

## New Progress in the Etiology of Cervical Cancer

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**Abstract:** Cervical cancer is one of the common gynecological malignancies, and the incidence rate is the second among female malignant tumors, only after breast cancer, but even in the first place in some developing countries. According to worldwide statistics, there are about 500,000 new cases of cervical cancer each year, accounting for 5% of all new cancer cases, 80% of which occur in developing countries. About 240,000 people die of cervical cancer every year in the world. In recent years, according to some countries and regions, the annual HPV infection rate of young women under 25 years old is 2.3%, of which 0.03% of infected people develop CIN III and cervical invasive cancer, and the younger HPV infection causes the etiology of cervical cancer. Research is especially important.

### 1. Introduction

In recent years, great progress has been made in the study of the etiology of cervical cancer. The etiology of cervical cancer has a long history. As early as the 1940s, an Italian doctor found from the analysis of death registration data that most women with cervical cancer were married women, few were unmarried, and nuns had almost no cervical cancer. In future studies, many risk factors associated with cervical cancer have been discovered. In summary, the risk factors for cervical cancer mainly include the following three aspects:

In recent years, breakthroughs have been made in the biological aspects of cervical cancer. Lavery (1977) observed the presence of human papilloma (HPV) particles in cervical cancer biopsies in electron microscopy, and Zurhausen suggested that HPV may be associated with cervical cancer. Domestic and foreign scholars have studied the relationship between HPV infection and cervical cancer. A large amount of research and many evidences have been obtained. The International Agency for Research on Cancer (IARC) Symposium (1995) clearly stated that HPV infection is the main cause of cervical cancer. 1. Relationship between HPV and cervical cancer: Studies of cervical cancer tissue specimens from around the world have found that HPV 16 and 18 have the highest infection rates. Cuzik et al reported that 94% of patients with cervical cancer had HPV infection, of which HPV16 accounted for 66%. Plummer summarized 10 058 authors: 100034 Peking University First Hospital, Department of Obstetrics and Gynecology, Cervical Cancer, of which 8 550 patients with cervical squamous cell carcinoma, cervical glandular epithelial cell carcinoma and cervical adenosquamous cell carcinoma The number of patients was 1 508. Their results also indicated that HPV16 infection was associated with cervical cancer. They also pointed out that the type of HPV is also related to the pathological type of cervical cancer. HPV16 type accounts for cervical squamous cell carcinoma. 46% to 63%, HPV18 accounted for 10% to 14%; while in cervical glandular epithelial cell carcinoma and cervical adenosquamous carcinoma, HPV18 predominantly, 37% to 41%, while HPV16 accounted for 26%. 36%. In most countries, HPV infection is very common. Among women worldwide, about 10% to 15% of new cases occur each year. Young sexually active women have the highest HPV infection rate, with a peak age of infection between 18 and 28 years. For women with active sexual activity, the lifetime cumulative probability of at least one HPV infection in the cervix is very high, up to 40%. Although most women have a shorter HPV infection period, they usually disappear within 8 to 10 months, but about 10% to 15% of women over the age of 35 have persistent infections. These women who

continue to contract HPV have a higher risk of developing cervical cancer. Many studies have found that the rate of cervical HPV infection decreases significantly with age, and this does not depend on women's sexual behavior, and may be related to immune function restriction or clearance. Epidemiological data combined with laboratory evidence has determined the etiological relationship between HPV and cervical cancer, that is, HPV infection is a necessary condition for cervical cancer. Recently, Manos et al. collected 1,008 cervical cancer biopsy specimens from 22 countries and found that the global HPV detection rate of cervical cancer was as high as 99.7%. This is the highest percentage of human tumor causing factors reported so far, and it is also indicated that HPV infection has a common meaning in relation to cervical cancer. In addition, there is strong evidence of HPV carcinogenesis in cytology and molecular biology.

The relationship between Chlamydia trachomatis infection and cervical cancer: common female sexually transmitted diseases -- Chlamydia trachomatis infection, can significantly increase women's chances of cervical cancer. There are several special types of Chlamydia trachomatis (called serotypes), and it has not previously been known which type of chlamydia is more important in inducing cervical cancer. Tarja Anttila et al. (University of Helsinki) recently published their research at the American Medical Association. Studies have shown that women with cervical cancer are twice as likely to be infected with chlamydia as women without cancer, and the former is twice as likely to be infected with HPV. The researchers also looked for specific serotypes of Chlamydia in 181 patients with invasive cervical cancer. They found that serotype G chlamydia increased the risk of cervical cancer most significantly, increasing risk by 6.6 times, serotype I increasing risk by 3.8 times, serum Type D increases the risk by 2.7 times. Jonathan Zenilman believes that the results of this study add a new dimension to the "effect of chlamydial infection". Checking for chlamydial infection is not only to prevent pelvic inflammatory disease and infertility, but also to prevent potential cervical cancer. Rokita et al performed colposcopy on 1,180 patients and found that Gardnerella vaginalis (GV) plays a major role in vaginal microbial infections. In addition to maternal and child infections in obstetrics, it is a disease of cervical cancer. enhancement. It mainly produces high concentrations of hydride (pH 6.0 or higher), which is clinically characterized as "fishy odor", which "erodes" the cervical mucosa and causes mucosal lesions. Our study shows that the use of spermidine and anaerobic bacteria alone can not cause malignancy in human embryonic cervical cancer cells infected with HPVE6E7, and the synergistic effect of the two can induce malignant transformation of cervical cells. Therefore, attention should be paid to vaginal bacteria, viruses and Chlamydia infectious diseases.

