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Epidemiology of Mucosal Human Papillomavirus (HPV) Infections Among Adult and Children

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1. Introduction

Human papillomavirus (HPV) infection is recognized today as the main causal factor for ~100% of cervical cancer cases in the world and of a substantial proportion of many other anogenital neoplasms (anal, vaginal, vulvar, and penile cancer). 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 epidemiology of mucosal human papillomavirus (HPV) has been well studied today, especially cervicovaginal HPV infection among young women and there are also more available epidemiologic data for older women, men and as well as for children. HPV infection is the most common sexually transmitted infections in the world. The predominant route of transmission is via sexual contact, although vertical and horizontal transmissions also occur. This chapter will review the epidemiology of mucosal HPV infections affecting genital, oral and conjunctival mucosa in adults and children. This chapter will detail the epidemiology of HPV in adult considering young versus older population rather than focussing on adolescent and adult populations separately because there is no universal cut-off age group to define high risk population as HPV is highly dependant on the onset of sexual activity.

2. Classification and carcinogenicity of HPVs

More than 100 HPV genotypes have been catalogued so far and can be classified according to the phylogenetics in genera and species (De Villiers et al, 2004) (table 1). The L1 protein is highly conserved among all HPV genotypes and is thus used for taxonomical purposes. Different genera of the Papillomaviridae (Alpha, Beta, etc.) share less than 60% nucleotide sequence identity in the L1 protein whereas species within a genus share between 60% and 70% nucleotide identity. A new HPV isolate is recognized as a new genotype when the nucleotide sequence of the L1 gene differs by more than 10% from the genotype with which it has greatest homology in DNA sequence.

Papillomaviruses can also be classified according to their tissue tropism (mucosal or cutaneous) and oncogenic potential (table 2) (De Villiers et al, 2004). Although it is possible

| Genus | Species | Genotypes of HPV |
|----------------------|----------|--|
| Alpha-papillomavirus | Alpha-1 | 42 |
| | Alpha-2 | 3, 10, 28,29, 77, 78, 94 |
| | Alpha-3 | 61, c62, 72, 81, 83, c87, c86, c89, 84, |
| | Alpha-4 | 2, 27, 57 |
| | Alpha-5 | 26, 51, 69, 82 |
| | Alpha-6 | 30, 53, 56, 66 |
| | Alpha-7 | 18, 45, 59, c85, 39, 68, 70 |
| | Alpha-8 | 7, 40, 43, c91 |
| | Alpha-9 | 16, 31, 33, 35, 52, 58, 67 |
| | Alpha-10 | 6, 11, 13, 44, 55, 74 |
| | Alpha-11 | 34, 73 |
| | Alpha-12 | RhPV1 |
| | Alpha-13 | 54 |
| | Alpha-14 | c90 |
| | Alpha-15 | 71 |
| Beta-papillomavirus | Beta-1 | 5, 8, 12, 14, 19, 20, 21, 24, 25, 36, 47, 93 |
| | Beta-2 | 9, 15, 17, 22, 23, 37, 80, |
| | Beta-3 | 38, 49, 75, 76 |
| | Beta-4 | 92 |
| | Beta-5 | 96 |
| Gamma-papillomavirus | Gamma-1 | 4, 65, 95 |
| | Gamma-2 | 50 |
| | Gamma-3 | 48 |
| | Gamma-4 | 60 |
| | Gamma-5 | 88 |
| Mu-papillomavirus | Mu-1 | 1 |
| | Mu-2 | 63 |
| Nu-papillomavirus | Nu | 41 |

Adapted from De Villiers et al, 2004.

