micropapillary) were grouped together as non-comedo DCIS (n = 119). While comedo necrosis has been shown to be associated with more aggressive biological behaviour in DCIS [2, 3], nuclear grade and tumour cell size also influence clinical outcome [2–5]. These parameters correlate with other prognostic variables in DCIS including c-*erb*B-2 expression [6], oestrogen receptor status [7] and DNA ploidy [8]. There is a correlation between architectural patterns and cytological characteristics, with comedo DCIS tending to be large cell in type and cribriform and micropapillary variants small cell [6] but this is not always the case [5]. In particular, solid DCIS appears to be cytologically heterogeneous, reflected in cell kinetic studies [9]. For these reasons a classification based primarily on the presence or absence of necrosis, with no reference to the cytological details of the component cells is somewhat of an oversimplification [10]. In the study reported by Silverstein two of the five recurrences in the non-comedo category occurred in the solid DCIS group (n = 25). Should these two cases be composed of large cells with high nuclear grade the recurrence rate based on cytological characteristics may be quite different to that observed using an architectural classification alone.

This work of Silverstein *et al.* constitutes one of the largest follow-up studies of DCIS and challenges the widely held view that histological appearances influence the biological behaviour of this disease. This has obvious implications for our understanding of the natural history and, in particular, the supposed heterogeneity of DCIS. Knowledge of the recurrence rate of DCIS based on the cytological characteristics of the component cells would be extremely useful.

1. Silverstein MJ, Cohlan BF, Gierson ED, *et al.* Duct carcinoma in situ: 227 cases without microinvasion. *Eur J Cancer* 1992, **28A**, 630–634.
2. Lagios MD, Margolin FR, Westdahl PR, Rose MR. Mammographically detected duct carcinoma *in situ*. Frequency of local recurrence following tylectomy and prognostic effect of nuclear grade on local recurrence. *Cancer* 1989, **63**, 618–624.
3. Ottesen GL, Graversen HP, Blichert-Toft M, Zedeler K, Anderson JA, on behalf of the Danish Breast Cancer Cooperative Group. Ductal carcinoma *in situ* of the female breast. Short term results of a prospective nationwide study. *Am J Surg Pathol* 1992, **16**, 1183–1196.
4. Patchefsky AS, Schwartz GF, Finkelstein SD, *et al.* Heterogeneity of intraductal carcinoma of the breast. *Cancer* 1989, **63**, 731–741.
5. Bellamy COC, McDonald C, Salter DM, Chetty U, Anderson TJ. Noninvasive ductal carcinoma of the breast: the relevance of histologic categorization. *Human Pathol* 1993, **24**, 16–23.
6. Bartkova J, Barnes DM, Millis RR, Gullick WJ. Immunohistochemical demonstration of c-*erb*B-2 protein in mammary ductal carcinoma *in situ*. *Human Pathol* 1990, **21**, 1164–1167.
7. Þur ME, Zimarowski MJ, Schnitt SJ, Baker S, Lew R. Estrogen receptor immunohistochemistry in carcinoma in situ of the breast. *Cancer* 1992, **69**, 1174–1181.
8. Killeen JL, Namiki H. DNA analysis of ductal carcinoma in situ of the breast. A comparison with histologic features. *Cancer* 1991, **68**, 2602–2607.
9. Meyer JS. Cell kinetics of histologic variants of *in situ* breast carcinoma. *Breast Cancer Res Treat* 1986, 7, 171–180.
10. van Dongen JA, Holland R, Peterse JL, *et al.* Ductal carcinoma *in situ* of the breast; second EORTC consensus meeting. *Eur J Cancer* 1992, **28A**, 626–629.

Correction

***De novo* Cisplatinum Resistance Does Not Influence Cellular Radiosensitivity** — This paper was published in *The European Journal of Cancer*, Vol. 29A, No. 9, pp. 1315–1320. Unfortunately, the following errors were included in the paper:

—In Table 1, the cell line for non-small cell lung cancer should be COR-L23.

—Also in Table 1, the histological type of cell line MOR should be adenocarcinoma.

—In reference 7, there should be only four authors: Britten RA, Warenius HM, Masters JRW and Peacock JH.