Dokumenttyp: journal article

Autor(en) des Beitrags: Aubele, M; Vidojkovic, S; Braselmann, H; Ritterswürden, D; Auer, G; Atkinson, MJ; Tapio, S; Höfler, H; Rauser, S; Bartlett, JM

Titel des Beitrags: Overexpression of PTK6 (breast tumor kinase) protein--a prognostic factor for long-term breast cancer survival--is not due to gene amplification.

Abstract: In a previous retrospective study, we demonstrated the prognostic value of protein tyrosine kinase 6 (PTK6) protein expression in breast carcinomas. Here, we analyzed PTK6 gene amplification using fluorescence in situ hybridization technique in a cohort of 426 invasive breast carcinomas and compared it with PTK6 expression level as well as with the clinical outcome of patients. Forty-five percent of tumors show increased PTK6 gene copy numbers when compared to normal tissue. Most of these, however, were related to chromosome 20 polysomy (30%), while gene amplification accounted for only 15%. Only “low level” amplification of the PTK6 gene, with up to eight signals per nucleus, was found. The PTK6 cytogenetic status (normal, gene amplification, polysomy 20) was not associated with histopathological parameters or with the protein expression of HER receptors. No statistical association was identified between PTK6 gene status and expression level. Further, the PTK6 gene status does not influence the disease-freesurvival of patients at t> or = 240 months. Based on these results, we state that the PTK6 overexpression is not essentially attributed to gene amplification, and the PTK6 protein expression-but not gene status-is of prognostic value in breast carcinomas. PTK6 protein overexpression may
result from polysomy 20 in a minority of the tumors. In a marked proportion of tumors, however, the overexpression is likely to be caused by posttranscriptional regulation mechanisms.