PREVALENCE OF HUMAN PAPILLOMAVIRUS (IgM) ANTIBODY AMONG FUOYE STUDENT

BY

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CERTIFICATION

This is to certify that this project work was carried out by AYODELE, FOLASADE JUSTINAH with MATRIC NO: MCB/15/3264.

This project work has been read and approved as meeting the requirement of the department of Microbiology, Faculty of Science, Federal University Oye-Ekiti. Ekiti-State.

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DEDICATION

I dedicate this project work to Almighty God who by his grace this project work is completed and also, to Mr Adewale David, Heritage, Goodness and Hephzibah Adewale for their support and encouragement.

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ABSTRACT

Human papilloma virus (HPV) infections are the most common sexually transmitted infections (STDs) in the world. This study was to document the prevalence of Human papilloma virus infection in Federal university Oye-Ekiti, Ekiti State, Nigeria. Urine samples were collected from ninety four participants and were screened for HPV (IgM) antibody using the HPV ELISA. Out of 94 samples analysed, 2 (2.1%) was positive for Human papilloma virus IgM, and higher prevalence were recorded in females (100%) compared to males. The age group prevalence was as follows 15 – 19 years (50.0%), 20 – 24 years (50%), 25 years and above (0%). It was observed that participants that have no permanent sex partner were 76.9% of the total number of participants positive for Human papilloma virus IgM, one sex partner were 19.2%, two partners were 0.0% and 3.9% for those with more partner. The result from this study reveals an association between Human papilloma virus infection and number of sex partners thus screening for HPV infection in asymptomatic patients to prevent the adverse consequences.

CHAPTER ONE

INTRODUCTION

Human papilloma virus (HPV) is a non-enveloped deoxyribonucleic acid (DNA) virus belonging to the family Papillomaviridae. This family includes more than 130 genotypes (Frazer, 2010), many of which infect the mucosal areas of the human upper digestive tract and the anogenital region through sexual contact (Touze, *et al.,* 2001; Ma *et al.,* 2013), leading to increased risk of development of cancer. These genotypes are grouped into "high-risk" and "low-risk" according to the degree of risk of development of cancer after infection. Infection with the high-risk serotypes of HPV can lead to cervical cancer and are associated with other mucosal anogenital, head and neck cancers. (Bosch *et a.,l* 1995; Chew *et al.,* 2005). Infection with the low-risk serotypes is known to cause benign or low-grade cervical tissue changes and genital warts (condyloma acuminata) on the cervix, vagina, vulva, and anus in women and on the penis, scrotum, and anus in men (CDC, 2012).

Genital HPV infection is one of the most common sexually transmitted infections in sexually active adolescents and young women (Richardson *et al.,* 2003). It has been estimated that at least 50% of sexually active adults have had a genital HPV infection (Di *et al.,* 2008) and that globally 75% of individuals (males and females) will experience an HPV infection at least once in their lifetime, with the highest rates of infection occurring in those under the age of 25 years (Tuin *et al.,* 2012) .In a recent meta-analysis, a global HPV prevalence of 11.7% was reported. The HPV prevalence in North America and Europe was estimated at 11.5% and 14.2%, respectively, while the prevalence in Africa was estimated at 21.1%, with sub-Saharan Africa topping the list at 24%

(Dahlstrom *et al.*, 2010).In Nigeria, the prevalence of HPV is high in all female age groups, and highest in women aged 15–23 years (Ezenwa *et al.*, 2013; Bruni *et al.*, 2014).

Studies have indicated that high-risk HPV genital infections in young females are transient and have little long-term significance (Hildesheim *et al.*, 1994; Hinchliffe *et al.*, 1995). However, when the infection persists, as in 5%–10% of infected women, there is a high risk of developing a precancerous lesion of the cervix, which can progress to invasive cervical cancer 15–20 years later (Goodman *et al.*, 2008). Persistent infection following acquisition of a high-risk HPV is generally defined by continued detection of cervical DNA of the same HPV type (Banister *et al.*, 2013).

