

Human Papilloma Virus Infection and Associated Risk Factors Among Pregnant Women Attending Outpatient Clinic In General Hospital Lapai, Niger State Nigeria

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Abstract- Cervical cancer caused by Human Papilloma Virus (HPV) has claimed the lives of many women worldwide. The study aimed at determining the prevalence and associated risk factors of HPV infection among pregnant women attending outpatient clinic in General Hospital, Lapai Niger State, Nigeria. A total of one hundred and eighty four (184) pregnant women were enrolled, blood samples were subjected to ELISA screening for HPV and further screened for HIV, Hepatitis B and C using determine and HBsAg/HCV Rapid test strip methods. While ABO blood grouping reagents were used to detect blood grouped types. Questionnaires were used to obtain data from participants and analyzed statistically. Of the 184 enrolled, the prevalence of HIV was 4 (2.17%) while the prevalence of HBV infection was 18 (9.78%), 7 (3.80%) positive to HCV and 43 (23.37%) positive to HPV infection, also, the prevalence of HPV/HIV infection was 2 (1.09%), 3 (1.63%) positive to HPV/HBV and lastly, 2 (1.09%) positive to HPV/HCV respectively. The result showed prevalence (28.79%) of HPV infection in women between 26-30 age groups, (28.24%) in women who had multiple sex partners and (27.73%) in women with multi-parity. Thus, Age, Marriage types and Parity shows statistically significant relationship between HPV infection ($P < 0.05$). Prevalence rate of HPV infection recorded high among other risk factors such as Civil Servant participants (26.32%), secondary school level participants (28.33%), women in Lapai Town (26.67%), no contraceptives use (24.42%) and women with history of blood transfusion (26.54%) respectively. With regard to blood group status, 'O' RhD+ve women, showed highest prevalent (27.54%). In relation to other viral infections, HPV/HBV recorded highest prevalence (1.63%). High HPV prevalence and associated risk factors observed among pregnant women attending prenatal clinic in General Hospital Lapai, regular HPV and other viral infections screening is recommended.

Index Terms- Prevalence, Associated risk factors, HPV infection, Pregnant women, Lapai.

I. INTRODUCTION

Cervical cancer caused by HPV, is the second most common cancer among women worldwide. HPV causes deformation of genomic integrity, control of cell cycle, cell

adhesion and apoptosis by suppressing tumor suppressor genes and interacting with cellular proteins via various proteins on carcinogenesis process (Jerome, *et al.*, 2018). The oncogenic human papilloma virus (HPV) types are the most significant risk factors in its aetiology (Quamrun. *et al.*, 2014). In a famous paper, the Italian physician Rigoni-Stern in 1760 to 1839 noted that cervical cancer was common among prostitutes, married women and widows but rare in virgins and nuns. Later, in 1898, McFadyean and Hobday successfully transmitted canine oral papillomatosis, while Codeac transmitted warts from horse to horse, in 1901. In 1934 Rous and Beard noted that papillomas of domestic rabbits frequently converted to squamous-cell carcinomas, although it was lately acknowledged in 1966. From this period, positive interest about role of HPV infection in cervical cancer began. In 1978, (Scheffner, *et al.*, 1993) in Italy, and Laverty *et al.*, in Australia, firstly demonstrated the presence of HPV virions within dense bodies of koilocytes, but deeper investigations were limited by the inability to propagate HPV in cultured cells or in simple animal models. Human Papilloma Virus (HPV) infection is a major health problem among women worldwide with a global prevalence of 11.7% resulting to Cervical Cancer.

