Prevalence of Human Papillomavirus infection by age and cervical cytology in Thika, Kenya.

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Financial Support

This work was supported by DAAD (German Academic Exchange Service), Africa Doctoral Research Fellowship (ADDRF), University of Heidelberg, Hologic™ and MTM laboratories AG

SUMMARY

Human papillomavirus (HPV) infections cause cervical cancer and premalignant dysplasia. Data on HPV and cervical cancer in Kenya are scarce. Type-specific HPV prevalence data provides a basis for assessing the impact of HPV vaccination programs on cervical cytology and how HPV based screening will influence cervical cancer prevention. To investigate HPV infections in a population in Kenya, we obtained cervical cells specimen from 498 women in a population in Thika district. We report HPV type specific prevalence and distribution data for 498 women (age range 18-74 years; mean age 36 years) recruited into the study in relation to age and cervical cytology. The study was conducted between January to May 2010. Pap smears were performed, HR HPV DNA were detected by Digene Hybrid capture 2® (hc2) test and HPV genotyping was performed with Multiplex Luminex HPV genotyping kit (Multimetrix, Progen, Germany). Samples from 106 women (21.3%) tested positive for HPV. Multiple HPV types were detected in 40 (37.7% of HC2-positive samples) and the rest had infection with single HPV type. The most common HR HPV type at all ages was HPV16, 52, 56, 66, and 18. There was a marked decline in the prevalence of HR-HPV with age.

The pattern of HR HPV distribution in this population was slightly different from existing literature, which has important consequences for HPV vaccination and prevention programs.

[Afr J Health Sci. 2011; 19:53-62]

Introduction

Human papillomavirus (HPV) infection has been recognized as the central causal agent cervical cancer which is the second most neoplastic malignancy of women worldwide [1]. More than 100 HPV types have been described and 40 can infect the anogenital tract [2]. Genital HPV types are categorized according to their association with cervical cancer [1]. About 20 are classified as high risk HPV (HR HPV) and are associated with cervical cancer and precancerous lesions, as well as low grade cervical pathology [3]. Low risk HPV (LR HPV) cause low grade cervical lesions, genital warts and recurrent respiratory papillomatosis [3].

Worldwide, HPV types 16 and 18 are responsible for approximately 70% of cervical cancer cases; HPV types 31, 33, 35, 42, 52 and 58 account for an approximately 20% of the cases [4]. However, there is substantial geographical variation in the relative frequency of different HR HPV types [4].

These findings have led to the development of two vaccines against HR HPV. A quadrivalent prophylactic vaccine against HPV types 6, 11, 16 and 18 was licensed in the USA [5] and more recent a bivalent vaccine against HPV 16 and 18 [6]. While commercial vaccines against HPV 16 and HPV 18 are now available, global variations in HPV type specific prevalence could affect their regional effectiveness [7]. Despite the widespread efforts to ascertain the burden of HPV infections in populations across diverse regions, little systematic data are available on the prevalence of HPV infections in Kenyan population.

The most prevalent type worldwide is HPV 16, and there is less variation in the geographical distribution of HPV 16 than in that of the other types [8]. The prevalence of HPV is higher in African women with a normal cervical cytology than in women in other regions of the world [4]. Yet, HPV 16 infections of women with a normal cytology are found more commonly than infections caused by other HPV types in regions of the world apart from sub-Saharan Africa, where infections by other oncogenic types, most significantly, HPV 35, may dominate [4]. Indeed, the latter study also showed that HPV 35 was as common in Africa as HPV 16 (in 8% of infections), followed by HPV 31, HPV 45, HPV 56, and HPV 58 (in 6% of infections) [4]. HPV testing rather than cytology test has been recommended for cervical cancer screening in some resource poor areas [9] and in areas with high prevalence of Human Immunodeficiency virus (HIV) [10].

Kenya has a population of 10.32 millions women ages 15 years and older who are at risk of accruing HPV infections and developing cervical cancer [11]. Current estimates indicate that every year 2635 new cases of cervical cancer are reported and 2111 deaths from the disease [12]. Cervical cancer ranks as the 1st most frequent cancer among women in Kenya, and the 2nd most frequent cancer among women between 15 and 44 years of age [11]. About 38.8% of women in the general population are estimated to harbour cervical HPV infection at a given time, and 60.9% of invasive cervical cancers are attributed to HPV 16 or HPV18 [12]. In Kenya, a clinic based study found the prevalence of HPV in HIV positive women was found to be 49% and 17% in HIV negative women ([13]. This is similar to previous findings of a clinic based study, HPV prevalence of 41% in HIV positive women and 14% in HIV negative women [14].