Cervical cancer is an important health problem worldwide, being the second most common cancer among women, and ranking first in many developing countries (Rock *et al.*, 2000; Castle *et al.*, 2009). Half a million women develop cervical cancer annually and more than half die from the disease (Di *et al.*, 2008). In 2008, more than 270,000 women died of cervical cancer worldwide, with nearly 85% of these deaths occurring in developing countries (Bruni *et al.*,2014). Cervical cancer is the second most common cancer in women aged 15–44 years in Nigeria and the incidence rate is 27/100,000 (Nnodu *et al.*, 2010). Current estimates indicate that every year 14,089 women are diagnosed with cervical cancer and 8,240 die from the disease in Nigeria (Bruni *et al.*, 2014). A prevalence of 26.3% for HPV in the general population has been reported in Southern Nigeria (Okolo *et al.*, 2010). The incidence of HPV in women with cervical cancer is reported to be 24.8% (Ezenwa *et al.*, 2013; Nnodu *et al.*, 2010), while HPV prevalence in the general population (among women with normal cytology) is 23.7% (Bruni *et al.*, 2014). Risk factors associated with HPV infection include heterosexuality, promiscuity, smoking (Matsumoto *et al.*, 2003; Simen *et al.*, 2008), high parity, early sexual debut (Ma *et al.*, 2013).

Infection with other sexually transmitted diseases (Matsumoto *et al.*, 2003) prolonged use of contraceptives, dietary factors, and genetic disorders such as WHIM (warts, hypogammaglobulinemia, immunodeficiency, myelokathexis) syndrome, (Kwasniewska *et al.*, 1998; Thomas *et al.*, 2004).

Infection with HPV, diagnosed by detection of antibodies to HPV in the serum or detection of HPV DNA, is the primary risk factor contributing to development of cervical intraepithelial neoplasia and invasive cervix carcinoma. Detection of anti-HPV has been shown to reflect the overall HPV infection rate in a population more effectively than detection of HPV DNA (Ma *et al.*, 2013).This study was conducted to determine the prevalence of HPV IgM among fuoye students.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 History of HPV

Human papilloma virus is a small, non-enveloped icosahedra sexually transmitted virus with a double-stranded DNA genome. These viruses are species specific and infect the basal epithelial cells of the skin and mucous membranes, causing different types of warts and anogenital cancers. Human papilloma virus (HPV) is the cause of 90%–95% of squamous cell cancers and majority of infections do not cause symptoms. However, Persistent infection with high-risk HPV (most frequently type 16 &18) may lead to precancerous lesions of the cervix in 5%–10% of infected women, if untreated these lesions can progress to invasive cervical cancer 15–20 years later. Cervical cancer is an important health problem worldwide, being the second most common cancer among women, and ranking first in many developing countries (Rock *et al.*,2000; Castle *et al.*, 2009). Half a million women develop cervical cancer annually and more than half die from the disease (Di *et al.*, 2008). In 2008, more than 270,000 women died of cervical cancer worldwide, with nearly 85% of these deaths occurring in developing countries. (Bruni *et al.*, 2014). Cervical cancer is the second most common cancer in women aged 15–44 years in Nigeria and the incidence rate is 27/100,000 (Nnodu *et al.*, 2010). Current estimates indicate that every year 14,089 women are diagnosed with cervical cancer and 8,240 die from the disease in Nigeria (Bruni *et al.*, 2014). A prevalence of 26.3% for HPV in the general population has been reported in Southern Nigeria (Okolo *et al.*, 2010). The incidence of HPV in women with cervical cancer is reported to be 24.8% (Ezenwa *et al.*, 2013; Nnodu *et al.*, 2010), while HPV prevalence in the general population (among women with normal cytology) is 23.7% (Bruni *et al.*, 2014).

Risk factors associated with HPV infection include heterosexuality, promiscuity, smoking (Matsumoto *et al.*, 2003; Simen *et al.*, 2008), high parity, early sexual debut (Ma *et al.*,, 2013).

2.2 Justification

Human papilloma virus infections account for the most common sexually transmitted diseases worldwide and have been associated with various epithelial cell cancers. Worldwide, cervical cancer is the fourth most frequent cancer in women with an estimated 530,000 new cases in 2012 representing 7.5% of all female cancer deaths. Of the estimated more than 270,000 deaths from cervical cancer every year, more than 85% of these occur in less developed regions (CDC, 2012).