More than 100 types of HPV genotypes have been isolated and characterized; among which 40 infect the mucosal epithelia, with 6.2 million new cases occurring annually. A prevalence rate of 24% has been recorded in Sub-Saharan Africa (IDSA. Journal of Infectious Diseases, 2010). The incidence rate of cervical cancer in Nigeria is reported as 25/100,000; prevalence rates for HPV in women being 44.9% (Musa, *et al.*, 2013), 26.3% (Thomas, *et al.*, 2004) and 24.0% (Okolo, *et al.*, 2012). HPV is also implicated in the genesis of several other cancers, such as head and neck (oral cavity, pharynx, and larynx) cancer and non-melanoma skin cancer and is suspected also to play a causal role in the genesis of a few other neoplasms (Trottier *et al.*, 2009). The predominant route of HPV transmission is via sexual contact and it is not easily detected at early stage of infection (Malloy, *et al.*, 2000), although HPV infection also may be transmitted by non-sexual routes (vertical and horizontal transmissions) such as transmission from a woman to a newborn infant at the time of birth (Center for Disease Control and Prevention 2010) and transmission from the anogenital region to hands via self-inoculation (Hernandez *et al.*,

2008).

II. MATERIALS AND METHODS

Study area

Present day of Lapai, was originally called Badeggi (meaning small area) and came into existence in 1800 founded by Abdullahi Nabadeggi, later renamed as Lapai after the migration of old Lapai to Badeggi in 1937 (Muhammadu Kobo 1980). Lapai is the Headquarters of one of the twenty five local government area in Niger State of Nigeria. It is bounded in the North – East by Paikoro and Gurara local government, on the West by Agaie local government and on the South by Federal Capital Abuja and Kogi State. The favourable climate conditions couple with the vast gentle slope environment make it possible for people to settle (Garba I.M. 2007).

Research Design

This research was a hospital based descriptive cross sectional survey conducted from the period of February, 2018 to May, 2018. Only consenting subjects were recruited and included in this study. Ethical approval was obtained from the ethical committee of the Hospital Management Board through the Medical Director of the Hospital. Informed consent and Demographic information was obtained from the participants using questionnaire.

Sample Collection and Storages

Blood samples used for this research were collected from pregnant women visiting prenatal clinic aseptically by vein puncture using 2ml sterile disposable syringes and needles. Drops of blood samples were placed each on the three depressions of the glass slides and blood grouped were determined, before dispensed into labelled specimen bottles. The samples were allowed to clot and centrifuged for approximately 20 minutes at 3000 rpm within 30 minutes after collection at 3,000 rpm for 5 minutes to separate the serum. The sera were collected into tubes and screened for HIV, Hepatitis B and C immediately before stored in a refrigerator at -80C to avoid loss of bioactivity and contamination. Thereafter, they were subjected to ELISA screening and HPV-IgG determination.

Blood Grouping Procedure:

All the materials required were set on the table and the Monoclonal Antibody (Mab) kit is placed in an Ice tray. Blood samples were brought to room temperature (18°C-25°C) A drop of blood sample was placed on the three depressions of the glass slide. A drop of Anti-A (blue bottle) was added into the 1st spot. A drop of Anti-B (yellow) was added into the 2nd spot. A drop Anti-D (colorless) was added into the 3rd spot. The content in each well was mixed using fresh mixing stick. Agglutination was observed in the form of fined red granules within 30seconds. (Anti RhD took slightly longer time to agglutinate compared to Anti A and Anti B).

HIV, HBV and HCV Rapid Test Determination:

Blood samples and buffer were brought to room temperature (15 – 30⁰c) before use. The test strips were removed from the sealed and the assay is performed immediately after

opening the foil pouch. 2 drops (approximately 50ul) of serum and 1drop of buffer were transferred to the sample pad of the HIV, HBV and HCV strips each with a disposable pipette and then the timer started. The results were read after red line(s) appeared in the control band at 10 minutes.

