Cervical Cancer: Open Access
ISSN: 2475-3173

Precision Cervical Cancer Prevention and Treatment: New Ideas and Clinical Consequences

Saria Dbar*

Abstract

The third most frequent cancer in women globally is cervical cancer, and ideas and knowledge about its treatment and prevention are quickly advancing. Cervical cancer has been linked heavily to the human papillomavirus (HPV), despite the fact that the virus alone cannot bring about the disease. Since most infections are temporary and may be naturally cured by the host immune system, HPV-driven malignancy is actually a low chance event. Years must pass after a persistent HPV infection before cervical cancer develops. The foundation of this clinical intervention is to understand the carcinogenic pattern and appropriate targets during HPV host contact, and this extended time frame thus presents a perfect opportunity. In this review, we cover the main causes of HPV and cervical carcinogenesis, as well as new concepts and technologies that are being developed for cancer interventions. More urgently, we cover how

early prevention, and early treatment.

cancer, provide blatant evidence of genetic factors contributing to cervical carcinogenesis.

HPV integration

One important genetic stage in the development of cervical cancer is the integration of the HPV genome into the host chromosome. Numerous investigations have demonstrated that HPV integration typically entails fragmenting the viral E1 and E2 open reading frames [8], leading to the overexpression of oncogenes E6 and E7. Each of the cellular targets that E6 and E7 have helps to induce malignant transformation. For instance, E6 attaches to and breaks down the pro-apoptotic protein BAK and the tumour suppressor p53, enhancing the host cell's resistance to apoptosis and enabling viral DNA repdce
On the other hand, E7 activates cyclin-dependent kinase 2 (CDK2)/cyclin A 58 and CDK2/cyclin E complex, reversing cell cycle arrest, and inhibits the tumour suppressor retinoblastoma 1 (RB1) to release E2F transcription factors [9].

DNA mutation of the host genome

e somatic mutations of the host genome during HPV-induced carcinogenesis have also been a crucial component in studying cervical carcinogenesis, in addition to HPV integrations into the human genome of the host genes. Analysis of DNA mutations is crucial for distinguishing between cancerous and healthy tissue as well as for determining the best course of treatment.

e most thorough genomic landscape research to date was released in Nature Journal, and it used NGS analysis to show both well-known and brand-new high frequency mutations. e scientists demonstrated that whereas PIK3CA (16%), ELF3 (13%), KRAS (8%), and CBFB (8%) were found in ACC 91, EP300 (16%), FBXW7 (15%), PIK3CA (14%), HLA-B (9%), and p53 (9%) were the most o en occurring mutations in SCC. Notable ndings include the discovery of driver mutations in the oncogenes HLA-B, EP300, and FBXW7 in cervical malignancies [10].

Conclusion

e precise prevention, detection, and treatment of cervical cancer are not only necessary but also now 194 due to the development of new concepts and technologies for cancer therapy. We will be able to forecast the prognosis of individuals with HPV infections at an earlier stage with the help of the molecular mechanism's elucidation that underlies HPV persistence and the associated cervical cancer. e future psychological and nancial burdens of cervical scr9er therapy. We TH7sts tr8 86gcervic