In developed countries, programmes are in place which enables women to get screened, making most pre-cancerous lesion identified at stages when they can easily be treated. Early treatment prevent up to 80% of cervical cancers in these countries (Okolo *et al.*, 2010). While in developing countries, limited access to effective screening means that the disease is often not identified until it is further advanced and symptoms develop. In addition, prospects for treatment for such late-stage disease may be poor, resulting in a higher rate of death from cervical cancer in these countries. The high mortality rate from cervical cancer globally (52%) could be reduced by effective screening and treatment programmes (Nnodu *et al.*, 2010).

Current estimates indicate that every year 14,089 women are diagnosed with cervical cancer and 8,240 die from the disease in Nigeria (Bruni *et al.*, 2014). A prevalence of 26.3% for HPV in the general population has been reported in Southern Nigeria (Okolo et al 2010). The incidence of HPV in women with cervical cancer is reported to be 24.8% (Ezenwa *et al.*, 2013; Nnodu *et al.*, 2010), while HPV prevalence in the general population (among women with normal cytology) is 23.7% (Bruni *et al.*, 2014).

2.3 Immunopathogenesis

During the early stages of an HPV infection, the host innate immune response becomes the first line of defense against the infection. Dendritic (DC), Langerhans (LC), natural killer (NK), natural killer T (NKT) cells and keratinocytes, among others, are important cells involved in promoting a good adaptive immune response against HPV infection and are the focus of this review. Most of these cell types can promote a cytokine-mediated pro-inflammatory process, which links the innate with the adaptive immune response. Moreover, NK cells are able to directly eliminate HPV infected cells (Renoux *et al.*, 2011). However, HPV can evade the immune response, mainly through the action of E6 and E7 proteins. The viral mechanisms of immune evasion range from modulation of cytokines and chemo-attractant expression to alteration of antigen presentation, and down-regulation of IFN-pathways and adherence molecules (Kanodia *et al.*, 2007). Evasion of the immune response by HPV is critical for a successful infection. Thus, stimulation of the innate immune response through strong adjuvants has turned out to be a promising therapeutic strategy for disrupting the evasion mechanisms of HPV and has been useful to understand the function of some innate immune cells during HPV infections.

HPV infects keratinocytes of the basal layer of the cervical epithelium (Sterling *et al.*, 1993; Stanley, 1994) and possibly stem cells (Maglennon *et al.*, 2011; Gravitt, 2012). As the main target of HPV, the keratinocyte plays an important role during the initiation of the HPV infection and subsequently becomes a link to promote an effective adaptive immune response. The keratinocytes are part of the innate immune defence system and have been considered as immune sentinels (Nestle *et al.*, 2009). They can function as non-professional antigen presenting cells, and are able to induce the expression of TH1 and TH2 type cytokines and cytotoxic responses in

CD4+ and CD8+ memory T cells, respectively (Black *et al.*, 2007). Keratinocytes in female genital tracts express several Toll-like receptors (TLRs), located either on the cell surface (TLR-1, TLR-2, TLR-4, TLR-5 and TLR-6) or in the endosomes (TLR-3 and TLR-9) (Nasu *et al.*, 2010).

2.4 Virology of HPV

HPV is a double-stranded DNA, non-enveloped capsid virus. It has 7900 base pairs which have 90% homology between the types (mandell *et al., 2009)* .The base pairs are arranged in a circle which includes the codes for two key proteins known as L1 and L2 . These two proteins act as the "immunogene" which is required for self-assembly and the infectivity protein, respectively (Yang *et al.*, 2003; Johnson *et al.*, 2009). The virus is transmitted between humans through breaks within the epidermis of the skin. Once the virus enters the skin, it attaches to a component of skin stem cells known as the tissue-specific heparin sulfate proteoglycans (selinka *et al.*, 2007; shafti–keramat *et al.*, 2003). Differentiation of the virus then occurs within the squamous keratinocyte (doorbar *et al.*, 2006), replicates, proliferates, and then moves to the next cell.

HPV genome is composed of 8k base pairs and divided into early and late gene.the early genome encodes E1,E2,E4, E5, E6 and E7 and the late genome encodes L1 and L2 oncoprotein E6 andE7 degrade tumor suppressor p53 and pRb, respectively. L1and L2 builds the structure of HPV capsid protein (Bosch *et al.*, 2001).

