Hpv-16 and its relation to cervical cancer literature review examples

Sociology, Population

Abstract

The purpose of this discussion is to determine the relationship between human papillomavirus type 16 and the incidence of cervical cancer in adult women. As such, prevalence and distribution of cervical cancer will be discussed, as will the current literature regarding the correlation between and potential causative effects of human papillomavirus type 16 on cervical cancer. Some of the studies examined in this discussion focus almost entirely on specific populations, but this discussion will focus on a more global, universal approach to the correlation between HPV-16 and cervical cancer. The literature reviewed in this discussion focuses primarily on discoveries and changes made in the field in recent years. Although other types of human papillomavirus may cause adverse effects in women, the focus of this discussion will center entirely around HPV type 16, and its relation to cervical cancer in women of all races, ethnicities, geographical locations, and ages. Cervical cancer is one of the diseases that is most commonly associated with the human papillomavirus, particularly the human papillomavirus type 16-- also known as HPV-16 (Marongiu, Godi et al., 2014). Human papillomavirus can lay dormant in the system for a significant period of time, meaning that women who contract this particular virus may be unaware that they carry it without being specifically tested for it (Hamlin-Douglas, Coutlee, et al., 2008). While certain types of HPV do not cause any adverse reactions in the carrier for the entirety of the individual’s life, there are certain types of HPV-- such as HPV-16-- that have a correlative and causative link with cervical cancer (Hamlin-Douglas, Coutlee, et al., 2008). These links are significant, and the expression of certain viral genes, viral load, and other important

factors all influence the development of cervical cancer for women who carry the type 16 human papillomavirus (Hamlin-Douglas, Coutlee, et al., 2008).

Viral Load and Incidence of Cervical Cancer

Marongiu, Godi et al. (2014) suggest that viral load is directly associated with the development of cervical cancer in female patients. The pathology of HPV-16 is such that viral load is an indicator of likelihood of developing cervical cancer, while viral integration is an extremely poor indicator (Marongiu. Godi et al., 2014). Petry, Schmidt et al. (2011) suggest that, " p16/Ki-67 Dual-stained cytology may identify women with a high probability of underlying CIN2+ and may efficiently complement HPV-based screening programs to prevent cervical cancer" (Petry, Schmidt et al., 2011). In short, determining viral load is an important task for those hoping to determine whether cervical cancer is likely in a particular patient.

The Marongiu, Godi et al. (2014) study suggests that women with an abnormally high viral load are much more likely to develop cervical cancer than those who have a relatively low, stable viral load (Marongiu, Godi et al., 2014). On the other hand, viral integration was not a consistent indicator for the prevalence of cervical cancer in patients (Marongiu, Godi et al., 2014). According to Marongiu, Godi et al. (2014), most patients demonstrated a pathology that showed a mixture of integrated and episomal forms of the virus, regardless of their cervical cancer stage (Marongiu, Godi et al., 2014). The study also noted that there was no significant difference between the average methylation levels-- 3'L1 levels-- in the samples that contained episomal or mixed viral loads (Marongiu, Godi et al., 2014).

HPV-16 Gene Expression and Cervical Cancer

One of the primary concerns for women who carry the HPV-16 virus is the issue of gene expression. The problem, Brandsma, Sun et al. (2009) suggest, lies in DNA methylation; dysregulated genes-- those which are undergoing carcinogenesis-- show high levels of DNA methylation. Varying degrees of DNA methylation were shown to be linked to different severity levels of cervical cancer. According to Brandsma, Sun et al. (2009):

Repeat sequencing of the HPV 16 PCR products revealed heterogeneous methylation at most methylated CpGs, as previously reported for smaller genomic regions by molecular cloning The existence of a limited number of patterns indicates that transfer and/or removal of methyl groups is not a random process, that cells with methylation (or not) at particular HPV16 CpGs have a selective growth advantage, and/or that the methylation of certain CpGs is incompatible with continued infection Pathologically, the samples with almost no HPV16 DNA methylation (pattern A) were the least severe, those with several methylated CpGs in the E1 and E6 ORFs were intermediate in severity (pattern B), and those with high frequency methylation, particularly in the E5/L2/ L1 region (pattern C), the most severe (Brandsma, Sun, et al., 2009).

