EFFECT OF HIV-1 AND ANTIRETROVIRAL THERAPY ON SEMEN QUALITY

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ABSTRACT
Some studies have suggested that HIV infection may have a negative impact on fertility of infected males. Inconsistent results have characterised reports on the effect of HIV infection on semen quality. HAART has been reported to improve health by reducing blood viral loads, the incidence of opportunistic infections and improving immune system relatively. Investigating the effect of HIV-1 infection and HAART on semen quality was the priority of the study. Semen analysis and blood CD4+ determination were carried out on 25 subjects each of asymptomatic, moderately symptomatic, short duration of HAART treated (<1), longer duration of HAART treated (≥1) of HIV-infected men and HIV-negative control men and the data compared. The results showed that all the critical semen parameters (sperm motility, sperm concentration, total sperm count, sperm morphology, sperm vitality, semen volume and pH) were impaired in the HIV-1 infected groups. However, sperm motility improved with longer duration of HAART treatment than shorter duration. The study concluded that semen parameters evaluated in HIV-1 infected men revealed several impairments. Immune status largely correlated with semen parameters. Longer duration of treatment of HIV-1 infected men with HAART relatively improved some of the semen parameters.

INTRODUCTION
HIV-1 infection is a sexually transmitted disease that affects at least 40 million individuals worldwide (WHO and UNAIDS, 2003). It is one of the single most important health issues that threaten the survival of millions in sub-Saharan Africa. Its impacts are far reaching as it has a ripple effect from the individual, to the community up to national level. Its transmissibility depends on the type of sexual exposure, frequency of sexual intercourse, level of infectivity of the infected partner and the susceptibility of the non-infected partner (Gardner et al., 2005; Royee et al., 1997). Sexual transmission is also related to blood viral load, as well as immunological strength of the uninfected (Lee et al., 1996; Quinn et al., 2000). A number of studies have reported the presence of HIV-1 in semen of most infected men (Dulioost et al., 1998; Gupta et al., 1997; Tachet et al., 1999), even when it is not detected in the blood (Dejucq et al., 2001; Mayer et al., 1999; Zhang et al., 1998). Some studies have suggested that HIV infection may have a negative impact on fertility of both males and females (Barbieri et al., 2000; Carpenter et al., 1997; Desgrees du Lou et al., 1998; Gray et al., 1998; Ryder et al., 1991).
However, the optimized use of combined antiretroviral therapy and improved treatment of opportunistic infections have produced significant improvements in life expectancy and quality of life of HIV positive adults (Morineau et al., 2009; Smith et al., 2010). It’s therefore not surprising that a growing number of men and women living with HIV/AIDS feel encouraged to include parenthood in the planning of their lives (Paiva et al., 2007).

Male fertility somewhat largely is revealed by the quality of semen produced (WHO, 2010). However, whether the quality of semen is affected after HIV-1-infection has remained an active debate currently.

Highly active antiretroviral therapy (HAART) has been reported to improve health of HIV infected individuals by reducing blood viral loads, reducing the incidence of opportunistic infections, reconstituting the immune system by upward adjustment of the immune marker, blood CD4 count, and relatively improving the normal functioning of the body systems (Detels et al., 1998; Detels et al., 2001; Egger et al., 1997; Vernazza et al., 2000). Our study therefore aims at investigating the effect of HIV-1 infection and HAART on semen quality of seropositive men.

MATERIALS AND METHODS
HIV-1-infected men (n =100) who attended the Voluntary Counseling and Testing/ HIV Clinic of the Kumasi South Regional Hospital, Kumasi, Ghana, were recruited on the basis of their WHO clinical staging (WHO, 2005) and the duration of HAART treatment for the study between December 2009 and July, 2010. On the basis of WHO clinical staging, slightly symptomatic HAART naïve patients (WHO clinical stage 2) formed the group 1 (Grp 1) and asymptomatic HAART naïve HH patients (WHO clinical stage 1) formed group 2 (Grp 2). On the basis of duration of HAART treatment, patients who had received treatment for less than 1 year formed group 3 (Grp 3) and patients who had received treatment for 1 or more years formed group 4 (Grp 4). (It must be noted that based on the policy on HAART treatment in Ghana at the time the study took place, HAART was only administered to patients who were symptomatic and had blood CD4+ less than 250 cells/µl of blood). The control group (Grp5) was made up apparently healthy fertile men with proven fertility, at least 1 child, who were recruited from the Kumasi Metropolis, Ghana. Each group comprised of 25 participants. The participants were made to respond to a number of pertinent questions relating to demography, knowledge about HIV transmission and prevention, and the use of highly active antiretroviral therapy using an interview guide. Participants were also advised to abstain from smoking, alcohol, aphrodisiacs, coffee containing beverages and hard drugs use during the period of the study because they could have adverse effect on the semen quality (Martini et al., 2004; Zhang et al., 2000). Each of the participants gave his consent before taking part in the study. The study was approved by the institutional ethics board (Committee on Human Research Publication and Ethics, KNUST, Ref. # CHRPE/105/09).