HPVgenomic DNA of 8kb

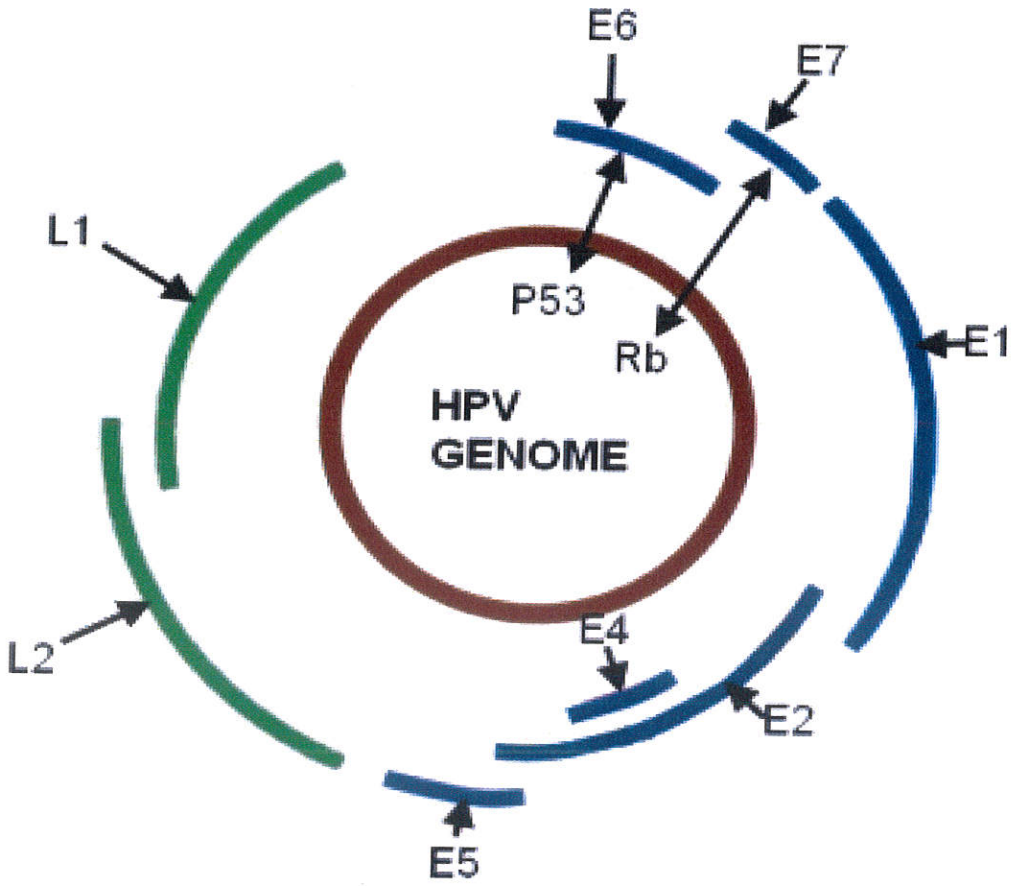

Figure 1- schematic representation of HPV genome (Lee, 2016)

2.5 Mode of Transmission

Human papilloma virus is mainly transmitted through sexual contact and skin- skin contact, and most people are infected with HPV shortly after the onset of sexual activities, and non- sexual transmission can occur through vertical and horizontal modes (Rombaldi *et al.*, 2008).

2.5.1 Vertical Transmission

Vertical transmission of HPV infection can occur from father or mother to the offspring. From the mother, the virus can be transmitted to the embryo, foetus or baby during pregnancy or childbirth. The infection can also occur at the time of fertilization via infected oocyte or spermatozoa (syrjanen, 2010). At present, there are no studies on HPV detection in oocytes, even though the virus has been detected in the seminal plasma and spermatozoa (Sabeena *et al.*, 2017). ThE main mode of transmission from the mother to child occurs during pregnancy and at the time of delivery.

I. Intrauterine Transmission

There is no viremic phase of HPV, and haematogenous spread from mother to foetus is unlikely. A foetus can be- come infected through the micro-tears in fatal membranes or through the placenta if the mother has genital HPV infection (Armbruster *et al.*, 1994). The detection rates of HPV-DNA in placental samples have varied from 0% to 42.5% in normal pregnant women (Rombaldi et al., 2008). Fedrizzi et al recently reported that HPV-DNA was 3.5-fold more frequent in the normal endometrial samples of smokers compared with non-smokers (Fedrizzi *et al.*, 2009). A Finnish family study noted a threefold higher HPV prevalence in the placental samples of smokers compared with non-smokers (Sarkola *et al.*, 2008).