Table 1. Phylogenetics of Papillomaviridae affecting humans

to find all these genotypes in both mucosal and cutaneous tissue, they are classified according to their tissue tropism as shown in table 2. This chapter will focus on the genotypes of HPV that infect the epithelial lining of the anogenital tract and other mucosal areas of the body (mucosal HPV). There are over than 40 genotypes of HPV that infect human mucosal from which 13–25 genotypes have been identified as probable or definite high-oncogenic risk (HR-HPV) according to their frequency of association with cervical cancer and other anogenital cancers (review in Trottier et al, 2006; IARC 2007; IARC 2011). Although, the vast majority of infected people will clear mucosal HPV infections without any clinical consequences, its role in the pathogenesis of malignant tumours has been well

described. HR-HPV is recognized unequivocally as the main causal factor for (~100%) cervical cancer, is responsible for a substantial proportion of many other (60-90 %) anogenital neoplasms (anal, vaginal, vulvar and penile cancers), and a non negligible portion (~30%) of head and neck cancers (oral cavity, pharynx, and larynx), and is suspected to play a causal role in many other neoplasms, such as conjunctiva carcinoma and lung cancer (Trottier et al, 2009). The latest classification published by the World Health Organization’s International Agency for Research on Cancer (IARC) referred HPV-16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 as HR-HPVs (IARC, 2011). This classification included also many other genotypes as probably carcinogenic such as HPV genotypes 26, 30, 34, 53, 66, 67, 68, 69, 70, 73, 82, 85, 97. Infection with low-oncogenic risk HPVs (LR-HPV), such as HPV-6 and 11, can cause benign lesions of the anogenital areas known as *Condylomata acuminata* (genital warts), a large proportion of low-grade squamous intraepithelial lesions (LSIL) of the cervix, oral papillomas as well as conjunctival papillomas. HPV-6 or 11 may also cause in rare instance recurrent respiratory papillomatosis, which in infants and young children can be very morbid and usually perinatally transmitted (Armstrong et al. 2000) whereas in adult it is usually sexually transmitted and less severe than in children (Kashima et al, 1992).

| Tissue tropism | Genotypes of HPV |
|----------------|---|
| Mucosal | High-risk: 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 Low-risk (or probably carcinogenic): 6, 11, 13, 26, 30, 32, 34, 42, 44, 53, 54, 61, 62, 66, 67, 68, 69, 70, 71, 72, 73, 74, 81, 82, 83, 84, 85, 86, 87, 89, 90 |
| Cutaneous | 1, 2, 4, 5, 8, 9, 12 ,1 4, 15 ,17, 19, 20, 21, 22, 23, 25, 27, 36, 37, 38, 41, 47, 48, 49, 50, 57, 60, 63, 65, 75, 76, 80, 88, 92, 93, 95, 96 |
| Both (mixte) | 3, 7, 10, 28, 29, 40, 43, 78, 91, 94 |

Adapted from De Villiers et al, 2004. Carcinogenic potential classification based on IARC monograph, Vol 100B, 2009.

Table 2. Classifications of HPVs according to their tissue tropism and oncogenic potential

3. Mode of transmission of mucosal HPV

3.1 Predominant route of transmission is via sexual contact

There is a strong and consistent association between sexual activity and mucosal HPV infections (Winer et al 2008). The number of lifetime and recent partners is one of the strongest risk factors for prevalent infection as well as acquisition in adult (Trottier et al, 2006). Further data supporting sexual intercourse as the primary route of genital HPV infection include documented transmission of genital warts between sex partners, concordance in sex partners for genotype-specific HPV infection, the rarity of genital HPV infection in virgin women, and increased risk of HPV acquisition following new and recent sex partners (Winer et al, 2008; Winer et al, 2008b; Burchell et al, 2006). The practice of anal intercourse is also associated with HPV detection in the anal canal in men who have sex with men and to a lesser degree for women (Dunne et al, 2006; Moscicki et al, 1999). Transmission may also occur via other sexual practices, such as oral sex, digital-vaginal sex and use of insertive sex toys (Edwards et al, 1998; Sonnex et al, 1999; Gervaz et al, 2003). For example, oral sex may explain why husband of women with cervical cancer are at higher

risk of upper aerodigestive track cancer (Hemminki et al, 2000). Studies on genital HPV infection between women who have sex with women also suggest the possibility of transmission between female sex partners (Marrazzo et al 2000). More studies are also available on the transmissibility of HPV; the evidence is that HPV is highly transmissible (Barnabas et al, 2006; Burchell et al, 2010). For example, Barnabas et al. (2006) estimated the per-partner male-to-female transmission probability as much as 60% for HPV-16.