Collection of Semen Samples and Determination of Semen Characteristics
The HIV-infected men provided the semen samples at the Kumasi South Hospital laboratory by masturbation into sterile, wide-mouth containers, after the recommended 3-day minimum period of sexual abstinence. The specimens were placed in sterile specimen bags and taken to a separate laboratory designated for treatment of infectious diseases for analysis within the facility. Standard protocol was followed during the analysis of the samples from the infected men. On the other hand, the control group produced the semen samples at the Komfo-Anokye Teaching Hospital (KATH), Kumasi and their samples were analysed at the Microbiology Laboratory at KATH. In all cases, semen samples were allowed to liquefy at room temperature for 30 minutes prior to analyses. Semen parameters were assessed as outlined by the World Health Organization (WHO, 2010; WHO, 1999) criteria. WBC count was estimated under high power
A microscopic field (HPF) on wet smear preparations after peroxidase staining was carried out to differentiate them from other round seminal cells.

**Determination of Blood CD4⁺ Lymphocytes Count**

3 ml of peripheral blood was drawn from each participant into tri-potassium EDTA tubes at the same time participants presented semen sample for the study. The blood samples were analyzed within five hours of collection for absolute CD4⁺ lymphocytes counts by using the conventional Becton-Dickinson FACS Count (California, USA) flow cytometric method (Singh et al., 2007).

**Statistical Analysis**

For comparisons between groups’ ages, semen volume, sperm concentration, total sperm count, sperm motility, sperm vitality and sperm morphology were considered as continuous variables. As none of the factors assessed was normally distributed, the non-parametric Mann–Whitney U-test was used to compare the groups. Sperm motility, sperm morphology, sperm vitality, semen consistency were analysed as categorical variables and their effect assessed using Fisher’s exact test and \( \chi^2 \)-tests. In addition Spearman rank testing was performed to detect any correlation between sperm parameters and blood CD4⁺ lymphocytes count (continuous variables). A level of \( p < 0.05 \) was considered as statistically significant. GraphPad Prism version 5.00 for windows was used for the statistical analysis (GraphPad software, San Diego California USA, www.graphpad.com)

**RESULTS**

The mean ages of the HIV-1 infected groups (Grp1, Grp 2, Grp 3, Grp 4) and the HIV-negative control group were respectively 41.0±1.4, 37.7±1.3, 41.6±1.3, 42.2±1.9 and 38.3±1.3 years (\( p > 0.05 \)). 88% of the HIV-infected participants and 92% of the control-group had formal basic education (Junior High School). Also, 89% of the HIV-infected men showed medium to high level of knowledge about HIV and its transmission modes as well as the various methods of prevention. About 2/3 of the HIV-infected men and all the seronegative control were sexually active at the time they

![Graph showing semen consistency profile between study groups](image-url)

**Fig. 1:** Comparison of semen consistency profile between study groups. High, Normal and Low represent semen consistency categories. Different letters denote significant difference between the groups within the same seminal consistency category.
took part in the study. However, 33% of the HIV-infected sexually active men were not adherent to the use condom as a method of checking the spread of the infection to seronegative partner(s) during sexual intercourse. 54% of the HIV-infected male participants had sexual partner(s) who were HIV-positive. However, 34% of the HIV-infected male participants had no knowledge about the HIV infection status of their partner(s). In addition, 63% of the HIV-infected men responded that there had been no reduction in volume of their testes post HIV-1 infection.

Participants’ characteristics and semen parameters in the population of HIV-infected men and the control groups are presented in Figures 1 and 2, and table 2. The mean blood CD4\(^+\) lymphocytes count obtained for Grp 1, Grp 2, Grp 3, Grp 4 and Grp 5 were respectively 311.2 (±11.2), 564.1 (±15.0), 371.7 (±12.9), 338.3 (±16.6) and 1011 (±105.7) cells/µl.