II. Perinatal Transmission

Most of the infections in newborns occur at the time of delivery. The concordance of type-specific HPV between mothers and newborns is suggestive of perinatal transmission. Perinatal transmission usually occurs from direct contact with the infected maternal genital tract or by ascending infection especially after premature rupture of membranes. In most of the studies, mothers were tested for genital HPV infection in the third trimester. In one recent study, healthy mothers were followed up in all three trimesters and then after delivery. In that study, the prevalence of HPV infection was lower in the last trimester than in the first two trimesters (Lee *et al.,* 2013). Genital HPV infection can lead to placental and cord blood positivity. There is a higher possibility of cord blood HPV detection when a mother has a history of genital warts. Women with genital warts often progress to persistent HPV infection, and there is a higher chance of intrauterine transmission to the foetus (Watts *et al.,* 1998). Another study found a lower chance of perinatal transmission (approx. 2.8%) in the context of pregnant women with clinical and laboratory evidence of HPV infection (Watts *et al.,* 1998). In a study by Park et al., (2012), complete resolution of the infection in new born babies occurred by 6months of age. The immunization of women prior to pregnancy is not recommended because the possibility of infection is lower, as per the epidemiological data (Smith *et al.*, 2010).The risk of infection in vaginal delivery is low, and caesarean section is not recommended if the pregnant woman is found to have genital HPV infection (Cason *et al.*, 1998). As per the systematic meta-analysis by Medeiros et al., the pooled relative risk (RR) of vertical transmission is 4.8, (Medeiros *et al.,* 2005). The possibility of the newborn testing positive for HPV is higher if the cord blood or placenta is positive for HPV-DNA (Sarkola *et al.,* 2008). There are controversial results regarding the persistence of HPV infection in babies infected at birth. The persistence of

infection acquired during birth is very rare among infants, and alternate modes of transmission by close contact or through fomites should be considered in children with HPV infection (Rombaldi *et al.*, 2009).

III .Transmission through Breast Milk

Even though there are reports of isolation of HPV-DNA including high-risk types from breast milk and colostrums, there was no concordance between the HPV- DNA isolated from breast milk and that detected in the cervical or oral samples of the mothers (Puranen *et al.*, 1996). A recent study on 21 HPV-positive and 11 HPV-negative mothers by Mammas et al. failed to demonstrate high-risk HPV- DNA in breast milk. The restriction of breast-feeding is not advised if the mother is found to be infected with HPV (Mammas *et al.*, 2011).

2.5.2 Horrizontal Mode of Transmission

Human papillomvirus can be transmitted horizontally among sexually unexposed adults and children by autoinoculation, heterosinoculation or via fomites. HPV infection in the mothers at post-partum clinics can lead to infection in babies by close contact (Castllsague *et al.*, 2009).

1. **Auto or Hetero-inoculation Transmission**

Human papillomavirus may be transmitted among the family members by kissing and digital contact. Adults with genital warts may transmit the genital HPV types to their sexual partners by finger–genital contact (Sonnex *et al.*, 1999). Non-penetrative sex or inoculation via fingers can infect the partner. Children acquire the infection from close family members and caregivers with hand warts during cleaning of the anogenital area and diaper changing (Surjanen & Puranen, 2000). Autoinoculation can also occur by scratching the genital area. Oral HPV infection plays

an important role in the viral transmission between family members. The virus persists longer in the oral cavity than in the genital area in children, and the most prevalent types of oral infections are low-risk types, HPV-6 and -11 (Surjanen, 2010). Even high-risk HPV-16 and -18 can be transmitted by autoinoculation (cason *et al.*, 1998).

2. Transmission Through Formites

According to the 1989 study by Ferenczy et al., HPV - DNA positivity in the hospital, surgical instruments and gloves is low after sterilization. Washing and soaking in 2% glutaraldehyde or heating to 100°C is sufficient for disinfection. The detection of HPV-DNA in asymptomatic individuals using sensitive assays alone does not mean infection, because the virus is ubiquitous. The HPV-DNA can be transmitted to the genital area via fomites .It is not possible, however, for children to become infected with HPV via swimming pools and the sharing of Western-style toilets at school or at home. A study conducted in Finland by Puranen et al. failed to detect HPV-DNA in the floors and seats of bathing resorts (Puranen *et al.*, 1996).

