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Association of Human Papillomavirus with Cervical Dysplasia and Carcinoma in Both Non-Pregnant and Pregnant Women

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Opinion Article

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DESCRIPTION

Papillomaviruses are icosahedral, non-enveloped, tiny (55 nm in diameter), double-stranded viruses with a DNA genome that is 8000 base pairs long. They cause cutaneous and mucosal tumours in all higher vertebrates, primarily in mammals and birds. Only humans and squamous stratified epithelia may harbour the over 100 different genotypes of the Human Papillomavirus (HPV). They can cause benign or malignant tumours.

For the infections and diseases they cause in the anogenital tract, a subset of about 40 of these viruses has taken on a significant medical and public health significance. The causal relationship between HPV and cancer is best demonstrated by the case of cervical cancer. Effective vaccines to prevent this disease and others have been available since 2006, which has been a significant advancement in public health whose breadth of application is anticipated to expand.

Although the Human Papillomavirus (HPV) is highly linked to cervical dysplasia and cancer in both pregnant and non-pregnant women, there is currently no conclusive evidence that pregnancy and HPV prevalence are significantly correlated. When the endocervical epithelium erupts, it is exposed to the acidic environment of the vagina, which leads to a significant amount of squamous metaplasia. Because HPV needs active cellular machinery to replicate and convert cells, this metaplasia is significant.

Schneider and colleagues used Southern blot hybridization to check for HPV DNA in the negative cervical smears of 92 pregnant and 96 non-pregnant, age-matched control subjects. The researchers found that there was both a higher incidence of HPV (predominantly the oncolytic HPV subtype 16) and a higher rate of viral DNA replication during pregnancy. Smith and colleagues discovered an increase in HPV prevalence with rising gestational age using the ViraPap/ViraType dot blot DNA hybridization method, indicating that pregnant women may be more susceptible to HPV infection as oestrogen levels rise. However, Kemp and associates and Chang-Claude and associates were unable to show a greater frequency of HPV infection during pregnancy using comparable hybridization techniques.

A prospective study was carried out in Barcelona by Castells ague and associates to measure the mother-to-child transmission of HPV subtypes. Pregnant mothers and their offspring who were 66 HPV-positive and 77 HPV-negative were included in this study. The researchers also conducted a similar screening study for the detection of cervical HPV DNA among 828 pregnant women in order to evaluate HPV prevalence and genotypic distribution in pregnancy. Exfoliated cells were obtained from the newborns' mouths and external genitalia at birth and at various intervals up to 2 years of age. At 418 newborn visits and a mean follow-up period of 14 months, 19.7% of infants born to mothers who tested positive for HPV and 16.9% of infants born to mothers who tested negative for HPV had tested positive for the virus at some point.

HPV-16 was the genotype that was most commonly found in both moms and neonates. Notably, there was a strong and statistically significant correlation between mother's and child's HPV status at the 6-week postpartum visit, with children of mothers who had the virus at the time of the visit being five times more likely to test positive for the virus than children of corresponding HPV-negative mothers (P=0.02). Hence it came to the conclusion that there is a low risk of vertical transmission of HPV genotypes and that other factors may contribute to infant HPV infections as well as vertical transmission. The possibility of horizontal mother-to-child transfer exists.