Table 1. Number of registrations of KS by sex and age and annual registration rates per 100 000. Vaud Cancer Registry, Switzerland, 1974–1990

	Males						Females					
	Age (years)				All-age standardised registration rates		Age (years)				All-age standardised registration rates	
Calendar years	0–14	15–39	40–59	≥ 60	Vaud*	World†	0–14	15–39	40–59	≥ 60	Vaud*	World†
1974–1982	0	0	0	0	0	0	0	0	0	0	0	0
1983–1984	0	5	1	0	1.2	1.0	0	0	0	0	0	0
1985–1986	0	3	3	0	1.2	1.0	0	0	0	0	0	0
1987–1988	0	8	4	2	2.6	2.1	0	1	0	1	0.33	0.30
1989–1990	0	7	11	0	3.1	2.6	0	3	1	0	0.66	0.65
Total (1983–1990)	0	23	19	2	2.1	1.7	0	4	1	1	0.26	0.25

*Age-standardised to the Vaud Canton population of 1983. †Age standardised to the world standard population.

To provide further information on the descriptive epidemiology of KS in Europe, we reviewed data collected from 1974 to 1990 by the Cancer Registry of the Swiss Canton of Vaud (whose population, according to the 1980 Census, was 530 000 inhabitants) [5]. The morphology code of the International Classification of Disease—Oncology (ICD-O) for KS (M9140/3) was used to identify cases.

Numbers of KS cases by age group and sex-specific incidence rates, standardised on both the Vaud population and the world standard population, are shown in Table 1. No cases of KS were registered in the resident population of the Canton of Vaud before 1983 (i.e. the year when the first cases of AIDS were reported in Switzerland) [6]. Afterwards, a steady increase in KS registration rates emerged to a total of 44 men and 6 women between 1983 and 1990. Among males, overall standardised (world standard) incidence rates were 1.0/100 000 men from 1983 to 1986, 2.1 in 1987–1988 and 2.6 in 1989–1990. Among females, the first two cases were registered in 1987–1988, corresponding to an overall rate of 0.3/100 000 women. Incidence rates in females raised to 0.7/100 000 in 1989–1990. The largest number of cases, in both sexes, was observed in young adults (15–39 years).

Since no cases of KS were registered in the Canton of Vaud before the spread of AIDS, incidence rates were compatible with the absence of the disease or with the extremely low rates in England and Wales, especially among natives [4]. A certain degree of underascertainment can be suspected but the traditionally very high proportion of histological verification of all suspected skin lesions [7] and the 100% verification rate for KS in the Vaud Cancer Registry is reassuring.

In conclusion, this study shows that before 1983 KS was not present in the resident population of the Swiss Canton of Vaud and thus suggests that the still uncertain causative agent for KS was virtually absent in this area prior to the AIDS epidemic. Conversely, over most recent years it has become the fourth leading site of cancer incidence in males aged 15–39 (after testis, Hodgkin's disease and melanoma).

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Histological Classification of Ductal Carcinoma *in situ*

Cecily M. Quinn

IN THEIR follow-up study of 227 cases of ductal carcinoma *in situ* (DCIS) Silverstein *et al.* [1] found no significant difference in disease recurrence rate according to histological subtype. Cases were broadly divided into two major categories based on architectural patterns including the presence or absence of necrosis. Cases showing comedo necrosis (n = 108) constituted one category and all other variants (solid, cribriform, papillary,

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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.

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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.