According to another study by de Martino et al., HPV can be transmitted through infected towels or other objects (De martino et al 2013). The role of fomites in the development of active infection is not well established (jayasinghe & Garland, 2013). In gynaecology clinics, transvaginal ultrasound probes and colposcopes can transmit the infection and most of the disinfectants are not sufficient to neutralize HPV (Ryndock & Meyers, 2014). A recent study found that nosocomial infection may be possible in invasive procedures such as transvaginal ultra sonogram; and probes tested positive for HPV-DNA and free virions even after treating with exonucleases (Gallay *et al.*, 2016).

2.6 Clinical Features

HPV infection commonly causes skin or mucous membrane growths (warts). Such as common warts, plantar warts, flat warts, female genital warts, male genital warts. Most HPV infection doesn't lead to cancer. But some types of genital HPV can cause cancer of the lower part of the uterus that connects to the vagina.

Genital warts: - these appear as flat lesions, small cauliflower-like bumps or tiny stem-like protrusion. In women, genital warts appear mostly on the valve but can also occur near the anus, on the cervix or near the vagina. In men, genital warts appear on the penis and scrotum or around the anus. Genital warts rarely cause discomfort or pain though they may itch.

Common warts; - common warts appear as rough, raised bumps and usually occur on the hand, fingers or elbows. In most cases, common warts are unsightly, but they can also be painful or susceptible to injury or bleeding

Plantar warts; - plantar warts are hard, grainy growths that usually appear on the heels or balls of the feet.

Flat warts; - flat wart are flat-topped, slightly raised lesions darker than the skin. They can appear anywhere, but children usually get them on the face and men tend to get them in the beard area, and women tend to get them on the legs.

Majority of HPV infection do not cause symptoms or diseases and resolve spontaneously. However, persistent infection with specific types of HPV (16 & 18) may lead to cervical cancer, but this progression usually takes many years. Symptom of cervical cancer tends to appear only after the cancer has reached an advanced stage and may include

- Irregular, intermenstral (between periods) or abnormal vaginal bleeding after sexual intercourse.
- Back, leg or pelvic pain
- Fatigue, weight loss or loss of appetite
- Vaginal discomfort or odorous discharge
- A single swollen leg

Disease	HPV type associated with the disease
Cutaneous	
Plantar Wart	1, 2
Common Wart	2, 1
Flat Wart	3, 10
Butcher's wart	7, 2
Genital	
Bowen's disease (intraepithelial neoplasm)	16 (Genital) 2, 3, 4, 16 (Extragenital)
Epidermodysplasia verruciformis (full skin thickness wart can become squamous cell cancer)	2, 3, 5, 8, 9 10, 12, 14, 15, 17
Condylomata acuminata (venereal or genital warts)	6, 11
Intraepithelial neoplasms	Low grade 6, 11 High grade 16, 18
Respiratory papillomatosis (transmitted from mother to baby)	6, 11

Table 1- HPV types associated with some diseases (source; okolo 2009)

CHAPRTER THREE

3.0 MATERIALS AND METHODOLOGY

3.1 MATERIALS

- Standard microtiter plate reader (450nm)
- Sterile universal bottles
- Precision pipettes & disposable pipettes tips
- 37°C incubator
- -20°C refrigerator
- Microtiter plates
- Test tubes for dilution
- Distilled water
- 100ml graduated cylinder
- Personal protective equipments
- Testing sample (urine)
- Sterile universal bottles
- Disposable pipette tips
- Graduated beaker
- Microtiter plates
- Squit bottles
- ELISA Research kit(melsin medical supplies china)

3.2 METHODS

3.2.1 Study population

The study population comprises of FUOYE undergraduates students (aged 15-30 years) who had enrolled for 2017/2018 academic session.

3.2.2 Study design

This study was conducted between June 2018 and July 2018. A structured questionnaire was administered to the participants to obtain information on socio-demographic variables such as age, sex, location within oye, no of sex partners. Only informed and consented participants were enrolled in the study.

3.2.3 Sample collection & storage for HPV screening

Urine samples were collected from 96 students visiting the university health care centre for HPV screening using ELISA research kit (melsin medical supplies, china). Urine samples were collected using sterile universal bottles and were stored in -20°C refrigerator until used.