Interpretation of HCV, HBsAg and HIV Rapid Test Results:

A colored band appeared in the control band region (C) and another colored band appears in the T band region = Positive Result. One colored band appeared in the control band region (C) and no band appeared in the test band region (T) = Negative Result. No colored band appeared in the control band region (C) either band appeared or not in the test band region (T) = Invalid Result

Determination of HPV-IgG in Blood Samples

All reagents and samples were brought to room temperature (18°C-25°C) naturally for 30min before starting assay procedures. The Microelisa Strip plate is detachable, detached unused strips from the plate frame, were returned to the foil pouch with the desiccant pack, and resealed for preventing damp. Positive Control wells, Negative Control wells and Sample wells were set. 50µl Positive Control was added to each Positive Control well and 50µl Negative Control 50µl were added to each Negative Control well. 10µl sera blood and 40µl Sample Diluents' were added to each of the Sample wells. 100µl of HRP-conjugate reagent was added to Positive Control wells, Negative Control wells and Sample wells, the wells were all covered with an adhesive strip and incubated for 60 minutes at 37°C. The Microtitre Plate was washed 4 times. 50µl Chromogen Solution A and B were added to each well and then gently mixed. The plate was protected from light and incubated for 15 minutes at 37°C. 50µl Stop Solution was added to each well and gently taps. The colored in the wells changed from blue to yellow. The Optical Density (O.D.) was read at 450nm using an ELISA reader within 15 minutes after adding Stop Solution (Around 5 minutes is the best time.).

Statistical Analysis

Data obtained from the questionnaires used to obtain data from participants were analyzed using Chi-square(X^2) and the test of significance was determined at $P < 0.05$.

III. RESULTS

Table 1 showed the highest prevalence (28.99%) of HPV infection between ages of 26 to 30 years old and the lowest prevalence (0.00%) between ages of 36 to 40 years old. Statistics revealed a significant relationship between Age and HPV infection in pregnant women attending outpatient clinic in General Hospital Lapai ($P=0.0260$).

Table 2 indicated a high prevalence (26.32 and 26.09%) of HPV infection among Civil Servants and Traders with the lowest prevalence (8.33%) among Teachers. $P=0.7867$ therefore, no significant relationship between Occupation and HPV infection. Prevalence of HPV infection among pregnant women with reverence to Educational status presented in Table 3 indicated high prevalence (28.33%) in secondary School level and lowest (9.38%) in Quranic level. $P=0.3306$ hence, no significant

relationship between Educational level and HPV infection among the women.

In relation to other risk factors, there was a higher prevalence (22.45%) of HPV infection among women who don't use contraceptives as compared to (24.42%) those who uses contraceptives. P=0.3633, no significant relationship between oral contraceptives use and HPV infection. Types of marriage recorded highest prevalence (28.24%) among women with multiple sex partner (Polygamy Marriages) and lowest prevalence (19.19%) among women with single sex partner (Monogamy Marriages). Statistics showed a significant relationship between the type of marriages and HPV infection (P=0.0240). In relation to area of domicile, results showed higher prevalence (26.57%) of HPV infection among women leaving in Lapai town and lower rate (20.21%) of women leaving in surrounded villages. P=0.3899 thus, no significant relationship between area of domicile and HPV infection. With respect to parity, the prevalence (26.05%) was highest among women with multi-parity pregnancies and lowest rate (18.46%) among primps pregnancies history. Statistics showed a significant relationship between numbers of pregnancies and HPV infection with P=0.0240. While history of blood transfusion recorded a higher prevalence (26.14%) in women who

had no history of blood transfusion and lowest rate (9.68%) in women who had transfusion history. P=0.9845 no significant relationship between transfusion history and HPV infection (Table 4).

Table 5 showed, highest prevalence (27.54%, 25.86% and 25.00%) of HPV infection among women with positives blood grouped respectively, but lowest prevalence (0.00%) were recorded among pregnant women who had negatives blood grouped. Though; no significant relationship between blood group type and HPV infection were recorded (P=0.1974).

Lastly, table 6 showed prevalence of HPV infection among pregnant women with other viral infections; out of 184 enrolled, the prevalence of HPV was (23.37%) while the prevalence of HBV infection was (9.78%), (3.80%) was HCV and (2.17%) was HIV infection respectively. There was a highest prevalence rate in women with HPV/HBV (1.63%) followed by HPV/HCV (1.09%) and lowest prevalence was recorded among women with history of HPV/HIV/HBV/HCV infections (0.00%). P=0.7274 therefore, no significant relationship between other viral infections and HPV infection among pregnant women visiting prenatal clinic in Lapai General Hospital.

