Table 6. Association of focal 14q13.3 amplification with disease progression.

| | n = 91 | 14q13.3 amplification status | | | |
|---------------------|--------|------------------------------|-----------------|-------------------------|-----------------|
| Clinicopathological | | Focal high-leve | I . | Wide low-level | |
| Characteristics | | Amplifications ($n = 17$) | | Gains_(n = 12) | |
| | | Correlation coefficient | <i>p</i> -value | Correlation coefficient | <i>p</i> -value |
| Stage | | | - | | |
| III | 18 | 0.257 | 0.013 | -0.031 | 0.774 |
| I and II | 73 | | | | |
| Recurrence | | | | | |
| Present | 40 | 0.200 | 0.056 | -0.083 | 0.432 |
| No | 51 | | | | |

Associations were tested using a dataset consisting of 91 lung adenocarcinomas (SI-Table 4). Of these 91 samples, seventeen were found by CGH to harbor focal (\leq 5 Mb) and high-level amplification (\log_2 ratio \geq 0.8) at 14q13.3. Twelve samples were found to contain wide and low-level DNA copy number gain at 14q13.3. Of the seventeen clinical parameters of this dataset, tumor stage was found to be significantly associated with the occurrence of focal high-level amplifications at 14q13.3. The presence of disease recurrence also associates with the occurrence of focal high-level amplifications but with less significance. These correlations were not found with tumors harboring wide low-level gains at 14q13.3.