3.2.4 Sample screening

Test samples (urine samples) were brought out of the -20°C refrigerator and allowed to attain room temperature. The samples were tested for HPV IgM using ELISA research kit (melsin medical supplies, china) which determines the concentration of the IgM antibody.

- The ELISA research kit were brought out of the -20°C refrigerator and allowed to attain room temperature and tests were carried out as described by the manufacturer (melsin medical co, Ltd)

Assay procedures

- $50\mu l$ of positive control and $50\mu l$ of negative control was added to the positive and negative well. $10\mu l$ of testing sample was added to the testing sample well, while the blank well was left empty.
- 100μl of HRP-conjugate reagent were added to each well and covered with an adhesive strip and incubated for 60minutes at 37°C .
- Each well were washed by filling each well with wash solution ($40\mu l$) using a squirt bottle.
- The process was repeated four times for a total of five washes, and after the last wash, remaining wash solution was removed by decanting. Inverting the plate and blot it against clean paper towel.
- $50\mu l$of chromogen A solution and $50\mu l$ of chromogen B solution were added to each well. The plate was gently mixed and incubated for 15minutes at 37°C. Following incubation, there is a colour changes from white to blue.
- $50\mu l$ of stop solution was added to each well and the colour in the wells changed from blue to yellow.
- Absorbance and optical density (O.D) was read at 450nm using a microtiter plate reader and assay results was analysed.

CHAPTER FOUR

4.0 DATA PRESENTATION, ANALYSIS AND RESULTS

4.1 RESULTS

A total of ninety six (96) FUOYE students aged between 15 to30 years participated in the study. Of the 96 Urine samples analyzed, two (2) were positive for HPV IgM antibodies giving a seroprevalence of [2.1%]. The socio-demographic characteristics distribution of study population is shown in Table 4.0. The age group, 15-24 years had the highest prevalence of HPV positives. It was observed that there is no statistically significant relationship among undergraduate students (X^2= 0.12, Pr=0.942), students age 15-19 years was positive of HPV by 50.0% and age 20-24 years by 50.0% compare to those that was negative of HPV. Also from this study, table 3 shows that there is significant relationship between sex of undergraduate students and HPV (X^2=4.28, Pr=0.039), whereby female were more prominent to be infected by HPV by 100.0% than male compare to those that were negative of HPV. There is also significant relationship between number of sex partner and HPV (X^2=30.64, Pr=0.000), students that had two partners and more than two partners were mostly infected by HPV by 50% each compare with those that were negative HPV.

It has also been observed from this study that there is no statistically relationship between undergraduate students that use condom and HPV (X^2= 0.72, Pr=0.698), whereby undergraduates that did not use condom were 100% infected by HPV compare those that were negative of HPV (Table 4.2.4). There is also no statistically relationship between undergraduate students that always travelled out of school and HPV (X^2= 0.19,Pr= 0.663), where those that

travelled always out of school and those that reported no were positive of HPV by 50% each compare to those that were negative of HPV.

4.2 DATA ANALYSIS

4.2.1: Distribution of HPV IgM antibody among FUOYE students.

Anti-HPV	IgM n (%)
Positive	2(2.2)
Negative	94(97.8)
Total	**96**

Table 4.2.2: Distribution of Respondents by Age and HPV

Background characteristics	Human Papilloma Virus		Total	Statistics
	Negative	Positive		
Age				
15 – 19 years	47 (50.0)	1 (50.0)	48 (50.0)	
20 – 24 years	42 (44.7)	1 (50.0)	43 (44.8)	X^2= 0.12
25 years and above	5 (5.3)	0 (0.0)	5 (5.2)	Pr=0.942
Total	**94 (100.0)**	**2 (100.0)**	**96 (100.0)**	

Table 4.2.3: Distribution of Respondents by Sex and HPV

Background characteristics	Human Papilloma Virus		Total	Statistics
	Negative	Positive		
Sex				
Male	65 (69.2)	0 (0.0)	**65 (67.7)**	X^2=4.28
Female	29 (30.9)	2 (100.0)	**31 (32.3)**	Pr=0.039
Total	**94 (100.0)**	**2 (100.0)**	**96 (100.0)**	