Table 1: Prevalence of HPV Infection among Pregnant Women with Respect to Age

Age	No Samples	No (%) Positive
15 - 20	16	2 (12.50)
21 – 25	47	13 (27.66)
26 – 30	69	20 (28.99)
31 – 35	49	8 (16.33)
36 – 40	3	0 (0.00)
Total	184	43 (23.37)

$X^2 = 11.0491$ df = 4 p-value = 0.02602

Table 2: Prevalence of HPV Infection among Pregnant Women with Respect to Occupation

Occupation	No Samples	No (%) Positive
Farmer	33	8 (24.24)
Trader	23	6 (26.09)
Teacher	12	1 (8.33)
Civil Servant	19	5 (26.32)
House Wife	97	23 (23.71)
Total	184	43(23.37)

$X^2 = 1.7223$ df = 4 p-value = 0.7867

Table 3: Prevalence of HPV Infection among Pregnant Women with Respect to Education

Education	No Samples	No (%) Positive
Non Formal Education	45	12 (26.67)
Quranic	32	3 (9.38)
Primary	21	5 (23.81)
Secondary	60	17 (28.33)
Tertiary	26	6 (23.08)

Total **184** **43**

$X^2 = 4.6018$ df = 4 p-value = 0.3306

Table 5: Prevalence of HPV Infection among Pregnant Women with Respect to Blood Group

Blood Group	No Samples	No (%) positive
A' Rhesus D+VE	36	9 (25.00)
B' Rhesus D+VE	58	15 (25.86)
AB' Rhesus D+VE	18	0 (0.00)
O' Rhesus D+VE	69	19 (27.54)
A' Rhesus D-VE		0 (0.00)
B' Rhesus D-VE	1	0 (0.00)
AB' Rhesus D-VE		0 (0.00)
O' Rhesus D-VE	2	0 (0.00)
TOTAL	184	43

$X^2 = 7.3278$ df = 4 p-value = 0.1974

Table 4: Prevalence of HPV Infection among Pregnant Women in Relation to other Risk Factors

Risk Factors	No Samples	No (%) Positive	X ²	p-value
Contraceptives Use				
Yes	86	21 (24.42)	0.8263	0.3633
No	98	22 (22.45)		
Type of Marriage				
One Sex Partner (Monogamy)	99	19 (19.19)	10.0380	0.01501
Multiple Sex Partner (Polygamy)	85	24 (28.24)		
Area of domicile				
Lapai Town	90	24 (26.67)	0.7394	0.3899
Surrounding Villages	94	19 (20.21)		
Parity				
Primps	38	12 (18.46)	12.9398	0.02395
Multi-parity	119	31 (26.05)		
Transfusion History				
Yes	31	3 (9.68)	0.1552	0.9845
No	153	40 (26.14)		

Table 6: Prevalence of HPV Infection among Pregnant Women with other Viral Infections

Samples Size	HPV	HIV	HBV	HCV	HPV/H IV	HPV/ HBV	HPV/H CV	HPV/HBV/H CV
184	43 (23.37)	4 (2.17)	18 (9.78)	7 (3.80)	2 (1.09)	3 (1.63)	2 (1.09)	0 (0.00)

$$X^2 = 1.3073 \text{ df} = 3 \text{ p-value} = 0.7274$$

IV. DISCUSSION

This study evaluated the prevalence of HPV infection among women in Lapai, Niger State of Nigeria with a total prevalence of 23.37% which is lower than prevalence (24.47%) reported among pregnant women attending antenatal care in General Hospital Minna, (Terese, *et al.*, 2015). However, record showed consistent HPV infection prevalence rates of 24.8%, 26.3% and 36.5% among women in Ibadan and Lagos (Thomas, *et al.*, 2004, Okolo, *et al.*, 2012 and Kehinde, *et al.*, 2017).

