Cancer Prevention: HPV Vaccination

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Objectives: *To provide an overview of human papillomavirus (HPV) vaccination as cancer prevention with current strategies that nurses can use to help patients and parents overcome barriers to HPV vaccination.*

Data Sources: *Peer-reviewed literature, presentation abstracts, and current immunization recommendations from the Advisory Council on Immunization Practice.*

Conclusion: *Nurses can help prevent cancer by encouraging HPV vaccination during routine immunization and make HPV vaccination normal and routine.*

Implications for Nursing Practice: *A vaccine to reduce/eliminate HPV-related cancers enables nurses' at all educational levels to advocate for cancer prevention through initiation and completion of the HPV vaccine series.*

Key Words: *cancer, prevention, nursing practice, HPV vaccination, oncology nursing, human papillomavirus.*

A number such as 3.7 billion dollars can be terrifying, and rightly so – but this gigantic number is the health care costs of cervical cancer. In addition, a staggering $252 million is spent on a yearly basis to manage and treat human papillomavirus (HPV)-related cancers and infections endured by both women and men.[1,2] HPV infections are associated with large percentages of various types of cancer: 96% to 99% of cervical cancers, 90% to 93% of anal cancers, 12% to 63% of oropharyngeal cancers, 36% to 40% of penile cancers, 40% of vaginal cancers,[3-6] and 40% to 51% of vulvar cancers.[3-7]

The two most common cancer-causing HPV DNA subtypes are types 16 and 18 and their distribution varies substantially, constituting approximately 79% of carcinomas in North America and 68% of carcinomas in Africa.[8] The pervasiveness of these kinds of infections in US males over the age of 19 living in high-risk populations is thought to be approximately 65% to 93%, with the nationwide prevalence for women estimated to be about 45% for ages 14 to 59 as of the year 2015.[9]

Pathophysiological Mechanisms of HPV Infection and Vaccine-Induced Protection Against Cancer

As a result of its unique mechanisms of infection, contact with high-risk HPV types 16 and 18

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most often lead to cancer development.[10,11] These papillomaviruses are the only known viral infections that self-initiate, interacting with the cell surface when it attacks. After that, it takes a mere 24 hours for HPV to fully assimilate the basal cell's nuclear DNA with its own. As a result, the transcription and translation of the infectious genetic material leads to the production of viral proteins that process the disease. Types 16 and 18 express proteins (L1 and L2) not found in low-risk HPV types that are diametrically linked to oncogenesis (cancer cell production) and the promotions of invaded skin cell growth.[12-14] L1 and L2 contribute to HPV's "outer cap layer" or "capsid layer" binding to the basal membrane, the basal cells of future hosts, and the base of the tongue and oral pharynx as with cervical cells.[10]

Cervarix (GlaxoSmithKline, Brentford, London) and Gardasil (Merck, Kenilworth, New Jersey, USA) vaccines were used to prompt the recognition of and the antibody protection of a person's natural immunity toward the L1 capsid protein. However, in accordance with the varied expressions of L1, defense is limited by the diminishing amount of cross-type protection from these kinds of vaccines.[15] Cervarix and Gardasil only induce immunity against types 16 and 18 and types 6, 11, 16, and 18, respectively.[16] Their antibody responses plateau 12 to 18 days after injection and decline until the subsequent injection in the series.[17]

With the help of statistical models, it is predicted that the immune response to L1 antibodies have sufficient memory to recognize and therefore respond to future HPV challenges for 32 years, saving boys and men from the ravages of cancer long after the vaccination series is completed.[18] Current evidence even suggests that the immune memory of L1 induced by the vaccine remains strong enough up to 7 years afterward that it can reliably respond to new forms of HPV.[19] Figure 1 demonstrates the difference between noninfectious and infectious HPV.

While 16 and 18 may be the most commonly occurring HPV genotypes, international distribution varies greatly, accounting for 79% of carcinomas in North America and 68% of carcinomas in Africa.[8] As such, a second generation of HPV vaccines, including Gardasil 9 (described below), are currently being developed with the intent of providing broader cross-type immunity, targeting L2 as it is more highly conserved (providing coverage) across HPV types, and most of all to cover more than just HPV types 6, 11, 16, and 18.[15,20]

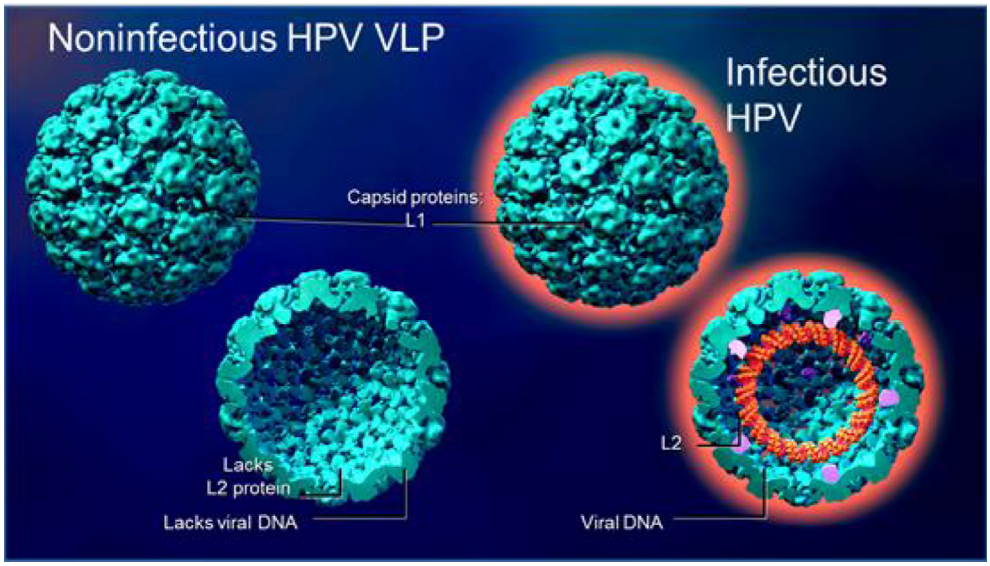

FIGURE 1. Noninfectious HPV VLP (vaccine) versus infectious HPV virus. HPV, human papillomavirus; HPV VLP, HPV virus like particles – resembles HPV but is non-infectious; Viral DNA, deoxyribonucleic acid: a main component of chromosomes and the material that transfers genetic characteristics, in this case the DNA of HPV; L2, L2 protein – protein expressed by HPV subtypes 16 and 18 that are linked to cancer cell production; capsid proteins, outer cap layer of the HPV also known as viral structural proteins which can result in the self-assembly of VPLs. © Timothy S. Baker, Professor, Departments of Chemistry & Biochemistry and Molecular Biology, University of California, San Diego; used with permission.