Supplementary materials

Table S1 sequence of TCF4 siRNA

Name	Target sequence
1A	GCGGGATAACTATGGAAAGAA
2A	CCCACATAAAGAAACCTCTTA
3A	GCGCTTTGGCCTTGATCAACA
4A	GCCTTGATCAACAGAATAACT
5A	GCAAATACTCCAAAGAAGTGT
6A	GGGCTTGACCGACAGACTTTA

Figure S1

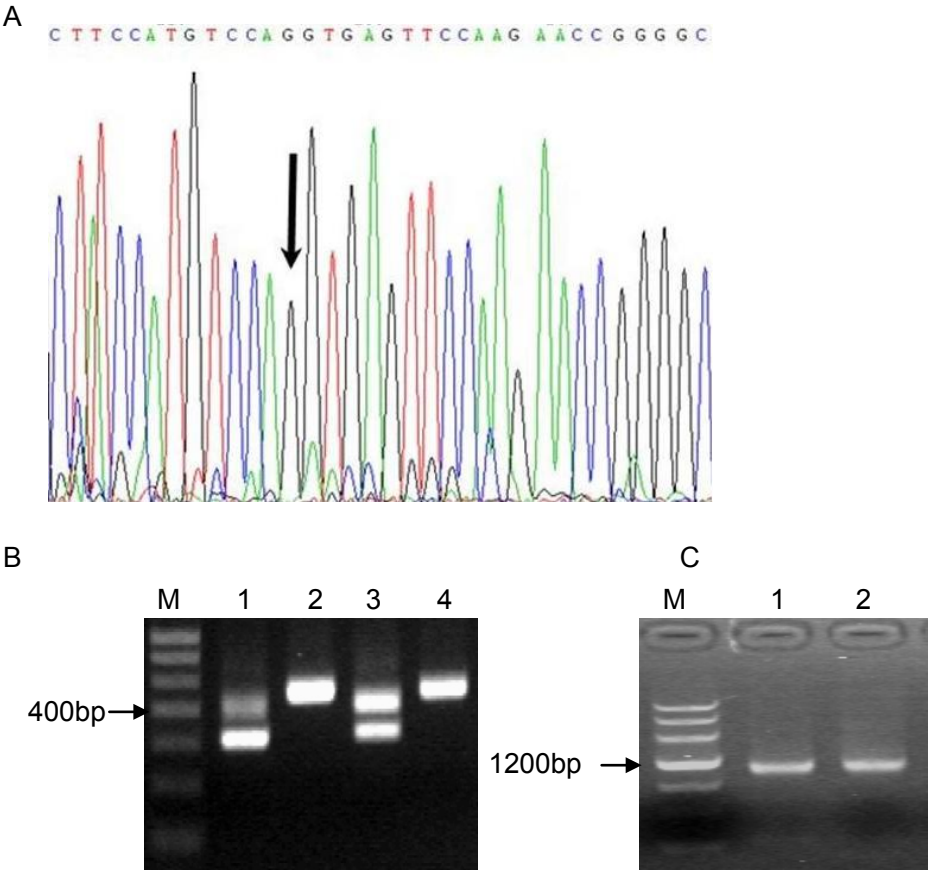

Figure S1 TCF4 expression in esophageal cancer cell line EC109. (A) Sequencing of the PCR product of TCF4N isoform in EC109. (B) RT-PCR analysis the splicing of exon 4 and 7~9 with primer P1F/R or P2F/R in EC109 cell (lane 1 and 2) and KESE150 cells (lane 3 and 4). (C) RT-PCR analysis the splicing variants spanning exon 3 to exons 17 in EC109 cells (lane 1) and KESE150 cells (lane 2) and the products were further cloned and sequenced.

Figure S2

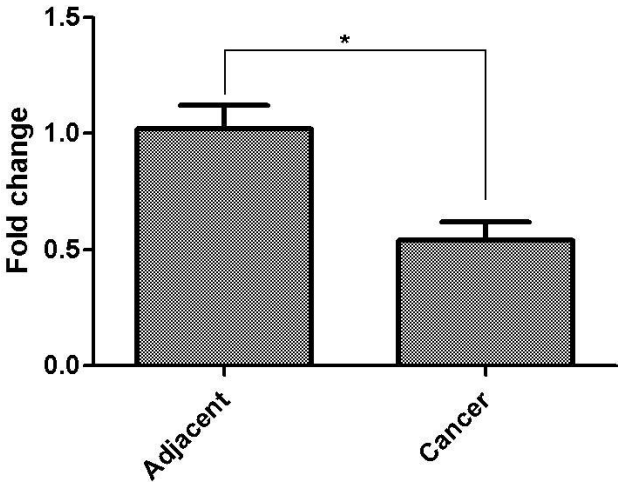

Figure S2 Gene expression of TCF4N variant in esophageal cancer and adjacent tissues. RNA was isolated from ten pairs of cancer and adjacent tissues, and the expression of TCF4N was detected by realtime RT-PCR. Fold changes were calculated as $2^{(\Delta\Delta Ct_{Cancer-Adjacent})}$. * $P<0.05$.

Figure S3

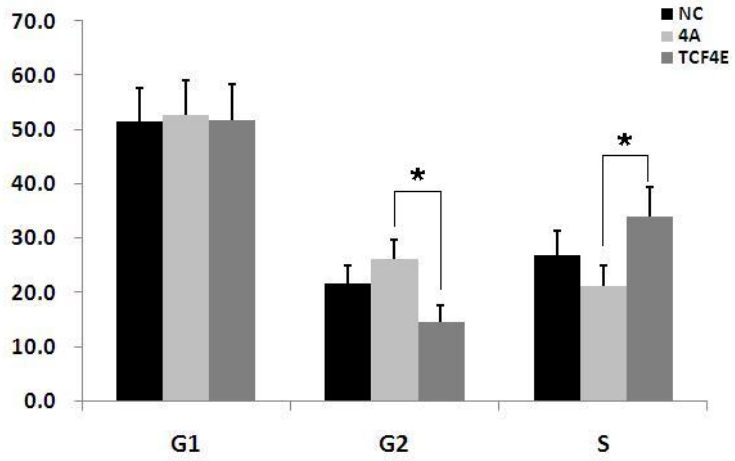

Figure S3 Cell cycle distribution of EC109 cells was determined by FACS as TCF4E expression was altered. Data were obtained from three independent experiments. NC: negative control; 4A: siRNA 4A knockdown group; TCF4E: Overexpression of TCF4E group. * P<0.05.

Figure S4

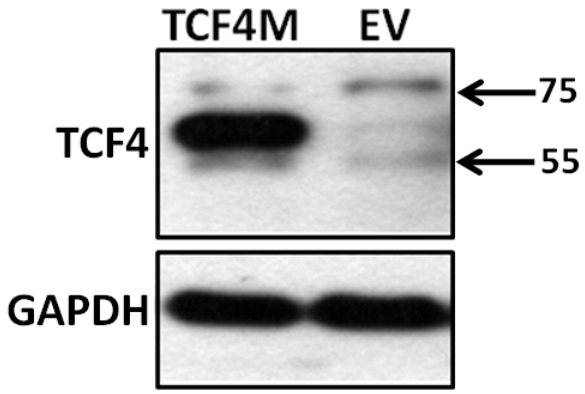

Figure S4 Overexpression of TCF4M isoform in EC109 cells. TCF4M construct was transfected into EC109 cells. 48 hours later, total proteins were isolated for Western blotting. GAPDH was used as controls for equal loading.