3.2 Vertical transmission

Non-sexual routes of transmission are believed to be far less common, but possible. Transmission of HPV from mother to child (perinatal infection) was first reported in 1956 (Hajek, 1956) in a case of juvenile laryngeal papillomatosis (JLP). Confirmation of the perinatal transmission of HPV in different mucosa (genital, oral) was subsequently supported by several studies although the route of transmission is not well understood (Syrjänen et al, 2000). Direct maternal transmission during vaginal delivery or at caesarean section following early membrane rupture is possible as well as in utero through semen or ascending infection from mother's genital tract (Syrjänen et al, 2000; Favre et al, 1998). Transplacental transmission is also possible since HPV DNA has been detected by PCR in amniotic fluid of HPV positive pregnant women. HPV 16 DNA has also been found in cord blood cells. See section on children for an estimate of the probability of transmission of HPV at birth.

3.3 Horizontal transmission and other route

Horizontal transmission had also been reported; possible routes of infection are the fingers and mouth, fomites and skin contact outside sexual contact. For example, transmission from the anogenital region to hands is possible via self-inoculation (Hernandez et al, 2008). Although possible, this non-sexual route of transmission is also believed to be far less common than sexual contact route, especially in adult. Obviously, the horizontal route is more important in children (excluding sexually abused children) than the sexual route although vertical transmission do occurs. For example, the presence of oral HPV DNA is detected in buccal cavities of 19–35% of healthy children aged 6–11 years (Puranen et al, 1997; Summersgill et al, 2001; Kojima et al, 2003). Blood transmission of HPV as well as transmission via breast milk is implausible since HPV infection does not produce viremia (Cason et al, 2005).

4. Epidemiology of HPV infection in adult

4.1 Anogenital HPV infection in women

4.1.1 Prevalence, incidence, duration, co-infection and re-infection

There are many studies that have reported on cervical HPV epidemiology. Studies on the prevalence of HPV around the world show that the prevalence of cervical HPV infection ranges from 2 to 44%. This wide variation in the prevalence estimates is largely explained by the age and the region of the populations studied. Typically, HPV prevalence increases rapidly following the onset of sexual activity (highest prevalence occurs among young women / adolescent). In fact, cervicovaginal HPV infection is rarely observed among

virgins, even among those who engage in sexual activity other than intercourse (Kjaer et al, 2001). The peak after sexual debut is usually followed by an age-related decline in prevalence, and occasionally a second but more modest peak in prevalence among older women (~45 years) (Trottier et al, 2006). The prevalence of cervical HPV infection is estimated at 5.2%, 8.7%, 12.9%, 14.3% and 25.6% in Europe, Asia, North America, South America and Sub-Saharan Africa, respectively (Clifford et al., Lancet 2005, Burchell et al. 2006). HPV-16 is the most common genotype in all regions of the world except in Eastern Africa, Japan and Taiwan, where HPV 52 is the most frequent genotype but overall, the top ranked genotypes are HPVs 16, 18, 31, 58, and 52 (de Sanjosé et al, 2007). Coinfection with multiple HPV genotypes is also a very common finding of many epidemiologic studies. For example, among the cohort of Brazilian women, between 1.9% to 3.2% were co-infected with multiples genotypes at a same visit (concurrently infected) whereas when considering cumulatively (period prevalence) during the first year and the first 4 years of follow-up, 12.3% and 22.3% were infected with multiple genotypes, respectively (Trottier et al, 2006b). We also have to take into account that positivity for HPV is typically higher in cervicovaginal than in exclusively cervical specimens (Bauer et al; 1991).

The incidence of cervical HPV infections has also been well studied in many cohorts of young or college-aged women. These cohort studies have shown that the cumulative incidence of cervical HPV infection exceeded 40% after 3 years among women who were initially HPV negative at enrolment (Trottier et al, 2006). These studies also had shown that the cumulative incidence is higher for high risk genotypes than for low-risk genotypes. As with prevalence, incidence rates of HPV in women tend to decline with age, although second peaks are sometimes observed in older women. In fact, over 75% of sexually active women will contract HPV in their lifetime and its detection is strongly and consistently associated with the number of sexual partners. In most cases, HPV infection is transient or intermittent; only a very small proportion of cervical HPV infection will persist and progress toward cervical cancer (Schiffman et al, 2003). The median duration of cervical infection for any HPV genotype appears to range between 4 and 20 months (Trottier et al, 2006).