Vaccination for the primary prevention of Human Papillomavirus infection in adolescent girls and use of methods to detect infection with carcinogenic HPV types allowing for early detection and treatment of precancerous cervical lesions are the main approaches of dealing with cervical cancer in recent years in the developed countries [15]. In Kenya there have been relatively few population based studies on the distribution of HPV genotypes. The recent and only available data in Kenya on HPV genotypes distribution is from a study in 2002 by Globoscan [12]. To address this lack of data, we examined HPV genotype distribution in 498 women who were between ages 18 and 74 who participated in the study. The findings report the type specific prevalence data for all HR HPV types in the population.

Materials and methods

The study was conducted in Thika District. A total of 498 samples were collected. Thika district is one of the administrative districts in Central province with the capital being Thika Town. The district is quite densely populated with a population of about 700,000 covering an area of 1960.2 sq km2. The population is both rural and urban and it's highly cosmopolitan. It is one of the leading industrial districts in the country as well as rich agricultural district. The poverty incidence is reported at 48.8%. The form of poverty include food and absolute poverty and are on the increase due to factors such as unemployment, collapse of agricultural centers and industries, poor infrastructure and the rise of HIV/AIDS. The total population at risk of developing cervical cancer is approximately 300,000 as per the population projections for 2010 [16].

Women who live in this region were invited through churches, posters placed at markets places and health centers to participate in the study. Sensitization meetings were held with the local doctors, public health officers, church leaders and local administration officers and this team was used to inform and request the communities to participate in the study. We also put up posters in the local shopping centers and local health centers and colleges. The local administration officers through the public health office were also requested to ask

the community to attend the screening and participate in the study.

Women were eligible for the study if they were (a) self identified as residing in the region; (b) were ages 18 and above (c) had an intact uterus an no current referral for a hysterectomy (d) Did not report the use of vaginal medication for the previous two days (e) did not report treatment for cervical disease for the previous 6 months and were not pregnant at the time of study. Ethical approval was obtained from the National Ethical Review Committee at the Kenya Medical Research Institute. The moderators explained to the participants the purpose of the research, detailed explanation of the procedures involved and assured them of confidentiality. Those who agreed to participate signed the informed consent forms. All the clinical examinations were conducted by female nurses and privacy was observed in all cases. Those who were found to have any symptoms of vaginal infection during the cervical examination and sample collection were referred to clinical officer in the reproductive health clinic for further examination and treatment. A semi structured questionnaire collected information on sociodemographic characteristics, reproductive history and some lifestyle factors.

Sample collection

Cervical cells were collected from the participants during the gynecology examination. The samples were collected using the ThinPrep® Pap test Kit as per the manufacturer's instruction and stored in the PreservCyt solution vial (Hologic, Bedford, USA). Once the samples were collected they were all transported in room temperature to the institute of pathology, university of Heidelberg for analysis.

High risk Human papillomavirus detection

After processing for cytology, residual ThinPrep® liquid based cytology samples four mls of the sample was used for HR HPV detection for all the samples. The HR HPV detection was carried out using the Digene Hybrid capture 2® (hc2) test according to the manufacturer's instructions as described previously [17]. A positive hc2 result was defined as RLU/Co $\geq$ 1, according to the manufacturer's criteria. Digene Hybrid capture 2® (hc2) test detects HR HPV types 16/18/31/33/35/39/45/51/52/56/58/59/68 and 5 low-risk types 6/11/42/43/44.

Human papillomavirus Polymerase chain reaction (PCR) and genotyping

HPV genotyping was performed on all samples that were positive in Digene Hybrid capture 2® (hc2) test. DNA was extracted from 2 ml of the PreservCyt sample solution using the Genfind™ DNA extraction kit as per the manufacturer's instructions. All reagents were equilibrated to room temperature prior to use and all prepared reaction mixtures were not stored for later use.10 mM Tris, pH 7.5, solution from a 2M Tris stock solution was prepared. The Lysis Buffer and Proteinase K (96 µg/mL) in an appropriate-sized conical tube was combined as per number of samples and mix by pipetting up and down. The extracted DNA was stored at -20° C.