As this study notes, the rate of DNA methylation in gene expression in carcinogenesis and cancerous cells was a distinct signifier for the severity of cervical cancer in patients. Individuals who demonstrated low levels of HPV-16 DNA methylation had significantly less severe cases than those with high frequency methylation (Brandsma, Sun, et al., 2009).

Brandsma, Sun et al. (2009) also note that there is a significant dip in rates

of cervical cancers when other types of HPV-- not HPV-16-- are present (Brandsma, Sun, et al., 2009). The methylation process for HPV-16 appears to be unique from many other types because it causes the viral DNA to mutate in such a way that it encourages carcinogenesis, particularly in the E5/L2/L1 region (Brandsma, Sun, et al., 2009).

Distribution, Prevalence, and Screening: HPV-16 and Cervical Cancer

Although global studies on the prevalence and pathology of HPV-16 and cervical cancer are rare, there are a number of localized, geographical studies that investigate the prevalence of HPV-16 and cervical cancer in a given area. According to Ronco, Dillner, et al. (2013), screenings for HPV-16 demonstrated a 60-70% protection rate against the development of malignant carcinomas in the four European studies surveyed (Ronco, Dillner et al., 2013). Indeed, screenings did an even more significant job preventing invasive, late-stage carcinomas than pre-malignant carcinomas (Ronco, Dillner et al., 2013). Ronco, Dillner et al. (2013) suggest that screening for HPV-16 provides a significant protection for women against the development of carcinogenesis (Ronco, Dillner et al., 2013).

The Ronco, Dillner et al. (2013) study suggests that, using four randomized trials, there are significant benefits to the medical profession in regards to pre-screening women for HPV-16. When screening for HPV-16, medical professionals are more likely to uncover issues that will become problematic in the future, and potentially lead to the development of severe cervical cancer (Ronco, Dillner et al., 2013).

Hamlin-Douglas, Coutlee, et al. (2008) note that there are certain

populations that seem to be at greater risk than others in terms of HPV-16 exposure and the subsequent development of cervical cancer. Hamlin-Douglas, Coutlee et al. (2008) write, “ Inuit women in Canada and Quebec show elevated incidence rates of cervical cancer and higher mortality rates when compared with the general Canadian and Quebec populations” (Hamlin-Douglas, Coutlee, et al., 2008). The Hamlin-Douglas et al. (2008) study did not make any attempts to determine why this incidence was higher in the Inuit population, but instead noted that extensive historical and demographic surveys were given to participants in hopes of determining their particular behaviors and habits (Hamlin-Douglas, Coutlee, et al., 2008). The study also noted that the grouping of women indicates that there is a higher prevalence of the virus and development of cervical cancer in some populations than others (Hamlin-Douglas, Coutlee, et al., 2008).

Conclusions

The incidence of cervical cancer is highly associated with the HPV-16 virus in women. Women who have acquired the human papillomavirus type 16 are at significant risk for cervical cancer; in addition, viral load is a significant indicator for women at risk. The methylation rates for HPV-16 viral DNA are also significant indicators for the presentation of cervical cancers; without the virus being present, it is almost unheard of for women to contract cervical cancers (Hamlin-Douglas, Coutlee, et al., 2008). Perhaps most significantly, HPV-16 clusters occur within specific populations; within these populations, women are more likely to develop cervical cancer. In addition, it should be noted that the level of viral DNA methylation is also associated

with the severity of the cancer that the woman experienced (Brandsma, Sun, et al., 2009). It can be concluded that, within the population afflicted with HPV-16, prevalence of cervical cancer rates are significantly higher than in the population carrying other types of human papillomavirus (Brandsma, Sun, et al., 2009).

References

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