Recent evidence shown that re-infection with HPV (with a different or either a same genotype) is a common occurrence (Trottier et al, 2010). Prior infection with HPV does not provide women with adequate immunity against subsequent infections. In fact, serum antibody levels after natural HPV infection when detectable are low and 40-50% of women do not develop measurable antibody response after HPV natural infection (Viscidi et al, 2004; Nonnenmacher et al, 1995; Park et al, 1998; Heim et al, 2002; Skjeldestad et al, 2008). Moreover, it has been shown that an infection with a specific genotype does not decrease the probability of being infected by a phylogenetically-related genotype (Thomas et al, 2000). Recent studies have shown that re-infection with a same genotype, as well as incident infection in older women who had multiple lifetime sexual partners, are associated with new sexual partners suggesting that infection in adult women may results not only from reactivation (infections acquired at a young age that never completely cleared but become undetectable and appeared later in life) but also from new exposure via sexual activity (Trottier et al, 2010; Munoz et al, 2004).

Relatively little is known about the epidemiology of anal HPV infection in women compare to cervical infection. However the few studies that reported on anal HPV infection shown that it is very common (Goodman et al, 2008; 2010; Shvetsov et al, 2009). When both cervical

and anal HPV testing is done, anal HPV is more common than cervical HPV (Williams et al, 1994; Palefsky et al, 2001). More recently, Goodman et al. (2010) reported that cervical and anal HPV infections do occur consecutively and that the risk of one increases the risk of the other and vice versa. They also reported on prevalence, incidence and clearance rates of genotype-specific anal HPV infection in women. The period prevalence of anal HPV was as much as 70% for a follow-up period that averaged 1.3 years (Goodman et al, 2008). The incidence of anal HPV infection was 50% through a follow-up period of average duration of 1.2 years whereas the median duration of anal HPV infection was 150 days (Shvetsov et al, 2009). In sum, data suggest that women's risk of anal HPV infection is at least as common (if not more common) as their risk of cervical HPV infection.

4.2 Anogenital HPV infection in men

4.2.1 Prevalence, incidence, duration

Compared to women, far fewer studies have been conducted among men but evidence suggests that the prevalence may even be more important in male. Depending of the anatomic sites (specifically the glans, corona, prepuce/foreskin and shaft, with improved HPV detection if a scrotal, perianal and/or anal canal sample is also obtained), the prevalence of anogenital HPV-DNA positivity among men ranges from 0 to 73% and is usually more than 20% (Giuliano et al, 2007; 2008; Dunne et al, 2006; Weaver et al, 2004). Also, HR-HPV appears to occur in a higher proportion of male than female infections (Giuliano et al, 2008b) and such as for women, penile HPV prevalence typically increases with the number of sex partners (Giuliano et al, 2007; Dunne et al, 2006; Weaver et al, 2004). Importantly and unlike for women, the available data do not indicate marked differences in HPV prevalence across age groups in men (Giuliano et al, 2008c). In fact, after the onset of sexual activity, HPV prevalence in men is relatively stable across age group.

Some cohort studies revealed that anogenital HPV incidence is at least as high among men as it is in women, with cumulative incidences ranging from 14 to 62% within 3 to 24 months (Dunn et al, 2006; Giuliano et al, 2008, Partridge et al, 2006; 2007). Although far fewer data are available for men, infection seems to be of short duration compare to women with a median duration of 5.9 months and no evidence for a difference in duration between oncogenic and non-oncogenic infections (Giuliano et al, 2008; Kjaer et al, 2005; Lajous et al, 2005). Only one study has reported on the risk of infection with multiple types in male and found that coinfection with multiple HPV genotypes was very common; the cumulative incidence of multiple genotypes after 24 months of follow-up of heterosexually active male university students 18–20 years was 35.6% (Partridge et al, 2007). There is no available study concerning the probability of re-infection with a same or a different genotype in men.