Age specific, findings revealed an increase rate (28.99 and 27.66%) of HPV infection among women between 26-30 years with decrease rate (16.33%) between ≥ 31 years. Although early onset of sexual activity (≤ 15 age), multi-parity and sexual promiscuity have been recognized as some of the significant risk factors for HPV infection (Nejo, *et al.*, 2018). Similar work carried out by Terese, *et al.*, (2015) in Minna, Nigeria is in line with this findings indicating a significant relationship ($P=0.0260$) between aged and HPV infection.

In relation to occupational and educational status, the study shows high incident (26.32%) among Civil Servant follow by Traders (26.09%) $P=0.7867$. Whereas, prevalence rate were high among pregnant women in Secondary (28.33%) $P=0.3306$. These findings contradicted similar study by Terese, *et al.*, (2015). Who reported high prevalence (12.77%) in no formal education and lowest (2.84%) among Civil Servant. Consequently, these results may be attributed to lack of awareness about HPV infection and its implications at all level. However, statistics shows no significant relationship in the occupational and educational status. Our present study showed that the prevalence of HPV infection was higher in Multiple sex partner (28.24%), which was in agreement with a study conducted in China (Yanru, *et al.*, 2016). The low prevalence of HPV infection among single sex partner may be due to the protective effect against HPV infection generated by living with a partner. Statistic shows a significant relationship ($P=0.0150$) between HPV Infection and type of marriage. In relation to contraceptives, higher prevalence (22.45%) was among women that don't use contraceptives as compared to (24.42%) those that use contraceptives. It is reported that contraceptives use was significantly associated with HPV infection, while some other studies have failed to confirm such an association (Olusola, *et al.*, 2015). In our study, we didn't find a significant relationship between the use of contraceptives and HPV infection ($P=0.3633$).

The prevalence of HPV co-infected with other virus among women showed prevalence of 1.63, 1.09 and 1.09% in HPV/HIV, HPV/HBsAg and HPV/HCV respectively. The prevalence of HPV infection was high, in contrast to a much lower prevalence of other sexually transmitted infections (STIs) in the same geographical area (Jerome, *et al.*, 2018). $P=0.7274$ no significant relationship between HPV infection with co-virus. Although, various prospective and cross sectional studies from other parts of the world have shown that among HIV-1 infected patients, active,

chronic and persistent HPV infection is more common in those with features of significant immunosuppression AIDS defined as $CD4 < 200$ cells/ul (Quamrun, *et al.*, 2014)

The number of pregnancies was significantly associated ($P=0.0150$) with HPV infection, since the higher proportion of HPV infection was prevalent among women with multi-parity (26.05%) but was less amongst primip women (18.46%). This agrees with (Cancer research UK, 2013) which stipulate that women who have had 7 or more children had double the risk of HPV infection than women with only 1 or 2 children. They also found a doubling of risk of squamous cell cervical cancer with 3 or more children, compared to no children. This indicates that the risk of getting infected with the virus doubles with number of times a woman gives birth because of the occurrence of cervical trauma at the time of delivery. When a woman gives birth, there are tendencies of weakening and rupture of epithelial cells around the cervix there by enhancing chances of infection when in contact with the virus.

High prevalence rate among women with blood Group A, B, and O Rhesus positives (25.0, 25.86 and 27.54%) and low rate 0.00% among all women with Rhesus Negatives were correlated with report by Yuzhalin, *et al.*, who revealed that carriage of non-O blood types increased the risk of ovarian cancer by 40-60% in premenopausal, but not in postmenopausal women (Yuzhalin, *et al.*, 2012). We did not observe a statistical significant relationship between ABO Blood group types and HPV infection in women ($P=0.197$).

