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Regarding the Aetiology of CIN and certain types of neoplasia, including Cervical CA

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INTRODUCTION

There are perhaps over one hundred or more Human Papilloma Virus (HPV)[1] types1 and other viruses including Herpes Simplex Virus (HSV) that are linked to possible mutations. HPV has been identified as the cause of almost all cervical cancers. Between twelve and fourteen HPV types (HPV16,18,31,33,35,39,45,51,52,56,58,59,66,68) have been identified as high-risk (HPV) for cervical cancer but even among these, the risk varies. This has been researched especially in the aetiology of CIN, cervical carcinoma, vulvo-vaginal neoplasia. anal and peri-anal neoplasia, oropharangeal neoplasia, etc. However, although millions of people have been exposed to such viruses, only a fraction of them develop neoplastic disease in those organs[2]. It is therefore clear that the cellular immunity of the individual or 'localised immunity' at the site of the viral infection was compromised in the first instance, which in turn became the substrate for the virus to cause the mutation and the subsequent neoplasia. Atypical organisms, especially Chalmydia mycoplasma, etc. have been shown to cause recurrent chronic inflammation, which is obviously due to a lowered resistance of immunity. There are high risk HPV strains for C.I.N. and cervical cancer.[2]

Those who have atypical organisms in certain areas such as the cervix of the uterus, vulva, vagina and other regions, contributed to the lowered "localised cellular immunity". This predisposes to the viral insult leading to mutations. It has been said that it is not understood why some people clear the infection while others develop CIN.[3]

High Risk HPV Strains [4]

HPV 16, HPV18, HPV 33, HPV58 mutated genes in cervical neoplasia are described as FATI, MLL3, MLL2, FADD.

Common genetic variations have been associated with CIN and cervical neoplasia. About 50-55% of gene mutations were seen in CIN and cervical cancer. Patients with HIV have a five times higher incidence of developing invasive cervical cancer.[5] Taking immunosuppressants also weakens the defenses against CIN and Cervical neoplasia, through HPV.[6] There are several other factors such as smoking[7], certain sexual activities, early age of sexual intercourse, multiple partners, poor socio-economic factors, etc.

In a study of a Hispanic population, it was found that there was an association of cervico-vaginal fungi and certain bacteria contributing to CIN. and high-risk HPV. They concluded that "the structural features of the cervico-vaginal ecosystem may be an important step in understanding of the biology of cervical neoplasia, to better develop therapeutic interventions that target the microbiota." [7]

The squamo-columnar junction of the cervix is especially vulnerable since there is a gradation of the cell structure from squamous cells of the ecto-cervix and the uterine endometrium. This makes it more vulnerable to the atypical organisms to induce a lowered resistance, providing a suitable substrate for the virus to enter the path of mutation.

A detailed double-blind randomised trial could be easily conducted, provided that there are sufficient patients. After a cervical smear is obtained and results show that an abnormality such as C.I.N. or early neoplasia is present, the groups are (under local analgesia) colposcoped and biopsied. Depending on the biopsy, the patients are identified and appropriately grouped. Half of them in each group is treated

[1] *WHO. Human Papillomavirus (HPV) and Cervical cancer.*
[2] *J. Med Gennet.2019; 56 (3): 186-194 Huang J et al*
[3] *Michelle Pugle /undated on April 13, 2004 Sexual Health> HPV Causes and risk factors of CIN*
[4] *J. Med Gennet.2019; 56 (3): 186-194 Huang J et al*
[5] *UNAIDS. The little-known links between Cervical cancer and HIV.*
[6] *Microbial Spectrum.2014; 4 (4): DM1H2-0026-2016. Overviews of infections in the immunocompromised host*
[7] *Microbiol. 2018 Oct 23,9:2523,Filipa Godoy – Victoria et al*

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with Doxycyclin or Azithromycin, along with their spouses. The other group receives a placebo. They are reassessed colposcopically and biopsied at three -month intervals, under local analgesia.

In a small group, it was observed that the group which had the antibiotic cover fared much better with no progression of the neoplastic process. Both groups had appropriate post-colposcopic care and instructions.

We believe that a double-blind, randomised study is warranted in this field as the aetiology for certain cancers of the female and male genital tract and perhaps peri-anal and oro-pharangeal lesions may have a similar origin.

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