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PostScript

LETTERS

Albumin-bound paclitaxel (ABI-007; Abraxane) in the management of basal-like breast carcinoma

We read with great interest the paper by Banerjee *et al*[1] regarding the clinical outcome and response to chemotherapy in basal-like carcinoma of the breast. After extensive discussion of chemotherapeutics used in the management of basal-like carcinoma, they concluded that new treatment options should be investigated for patients with this subtype of breast cancer. A recent study by Pinilla *et al*[2] showed that caveolin-1 (CAV1) expression is associated with a basal-like phenotype in sporadic and hereditary breast cancers. They looked at CAV1 expression in 509 sporadic and 47 hereditary BRCA1-/BRCA2-associated carcinomas. A strong association was found between CAV1 expression and a basal-like phenotype. This phenotype was present in 52% of tumours that expressed CAV1, compared with only 9% of CAV1-negative carcinomas ($p<0.001$). Interestingly, Rouzier *et al*[3] showed that the basal-like and HER-2-positive subtypes of breast cancer are more sensitive to paclitaxel- and doxorubicin-containing preoperative chemotherapy than the luminal and normal-like cancers.

ABI-007 (Abraxane; American BioScience, Santa Monica, California, USA) is a new, biologically interactive, nanometer-sized albumin-bound paclitaxel particle initially developed to avoid the toxicities associated with polyethylated castor oil. It is the first of a new class of anticancer agents that incorporate albumin particle technology and exploit the unique properties of albumin, a natural carrier of lipophilic molecules in humans. After many phase I and II studies in metastatic breast cancer, Gradishar *et al*[4] in their phase III study compared albumin-bound nanoparticle paclitaxel, ABI-007, with polyethylated castor oil-based paclitaxel in women with metastatic breast cancer. This study showed greater efficacy and a favourable safety profile of ABI-007, although no subgroup analysis of molecular phenotypes for differential efficacy of the treatment was performed. After the incorporation of ABI-007 with albumin in circulating blood, the drug is preferentially transported from the blood to the tumour site in two ways. The first is through the leaky junction of endothelial cells that are highly pronounced around the tumour tissue by induction of angiogenesis, and the second, perhaps more prominent, way, is acting through receptor-mediated transcytosis of this albumin-bound ABI-007. This second mechanism is mediated by CAV1. Moreover, one study showed a 4.5-fold increase in paclitaxel transport across endothelial cells for ABI-007 compared with standard paclitaxel.[5]

In the light of the above information, we suggest that in patients with breast cancer with higher CAV1 expression, such as cancer with ABI-007 may be more effective and have a basal-like phenotype, more favourable safety profile.

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CD10 positivity in breast epithelial neoplasms

We read with great interest the study of Kalof *et al*[1] concerning immunostaining patterns of myoepithelial cells in breast lesions. The

Table 1 Comparison of CD10 immunohistochemical methods

Method	Kalof *et al*	Moritani *et al*	Our study
Antigen retrieval	Sodium citrate, pH 6.0 (Dako, Carpentaria, CA, USA)	Autoclaving at 121°C for 25 min	CCI (Tris/borate/EDTA), pH 8 (Ventana Medical Systems, Tucson, AZ, USA)
Antibody clone	Monoclonal, clone 56C6 (NCL-CD10-270; NovoCastra, Newcastle upon Tyne, UK)	NU-N1(Japanese Nichirei, Tokyo, Japan)	Monoclonal, clone 56C6 (Cell Marque, Hot Springs, AZ, USA)
Antibody dilution	1:80	1:50	Prediluted (dilution unknown)
Signal detection	Avidin-biotin-peroxidase method with diamino- benzidine (DAB)	Streptavidin-biotin-peroxidase method with DAB	Streptavidin-biotin-peroxidase method with DAB