 43(3), 456-478.
33. Williams, T., and Richards, J. (2017). Hormone timing and reproductive outcomes. Journal of Clinical Endocrinology and Metabolism*,* 102(8), 2834-2841.
34. World Health Organization. (2020). Infertility definitions and terminology. World Health Organization.
35. Zhang, L., Wang, H., and Liu, J. (2022). Analytical performance of MAGLUMI chemiluminescence immunoassay system*.*Clinical Chemistry and Laboratory Medicine, 60(7), 1023-1030.
36. Zegers-Hochschild, F., Adamson, G. D., Dyer, S., Racowsky, C., de Mouzon, J., Sokol, R., ... and Simpson, J. L. (2017). The international glossary on infertility and fertility care. Human Reproduction, 32(9), 1786-1801.