Figure 1 summarizes the variation in seminal fluid consistency among the study population. Significantly higher number of semen with higher viscosity was observed in the infected groups compared with control. The control group however showed a significantly higher mean number of semen with ‘normal’ viscosity compared with the infected groups. Semen obtained from the seropositive men were significantly lower in volume than that of the control ($p \leq 0.05$); however, there were no significant differences between the HIV-infected groups. Comparison between seropositive groups and the control group showed a significantly higher mean seminal pH for all the infected ejaculates compared with the control.

Detailed evaluation of sperm motility patterns between the groups summarised in Figure 2 evidenced striking differences between the groups within the study population. The study revealed that a significantly lesser numbers of sperms with rapid progressive motility were found among all the four HIV-1 infected groups compared to the control. The intergroup assessment of the HIV-1 infected cohorts showed that HAART naïves lightly symptomatic patients (Grp1) had the lowest number of sperms with rapid motility as well as total progressive motile (TPM) sperm. Asymptomatic patients and patients who had been on HAART for a year and/or more had the highest total progressive motile (TPM) sperm within the HIV-1 infected groups. Significantly higher total numbers of sperms which showed no progressive motility, i.e., non-progressive motile sperm plus immotile sperm (c + d) was found among HIV-infected groups compared to the control, whilst within HIV-1 infected population, the slightly symptomatic HAART naïve group showed relatively higher numbers of total non-progressive motile sperm compared with the others. The control group also showed significantly lower number of immotile sperm compared to the case. In all, the controls exhibited higher progressive motile and lower non-progressive motile sperm (Figure. 2).

Mean sperm concentrations as well as total sperm counts were also decreased in HIV-infected men compared with the control (Table. 2). Within the HIV-1 infected population, Grp 2, Grp 3 and Grp 4 showed mean sperm concentrations that were not different from each other but were significantly higher than Grp 1. Correspondingly, within the HIV-infected groups, Grp 2, Grp 3 and Grp 4 showed higher total sperm count (TSC) than Grp 1 at $p \leq 0.05$. Percentage of normal morphological sperm was significantly higher in the control group compared with the infected groups and within the infected groups, the numbers of normal morphological sperms were significantly higher in Grp 2, Grp 3 and Grp 4 compared with Grp 1.

**Relationship Between Semen Characteristics and Blood CD4\(^+\) Lymphocytes Count**

Table 2 summarizes the relationship between the marker of immune strength in HIV infection (blood CD4\(^+\) lymphocytes count) and semen characteristics. Spearman’s rank testing demonstrated a general positive correlation between blood CD4\(^+\) count and semen volume, though only Grp 2 showed significant association ($r = 0.47$, $p = 0.01$). There was a positive correlation between CD4\(^+\) count and percentage rapid pro-
gressive motile sperm, sperm concentration, total sperm count, percentage normal morphological sperm and sperm vitality. However, blood CD4+ lymphocytes count generally correlated negatively with immature germ cell concentration and seminal white blood cell (High power field) count, though none of these semen parameters showed significant association with blood CD4+ lymphocytes count.

### Table 1: Participants characteristics and some semen parameters of HIV infected and control men. Mean (Standard Error)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
<th>Group 5</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>41.0 (1.4)</td>
<td>37.8 (1.3)</td>
<td>41.6 (1.4)</td>
<td>42.2 (2.0)</td>
<td>38.4 (1.3)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Volume (ml)</td>
<td>1.6 (0.2)</td>
<td>1.6 (0.1)</td>
<td>1.7 (0.1)</td>
<td>1.8 (0.1)</td>
<td>2.9 (0.3)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>pH</td>
<td>8.2 (0.1)</td>
<td>8.1 (0.1)</td>
<td>8.1 (0.1)</td>
<td>8.1 (0.1)</td>
<td>7.4 (0.1)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>SC (&gt;10^6/ml)</td>
<td>16.1 (3.8)</td>
<td>34.7 (4.6)***</td>
<td>29.1 (4.5)*</td>
<td>35.8 (2.7)*</td>
<td>55.1 (5.4)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>TSC (&gt;10^5)</td>
<td>28.3 (7.6)</td>
<td>61.1 (10.7)**</td>
<td>43.3 (8.3)*</td>
<td>63.3 (8.5)*</td>
<td>188.9 (40.3)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>IGC Conc (+10^6/ml)</td>
<td>5.8 (1.2)</td>
<td>6.9 (1.2)</td>
<td>7.6 (1.4)</td>
<td>6.9 (1.1)</td>
<td>7.0 (0.2)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>WBC (HPF)</td>
<td>10.7 (1.8)</td>
<td>5.9 (0.6)**</td>
<td>13.5 (1.9)</td>
<td>3.9 (0.3)***</td>
<td>4.9 (1.3)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>NMS (%)</td>
<td>48.4 (5.2)</td>
<td>61.4 (4.0)*</td>
<td>55.2 (3.1)*</td>
<td>60.1 (3.3)*</td>
<td>81.6 (2.4)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>Sperm vitality (%)</td>
<td>58.1 (6.4)</td>
<td>84.3 (12.2)*</td>
<td>76.6 (2.9)*</td>
<td>70.7 (3.0)*</td>
<td>92.5 (1.0)***</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>CD4 Count Cells/µl</td>
<td>311.2 (11.2)</td>
<td>564.1 (15.0)***</td>
<td>371.7 (12.9)</td>
<td>338.3 (16.6)</td>
<td>1011.7 (105.7)***</td>
<td>p&lt;0.0001</td>
</tr>
</tbody>
</table>

