題名:Type specific distribution of HPV along the full spectrum of cervical carcinogenesis in Taiwan; an indication of viral oncogenic potential

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摘要:Objective: The distributional trend of different types of human papillomavirus (HPV) along the spectrum of cervical diseases from nonneoplastic

HPV infection (Normal), cervical intraepithelial neoplasia 1 (CIN1), cervical intraepithelial neoplasia 2/3 (CIN2/3) to invasive

cancer (CC) reflects the transformation potential of each HPV type.

Study design: Type-specific distribution of HPV in four hospital- and population-based HPV surveys in Taiwan was analyzed.

Results: Among the 1605 (out of 6356) women positive for HPV, the prevalence of HPV infection in Normal, CIN1, CIN2/3, and CC was

10%, 60%, 70%, and 86%, respectively. The order of type-specific prevalence was HPV 52 (20.9%), HPV 16 (16.9%), HPV 58 (9.0%), and

HPV 18 (8.5%) in Normal; HPV 52 (16.5%), HPV 16 (10.2%), HPV 51 (8.4%), and HPV 58 (6.5%) in CIN1; HPV 16 (29.8%), HPV 52

(17.1%), HPV 58 (16.4%), and HPV 33 (10.3%) in CIN2/3; and HPV 16 (50.7%), HPV 18 (11.9%), HPV 58 (10.1%), and HPV 33 (8.4%) in

CC. We compared the step-wise distributional changes of five major HPV types along the spectrum of cervical neoplasia. The CIN1 vs.

Normal distributional ratio of each HPV type varied from 0.41 to 0.97, indicating a relatively similar chance of

giving rise to CIN1. In the CIN2/3 vs. CIN1 and CC vs. CIN2/3 comparisons, the distributional changes varied dramatically among different HPV types. Upon progression to CIN2/3, the distributional proportions of HPV 16, 33, and 58 became 2.1- to 5.4-fold higher, and that of HPV 18 became 0.3- to 0.5-fold lower than their CIN1 and normal counterparts. In the CC vs. CIN2/3 comparison, the change in distributional proportion was highest in HPV 18 (5.7-fold), followed by HPV 16 (1.7-fold), HPV 33 (0.8-fold), HPV 58 (0.6-fold), and HPV 52 (0.18-fold).

Conclusion: The distribution-defined progression from subclinical infection to CC was highest for HPV 16, followed by HPV 33, 18, 58, and

52. The differential disease progression potential of different HPV types reflects their transformational capability at different steps of cervical carcinogenesis and warrants the clinical attention of HPV infection type specifically.