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Expression of c-kit in common benign and malignant breast lesions

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ABSTRACT

Aims and background. c-kit (CD117) is a transmembrane tyrosine kinase that acts as a type III receptor for mast cell growth factor. In recent years, the role of c-kit in the development of preinvasive and invasive breast carcinomas has been investigated. The aim of our study was to detect c-kit expression in the entire spectrum of common benign and malignant breast lesions in correlation with a well-studied myoepithelial or stem-cell like marker (p63).

Methods and study design. We evaluated 270 cases of benign and malignant breast lesions including fibrocystic disease, fibroadenoma, sclerosing adenosis, atypical ductal hyperplasia, ductal/lobular carcinoma *in situ*, and ductal/lobular/mixed type carcinoma. C-kit staining was evaluated in the cytoplasm/cell membrane in epithelial and myoepithelial cells and p63 in the nuclei of myoepithelial cells.

Results. c-kit was highly expressed (85.3%) in benign lesions (fibrocystic disease, sclerosing adenosis, fibroadenoma), and p63 expression was 95.5% in the aforementioned lesions. c-kit distribution in preinvasive and invasive lesions was as follows: ductal/lobular carcinoma *in-situ*, 43%/35%; ductal/lobular carcinoma, 36%/39%; and mixed type carcinoma, 20%. c-kit was highly expressed in myofibroblast/fibroblast cells only in grade III ductal/lobular carcinomas. c-kit was totally absent in stromal cells in benign lesions and *in situ* carcinomas whereas expression was weak in grade I and II carcinomas.

Conclusions. Combined overexpression of c-kit and p63 is indicative of benign breast lesions. In contrast, there is reduced expression of c-kit in *in situ* and invasive breast carcinomas, with simultaneous overexpression in the stromal cells. This suggests that c-kit may play a role in breast cancer progression. Free full text available at www.tumorionline.it

Key words: breast cancer, c-kit (CD117), fibroblasts, fibrocystic disease, myofibroblasts, p63.

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