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ORIGINAL ARTICLE

# Genotype-specific prevalence and distribution of human papillomavirus genotypes in underserved Latino women with abnormal Papanicolaou tests

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**KEYWORDS**
Human papillomavirus (HPV);
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HPV distribution;
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Underserved population

**Introduction** Knowledge about the prevalence and distribution of human papillomavirus (HPV) genotypes in cervical premalignant and malignant lesions is crucial to guide development of clinical management strategies and prophylactic vaccines. The aim of this study was to determine HPV genotype-specific prevalence and distribution in an underserved cohort of Latino women.

**Materials and methods** From December 2009 to April 2011, 808 SurePath cervicovaginal specimens were collected from women who were referred from charity clinics for abnormal Papanicolaou tests. The patients' average age was 36.5 years (range 19-85 years). The specimens were tested for HPV genotypes by DNA microarray and sequencing assays.

**Results** The HPV infection rate was extremely high (93% for any HPV and 64% for high-risk [HR]-HPV), with frequent multiple-strain infection (39%). Younger age (<30 years) was associated with frequent HR-HPV infection, multiple strain infections, and cytologic abnormalities. When compared with previous reports, HPV 16 remained the most common genotype (44.6%) in women with high-grade squamous intraepithelial lesion; however, a significant increase in HPV 31 (17.9%) and 45 (10.7%) and a decrease in HPV 35, 52, 33, and 66 were observed.

**Conclusions** The HPV genotype-specific prevalence and distribution pattern in this cohort of underserved Latino women differed significantly from previously published data in the United States.

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Understanding the potentially changing trends in HPV distribution pattern will help guide the development of appropriate preventive and therapeutic strategies for both underserved and general populations.
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## Introduction

Human papillomavirus (HPV) infection is common in the United States. A recent survey documented a 42.5% overall prevalence of anogenital HPV infection in females aged 14 to 59 years.[1] The majority of HPV infections are transient and do not lead to high-grade dysplasia or cancer. Only 3% to 10% of HPV-infected women will fail to clear the viral infection and become persistent HPV carriers, constituting a high-risk group for progression to cervical cancer.[2]

The common anogenital HPV genotypes include more than 40 closely related species that are conventionally categorized as high-risk (HR) or low-risk (LR) HPV based on current understanding of their carcinogenic potential.[3,4] Of these HR-HPV genotypes, HPV 16 and 18 are responsible for more than 70% of all cases of cervical cancer worldwide.[5] On the other hand, the LR-HPV genotypes, led by HPV 6 and 11, are causally related to about 90% of anogenital warts and essentially all cases of recurrent respiratory papillomatosis.[6] These data were the cornerstone in formulating the current strategy on HPV vaccination, which includes the most common disease-related HPV genotypes.[7]

Geographic and ethnic variation of HPV infection is well recognized.[8-11] Worldwide, the most prevalent anogenital HPV genotype is HPV 16, followed by HPV 18 and 31.[8] Notably, there are different levels of disease risk associated with specific HPV types, and persistent infection with certain carcinogenic types is a strong predictor of HPV-related cancers. Considering the dynamic nature of the world population today due to ease of travel and relatively porous borders, it would not be surprising to see changes in the prevalence and distribution of HPV genotypes in a given population. For example, a recent study from Hong Kong showed that the proportion of HPV 16−positive cervical squamous cell carcinoma significantly increased over the past 35 years, from 45.2% in the 1972 to 1973 period to 58.8% in the 1984 to 1986 period, and to 61.2% in the 1997 to 2007 period. Within the same period, HPV 52−positive cervical squamous cell carcinoma decreased from 30.1% to 14.7%.[12]

The Latino population in the United States has grown substantially in the last decade, now surpassing 50 million.[13] In the early 1990s, it was determined that approximately one-third of Latinos in the United States did not have health insurance, and those Latinos of low income were among the least likely, when compared with all other groups, to have a regular source of primary care.[14] Based on the most recent US census, the Latino population in Harris County, where Houston is located, grew by nearly 50% to 1.7 million in the past decade.[13] Recent demographic changes may cause a considerable shift in prevalence and distribution of HPV genotypes in the United States, especially in regions with a significant demographic change. Currently, large-scale studies on anogenital HPV infection have not been done in this particular population, and little is known about the differences in HPV genotype-specific prevalence and distribution compared with the general population in the United States. As a result, there are insufficient data at the current time to predict the future trend of anogenital HPV infection and to estimate the possible impact of the demographic change on cervical cancer prevention and treatment in the postvaccination era. This information is critical in formulating future strategies for cervical cancer outreach education, screening, vaccination, and treatment in affected regions.

## Materials and methods

### Study population and cytology classification

The study was conducted with approval from the Institutional Review Board of The Methodist Hospital Research Institute (IRB1210-0221). The study included 808 women who were referred to our hospital from 84 charity clinics in the greater Houston region for abnormal Papanicolaou (Pap) tests from December 1, 2009 to April 30, 2011. The women were almost exclusively Latino who had no medical insurance and did not qualify for Medicare or Medicaid programs at the time of the study. Liquid-based Pap tests (SurePath, BD, Franklin Lakes, N J) were performed during evaluation, and the findings were interpreted according to the criteria set by the 2001 Bethesda System[15] with minimal modification to include an additional category of low-grade squamous intraepithelial lesion-cannot exclude, high-grade squamous intraepithelial lesion (LSIL-H).

### HPV genotyping by DNA microarray and DNA sequencing

HPV DNA was extracted from the residual SurePath specimens and amplified with polymerase chain reaction in the L1 region of the HPV genome before being labeled with Cy5 and hybridized with a HPV DNA microarray chip with 40 HPV genotypes (GG HPV DNA Genotyping Chip Kit, GoodGene Inc., Seoul, Korea). The signal was visualized using a GenePix 4000B Microarray Scanner (Molecular Devices, Inc., Sunnyvale, Calif). Direct DNA sequencing was also performed in all samples. The sequence data obtained by automated DNA sequencing were analyzed for

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