We recorded higher prevalence (26.14%) in women without history of blood transfusion as against lower prevalence (9.68%) in women with history of blood transfusions. This suggests that blood transfusion has nothing to do with increase HPV infection rate. This result was correlated with report in 2005, by Sohrab, *et al.*, which demonstrated that the HPV16 genome exists in HIV patients who acquired HIV infection via transfusion and vertical transmission and who were, according to clinical history, sexually naive. Further study demonstrated that the HPV16 genome was also present in blood of healthy blood donors, suggesting a potential for transmission via the bloodstream. However, blood transmission of HPV as well as transmission via breast milk is implausible since HPV infection does not produce viremia (Cason *et al.*, 2005). $P=0.9845$ no statistical significant relationship between HPV infection and blood transfusion history. HPV infection prevalence among pregnant women in relation to area of domicile, recorded high prevalence (26.67%) in women leaving in Lapai town as compared to surrounding villages (20.21%). This study agreed with report of high HPV prevalence and associated risk factors observed in the continuous transmission of the virus in Southwest Nigeria as against other part of Nigeria. (Nejo, *et al.*, 2018). $P=0.9845$ no statistical significant difference recorded.

V. CONCLUSION

The prevalence of HPV infection was 23.37% which demonstrates an alarming rate among pregnant women in Lapai considering the danger of infection posed to the health of the woman and her baby it is recommended among others, that there is urgent needs for advocacy for women of childbearing age to undergo regular HPV screening and also increase the availability and affordability of immunization facilities across the country.