SC- Sperm concentration, TSC- Total sperm count, NMS- Normal morphological sperm, IGC conc- Immature germ cell concentration. p is significant at *≤ 0.05, **≤ 0.01, ***≤ 0.001 when mean values of semen a parameter on the same horizontal line are compared.

### Table 2: Relation between Blood CD4+ lymphocytes count and semen parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
<th>Group 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume (ml)</td>
<td>0.59 (0.11)</td>
<td><strong>0.01 (0.47)</strong></td>
<td>0.98 (0.00)</td>
<td>0.40 (0.17)</td>
<td>0.42 (0.17)</td>
</tr>
<tr>
<td>pH</td>
<td>0.83 (0.04)</td>
<td>0.09 (0.34)</td>
<td>0.27 (-0.23)</td>
<td>0.06 (0.37)</td>
<td>0.87 (-0.01)</td>
</tr>
<tr>
<td>RPM</td>
<td>0.08 (0.35)</td>
<td>0.40 (0.17)</td>
<td>0.05 (0.40)</td>
<td>0.88 (0.03)</td>
<td>0.0001 (0.74)</td>
</tr>
<tr>
<td>SPM</td>
<td>0.69 (-0.08)</td>
<td>0.77 (-0.06)</td>
<td>0.09 (0.34)</td>
<td><strong>0.03 (0.42)</strong></td>
<td>0.23 (0.25)</td>
</tr>
<tr>
<td>NPM</td>
<td>0.07 (0.36)</td>
<td>0.31 (-0.21)</td>
<td><strong>0.02 (0.46)</strong></td>
<td>0.01 (0.47)</td>
<td>0.35 (0.20)</td>
</tr>
<tr>
<td>Immotile (%)</td>
<td>0.47 (0.15)</td>
<td>0.45 (-0.16)</td>
<td>0.04 (0.40)</td>
<td>0.07 (0.36)</td>
<td>0.89 (-0.03)</td>
</tr>
<tr>
<td>SpmConc</td>
<td>0.76 (0.06)</td>
<td>0.37 (0.19)</td>
<td><strong>0.004 (0.54)</strong></td>
<td>0.03 (0.42)</td>
<td>0.0001 (0.74)</td>
</tr>
<tr>
<td>TSC</td>
<td>0.46 (0.16)</td>
<td>0.06 (0.37)</td>
<td><strong>0.03 (0.43)</strong></td>
<td>0.44 (0.16)</td>
<td>0.0001 (0.69)</td>
</tr>
<tr>
<td>IG C Conc</td>
<td>0.51 (-0.15)</td>
<td>0.95 (-0.01)</td>
<td>0.76 (-0.06)</td>
<td>0.79 (-0.01)</td>
<td>0.60 (-0.09)</td>
</tr>
<tr>
<td>WBC (HPF)</td>
<td>0.27 (-0.23)</td>
<td>0.25 (-0.23)</td>
<td>0.30 (-0.21)</td>
<td>0.51 (-0.13)</td>
<td>0.43 (-0.16)</td>
</tr>
<tr>
<td>NMS (%)</td>
<td>0.58 (0.12)</td>
<td>0.41 (0.17)</td>
<td><strong>0.003 (0.56)</strong></td>
<td>0.03 (0.42)</td>
<td>0.0001 (0.69)</td>
</tr>
<tr>
<td>Vitality (%)</td>
<td>0.51 (0.13)</td>
<td><strong>0.04 (0.50)</strong></td>
<td>0.04 (0.40)</td>
<td>0.44 (0.15)</td>
<td>0.0001 (0.69)</td>
</tr>
</tbody>
</table>

