Relevance of E-cadherin expression to EGFR-TKI molecular targeted therapy sensitivity/resistance and its clinical significance

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ABSTRACT. We examined the effect of E-cadherin expression on epidermal growth factor receptor-tyrosine kinase inhibitor (EGFR-TKI) molecular targeted therapy sensitivity/resistance. We treated MCF-7, MDA-MB-231, T24, SiHa, H460, SK-HEP-1, MHCC97-H, and THP-1 cells with the EGFR-TKIs PD153035 and gefitinib, and then tested the drug-resistance and sensitivity using the MTT method, calculated IC50 values for each cell line, and compared the results to E-cadherin content. The MTT assay was used to determine the survival rates of
MCF-7, MDA-MB-231, T24, SiHa, H460, SK-HEP-1, MHCC97-H, and THP-1 cells upon the action of EGFR-TKI (PD153035, gefitinib). For PD153035, the IC\textsubscript{50} in MCF-7, MDA-MB-231, T24, and SiHa cells differed from that of H460, SK-HEP-1, MHCC97-H, and THP-1 (P < 0.05). Following gefitinib treatment, the IC\textsubscript{50} values of MCF-7, MDA-MB-231, T24, and SiHa cells differed from those of H460, SK-HEP-1, MHCC97-H, and THP-1 cells (P < 0.01). The survival rate of MCF-7, MDA-MB-231, T24, and SiHa cells clearly decreased with increasing drug concentration, indicating the cells were sensitive to the drugs and that E-cadherin expression was positive; however, H460, SK-HEP-1, MHCC97-H, and THP-1 cells showed no significant decreased with increasing drug concentration, indicating that they were resistant to the drugs and that E-cadherin expression was negative. The survival rate of epithelial tumor cells through the action of EGFR-TKI is related to E-cadherin expression. E-cadherin may play a significant role in the sensitivity regulation of EGFR molecular targeting treatment. E-cadherin may provide important clues for selecting proper EGFR-TKI molecular targeting treatment.

**Key words:** Clinical significance; E-cadherin; Molecular treatment; Epidermal growth factor receptor-tyrosine kinase inhibitor; Resistance