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REFERENCES

- [1] Bauer H.M, Ting Y, Greer CE, Chambers JC, Tashiro CJ, Chimera J, Reingold A, Manos MM (1991): Genital human papillomavirus infection in female university students as Determined by a PCR-based method. *JAMA* 1991; 265:472-7.
- [2] Bennani B., C. Bennis, C. Nejjari, L. Ouafik, M.A. Melhouf, K. El Rhazi, K. Znati, H. Chaara, C. Bouchikhi and A. Amarti Riffi. (2012): Cervical Cancer Early Detection *J Infect Dev Ctries*, 6(7): 543-550.
- [3] Bodaghi, L.V. Wood, G. Roby, C. Ryder, S.M. Steinberg, and Z. Zheng. (2005): *J Clin Microbiol*, 43(11):5428–5434.
- [4] Cancer research UK, Cervical cancer risks and causes. (2013): <http://www.cancerresearchuk.org>
- [5] Centers for Disease Control and Prevention “CDC” (2010): FDA licensure of bivalent human papillomavirus vaccine (HPV2, Cervarix) for use in females and updated HPV vaccination recommendations from the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep*; 59:626-9.
- [6] Dutra I, Santos MR, Soares M, Couto AR, Bruges-Armas M, Teixeira F, Monjardino L, Hodgson S, Bruges-Armas J (2008): Characterisation of human papillomavirus (HPV) genotypes in the Azorean population, Terceira Island. *Infect Agent Cancer* 3:6.
- [7] IDSA. *Journal of Infectious Diseases*(2010): 202(12): 1789-1799.
- [8] Garba Ibrahim Magaji (2007): historical background of badeggi. Unpublished page 15-17.
- [9] Jerome Bigoni, Rosa Catarino, Caroline Benski, Manuela Viviano, Maria Munoz, Honoré Tilahizandry, Patrick Petignat, and Pierre Vassilakos (2018): High Burden of Human Papillomavirus Infection in Madagascar: Comparison with Other Sexually Transmitted Infections: *Arch Basic Appl Med. Feb*; 6(1): 105–112. Published online May 4 PMID: PMC5997288 NIHMSID: NIHMS968798 PMID: 29905313.
- [10] Kashima HK, Shah F, Lyles A, Glackin R, Muhammad N, Turner L, Van Zandt S, Whitt S, Shah K (1992): A comparison of risk factors in juvenile-onset and adult-onset recurrent respiratory papillomatosis. *Laryngoscope* 1992; 102(1):9-13.
- [11] Kehinde Sharafadeen Okunade, Chidinma Magnus Nwogu, Ayodeji Ayotunde Oluwole and Rose Ihuoma Anorlu (2017): Prevalence and risk factors for genital high-risk human papillomavirus infection among women attending the out-patient clinics of a university teaching hospital in Lagos, Nigeria. Published online Nov 14. doi: [10.11604/pamj.2017.28.227.13979] PMID: PMC5882206 PMID: 29629013
- [12] Keita N, Clifford G.M, Koulibaly M, Douno K, Kabba I, Haba M, Sylla B.S, van Kemenade F.J, Snijders P.J, Meijer C.J, Franceschi S (2009): HPV infection in women with and without cervical cancer in Conakry, Guinea. *Br J Cancer* 101:202-208.
- [13] McLaughlin-Drubin, M.E.; Crum, C. P. & Munger, K. (2011): Human papillomavirus E7 oncoprotein induces KDM6A and KDM6B histone demethylase expression and causes epigenetic reprogramming. *Proc.Natl.Acad.Sci.U.S.A.*, Vol.108, pp. 2130-2135
- [14] Musa J, Taiwo O, Achenbach C. Achenbach, S. Olugbenga, B. Berzins, A.S. Sagay, J.A. Idoko, P.J. Kanki and R.L. (2013): *Murphy. Arch Gynecol Obstet*, 288(6): 1365-1370.
- [15] Muhammadu Kobo (1980): Brief History of Lapai Emirate; first edition. Published by “FOURTH DIMENSION PUBLISHERS”. 179 zik avenue Enugu, Nigeria. Printed by Gaskiya corporation Ltd Zaria.
- [16] Ndams I.S, Joshua I.A, Luka S.A, Sadiq H.O. and Ayodele S.B. (2010): *Ann Nigerian Med Journal*, 4(1):14-17.
- [17] Nejo Y.T, Olaleye D.O, and Odaibo G.N (2018): Prevalence and Risk Factors for Genital Human Papillomavirus Infections Among Women in Southwest Nigeria. The publisher's final edited version of this article is available at *Arch Basic Appl Med*.
- [18] Ogedegbe G, Cassells AN, Robinson CM, DuHamel K, Tobin JN, Sox CH, Dietrich AJ (2005): Perceptions of barriers and facilitators of cancer early detection among low-income minority women in community health centers. *J Natl Med Assoc* 97:162-170.
- [19] Okolo C, Franceschi S, Adewole I, Thomas J.O, Follen M, Snijders P.J, Meijer C.J, Clifford G.M (2012): Human papillomavirus infection in women with and without cervical cancer in Ibadan, Nigeria. *Infect Agent Cancer* 5:24.