HPV genotyping was done using Multiplex. A 10 µl volume of extracted DNA was added to an equal volume of reaction mixture containing 2µl primer set 1, 0.5µl primer set 2, 5µl PCR gold buffer, 7µl of 25Mm MgCL2, 7µl DNTP mix (10mM each) and 0.2µl DNA polymerase (5 U/ µl). PCR grade water was added to 40 µl per sample. The PCR tubes were then placed in a thermocycler with the following protocol: 94°C for 15 minutes, (94°C for 20 seconds, 38°C for 40 seconds, 71°C for 80 seconds) * 40, and 71°C for 4 minutes.

Genotyping was performed using a Multiplex HPV genotyping kit (Multimetrix, Progen, Germany) as per the manufacturer's instructions. Each 200ng DNA was subjected to PCR using HPV consensus primers for amplification of the HPV L1 gene. Amplified DNA was genotyped by applying a bead based hybridization with HPV type-specific probes using the Luminex technology, allowing for typing of the high risk types HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, 68, 70, 73, and 82. PCR primers and Luminex reagents are both used from the Multimetrix HPV genotyping kit (Progen, Heidelberg). Two sets of primers are provided by the manufacturer. Primer set 1 contained all HPV primers: 9 forward and 3 biotinylated reverse primers necessary to amplify the HPV types under investigation. Primer set 2 (DNA quality control primers) contained primers for the amplification of a β-globin gene fragment which were included in each run to ensure sufficient DNA integrity. After resuspending the beads, the read-out in the Luminex analyzer was performed. HPV types are discerned according to the unique bead signature, whereas the presence of PCR products is determined by phycoerythrin fluorescence. An analytical

sensitivity cut-off was calculated based on the negative control.

Cytology

All cytology was read independently of the HPV results at the Institute of Pathology, Mannheim. Slides were read according to the routine laboratory protocol and reported as such. National guidelines were adhered to, thus high grade abnormalities were referred for colposcopy and biopsy. In women with low grade abnormalities cytology was to be repeated at 6 months.

Data analysis

All the data collected was double entered into a computer database designed using MS-Access application. Back up was performed regularly to avoid any loss or tampering of data. Data cleaning and validation was performed to achieve a clean set that was exported to a Statistical Package for Social Sciences (SPSS) 12.0 file. Hard cover books were used to document any vital information collected and observed during the study period. All the filled questionnaires and tapes were arranged in box files and properly stored in lockable drawers for confidentiality.Univariate analysis was performed. All variables were subjected to descriptive data analysis. Descriptive statistics such as median, minimum, and maximum was used to summarize continuous variables while categorical variables was summarized using proportions

Results

Characteristics of the study population

A total of 498 women met the eligibility criteria. Mean age of the study participants was 36 $\pm$ 10 ranging between 18 and 74 years. Distribution of age among the participants revealed comparable proportions in every category. The highest proportion seen in those aged 30 - 34 years (18.1%; 90) followed by 35 - 39 years (17.5%; 87). The lowest proportion observed (9.2%; 46) was 45 - 49 years old. The mean number of full term births for those that had given birth to at least one child was 3 $\pm$ 1 ranging between 0 and 10 children. The highest proportion (62.2%; 310) of the participants had 1 – 3 children with 18.9% (95) having more than three. A significant majority (66.9%; 333) of the study participants was single. Level of education varied between none and university education, with a very small proportion (1.0%; 5) having no formal education. The highest proportion of participants (37.1%; 185) had secondary education. Source of income among the study participants was characterized by high self employment (39.4%; 196) and formal employment (30.9%; 154). Level of unemployment was relatively high (23.3%; 116) while 5.2% (26) of the participants being students/housewife. Six of the participants (1.2%) declined to mention their source of income.

A total of 498 women were tested using Digene (hc2) procedure at recruitment. Samples from 106 women (21.3%) tested positive for HPV. Out of all the samples that were analyzed 22 were classified as HC2-positive but HPV negative. The remaining 103 HC2-positive samples (82.4%) were positive for one or more of the 24 HPV types. Multiple HPV types were detected in 40 (37.7% of HC2-positive samples) and infection with single HPV type was detected in 63 (59.4%) of the HC2-positive samples. Most women had normal cytology (89%), (3%) had LSIL, (5.8%) ASCUS and (2.2%) had HSIL.

