

Table 2.13. Case-control studies of HPV and vaginal cancer

Reference, study location	Sites included	No. of cases	No. of controls	Method of detection	HPV prevalence (%)		Odds ratio (95% CI)	Comments
					Cases	Controls		
Carter <i>et al.</i> (2001), USA	Sera	35	1804	Seropositivity to HPV16 and HPV18 capsid proteins by ELISA (L1)	N=15/35 (42.9%) HPV16 L1 Ab	N=264/1804 (14.6%) HPV16 L1 Ab	4.8 (2.4–9.5)	Sera
					N=4/8 (50%) HPV16 L1 Ab + HPV16 DNA	N=264/1804 (14.6%) HPV16 L1 Ab + HPV16 DNA	6.3 (1.6–25.6)	
					N=8/35 (22.9%) HPV18 L1 Ab	N=258/1802 (14.3%) HPV18 L1 Ab	1.8 (0.8–4.0)	
					N=1/2 (50%) HPV18 L1 Ab + HPV18 DNA	N=258/1802 (14.3%) HPV18 L1 Ab + HPV18 DNA	–	
Daling <i>et al.</i> (2002), USA	Sera from patients with no history of vaginal cancer	37	2041	Seropositivity to HPV16 and HPV18 capsid proteins by ELISA (L1)	N=15/35 (42.9%) HPV16 L1 Ab	N=263/1800 (14.6%) HPV16 L1 Ab	4.3 (2.1–8.7)	Sera
					N=6/14 (42.9%) HPV16 L1 Ab + HPV16 DNA	N=263/1800 (14.6%) HPV16 L1 Ab + HPV16 DNA	4.4 (1.5–13.0)	
					N=8/35 (22.9%) HPV18 L1 Ab	N=257/1798 (14.3%) HPV18 L1 Ab	1.6 (0.7–3.6)	
Madsen <i>et al.</i> (2008), Denmark	Controls were Corpus Uterine Cancer samples	27	17	GP5+/6+ PCR-EIA assay and type-specific for HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66 & 68 and other 23 low risk HPV.	N=24 (89%)	N=0 (0%)	–	Formalin-fixed paraffin-embedded tissue samples. [Controls are unlikely to harbour hPV].