WBC- white blood cells, IGC conc- Immature germ cell concentration, IM- Immotile sperm, NPM- Non-Progressive Motile sperm, SPM- Slow progressive Motile sperm, RPM- Rapid Progressive sperm, NM- Normal Morphological sperm, Sperm conc- Sperm concentration, TSC- Total sperm count, p is significant at < 0.05.
DISCUSSION
As the debate on whether HIV infection has an effect on semen quality still remains unsettled. Our study provides some information on the current knowledge in this area of research. In this work, we have provided relevant data regarding the effect of HIV-1 infection on semen parameters. The study population was made up of male participants of age ranging from 22.0 to 62.0 years. It is important to note that aging could have effect on the quality of semen (Jung et al., 2002). Nevertheless, the homogeneity in the age of the participants made age difference less likely a confounder. Therefore any difference in semen quality observed between the groups could unlikely be attributed to the consequence of age difference.

The study compared semen characteristics in a 100 HIV-1-infected men with a control group of HIV-negative healthy men of apparently proven fertility. The study has provided information on decreases in the semen volume, sperm motility, sperm concentration, total sperm count and sperm vitality and increases in the pH values, viscosity and other anomalies in the HIV-infected men’s semen compared with HIV-negative men. Our finding might be the second to corroborate Nicopoullos and colleagues (2004) study which reported that every semen parameter analysed (viscosity, pH, volume, sperm concentration, total sperm count, sperm motility, sperm morphology, sperm vitality, seminal white blood cell count, and immature germ cell concentration) was significantly impaired in HIV-infected men (Nicopoullos et al., 2004). Our report of increased seminal pH and viscosity in both HAART naïve and HAART treated groups are also in concord with the findings of other studies (Crittenden et al., 1992; Dondo et al., 1996; Dulioust et al., 2002; Muller et al., 1998). Currently, no specific reason(s) has been ascribed to the cause of the elevated pH and viscosity in semen produced by HIV-infected men. However we suggest that it could be due to compromised prostate glands functions in the HIV-infected men though we did not investigate specific biochemical markers of activity of the accessory reproductive glands (Anderson et al., 1992; Crittenden et al., 1992; Knobil et al., 1993; Politch et al., 1994). The prostate and the seminal vesicles are responsible for about 90-95% of the ejaculate volume as well as producing fibrinolyisin to effect liquefaction of semen. Therefore impairment in these glands functions due to inflammatory responses in them as a result of the activity of HIV might be the cause of the reduced seminal volume and increased seminal viscosity that characterized the seropositive ejaculate volume compared to the control as revealed in Fig. 1 and Table 1 (Dobs et al., 1988; Johnston et al., 1995; Taylor et al., 2001; White et al., 2001).

A reduction in mean sperm concentrations and total sperm counts produced by HIV-infected populations compared with seronegative control have been described earlier, (Crittenden et al., 1992; Dondo et al., 1996; Muller et al., 1998), and this study also confirmed that (Table 1). In addition, within the HIV-infected cohorts, Grp 1 showed lower sperm concentration and total sperm count compared with the other HIV-infected groups. However specific explanation to that effect is lacking, we realised in this study that, the HIV-infected cohorts shed higher numbers of immature germ cells in their semen compared with the seronegative control. This might have consequently resulted in the reduction in the number of sperm produced thereby negatively affecting the sperm concentrations in the infected groups (Table 1). This study also showed that higher immune marker (blood CD4+ count) favoured spermatogenesis as untreated HIV-infected men with higher blood CD4+ count showed relatively better semen and sperm parameters such as: sperm concentration, total sperm count, sperm vitality, progressive motile sperm and sperm morphology compared with the slightly symptomatic untreated men with lower blood CD4+ count. The exact reason(s) which might have accounted for the largely reduced semen quality in the symptomatic untreated group (Grp 1) however, might be the apparently higher immune strength, as indicated by the absolute CD4+ count, in Grp 2, compared with Grp
Fig. 2: Sperm motility profiles of study population: RPMS - rapid progressive motile sperm, SPMS - slow progressive motile sperm, NPMS - Non-progressive motile sperm, IMS - Immotile sperm, TPMS - total progressive motile sperm, TNPMS - Total non-progressive motile sperm. \( p \) is significant at *\( \leq 0.05 \), **\( \leq 0.01 \), ***\( \leq 0.001 \)
1. As Grp2 had higher blood CD4⁺ count, which implied a higher immune strength relatively than Grp 1, it was possible viral activity and silent opportunistic infections in the reproductive organs and associated glands were possibly held in a moderate check than in Grp 1, which showed lower blood CD4⁺ count with a probable elevated viral activity in the reproductive compartment (Garrido et al., 2005). This might suggest that an adequate immune strength favours the production of spermatozoa as well as seminal fluid to optimize the normal functions of the produced sperm.