## **2. Behavioral risk factors and cervical cancer**

Behavioral risk factors include premature sexual life, multiple sexual partners, oral contraceptives, smoking, multiple pregnancies, low socioeconomic status, malnutrition, and spousal disorders. It is well known that cervical squamous epithelium has a period of active development at an early age. At this time, if repeated exposure to infection, loss factors or stimulation by sperm, potential cell variability may occur, and cancer may occur after a few years of incubation. As early as 1976, Singer et al. suggested that human semen might play a role in the development of cervical cancer. Jeremias To evaluate the effects of semen on the potential oncogenes of cervical cells, the levels of mRNA transcription of matrix metalloproteinases MMP-2 and MMP-9 in HeLa cells and cervical cancer epithelial cells (HPV18 infection) were examined. The ability to inhibit MMP-2 production, while semen has an inducing effect on MMP-9, and there are individual differences. This suggests that the induction of MMP-9 by semen increases the risk of cervical cancer during sexual activity, especially in women who have been infected with HPV. The enhancement of MMP-9 expression in cervical epithelial cells in vivo and in vitro is closely related to invasive behavior. In addition, in vivo and in vitro experiments have shown that semen affects cervical epithelial cells and lymphocytes in women, demonstrating that semen supernatant is an inducer of heat shock protein and IL-10 gene transcription, and is an inhibitor of interferon gamma gene transcription. Agent. Michael et al. demonstrated that semen inhibits the recognition and killing of EB virus by lymphocytes, leading to the reproduction of EB virus in cervical epithelial tissues. We

used ra ji cells to detect polyamines in human semen. Studies have shown that polyamines can activate the expression of early antigens of EB virus in raji cells. Therefore, the use of barrier contraception (vaginal diaphragm, condom) can reduce the risk of cervical cancer. Smoking may be one of the causes of cervical cancer. Epidemiological investigations have shown that smokers are closely related to genital squamous cell carcinoma. The current study found the following relationship: 1 Recent studies have controlled the number of sexual partners and other confounding factors, found that the OR value of cervical cancer and smoking is 1.8 (95% confidence interval is 1.7 ~ 1.9); 2 Smoking is not associated with cervical adenocarcinoma, cervical cancer is associated with various aspects of sexual life and HPV-specific types; 3 a small number of P450 enzymes (CYP1A1, CYP1A2, CYP2D6 and CYP2E1) are expressed in HPV16 immortalized epithelial cell lines compared to primary cells. The line is high, and the P450 enzyme has been shown to play an important role in activating specific oncogenes in tobacco; 4HPV16 or HPV18 immortalized cervical cell lines are malignant after tobacco concentrate treatment; 5 recent studies have shown that smoking can cause cervix Injury of epithelial DNA; 6 found in smoker cervical mucosa tissue contains a strong carcinogen specific to tobacco: nitrosamine, 4-(methyl-1-nitroso)-1-(3-pyridyl -1 - Butanone; 7 Glutathione S Transferase M1 is involved in the detoxification of polycyclic aromatic hydrocarbons in smokers, but glutathione S transferase M1 is active in HPV16 transfected human cervical keratinocytes Reduced. Therefore, the cancer-promoting effect of smoking on HPV should be further studied.

### 3. Research progress in cervical cancer screening methods

With the development of the etiology of cervical cancer, its prevention and treatment methods are constantly improving and developing. Screening remains the primary means of preventing and controlling cervical cancer before the HPV vaccine is used in the population. Practices in various countries have proved that census can reduce the occurrence and death of cervical invasive cancer. The reason is that cervical cancer has a series of precursor lesions. Its occurrence and development are from quantitative to qualitative change, to gradual change to mutation, usually by cervix dysplasia (light ※In the case of the in-situ cancer ※The early invasive cancer ※The continuous development process of the invasive cancer. These precursor lesions can exist for many years, and the cervix has a favorable anatomical basis, which is easy to expose, easy to observe, palpate, and take. If it can be diagnosed in the precursor lesion stage, it can be further treated or monitored. The treatment effect of early cervical lesions is much better than that of cervical cancer. It is reported that the five-year survival rate of cervical invasive cancer is 67%, that of early cervical cancer is 90%, and that of cervical carcinoma in situ is almost 100%. Therefore, through the census, early diagnosis and early treatment can be achieved, and the incidence and mortality of cervical invasive cancer can be reduced.

Virological detection HPV DNA assay: Many scholars have proposed to detect HPV infection as a screening tool for cervical cancer. At present, HPV detection methods include cytology, dot blotting, fluorescence in situ hybridization, in situ hybridization, Southern hybridization, PCR, and hybridization capture. However, the Hybrid Capture (HC) test is a newly developed and FDA-approved technology for the detection of HPV DNA in the United States. Hybrid capture generation trials can detect nine high-risk HPVs, including 16, 18, 31, 33, 35, 45, 51, 52, and 56. The hybrid capture second-generation test can simultaneously detect 13 high-risk HPVs, 16 , 28 , 31 , 33 , 35 , 39 , 45 , 51 , 56 , 58 , 59 , and 68 .

### 4. Conclusion

In the near future, liquid-based cytology and HPV testing will be heavily incorporated into cervical cancer screening programs for women over the age of 30, but the most desirable and fundamental solution to cervical cancer is to use vaccines for the cause. prevention. Nowadays, the world is working on HPV vaccine research. Experts predict that in the near future, cervical cancer will be the first malignant tumor that humans can comprehensively prevent and eradicate by

immunization.

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