of concurrent viral plaques. Despite rapid cell-to-cell spread of HSV-2, infected cells are eliminated by localised CD8+ T-cells within 24 h of plaque initiation. Moreover, the extent of secondary plaque formation prior to episode termination is determined by spatial CD8+ T-cell density surrounding the site of infection.

**Conclusions** Genital HSV-2 utilises three kinetically distinct methods of spread to initiate and sustain prolonged shedding episodes. The extent and severity of secondary plaque formation is determined by spatial immune cell density.

**Materials and Methods** One hundred thirty-one invasive oral SCCs were tested for HPV using laboratory-developed PCR assays for HPV16. F53 expression, tumour angiogenesis (CD-31 staining) and proliferation (MBI-1) were also assessed by immunohistochemistry in paraffin embedded tissue. Patients mean age was 58.09±10.41, median 59 (116 men and 15 women). Clinical Stage distribution was: I (17 cases); II, 56; III, 32; IV, 26. Most tumours were histological grade I (39) and II (74). Patients with pathological stage I-II were referred to surgery (65 cases) and patients with Stage III-IVA were referred to surgery and postoperative radiation therapy (66 cases). Mean radiotherapy given doses were 62.13±7.74, median 65 Gy in 1.8-2 Gy fractions. No chemotherapy was used in any case.

**Results** 41 cases (31.3%) were HPV 16 (+). No relation was found with age, gender, or tumour characteristics. In fact no relation was found to p53 expression, tumour proliferation or angiogenesis. 15-year DFS was 62.20% in HPV (+) patients was, compared to 37.3% in the HPV(−) group (p<0.076). In stage III-IV cases (treated by surgery and radiation therapy) this difference reached statistical significance (15 y DFS 72.4% vs 56.0% p<0.020). Similar results were found for Cause Specific Survival (15 y DFS 68.4% vs 26.2 p<0.054).

**Conclusion** These data show that the HPV status is a good predictor of DFS and survival in patients treated with radical surgery and adjuvant radiotherapy in oral carcinomas. This prognostic advantage seem to be independent of tumour proliferation, p53 status or angiogenesis. Other molecular processes could be implicated in the different response to radiotherapy.

**Basic sciences oral session 2—Immunity and animal models**

**Objective** Neisseria gonorrhoeae and Chlamydia trachomatis cause similar urogenital diseases and up to 70% of individuals with gonorrhoea also have chlamydia. Using a newly developed female mouse model of coinfection, we recently reported that higher numbers of N gonorrhoeae were recovered from mice with a pre-existing C. trachomatis infection, the mouse strain of Chlamydia, compared to mice infected with N gonorrhoeae alone. Recent studies on the host response to N gonorrhoeae implicate toll-like receptor 4 (TLR4) and IL17 responses as being protective against N gonorrhoeae. Here we tested the hypothesis that the immune response to chlamydial infection makes the genital tract more permissive to N gonorrhoeae.

**Methods** Using an immune-targeted RT-PCR array we screened for alterations in host gene expression during chlamydial infection of BALB/c mice that may account for the observed increase in gonococcal colonisation. Mouse genital cells were collected by vaginal swab and analysed for TLR4 expression by Flow cytometry. Coinfection studies were performed in BALB/c (TLR4 wild type) and C57-LPS-d/I (TLR4 mutant) mice and the number of viable chlamydiae and gonococci recovered from each group was determined by immunofluorescence using L929 cells and quantitative culture on GC agar, respectively.