The prevalence rates for specific HPV type are shown in Table 1, overall and by age group. The most common HPV type at all ages was HPV16 (overall prevalence 4.6%), followed by HPV52 (overall prevalence 3.8%), HPV56 and HPV66 (both 2.4%), HPV18 and HPV35 (both 2.0%), HPV51 (1.8%), HPV42, HPV68 and HPV73 (all three at 1.6% each), HPV6 and HPV45 (both1.4%), HPV11 and HPV59 (both 1.2%), HPV31 and HPV53 (both 1.0%). There was a marked decline in the prevalence of HPV with age, both overall (28.1% below age 30 years and 17.4% at 30 years or above) (Table 1).

A total of 498 women were tested using Digene (HC2) procedure at recruitment. Samples from 106 women (21.3%) tested positive for HPV. Out of all the samples that were analyzed 22 were classified as HC2-positive but HR-HPV negative. The remaining 103 HC2-positive samples (82.4%) were positive for one or more of the 24 HR types. Multiple HPV types were detected in 40 (37.7% of hc2-positive samples) and infection with single HPV type was detected in 63 (59.4%) of the hc2-positive samples. Most women had normal cytology 443 (89%), 15 (3%) had LSIL, 28 (5.8%) ASCUS and 11 (2.2%) had HSIL.

The prevalence rates for specific HPV type are shown in Table 1, overall and by age group. The most common HPV type at all ages was HPV16 (overall prevalence 4.6%), followed by HPV52 (overall prevalence 3.8%), HPV56 and HPV66 (both 2.4%), HPV18 and HPV35 (both 2.0%), HPV51 (1.8%), HPV42, HPV68 and HPV73 (all

three at 1.6% each), HPV6 and HPV45 (both1.4%), HPV11 and HPV59 (both 1.2%), HPV31 and HPV53 (both 1.0%).

There was a marked decline in the prevalence of HPV with age, both overall (28.1% below age 30 years and 17.4% at 30 years or above) (Table 1). Similarly, there was a marked decline in the prevalence of HPV with CIN status, both overall (81.8% of women who tested positive for any type of CIN and 19.3% of those who tested negative i.e. normal) and for each HPV type (Table 2).

The overall HPV prevalence in women with normal cytology was 78(17.6%), ASCUS 5(10.3%), LSIL 11(73.3%) and with HSIL 9(81.3%) as shown in Table 2. HPV 16 was most common amongst the ASCUS and HSIL cytology groups. In LSIL cytology group HPV 42, 52 and 56 were the most prevalent while in those with normal cytology, HPV 52, 16 and 66 were the most common (Table 2).

Table 1: Prevalence of HR-HPV overall and as a proportion of HR-positive women by age group

	<= 29 years			30 - 39 years			40 - 49 years			50 or more years			All ages		
HR-HPV type	n	% of all women	% of HR-HPV + women	n	% of all women	% of HR-HPV + women	n	% of all women	% of HR-HPV + women	n	% of all women	% of HR-HPV + women	n	% of all women	% of HR-HPV + women
6	5	3.3	11.6	2	1.1	5.6	0	0.0	0.0	0	0.0	0.0	7	1.4	6.8
11	3	2.0	7.0	1	0.6	2.8	2	1.7	12.5	0	0.0	0.0	6	1.2	5.8
16	9	5.9	20.9	9	5.1	25.0	2	1.7	12.5	3	5.8	37.5	23	4.6	22.3
18	3	2.0	7.0	3	1.7	8.3	2	1.7	12.5	2	3.8	25.0	10	2.0	9.7
26	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
31	2	1.3	4.7	2	1.1	5.6	1	0.9	6.3	0	0.0	0.0	5	1.0	4.9
33	0	0.0	0.0	1	0.6	2.8	0	0.0	0.0	0	0.0	0.0	1	0.2	1.0
35	5	3.3	11.6	4	2.3	11.1	1	0.9	6.3	0	0.0	0.0	10	2.0	9.7
39	1	0.7	2.3	1	0.6	2.8	0	0.0	0.0	1	1.9	12.5	3	0.6	2.9
42	4	2.6	9.3	3	1.7	8.3	1	0.9	6.3	0	0.0	0.0	8	1.6	7.8
43	2	1.3	4.7	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	0.4	1.9
44	1	0.7	2.3	2	1.1	5.6	1	0.9	6.3	0	0.0	0.0	4	0.8	3.9
45	2	1.3	4.7	3	1.7	8.3	2	1.7	12.5	0	0.0	0.0	7	1.4	6.8
51	2	1.3	4.7	6	3.4	16.7	0	0.0	0.0	1	1.9	12.5	9	1.8	8.7
52	11	7.2	25.6	5	2.8	13.9	3	2.6	18.8	0	0.0	0.0	19	3.8	18.4
53	2	1.3	4.7	2	1.1	5.6	1	0.9	6.3	0	0.0	0.0	5	1.0	4.9
56	7	4.6	16.3	3	1.7	8.3	2	1.7	12.5	0	0.0	0.0	12	2.4	11.7
58	1	0.7	2.3	0	0.0	0.0	0	0.0	0.0	1	1.9	12.5	2	0.4	1.9
59	1	0.7	2.3	4	2.3	11.1	1	0.9	6.3	0	0.0	0.0	6	1.2	5.8
66	8	5.2	18.6	2	1.1	5.6	2	1.7	12.5	0	0.0	0.0	12	2.4	11.7
68	3	2.0	7.0	2	1.1	5.6	2	1.7	12.5	1	1.9	12.5	8	1.6	7.8
70	1	0.7	2.3	1	0.6	2.8	1	0.9	6.3	0	0.0	0.0	3	0.6	2.9
73	4	2.6	9.3	4	2.3	11.1	0	0.0	0.0	0	0.0	0.0	8	1.6	7.8
82	0	0.0	0.0	2	1.1	5.6	2	1.7	12.5	0	0.0	0.0	4	0.8	3.9
16 and/or 18	12	7.8	27.9	10	5.6	27.8	4	3.4	25.0	5	9.6	62.5	31	6.2	30.1
Any HR-HPV	43	28.1	100	36	20.3	100	16	13.8	100	8	15.4	100	103	20.7	100
HC2+ No HR-HPV	11	7.2	-	7	4.0	-	3	2.6	-	1	1.9	-	22	4.4	-
HC2 -	110	71.9	-	139	78.5	-	98	84.5	-	45	86.5	-	392	78.7	-
All women	153	100	-	177	100	-	116	100	-	52	100	-	498	100	-

