Alteration in expression of the rat mitochondrial ATPase 6 gene during Pneumocystis carinii infection

ABSTRACT

Over-expression of the ATPase 6 gene occurs in type II pneumocytes and Clara cells during infection with P. carinii.

INTRODUCTION

This paper presents a comparative study of the effect of Pneumocystis carinii infection on mitochondrial ATPase gene expression in patients with lung cancer and healthy controls. The study was conducted on patients with lung cancer and healthy controls and compared the expression level of the mitochondrial ATPase gene in the lung cancer cells and the healthy controls. The results showed that the expression of the mitochondrial ATPase gene in the lung cancer cells was significantly higher than in the healthy controls in comparison to the lung cancer cells. The mRNA level of the mitochondrial ATPase gene in the lung cancer cells was significantly higher than in the healthy controls. The expression level of the mitochondrial ATPase gene in the lung cancer cells was significantly higher than in the healthy controls. The mRNA level of the mitochondrial ATPase gene in the Pneumocystis carinii, an immunocompromised pathogen with a high mortality rate and endemic lung disease, is responsible for pneumonia in patients with severe morbidity and mortality, but the specific relationship between this organism and the host cell is unclear. It is hypothesized that P. carniivisie interacts with the type I pneumocyte and after interacting with it, it forms trophozoites that act as binding mediators to the target cell. The major surface glycoprotein of The first line of defense against infection is the lungs, and alveolar macrophages interact with P. carinii to initiate this process. Phemotaxis, which is a major surface glycoprotein, has been shown to be chemotactic and to mediate the growth of macromolecules like phagocytis in vitro. When P5. The attachment of P. carinii organisms to type II pneumocytes is uncertain, but they do maintain the structural integrity of alveoli for gas exchange. They produce alvéolar surfactant and differentiate into type I pneumatocyte after lung injury. Type II pneumonia (PcP) also raises the production of surficial-A (SP-AA) in patients with PCA, and the secretion of phosphatidylcholine from type III cells is decreased, leading to a deficiency of PS2-based surigma PcC in people with an individual This study aimed at finding the changes in host cell gene expression that are altered by infection with pseudofungus (P. carinii bacteria) We have used mRNA differential display to compare these two models and found that the mitochondrial ATPase 6 gene is over-expressed in P.carinichiosis-infected cells, while in rats it was found only that this gene expressed more freely than in mice and then increased in type II pneumocytes and Clara cells

CONCLUSION

Remarkable conclusions Using the method of mRNA differential display, we were able to identify genes that have been altered in Pneumocystis carinii-infected hosts. The nucleotide sequence of one of these fragments was identical to the gene that encoded the F0F1-ATPase 6 complex, and the resulting overexpression of the same gene was detected by Northern blot analysis of total RNA extracted from P. cardiniis-infused rat lung compared to those of Mraxrax C type II