- [20] Olusola Anuoluwapo Akanbi, Abiodun Iyanda, Folakemi Osundare, and Oluyinka Oladele Opaleye (2015): Perceptions of Nigerian Women about Human Papilloma Virus, Cervical Cancer, and HPV Vaccine. Department of Medical Microbiology and Parasitology, College of Health Sciences, Ladoko Akintola University of Technology, PMB 4400, Osogbo, Nigeria.
- [21] Onuki M, Matsumoto K, Satoh T, Oki A, Okada S, Minaguchi T, Ochi H, Nakao S, Someya K, Yamada N, Hamada H, Yoshikawa H (2009): Human papillomavirus infections among Japanese women: age-related prevalence and type-specific risk for cervical cancer. *Cancer Sci* 100:1312-1316.
- [22] Quamrun Nahar, Farhana Sultana, Anadil Alam, Jessica Yasmine Islam, Mustafizur Rahman, Fatema Khatun, Nazmul Alam, Sushil Kanta Dasgupta, Lena Marions, Ashrafunnessa, Mohammed Kamal, Alejandro Cravioto, and Laura Reichenbach (2014): Genital Human Papillomavirus Infection among Women in Bangladesh: Findings from a Population-Based Survey. Department of Pathology, Singapore General Hospital, Singapore. *Int J STD AIDS. Dec*;25(14):1013-21. doi: 10.1177/0956462414528315. Epub Mar 19. *PLoS One*; 9(10): 107675. tay.sun.kuie@sgh.com.sg. journal.pone.0107675 PMID: PMC4182674 PMID: 25271836.
- [23] Scheffner, M.; Huibregtse, J. M.; Vierstra, R. D. & Howley, P. M. (1993): The HPV-16 E6 and E6-AP complex functions as a ubiquitin-protein ligase in the ubiquitination of p53. *Cell.*, Vol.75, pp. 495-505
- [24] Smith, J.S., Lindsay, L., Hoots, B., Keys, J., Franceschi, S., Winer, R. and Clifford, G.M. (2007): Human papillomavirus type distribution in invasive cervical cancer and high-grade cervical lesions: a meta-analysis update. *Int J Cancer*, 121, 621-632.
- [25] Swarz T.F (2007): Human papillomavirus -16/18 candidate vaccine adjuvanted with AS04 and its impact on the incidence of cervical cancer. *Expert Rev Obstet Gynecol* 2:1320-1323.
- [26] Syrjanen K, Shabalova I, Petrovichev N, Kozachenko V, Zakharova T, Pajanidi J, Podistov J, Chemeris G, Sozaeva L, Lipova E, Tsidavaeva I, Ivanchenko O, Pshepurko A, (2006): Oral contraceptives are not an independent risk factor for cervical intraepithelial neoplasia or high-risk human papillomavirus infections. *Anticancer research*; 26:4729–4740. *Crow JM. HPV: The global burden. Nature. 2012*;488:S2–3.
- [27] Terese E. Azua, Priscilla Y. Orkaa and Regina A.I. (2015): Prevalence of Human Papilloma Virus (HPV) Infection among Pregnant Women attending Antenatal Care at General Hospital, Minna – Nigeria. *Journal of Microbiology and Biotechnology: Scholars Research Library. J. Microbiol. Biotech. Res.*, 2015, 5 (3):38-44 (<http://scholarsresearchlibrary.com/archive.html>) ISSN; 2231 –3168 CODEN (USA). JMBRB438.
- [28] Tseng, C. J., C. C. Pao, J. D. Lin, Y. K. Soong, J. H. Hong, and S. Hsueh. (1998): Detection of human papillomavirus types 16 and 18 mRNA in peripheral blood of advanced cervical cancer patients and its association with prognosis. *J. Clin. Oncol.* 17 : 1391-1396.
- [29] Thomas J.O, Herrero R, Omigbodun A.A, Ojemakinde K, Ajayi IO, Fawole A, Oladepo O, Smith J.S, Arslan A, Munoz N, Snijders P.J, Meijer C.J, Franceschi S (2004): Prevalence of papillomavirus infection in women in Ibadan, Nigeria: a population-based study. *Br J Cancer* 90:638-645.
- [30] Xi L.F, Toure P, Critchlow C.W, Hawes S.E, Dembele B, Sow PS, Kiviati N.B (2003): Prevalence of specific types of human papillomavirus and

cervical squamous intraepithelial lesions in consecutive, previously unscreened, West-African women over 35 years of age. *Int J Cancer* 103:803-809.

- [31] Yanru Zhang, Yueyun Wang, Li Liu, Chun Guo, Zihua Liu, and Shaofa Nie (2016): Prevalence of human papillomavirus infection and genotyping for population-based cervical screening in developed regions in China. Published online Aug 22. doi: [10.18632/oncotarget.11498] PMID: 27566561.
- [32] Yuzhalin AE, Xuefeng Liu and Ryder (2012): *Asian Pac J Cancer Prev: Research Institute for Complex Issues of Cardiovascular Diseases under Siberian Branch of Russian Academy of Medical Sciences, Kemerovo, Russian Federation.*
- [33] Zur Hausen, H. (2002): Papillomaviruses and cancer: from basic studies to clinical application. *Nat. Rev. Cancer*, 2: 342-350.

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