4.2.2 Special case for men who have sex with men

Men who have sex with men (MSM) have been observed to have a particularly high prevalence of HPV infections (Chin-Hong et al, 2004; Palefsky et al, 1998) and especially HIV positive MSM (de Pokomandy et al, 2009). Cohort studies of HIV-positive MSM revealed that the prevalence of anal HPV is more than 95% in these men, with high rates of multiple HPV genotype infections and lower genotype-specific HPV clearance rates (de Pokomandy et al, 2009; Kiviat et al, 1990; Palefsky et al 2005).

4.3 Oral HPV infection in adults

It is clear that oral mucosa act as a reservoir for HPV. A systematic review (Kreimer et al, 2010) of studies on oral HPV infection among 4070 healthy and cancer free individuals estimated the prevalence of oral HPV infection (any genotypes) of 4.5%. More specifically, 1.3% had oral HPV-16 and 3.5% had carcinogenic HPV. In this systematic review oral HPV-16 accounted for 28% of all HPV detected in the oral region and there was no difference in the oral prevalence between men and women. Other recent studies had shown nearly the same prevalence. A study among 1,688 healthy men aged 18 to 74 (median = 31 years) in United States, Mexico, and Brazil shown that the prevalence of oral HPV infection was 4% (Kreimer et al, 2011) whereas the study of Matsushita et al (2011) estimate oral HPV prevalence at 6.1%. Genotypes mostly detected included HPVs -16 and -18 and the tonsil appears to be the site with the highest prevalence.

4.4 Conjunctival HPV infection in adult

It is also clear that conjunctival mucosa act as a reservoir for HPV. LR-HPV such as HPVs -6 and -11 as well as HR-HPV, such as genotypes 16 and 33 are associated with the occurrence of conjunctival papilloma (Sjö et al, 2007). Ateenyi-Agaba et al. (2010) have tested conjunctival biopsy samples from healthy individuals to estimate the prevalence of HPV. The prevalence of mucosal HPVs was 3.5%, the prevalence of cutaneous HPVs was 10.5% whereas the prevalence multiple-genotype infections was present in 13.3% (Ateenyi-Agaba et al, 2010). It is possible that conjunctival tissues are more likely to be infected with cutaneous genotypes because of horizontal transmission.

5. Epidemiology of HPV infection in infant and children

The few studies available on genital and oral HPV infection in children have shown clear evidence of HR-HPV infections in healthy children (Kojima et al, 2003; Summersgill et al, 2001; Smith et al, 2007; Rice et al, 2000; Syrjänen et al, 2000). Sexual abuse and vertical and horizontal transmission may explain the positivity in children. This chapter focuses on the evidence about the epidemiology of HPV in children and not on the possible route of transmission since it is impossible to distinguish between both routes based simply on the clinical/epidemiological data. What is well known is that both routes (sexual and non-sexual) are possible. For example, perinatal HPV transmission has been unequivocally identified as a possible cause for the rare disease called Juvenile-Onset Recurrent Respiratory papillomatosis (JORRP) (Wiatrak et al, 2004).

Confirmation of the perinatal transmission of HPV in different mucosa (genital, oral) is supported by several studies. These studies have reported widely varying probability of infection in newborns, with estimates from the first couple of days of life ranging from 4 to 79% among infants born to mothers testing positive for HPV DNA during pregnancy (Table 3). Although perinatal transmission may be observed in baby born by caesarean, it is usually admitted that caesarean decreases the risk of perinatal infection. For example, a significantly higher rate of HPV 16/18 infection was found at birth when infants were delivered vaginally than when infants were delivered by cesarean (51.4% versus 27.3%) (Tseng et al, 1998). A systematic review (Medeiros et al, 2005) reported a higher risk of HPV infection after vaginal delivery than after cesarean section (RR: 1.8; 95%CI: 1.3-2.4). The risk of transmission has also

been identified as increasing with the rupture of membranes; the longer time rupture of membranes occurred before delivery, higher risk of transmission (Tenti et al, 1999).

