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CORRECTION

Correction to: The physical state of HPV16 infection and its clinical significance in cancer precursor lesion and cervical carcinoma

Wei Li[1] · Wei Wang[1] · Mani Si[1] · Linfei Han[1] · Qinglei Gao[1] · Aiyue Luo[1] · Yan Li[1] · Yunping Lu[1] · Shixuan Wang[1] · Ding Ma[1]

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The authors would like to correct Fig. 2a, as the error was introduced in the preparation of this figure for publication. We sincerely apologize for having this error in the article, the authors have provided corrected version of Fig. 2 here.

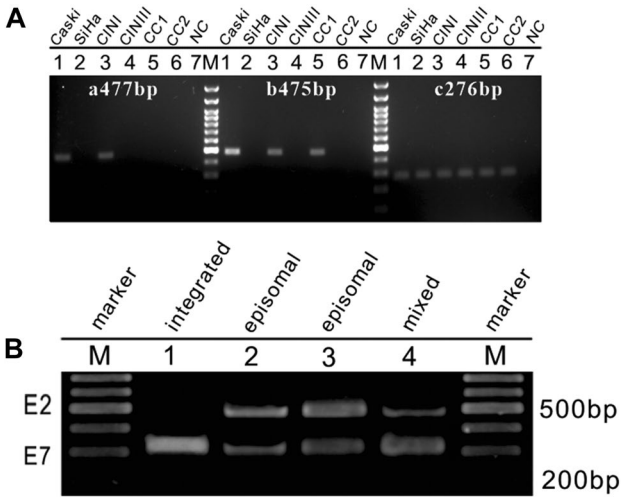

Fig. 2 Detection for physical status of HPV16 infection. **a** Characterization of HPV-16 integration. Gel photograph of PCR performed on DNA extracted from different tissues, demonstrating failure of amplification of those different fragments (amplimer a, b, c, shown as 475-bp, 477-bp, 276-bp products, respectively) of the E2 gene in CIN and CC lesions. M marker (100-bp DNA ladder), 1 Caski cell, 2 SiHa cell, 3 a CIN I lesion, 4 a CIN III lesion, 5–6 CC lesions (integration), 7 water blank negative control. **b** Discrimination between episomal infection and mixed infection. Gel photograph of multiplex PCR performed on DNA extracted from paraffin-embedded cervical lesion tissue samples with primers located within the E7 region (315-bp product) and E2 region (amplimers B, 477-bp product) of the HPV-16 genome. M marker, 1 integrated infection (CC), 2 episomal infection (normal cervix), 3 episomal infection (CIN III), 4 mixed infection (CC)

Reference

Li W, Wang W, Si M, Han L, Gao Q, Luo A, Li Y, Lu Y, Wang S, Ma D. The physical state of HPV16 infection and its clinical significance in cancer precursor lesion and cervical carcinoma. J Cancer Res Clin Oncol. 2008 Dec;134(12):1355–61.

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✉ Shixuan Wang
sxwang@tjh.tjmu.edu.cn

✉ Ding Ma
dma@tjh.tjmu.edu.cn; dingma424@yahoo.com

[1] Cancer Biology Research CenterTongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430030, Hubei, People's Republic of China