The expression of vimentin in epithelial cells from human nasal mucosa

M. Kasper and P. Stosiek
Pathological Institute, District Hospital, 0-8900 Görlitz, Federal Republic of Germany

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Summary. The results of an immunohistological study of the normal human nasal mucosa show that there are frequently vimentin-positive cells detectable in addition to cytokeratins in the respiratory epithelium. The vimentin cells are probably ciliated and/or goblet type in origin. Furthermore, some co-expressing cells were found in basal parts of the submucous glands.

Key words: Cytokeratin – Vimentin – Human nasal mucosa

Introduction

It is now generally accepted that epithelial cells of various animal and human tissues and of different histogenetic origin can express two or more types of intermediate filaments, in contrast to previous considerations [17]. The list of epithelia which co-express cytokeratins and vimentin is growing (Table 1). This phenomenon is detectable in simple, atypical and differentiated squamous epithelia. Recently examined examples have been human enamel epithelium and pituitary epithelia [12, 13]. An explanation for the unusual co-expression of vimentin cannot yet be given, but it appears to be related to functional requirements rather than to germ layer derivation [18].

The present preliminary study used immunoperoxidase techniques with a panel of monoclonal antibodies and by double immunofluorescence methods to show that cytokeratin and vimentin are also expressed simultaneously in a subpopulation of nasal mucosal epithelial cells.

Materials and methods

Human nasal mucosa (n = 10) was used following tissue sampling for investigating dysplastic and polypoid alterations in hospitalized patients. Only histologically normal-appearing areas of the respiratory epithelium without any pathological changes were examined in this study. Furthermore, four samples of autopsic nasal tissues (8–12 h postmortem) from patients aged 41–72 years without clinically known respiratory dysfunction were included in this investigation.

Offprint requests to: M. Kasper

Figs. 1, 2. Human nasal mucosa. Immunofluorescent reactions using the polyclonal anti-cytokeratin antibody 1146 (Fig. 1) and immunostaining of the epithelial cells with the monoclonal antivimentin antibody V9, using indirect immunoperoxidase (Fig. 2). The dotted lines indicate the basal membrane. × 200
Fig. 3a, b. A human submucous gland. Double-label immunofluorescence for demonstrating vimentin a antibody V9 and b cytokeratin antibody 1146. × 400

Biopsy specimens were immediately embedded in phosphate-buffered saline (PBS) and frozen in liquid nitrogen. The immunoperoxidase methods were performed on 4-μm-thick frozen sections, as described previously [12].

The following monoclonal antibodies were used: anti-vimentin V9 (Boehringer, Mannheim, FRG); anti-vimentin VIM 3B4 (Progen, Heidelberg, FRG; a gift from Dr. R. Moll, Mainz, FRG); anti-cytokeratin A45-B/B3 [9]; anti-cytokeratin 18 (RCK 106, a gift from Prof. F. C. S. Ramackers, Nijmegen, The Netherlands); anti-collagen IV (1043 [8], kindly provided by Dr. J. P. M. Cleutjens, Maastricht, The Netherlands); anti-peroxidase [22] (kindly provided by Dr. P. Jantscheff, Berlin-Buch, GDR); anti-human secretory component (Dr. Behn, Sektion Biowissenschaften, Karl Marx University, Leipzig, GDR).

For double immunofluorescence to study the cytokeratin-vimentin sequence we used a rabbit polyclonal, cytokeratin antiserum 1146 (kindly provided by Prof. G. N. P. van Muijen, Nijmegen, The Netherlands), diluted 1:50 in PBS, monoclonal vimentin antibody V9, diluted 1:200, and FITC-coupled swine antibodies to rabbit (SEVAC, Prague, Czechoslovakia) and Texas-red-labelled goat antibodies to murine Ig (Dianova, Hamburg, FRG; kindly provided by Prof. H. Denk, Vienna, Austria). Both of the second antisera were diluted 1:20 in PBS. The details of the method have been described previously [10]. Negative controls were performed by replacing the primary antibody with PBS or non-related hybridoma supernatants.

Results

The human respiratory and glandular nasal epithelia displayed a diffuse immunoreaction for cytokeratins, using the broad range monoclonal antibody A45-B/B3, the CK-18 specific antibody RCK-106, and the polyclonal antiserum (Figs. 1, 4a).

The collagen IV specific monoclonal antibody marked the basal membrane (not shown), indicating the pres-