| Study, year; country | Genotype of HPV | Sample (number of HPV+ pregnant mother) | Transmission rate at birth | Follow-up |
|----------------------------------|---|---|--|-----------|
| Tseng et al, 1998; Taiwan | 16, 18 | 68 | 40 % (buccal and genital) | delivery |
| Puranen et al, 1997; Finland | 6, 11, 16, 18 | 42 | 79% (nasopharyngeal) | delivery |
| Chatterjee et al, 1998; India | 6, 11, 16, 18 | 12 | 42% (buccal) | delivery |
| Tenti et al, 1999; Italy | 6, 11, 16, 18, 31, 33, 35, 39, 51, 54, 56, 58, 59, 66, 68, 69, 70, 83, 84 | 37 | 30% (oropharyngeal) | 18 mths |
| Bandyopadhyay et al, 2003; India | 6, 11, 16, 18, 31, 33 | 38 | 18% (buccal) | 12 mths |
| Smith et al, 2004; United States | 6, 11, 16, 18, 31, 33, 53, 66 | 172 | 4% (buccal, genital) | 6 mths |
| Rintala et al, 2005; Finland | 16, 18, 31, 33, 35, 39, 45, 51, 52, 54, 56, 58 | Include 77 newborns | (15% and 9% of newborns had genital or oral HPV infection, at birth, respectively) | 2 yrs |
| Rombaldi et al, 2009; Brazil | 6/11, 16, 18, 31, 33, 42, 52, 58 | 49 | 20% (buccal, axillary and inguinal regions) | 1 yr |
| Castellsagué et al, 2009; Spain | 6, 11, 16, 18, 31, 33, 39 | 66 | 19.7% (mouth and anogenital exfoliated cells) | 2 yrs |

Table 3: Cohort studies on HPV perinatal transmission

Only a few studies have analysed the probability of persistence among babies born to HPV-infected mothers such as Rombaldi et al (2009) and Watts et al. (1998) who have reported a very low proportion of persistent infection in infants (reported 0% in a 1 and 3-year follow-up study, respectively) whereas some reported very high proportions ranging from 27 to

56% (Fredericks et al, 1993; Kaye et al, 1994; Pakarian et al, 1994; Cason et al, 1995, 2005; Syrjänen et al, 2000). These studies have shown that perinatally acquired HPV can persist for at least 2 years and that HPV is mostly prevalent during the first year of infancy reaching a peak at 6 months of age.

Anogenital warts may also be transmitted perinatally (Jayasingue et al, 2006; Sinal et al, 2005; Marcoux et al, 2006; Sinclair et al, 2005; Jones et al, 2007). Boyd (1990) has shown that at least 20% of anogenital warts occur because of perinatal transmission. Although the incubation period for children is not known, a period of several months typically elapses between viral infection at delivery and clinical manifestations (Monk et al, 2007). A review of studies of the HPV genotype distribution in anogenital warts in children has shown that 75% are caused by genotypes 6 and 11, 11% by HPV-2, 6% by HPV-16 and -18 and 3% by HPV-27 and -57 (Syrjänen et al, 2000; Sinal et al, 2005; Marcoux et al, 2006; Aguilera-Barrantes et al, 2007). Since the 1990s, the incidence of anogenital warts has dramatically increased in adults as well as in children (Syrjänen et al, 2000). To summarize, it is clear that perinatal transmission of HPV occurred although the frequency at which it occurred and persist remains controversial.

It is also clear that infants and children might acquire oral and genital HPV infection postnatally from a variety of sources such as direct transmission (person-to-person or auto-inoculation), indirect transmission (via contaminated objects) and sexual abuse (Syrjänen et al, 2000). For example, in their longitudinal study of the prevalence of HR-HPV in oral and genital mucosa of infants during their first 3 years of life, Rintala et al. (2005b) found that 42% of infants (negative at birth) had acquired an oral HR-HPV infection (from which 10% had persistent infection) and 36% had acquired a genital HR-HPV infection (from which 1.5% had persisted). Some cross-sectional studies have estimated the prevalence of HPV among children of different age-groups. The estimates of prevalence of HPV (detected by PCR) in oral swabs from children aged 0-13 years range from 32 to 52% (Rice et al, 2000; Syrjänen et al, 2000). In their review of studies that analyzed PCR-detected HR-HPV during infancy and childhood, Cason et al. (2005) reported HPV prevalences ranging from 9 to 55%. However, oral HPV infection is likely to decrease with age. The study of Marais et al. (2006) had compared oral HPV prevalence among children, adolescent and adult. They found that oral HPV infection was highest in children (7.9%), followed by adolescents (5.1%), and lowest in normal adults (3.5%). Mamas et al. (2011) also shown the presence of HPV in the lower respiratory tract of healthy children; 8% of bronchoalveolar lavage of children (2-12 years old) they tested were positive for HPV. Finally, there are no available studies on the prevalence of conjunctival HPV in children although papilloma represents 7-10% of conjunctival benign tumors in childhood where HPV-6 and -11 are the major genotypes responsible for conjunctival lesions (Okan et al, 2010).

