Summary: Susceptibility of high-risk human papillomavirus type 16 to clinical disinfectants

Meyers, J., et al., Susceptibility of high-risk human papillomavirus type 16 to clinical disinfectants. J Antimicrob Chemother, 2014.

Human papilloma virus (HPV) causes cervical and anogenital cancers

Human papilloma virus (HPV) is the causative agent of cervical and anogenital cancers, with the HPV16 strain responsible for up to 60% of all HPV-associated cancers.[1] Little is known about HPV susceptibility to disinfection; this is of concern since in addition to well documented sexual transmission, non-sexual transmission may also be important. Using a novel method of virus production the researchers demonstrate that glutaraldehyde (GTA) and *ortho*-phthalaldehyde (OPA), two commonly used disinfectants, are unable to inactivate HPV in a 45 min liquid suspension test. [1]

Disinfectant efficacy against HPV could not previously be tested

The activity of disinfectants against HPV has not previously been tested due to the lack of a method to produce sufficient quantities of native HPV virus in the laboratory. This changed recently when Professor Craig Meyers from Pennsylvania State University and Professor Richard Robison from Brigham Young University developed a novel method to produce natural, infectious HPV. This novel HPV production method is the first to produce native HPV16 viruses, called virions. Virions are different to quasi-viruses (or pseudo-virions) used in previous studies. Quasi-virus is easily produced in the laboratory, however it is unable to replicate like native virions. A highly specialised method to produce native HPV16 virions that have full replication capability was developed.

The native virions are produced by inserting the virus DNA into human foreskin cells. These infected cells grow and multiply amongst a rat collagen matrix which sits on a stainless steel grid. The grid is lifted on the surface of a nutrient rich medium, feeding the infected cells by diffusion. The mature virus is extracted by breaking open the cells. The virucidal activity of 11 commonly used clinical disinfectants were tested in both quasi and native HPV16 virions.

The suspension test involved mixing HPV16 quasi-virus or native virions with the clinical disinfectant, and leaving the mixture for 45 min at room temperature. After incubation, neutralisers were added and the remaining quasi-viruses or native-virions filtered out of the solution. These virus particles were added to skin cells and left for two days in an infectivity test. Detection of HPV DNA in the skin cells after two days indicated failure of the clinical disinfectant to eradicate infectious HPV. The clinical disinfectants tested were ethanol (70% and 95%), isopropanol (70% and 95%), GTA (2.4% and 3.4%), OPA (0.55%), phenol, PAA-silver (0.25% and 1.2%) and hypochlorite (0.525%).

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Glutaraldehyde and OPA shown to be completely ineffective against HPV

Of all the clinical disinfectants tested, only 1.2% PAA-silver and 0.525% hypochlorite achieved a >4 $\log_{10}$ reduction in infectivity after disinfection of native virions. They also achieved this reduction when tested against quasi-virus, as did isopropanol (both concentrations) and phenol. GTA (both concentrations) and OPA were ineffective in producing any significant reduction in infectivity in both virus types. This is of concern – OPA is currently accepted as a high level disinfectant widely used to reprocess semi-critical items including intracavity ultrasound probes and GTA is used as a sterilant in medical and dental healthcare facilities. As a result, the contact times for 3.4% GTA and 0.55% OPA were extended from 45 min to 24 h, which did not result in a decrease in viral infectivity. The findings suggest that a review of clinical disinfectants and disinfection standards as related to high level disinfection may be warranted.

References

1. Meyers J, Ryndock E, Conway MJ, Meyers C, Robison R. Susceptibility of high-risk human papillomavirus type 16 to clinical disinfectants. The J. Antimicrob Chemother. 2014;69:1546-50

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