Table 4.2.4: Distribution of Respondents by Number of Sex Partner and HPV

Background characteristics	Human Papilloma Virus		Total	Statistics
	Negative	Positive		
Number Of Sex Partner				
0	74 (78.7)16	0 (0.0)	**74 (77.1)**	
1	(17.0)	0 (0.0)	**16 (16.7)**	X^2=30.64
2	2 (2.1)	1 (50.0)	**3 (3.1)**	Pr=0.000
More	2 (2.1)	1 (50.0)	**3 (3.1)**	
Total	**94 (100.0)**	**2 (100.0)**	**96 (100.0)**	

Table: 4.2.5: Distribution of Respondents by Condom use and HPV

Background characteristics	Human Papilloma Virus		Total	Statistics
	Negative	Positive		
Do you use condom?				
Yes	21 (22.3)	0 (0.0)	**21 (21.9)**	X^2= 0.72
No	69 (73.4)	2 (100.0)	**71 (74.0)**	Pr=0.698
Occasionally	4 (4.3)	0 (0.0)	**4 (4.2)**	
Total	**94 (100.0)**	**2 (100.0)**	**96 (100.0)**	

Table 4.2.6: Distribution of Respondents that Travelling out of School and HPV

Background characteristics	Human Papilloma Virus		Total	
	Negative	Positive		
Do you always travel out of school?				
Yes	33 (35.1)	1 (50.0)	**34 (35.4)**	X^2=0.19
No	61 (64.9)	1 (50.0)	**62 (64.6)**	Pr=0.663
Total	**94 (100.0)**	**2 (100.0)**	**96 (100.0)**	

CHAPTER FIVE

5.0 DISCUSSION, RECOMMENDATION AND CONCLUSIONS

5.1 DISCUSSION

In this study, HPV IgM antibodies were detected in urine samples from 2 of 96 students studied, giving a seroprevalence of 2.1%. This percentage is lower than the 26.3% reported in Ibadan, Nigeria (okolo *et al.*, 2010). The lower seroprevalence in this present study compared with that in a previous study report from Nigeria may be due to a difference in the study population. Another reason could be the high sensitivity of the enzyme-linked immunesorbent assay that permits detection of the HPV antibody in samples with low HPV antibody which would probably otherwise have been scored as negative for HPV.

The high seroprevalence in the previous study could be due to the lifestyle of the local population in the study area, where people are constantly exposed to the virus, by means of early sexual debut, early marriage, multiple sexual partners due to polygamy, and high divorce rates. Acquisition of HPV infection has been shown to be strongly related to sexual behaviour, and the prevalence of HPV increases with increasing number of sexual partners and early sexual debut.

Subjects in the previous study area often marry as young as age 15 years, and it has been reported that the age of sexual debut in Nigeria is 9–10 years (kolawole, 2008). However, detection of antibodies to HPV, which signifies infection, does not mean eventual development of cervical cancer. This is because most HPV infections in younger women are transient or asymptomatic, often spontaneously regress (cuzick *et al., 1999*) and have little long-term

significance. Moreover, the incidence of cervical cancer in women younger than 30 years is very low, and 70% of cases of HPV infection resolve in one year and 90% in 2 years (Goldstein 2009). However, persistent infection with one or more high-risk types of HPV is an important etiologic factor in the development of cervical intraepithelial neoplasia and progression to cervical cancer (ho GY, 1998) In addition, virologic, environmental, immunologic, and genetic factors have also been implicated in the development of cervical cancer.(Tobara *et al.*, 2009)

5.2 CONCLUSION

HPV IgM antibody which is a marker of recent HPV infection was detected among FUOYE students visiting the university health care centre. The overall prevalence rate was approximately 2.1% (2/96). Although, the prevalence rate is relatively low, however, this study showed the evidence of HPV infection among the undergraduates. The high level of IgM is an indication of resents infection and the subjects may still be actively sexually involved.

5.3 RECORMENDATIONS

- It is recommended that HPV infection among the undergraduates needs to be studied in further details incorporating a larger population size, and carrying out RT-PCR to enable us understand the viral load (rate of infectivity) as well identifying the HPV genotype(s) possibly circulating among them.
- Public enlightenment about the infection and mode of transmission may be useful in keeping the rate of HPV infection very low.

- In addition, HPV routine screening together with other viral agents should be in included in the university requirements before given admission to know their health status.

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