There are some studies that reported cases of squamous cell carcinoma (SCC) involving the larynx/lung in childhood which transformed from the recurrent respiratory papillomatosis with HPV (Lin et al, 2010; Katsenos et al, 2011) but cancers associated with HPV in childhood are not frequent. However, although rare, cancers related to HPV are increasing in recent years in children and this increasing correlates with increased prevalence of HPV in the community (Chow et al, 2007). Moreover and importantly, there are no longitudinal studies available to clarify whether children exposed to HPV (oral, anogenital or conjunctival) are at risk of developing carcinoma in adulthood. A better understanding of the natural history of HPV infection in children is clearly needed.

6. Conclusion

Anogenital HPV infection is very common with high prevalences found in both females and males. Typically, anogenital HPV prevalence increases rapidly in adolescents/young adults following sexual debut, and the highest prevalence occurs among this population (Kjaer et al, 2001). The probability of finding a cervicovaginal HPV infections in women decrease after that according to age with a possible peak at older age whereas it is relatively stable according to age for men. The predominant route of HPV transmission is through sexual contact although vertical and horizontal transmissions are possible. Most sexually-active individuals are likely to be exposed to anogenital HPV infection during their lifetimes and most infections will be cleared spontaneously within a year. A small fraction of people will have persistent infection with HR-HPVs, which is unequivocally established as a necessary cause of cervical cancer and is likely to be responsible for a substantial proportion of other anogenital neoplasms and non negligible number of head and neck cancers. Persistent infection result in substantial morbidity and invoke high costs associated with the treatment of clinically relevant lesions. Oral and conjunctival infections may also exist in adults although the epidemiology of these infections has been less studied. Children are also exposed to HPV as anogenital and oral samples of healthy children have often been found positive. The incidence of HPV-associated diseases, such as squamous cell carcinomas, has increased in children in recent years which may be, in part, related to an increase in HPV prevalence (Syrjänen et al, 2000; Chow et al, 2007). HPV vaccination, one of the most remarkable discoveries of the past decade, is currently implanted all around the world and is expected to prevent a substantial proportion of cervical and other HPV-related cancers in the future.

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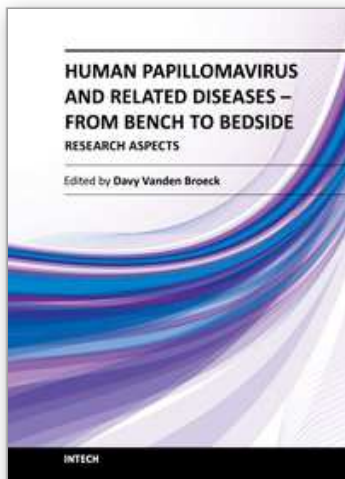
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Cervical cancer is the second most prevalent cancer among women worldwide, and infection with Human Papilloma Virus (HPV) has been identified as the causal agent for this condition. The natural history of cervical cancer is characterized by slow disease progression, rendering the condition, in essence, preventable and even treatable when diagnosed in early stages. Pap smear and the recently introduced prophylactic vaccines are the most prominent prevention options, but despite the availability of these primary and secondary screening tools, the global burden of disease is unfortunately still very high. This book will focus on epidemiological and fundamental research aspects in the area of HPV, and it will update those working in this fast-progressing field with the latest information.

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