

with antigen processing (TAP) deficiency is however missing. TAP proteins are crucial for peptide loading on newly synthesised Human Leukocyte Antigen (HLA) class I molecules, and the consequence of TAP deficiency is a very low cell-surface expression of these HLA class I molecules.^{4 5} Clinically, besides skin ulcers, the patients suffer from chronic bacterial infections of the upper and lower airways with bronchiectasis.^{4 5} Although the known cases of this immune deficiency are rare (around 30 patients reported in the literature), there might be much more, as the diagnosis of idiopathic bronchiectasis might be made in most instances without having checked for TAP deficiency (by staining of patients' peripheral blood mononuclear cells with an anti-HLA class I antibody which reveals by flow cytometry a very low expression level compared to healthy control donors, and/or the absence of HLA class I molecules by serotyping).

In our opinion, it is important to detect TAP deficiency as soon as possible in order to optimise the treatment of the patients, and thus it should be systematically included in the diagnostic algorithms applied to the exploration of chronic respiratory infections and bronchiectasis.

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TAP deficiency is also a cause of bronchiectasis

We read with interest the recent bronchiectasis review by Whitters and Stockley.¹

The authors provide a comprehensive overview of the changes in the immune response that might favour bacterial colonisation of the lower airways and in a second step initiate the vicious cycle leading to chronic inflammation and recurrent infections. Their description of the innate immune system in the lung includes antibacterial peptides, neutrophils and toll-like receptors. Although this is correct, Natural Killer (NK) cells might also be mentioned, as their role in the immune response to bacteria (besides their antiviral and antitumor functions) has been

repeatedly documented.^{2 3} NK cells participate in innate immune defence mechanisms through (i) the release of cytotoxic granules containing, among others, the antibacterial factors granzysin, α -defensins and cathelicidin,² and (ii) cytokine production that stimulates both innate and adaptive immunity. In addition, they have immunoregulatory properties able to limit excessive immune responses.³ Thus, it is highly likely that NK cells are one of the actors participating in the pathogenesis of bronchiectasis. The same may hold true for the more recently discovered (non NK) innate lymphoid cells, but more work will be necessary to establish this.

Another important point of the review is the useful list of the causes of bronchiectasis in which transporter associated

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