Gene therapy for head and neck SCC using KITENIN-Antisense Joon Kyoo Lee, MD, PhD
Department of Otolaryngology-Head and Neck Surgery, Chonnam National University Medical School and Hwasun Hospital, 160 Ilsimi, Hwasun-eup, Hwasungun, Jeonnam, Korea 519-809

ABSTRACT

INTRODUCTION

Western blot analysis

RESULTS

Cell invasion assay

Figure 2 Cell invasion assay: The number of invading si-KITENIN-transfected SNU-1041 cells was 64.2 ± 19.2, whereas for the scrambled DNA-transfected SNU-1041 cells the number was 96.5 ± 10.2 as measured by 10 random squares of 0.5 × 0.5 on the microscopic field of view under conditions with 20 µg/ml of fibronectin; the difference between the two groups was statistically significant (p < 0.001) (Figure 2).

Figure 3 Cell migration assay: The artificial wound gap became significantly narrower in the control group as time passed by 20, 28, 44 hours, compared to the si-KITENIN group (p < 0.001) (Figure 3). On the third day, the gap was nearly filled within 44 hours. However, a remained wide open in the si-KITENIN group

Figure 4 Cell proliferation assay: The proliferating cells, as determined by the absorbance, were significantly decreased in the si-KITENIN group compared to the scrambled group. On the third day, the absorbance was 0.4933 ± 0.0484 in the si-KITENIN group and 0.6023 ± 0.0319 in the scrambled group (p < 0.001).

Gene therapy using anti-KITENIN strategies may help or delay the progression (invasion, migration and proliferation) of head and neck squamous carcinoma.