HR-HPV – High-risk human papillomavirus

Table 2: Prevalence of HR-HPV overall and as a proportion of HR-positive women by Pap smear

HR-HPV type	ASCUS			LSIL			HSIL			Normal			All Pap test Diagnosis		
	n	% of all women	% of all HR-HPV+ women	n	% of all women	% of all HR-HPV+ women	n	% of all women	% of all HR-HPV+ women	n	% of all women	% of all HR-HPV+ women	n	% of all women	% of all HR-HPV+ women
6	0	0.0	0.0	1	6.7	9.1	0	0.0	0.0	6	1.4	7.7	7	1.4	6.8
11	0	0.0	0.0	1	6.7	9.1	1	9.1	11.1	4	0.9	5.1	6	1.2	5.8
16	2	6.9	40.0	2	13.3	18.2	6	54.5	66.7	13	2.9	16.7	23	4.6	22.3
18	1	3.4	20.0	1	6.7	9.1	1	9.1	11.1	7	1.6	9.0	10	2.0	9.7
26	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
31	1	3.4	20.0	1	6.7	9.1	0	0.0	0.0	3	0.7	3.8	5	1.0	4.9
33	0	0.0	0.0	1	6.7	9.1	0	0.0	0.0	0	0.0	0.0	1	0.2	1.0
35	1	3.4	20.0	1	6.7	9.1	1	9.1	11.1	7	1.6	9.0	10	2.0	9.7
39	1	3.4	20.0	0	0.0	0.0	0	0.0	0.0	2	0.5	2.6	3	0.6	2.9
42	0	0.0	0.0	3	20.0	27.3	2	18.2	22.2	3	0.7	3.8	8	1.6	7.8
43	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	0.5	2.6	2	0.4	1.9
44	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	4	0.9	5.1	4	0.8	3.9
45	0	0.0	0.0	2	13.3	18.2	1	9.1	11.1	4	0.9	5.1	7	1.4	6.8
51	1	3.4	20.0	1	6.7	9.1	2	18.2	22.2	5	1.1	6.4	9	1.8	8.7
52	0	0.0	0.0	3	20.0	27.3	2	18.2	22.2	14	3.2	17.9	19	3.8	18.4
53	0	0.0	0.0	1	6.7	9.1	1	9.1	11.1	3	0.7	3.8	5	1.0	4.9
56	0	0.0	0.0	3	20.0	27.3	1	9.1	11.1	8	1.8	10.3	12	2.4	11.7
58	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	2	0.5	2.6	2	0.4	1.9
59	0	0.0	0.0	0	0.0	0.0	2	18.2	22.2	4	0.9	5.1	6	1.2	5.8
66	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	12	2.7	15.4	12	2.4	11.7
68	0	0.0	0.0	1	6.7	9.1	0	0.0	0.0	7	1.6	9.0	8	1.6	7.8
70	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	3	0.7	3.8	3	0.6	2.9
73	0	0.0	0.0	0	0.0	0.0	1	9.1	11.1	7	1.6	9.0	8	1.6	7.8
82	0	0.0	0.0	0	0.0	0.0	1	9.1	11.1	3	0.7	3.8	4	0.8	3.9
16 and/or 18	3	10.3	60.0	3	20.0	27.3	6	54.5	66.7	19	4.3	24.4	31	6.2	30.1
Any HR-HPV	5	17.2	100	11	73.3	100	9	81.8	100	78	17.6	100	103	20.7	100
HC2+ No HR-HPV	1	3.4	-	2	13.3	-	1	9.1	-	18	4.1	-	22	4.4	-
HC2 -	23	79.3	-	3	20.0	-	1	9.1	-	365	82.4	-	392	78.7	-
All women	29	100	-	15	100	-	11	100	-	443	100	-	498	100	-

