

The Pathogenesis of Human Papillomavirus (HPV) in the Development of Cervical Cancer:

Are HPV vaccines a safe and effective management strategy for this disease?

Whilst it can be claimed that HPV infection is a necessary pre-cursor to most cervical cancer, it must also be emphasized that most high risk HPV infections do not progress to cervical cancer^{1,2}. Scientists recognize that HPV infection with one of 15 or more high-risk strains is not sufficient on its own to induce cervical cancer^{1,2,3}. Several environmental and lifestyle factors have been identified as necessary for the progression of normal epithelial cells to carcinoma^{1,2,3}. In 2006, when scientists declared Human Papillomavirus (HPV) Type 16 and 18 to be the determining cause of cervical cancer⁴, it was known that HPV infection on its own was not sufficient to cause cervical cancer. Over the past century several co-factors have been identified as necessary for the progression of normal epithelial cells to carcinoma. Whilst there is still doubt about the identity of some co-factors, those that are confirmed are: multiple partners for the male and female, presence of HPV plus other viruses (for example HPV + Herpes Simplex Virus Type 2), prostitution, sex without a condom/microbicides, low socioeconomic status (poor hygiene/sanitation/nutrition conducive to sexually transmitted diseases), immunosuppression, smoking, and oral contraceptives^{1,2,3}.

The biological plausibility of an etiological theory requires that the incidence of the causal agent varies with the incidence and mortality of the disease⁵. Yet it is noted that the incidence and mortality of cervical cancer does not vary with the incidence of infection with HPV strains 16 and 18 worldwide^{6,7}. HPV 16 and 18 are *less prevalent* in the countries with the highest rates of cervical cancer: the developing countries⁷. Clifford et al (2003) state a vaccine targeting HPV strains 16 and 18 may prevent more invasive cervical cancer in developed nations where cervical cancer rates are low, than in the developing countries which carry the highest burden of this disease⁷ (Figure 1). HPV infection is prevalent in all countries yet cervical cancer rates vary significantly from country to country¹. This is evidence that a co-factor is a determinant, in *conjunction with HPV* in the progression to cervical cancer.

The trials for the Human Papillomavirus (HPV) vaccine did not observe that the vaccine would prevent any cases of cervical cancer⁸. The clinical trials for this vaccine observed pre-cancerous lesions in women 16 – 26 years of age⁸. This was an inadequate surrogate for cervical cancer because studies show that eighty percent of lesions in this demographic clear quickly without requiring treatment². In addition, it is a demographic which rarely gets cervical cancer and the participants in the trials were not selected for the risk factors known to progress HPV infection to cervical cancer in later years⁹.

This research concludes that the decision to use a vaccine to prevent cervical cancer was based upon the assumption that it would prevent cervical cancer: circumstantial evidence and not empirical evidence. HPV vaccines have been promoted to women on misleading information and the long-term risks of the vaccine have not been determined. A reduction in the burden of cervical cancer globally would be assisted by targeting the risk factors for this disease. This has already been demonstrated in China and other developed nations where the incidence of cervical cancer has been very low since the seventies⁶. This was achieved by altering the environmental/lifestyle factors listed above and introducing screening programs.

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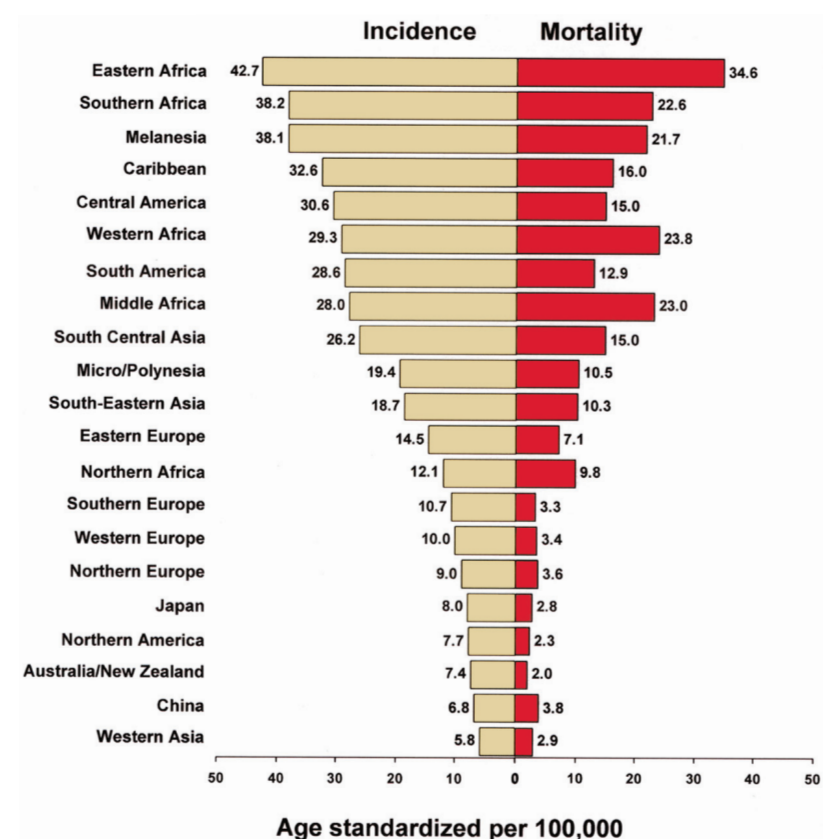
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Figure 1: Global Incidence and Mortality Rates for Cervix Uteri Cancer



Ref: Figure 11: Age-Standardised Incidence and Mortality Rates for Cervix Uteri Cancer, Parkin D, Bray F, Ferlay J and Pisani P, Global Cancer Statistics 2002, CA Cancer J Clin 2005; 55: 74 - 108

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