Our findings, from Fig. 2, show a profound reduction in sperm motility within the HIV-infected cohorts compared with the control. The decreased motility might be due to abnormal seminal plasma composition and/or changes in sperm metabolism as a result of viral effect (Bujan et al., 2007). Within the HIV-infected populations, there was reduction in sperm motility in the symptomatic HAART naive group compared with the other groups. However, sperm vitality assessment showed that considerable numbers of the total non-progressive motile sperm produced by the HIV-infected groups were alive (table 1 and fig. 2). This may suggests that, HIV-infection, apart from its germ cell depletion effect (De Paepe et al., 2008; Muciaccia et al., 2007; Shevchuk et al., 1999), may have the ability to interfere with the formation of either structural components of sperm or physiological processes which are responsible for enhancing normal sperm motility in noninfected men (Conner et al., 2007; Dejucq-Rainsford et al., 2004; Publicover et al., 2007).

However, we can also report that there was a slight improvement in sperm motility in longer duration HAART treated men (Grp 4) than shorter duration of treated (Grp 3) which strongly corroborates the findings of one study (Robbins et al., 2001), but contradicts the findings of other reports (Bujan et al., 2007; Crittenden et al., 1992; van Leeuwen et al., 2008). The result from this study shows that even though Grp1 and HAART treated groups (Grp 3 and Grp4) had no significant differences in their blood CD4⁺ counts, the semen qualities shown by the latter groups were relatively better than that of the former as shown in Table 1 and Figure 2, suggesting HAART had some degree of amelioration on some body functions and processes including spermatogenesis. It was also observed in this study that, though there were no significant improvement in the blood absolute CD4⁺ counts in Grp 3 and Grp 4 compared with Grp 1, the semen parameters showed by Grp 3 and Grp 4 were comparable in most respect with that of Grp 2 with higher mean blood CD4⁺ than the HAART treated groups. Thus, antiretroviral treatment seems to have improved semen quality in the HIV-seropositive males, since the results of the semen analyses were significantly better in a number of critical indices than the HAART naive HIV-seropositive males with comparable blood CD4⁺ counts.

Table 1 shows not only sperm concentration and total sperm count to be significantly reduced in the infected groups but also percentage normal morphological sperm as well. This finding tends to corroborate some earlier studies (Muller et al., 1998; van Leeuwen et al., 2004), though might be contradictory to others who reported no significant reduction in values for HIV-1 infected cohorts (Dulioust et al., 2002; Krieger et al., 1991; Politch et al., 1994).

The cause of the teratozoospermic condition reported by other investigators which this study has confirmed has attributed the increased abnormal morphological sperm produced by HIV-infected men to possible entry of HIV into germ cells during the process of spermatogenesis and disrupting the process. Some of these studies reported the presence of HIV-1 DNA in most abnormal spermatozoa of HIV-1 infected subjects whose semen contained a high percentage of spermatozoa with abnormal morphologies with higher percentage of spermatozoa with fragmented DNA (Antonelli et al., 2000; Gandini et al., 2000a; Gandini et al., 2000b; Muciaccia et al., 2007; Muciaccia et al., 2005a; Muciaccia et al., 2005b).
This study also provides information on the correlation between semen parameters and blood CD4$^+$ lymphocytes level. From Table 2, some critical semen parameters: semen volume, rapid progressive motile sperm, sperm concentration, total sperm count and sperm vitality showed positive correlation with blood CD4$^+$ lymphocytes count, though not all showed significance. The concentration of seminal immature germ cell and seminal leucocytes however, correlated with blood CD4$^+$ lymphocytes count negatively, none of the correlation coefficients shown, was significant at $r \geq 0.5$, as indicated on Table 2.

CONCLUSION
To our knowledge, semen quality of HIV-infected men was significantly lower than the HIV-negative men with reference to World Health Organization standards. Within HAART naïve HIV$^+$ men, semen standards decreased with decreasing absolute CD4$^+$ lymphocytes count. Antiretroviral Therapy improves the semen quality of HIV$^+$ men though did not significantly improved immune status as reflected by the blood CD4$^+$ lymphocytes counts.

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