

nasal allergy

NIELS MYGIND MD

*Senior Lecturer in Otopathology
Otopathological Laboratory
University Ear Nose and Throat Clinic
and Institute of Pathological Anatomy
Rigshospitalet, Copenhagen, Denmark*

FOREWORD BY

A.W.FRANKLAND
MA, DM

*Guy's Hospital, London
Formerly Director, Allergy Department
St Mary's Hospital, London
President, European Academy of
Allergology and Clinical Immunology*

SECOND EDITION

BLACKWELL SCIENTIFIC PUBLICATIONS
OXFORD LONDON EDINBURGH BOSTON MELBOURNE

Contents

Foreword by <i>A.W. Frankland</i>	vii
Introduction	ix
Acknowledgements	x
Abbreviations	xii
PART I: ULTRASTRUCTURE	
1 Structure and ultrastructure of the nose	3
2 Applied physiology of the nose	39
PART II: IMMUNOLOGY	
3 The immune system	59
4 Allergens and allergen extracts	84
5 Genetics and prevalence of atopic allergy	103
6 Sensitization	106
7 Mast cell degranulation	116
8 Significance of IgG antibodies	133
9 Non-immunological factors	140
10 Inflammatory reactions	155
11 Eosinophil leucocytes	170
12 Allergy diagnosis	182
13 Immunology of nasal secretion	199
PART III: DISEASE	
14 Hay fever	219
15 Perennial rhinitis	224
16 Nasal polyps	233
17 Secretory otitis media	239



Fig. 1.2 (cont.)

(c) Partly ciliated pseudostratified epithelium. Some cells are covered by microvilli and others also by 50-100 cilia. The central cell is a filled goblet cell with central displacement of microvilli ($\times 3,300$). From Mygind (1975). By courtesy of *Rhinology*.

being very complex (Fig. 1.4). Formerly it was believed that the central microtubules act as a 'skeleton' and the peripheral microtubules as 'muscles' for the ciliary motion. This is not correct as the microtubules are not contractile, but they are involved in the mechanism of ciliary motion (Satir 1974).

The energy for moving the cilium is supplied, as in other cellular work processes, by the breakdown of adenosin triphosphate (ATP). The enzyme that accomplishes this breakdown in cilia is an ATP-ase, named dynein. It is localized to a series of projections called dynein arms that protrude from one side of each of the nine outer doublets (Fig. 1.4). According to Satir (1974) energy production in the dynein arms causes peripheral microtubules to slide past one another, and shear resistance in the

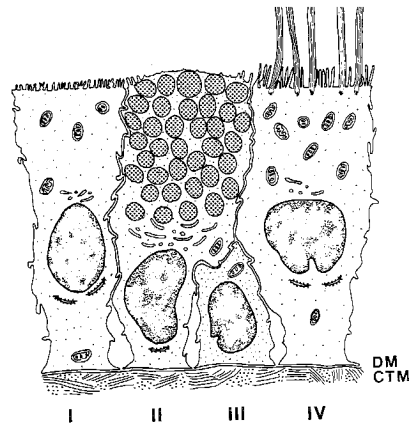


Fig. 1.3. Transmission electron microscopic diagram of the four cell types in nasal respiratory epithelium.

I: Non-ciliated columnar cell, covered by microvilli of uniform length. II: Goblet cell, packed with mucigen granules. Slight prominence of the cell at the luminal surface, and disappearance of most microvilli. III: Basal cell. IV: Ciliated columnar cell, covered by cilia and microvilli of uniform length. The cilia contain microtubules and are anchored to the cell surface by the basal bodies. Many mitochondria in the luminal part of the cell.

DM: Double-membrane, which constitutes the electron microscopic basement membrane. CTM: Connective tissue membrane, which together with the double-membrane constitute the light microscopic basement membrane.

cilium changes sliding to bending. The hypothesis that this is the mechanism of ciliary motion is called the *sliding-microtubule hypothesis*.

In a few individuals a hereditary *lack of dynein arms causes ciliary non-motility* and result in severe chronic airways infection (Fig. 1.5) (Pedersen & Mygind 1976).

The cilia in the human nose are $0.3 \mu\text{m}$ in diameter, $4-6 \mu\text{m}$ long and there are about 100 per cell.

There exists some doubt regarding the number of cilia per cell. Transmission electron microscopy of human trachea showed the number to be 250 (Rhodin 1974). Estimated by scanning electron microscopy the number is 50-100 in the anterior part of the human nose (Fig. 1.2c) (Mygind & Bretlau 1974a), and 40-50 in the middle ear (Shimada & Lim 1972). Differences in the methods used and the mucosa studied may account for the discrepancies.