

Speaker Abstracts

HPV associated cancers on the rise —a growing problem

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Viral infections cause at least 15% of human cancers. One of the most important oncogenic viruses is human papillomavirus (HPV); a causal agent in about 5% of all cancers. HPVs are a large group of viruses that infect both cutaneous and mucosal squamous epithelia and have an exclusively intra-epithelial infectious cycle. About 15 mucosal types are high risk or cancer-causing HPVs, with HPV type 16 and HPV type 18 the most important. Infection with one of these oncogenic HPVs can cause carcinoma of the cervix in women, which is the third most common cancer in women worldwide. Secondary intervention by screening effectively controls this disease in developed countries, but not in the developing world—which bears 86% of the cervical cancer burden. Projections of population growth indicate that without primary or secondary intervention such inequality in disease burden will increase in the coming 3 to 4 decades.

HPV-associated cancers are not confined to the cervix and HPV infection is implicated in the development of vaginal, vulval, anal, penile, and head and neck cancers. Importantly, the incidence of HPV-related cancers in these sites, particularly anal carcinomas and tonsillar carcinomas, is increasing.

Cervical-cancer screening following prophylactic human papillomavirus vaccination

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The recognition that infection with certain types of human papillomavirus (HPV) is a necessary cause of cervical cancer has opened new fronts for the prevention of this disease. Primary prevention is now possible via immunisation with highly efficacious HPV vaccines, and secondary prevention has gained impetus with the advent of sensitive HPV DNA testing to improve traditional cytology-based screening programmes. Although universal vaccination of teenagers and young women is desirable, cost remains a key obstacle. Even with high uptake, a statistically detectable reduction in the burden of cervical cancer via HPV vaccination is unlikely to be observed for at least 10–15 years. To achieve cost-effective reductions in the burden of cervical-cancer prevention, initiatives must consider screening and immunisation as integrated and organised approaches that take advantage of HPV testing as primary screening tests, followed by triage with cytology. On the basis of preliminary findings from the vaccination trials, as successive cohorts of vaccinated young women reach screening age, reduction in cervical lesions will lead to a decrease in rates of colposcopic referral to about 40–60% of the current case loads in most Western countries. These reductions are likely to translate into initial savings for the health-care system, but the vaccine-induced decrease in cervical lesions might lead to a degradation of performance characteristics of cytology because of a decreased rate of abnormalities. The positive predictive value of cytology will decline in vaccinated women because clinically relevant lesions will become less common. A decline in performance will occur because of a decrease in the signal (squamous abnormalities) to noise (inflammation and reactive atypias) ratio that will have a negative impact due to the subjective and tedious nature of reading and interpreting smears.

Another issue is whether screening algorithms can be different for vaccinated versus unvaccinated individuals; this will be particularly important where vaccine coverage is intermediate (30–70%). Knowledge of vaccine status, and registries and other mechanisms are needed if the attendant savings associated with reduced screening are to be realised.