Insulin-like growth factor 1 receptor (IGF1R) was proved to be associated with longer patients' survival, while - on the contrary - STAT3 (signal transducer and activator of transcription) was recognized as factor of poor prognosis in breast cancer. Surprisingly, these two agents of adverse prognostic significance were reported to act in accord. Namely, STAT3 was also found to mediate IL-6 dependent upregulation of IGF1R during constitution of malignant phenotype. The aim of this study was to examine if there is any link between expression of STAT3 and IGF1R and compare STAT3 expression with glucose transporter 1 (GLUT1). IGF1R and STAT3 were examined in 106 (GLUT1 in 67) human, female, primary, invasive, ductal breast cancers with immunohistochemistry. STAT3 correlated with IGF1R in all patients ($r=0.3385$, $p<0.001$) and also was preserved in node positive tumors (N+) ($r=0.3182$, $p=0.014$), node negative tumors (N-) ($r=0.4215$, $p=0.003$), patients after chemotherapy (Ch+) ($r=0.5164$, $p=0.001$) and patients without chemotherapy (Ch-) ($r=0.2469$, $p=0.042$). Relationship between STAT3 and GLUT1 almost reached statistical significance ($r=0.2340$, $p=0.057$) in all patients and was significant in chemotherapy spared patients ($r=0.4173$, $p=0.022$). Our findings could confirm functional dependence between IGF1R as membranous receptor and STAT3 as an intracellular messenger and upregulator of IGFR1 in human ductal breast cancer. Due to adverse reports on prognostic role of IGF1R and STAT3, further studies are required to determine more reliably their impact on patients' outcome. Besides IGFR1, another molecule that involves glucose metabolism - GLUT1 also seems to be co-expressed within production of STAT3 in breast cancer. The pattern of IGFR1, STAT3 and GLUT1 co-expression and overexpression reflects hypermetabolic state of breast cancer cell.