HR-HPV – High-risk human papillomavirus

This study report describes the prevalence and distribution of HPV types within the general population in a Kenyan population. The data represent a starting point for understanding the burden of HPV infections, which may help to assess the public health impact of HPV DNA genotyping in cervical cancer screening and HPV vaccination among Kenyan women. In our study sample the total HPV prevalence was 21.3% in the general population. Previous prevalence studies of HPV from selected populations have been documented at 14% and 17% ([14], [13]). Elsewhere in the world, the prevalence of HPV types from unselected population has been reported from previous studies as, 21% in Honduras, 7.6% in Costa Rica, and 16% in Thailand ([18], [19], [20].

In this study HPV 16 was the most prevalent (4.6%). This is similar to what is documented world wide [8]. HPV 16 was not the sole predominant type in or study population. The second most prevalent was HPV 52 (3.8%), 56 and 66 (2.4%) and 18 and 35(2.0%). HPV 52, 56 and 66 were more prevalent than HPV 18. This is in contrast with most other studies conducted in both random and selected cervical cancer and pre cancer populations [8]. In general, however, HPV 52 is amongst the least prevalent HR HPV type exciting at rates of 0% in HPV positive women in Thailand, 6% in Philippines and 5% in Paraguay [21].

Available literature indicates that HR HPV 52 has only been reported to be the most or second most prevalent in Nairobi 2003 and Thailand 2000 respectively [22], [23]. It is important to note that we describe these comparisons with caution as we realize that these studies have used different HPV primers that can have different sensitivities for different HPV types. Multiple types' infection was common in our population with a prevalence of 37.7%.

There was a marked decline in the prevalence of HPV with age both overall and for each HR HPV type. This is similar to other findings in Kenya and the region [22], [3]. This supports the fact that about 1 out of 4 women younger than 30 years of age have prevalent HPV infection [24].

Most of the infections regress spontaneously due to natural immune responses that apparently develop during the normal course of these infections. The rate of prevalent infections in women older than 30 years of age thus substantially decreases and ranges somewhere between 5 to 10% [24]. There was also a noted increase on HR HPV prevalence in women who tested positive for any CIN than those who tested normal similar to what is documented world wide [25].

This study provides information on the cervical HPV types infecting a large number of Kenyan women. We feel the women who participated in this study are a representative of those in other settings in Kenya and East Africa. Therefore, the prevalence found reflects an estimate of HPV prevalence and distributions of specific HPV types within women in Kenya.

Conclusion

The study demonstrates that HPV is highly prevalent in this region and confirms the findings of other African studies. The data provided here may help to inform efforts to integrate HPV genotyping into cervical cancer screening and to develop vaccination strategies for this high risk population. It is important to not that the distribution of HPV types varies substantially within regions and the importance of types 52, 56, 66 and 35 has been demonstrated within this study. Therefore, further epidemiological studies are warranted to identify predominant HPV types for sufficient development of efficient HPV vaccines for this region.

Acknowledgement

We greatly acknowledge all the women who participated in the study, the staff at Thika District Hospital, Field assistants, Kiarie K and Ngugi Paul, the Director and staff members at the Department of Applied Tumor virology, university of Heidelberg.

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