Table 1. *Intensity of* p53 *immunostaining*

Melanocytic lesions	Staining intensity 0/(+)	+	++/+++	Total
Regular nevi (n = 10)				
PAb-1801	0	7	3	10
PAb-240	0	8	1	9
Dysplastic nevi (n = 11)				
PAb-1801	2	7	2	11
PAb-240	4	6	1	11
Superficial spreading Melanoma (n = 12)				
PAb-1801	2	6	4	12
PAb-240	4	7	1	12
Nodular melanoma (n = 10)				
PAb-1801	1	6	3	10
PAb-240	0	6	3	9

histochemical detection of a reliable tumour marker would therefore be a great advantage.

Immunostaining of p53 protein was examined in 43 benign and malignant melanocytic lesions using two common antibodies (Oncogene Science, NY): PAb-1801 against human wild-type and mutant p53 and PAb-240 against mutant p53 protein. Formalin-fixed and paraffin-embedded routine specimens were used to examine the practical value of p53 immunostaining in diagnostic evaluation of these lesions.

Table 1 shows that there was no significant difference between benign, premalignant and malignant melanocytic lesions with respect to intensity of p53 immunostaining (χ^2 test), although some of the malignant melanomas showed a strong, dot-like staining in the cytoplasm of the tumour cells. As a rule, both nuclear and cytoplasmic positivity were present, corresponding to the results with fresh tissue [4]. These findings indicate that immunopositivity does not discriminate between benign and malignant melanocytic proliferations.

The positive staining in a large proportion of regular nevi was surprising. However, recent reports stress that deterioration of the p53 protein may be introduced by formalin fixation [5], and positivity may therefore be artificial and without biological significance in some cases. For these reasons, p53 immunostaining using the present antibodies on formalin-fixed, paraffin-embedded specimens should not be used in the routine evaluation of melanocytic lesions.

1. Hall PA, Ray A, Lemoine NR, Midgley CA, Krausz T, Lane DP. p53 immunostaining as a marker of malignant disease in diagnostic cytopathology. *Lancet* 1991, **338**, 513.
2. Heyderman E, Dagg B. p53 immunostaining in benign breast disease. *Lancet* 1991, **338**, 1532.
3. Clemente C, Cochran AJ, Elder DE, *et al*. Histopathologic diagnosis of dysplastic nevi. *Human Pathol* 1991, **22**, 313–319.
4. Stretch JR, Gatter KC, Ralfkiaer E, Lane DP, Harris AL. Expression of mutant p53 in Melanoma. *Cancer Res* 1991, **51**, 5976–5979.
5. Kawasaki Y, Monden T, Morimoto H, *et al*. Immunohistochemical study of p53 expression in microwave-fixed, paraffin-embedded sections of colorectal carcinoma and adenoma. *Am J Clin Pathol* 1992, **97**, 244–249.

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Unexpected *Prad*-1 Amplification in Multiple Simultaneous Localisations of Squamous Cell Carcinoma of the Head and Neck

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PRAD-1 is the most recent addition to the list of genes localised in 11q13 region. This gene may be equivalent to the cyclin D1 and it has been proposed that *Prad*-1 plays a key role in the regulation of cell growth [1]. Its clinical significance is not yet defined, but its amplification has been described in parathyroid adenomas, centrocytic lymphomas, breast adenocarcinomas, as well as in squamous cell carcinoma cell lines [2–5]. Analysed by polymerase chain reaction (PCR) techniques, *Prad*-1 is often coamplified with *hst*1 and *int*-2 genes, and in squamous cell carcinomas, these amplifications show a trend to be associated with poor prognosis [6]. In our previous multiparametric and prospective study, we found *Prad*-1 amplifications (with dopamine receptor gene as a control) in 27 out of 51 (53%) squamous cell carcinomas, and amplifications were related to small T volume and tumour vascularisation [7]. 7 other patients, presenting multiple simultaneous head and neck localisations, and biopsied at the principal tumour site, were analysed by PCR. Five tumours were amplified (three non-amplified normal

Table 1.

Patient number	Localisation	TNM (AJC-UICC 86)	Number of *Prad*-1 copies
5	Oral cavity and piriform sinus	T4N1	4
45	Hypopharynx and oesophagus	T1N0	2 (not amplified)
76	Oral cavity and oropharynx	T4N0	7
93	Oral cavity and oropharynx	T4N2c	4
114	Hypopharynx and oesophagus	T4N0	2 (not amplified)
120	Oral cavity and oropharynx	T3N0	12
141	Hypopharynx and oropharynx	T3N2c	7

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mucosas being control). Interestingly, amplifications were detected in primary tumours except in association with oesophageal localisations (Table 1). Our previous results suggest that *Prad*-1 may play an important role in the early steps of carcinogenesis in squamous cell carcinoma and may also be implicated in the pathogenesis of multilocalisations.

1. Mokotura T, Bloom T, Kim HG, *et al.* A novel cyclin encoded by a *bcl*1-linked candidate oncogene. *Nature* 1991, **350**, 512–515.
2. Rosenberg CL, Kim HG, Shows TB, *et al.* Rearrangement and overexpression of D11S287E, a candidate oncogene on chromosome 11q13 in benign parathyroid tumors. *Oncogene* 1991, **6**, 449–454.
3. Rosenberg CL, Wong E, Petty EM, *et al.* PRAD-1, a candidate BCL1 oncogene: mapping and expression in centrocytic lymphoma. *Proc Natl Acad Sci USA* 1991, **88**, 9638–9642.
4. Schuuring E, Verhoeven E, Mooi WJ, Michalides JAM. Identification and cloning of two overexpressed genes, U21B31/PRAD1 and EMS1, within the amplified chromosome 11q13 region in human carcinomas. *Oncogene* 1992, **7**, 355–361.
5. Lammie GA, Fantl V, Smith R, *et al.* D11S287, a putative oncogene on chromosome 11q13, is amplified and expressed in squamous cell and mammary carcinomas and linked to BCL-1. *Oncogene* 1991, **6**, 439–444.
6. Kitagawa Y, Ueda M, Ando N, *et al.* Significance of *int-2*/*hst*-1 coamplification as a prognostic factor in patients with esophageal squamous cell carcinoma. *Cancer Res* 1991, **51**, 1504–1508.
7. Klijanienko J, Bianchi AB, Cvitkovic E, *et al.* Prad-1 gene amplification in head and neck squamous cell carcinoma. Histoclinical analysis. *Human Path* (submitted).