Development of porcine embryos reconstituted with somatic cells and enucleated metaphase I and II oocytes matured in a protein-free medium

ABSTRACT
After nuclear transfer, somatic cells from porcine M I oocytes may develop into blastocysts.

INTRODUCTION
Pneumocystis carinii, an immunodeficient organism that causes high morbidity and mortality rates, is responsible for pneumonia in patients with a severe immune response. The type I pneumocyte is considered the target cell for this organism, and its trophozoites attach to the host cell upon contact with it. The up-regulation of integrin expression on the surface of cultured lung cells by the major surface glycoprotein of P. carinii was found to facilitate attachment to type I pneumocytes. Other possible mediators include laminin, vitronectin and mannose. Alveolar macrophages interact with Prion bacteria as the first line of defense against infection. The primary surface glycoprotein of P. carinii has been shown to act as a chemotactic agent for macrophages and monocytes in vitro, while alveolar macrofemoral macrocytization is achieved by its mediated interaction with fibronectin. Alveolar macrophages have been shown to release TNF-, prostaglandin E2 and leukotriene B4 as well as other cytokines which are potent inhibitors of lung inflammation and lung injury; these events are early markers in the acute response to infection and the expulsion of P. carinii organisms out of the lungs, although it is unclear whether P Carinii organisms can be either reliant on P. carinae or maintain the structural integrity of alveoli for gas exchange, producing alvéolar surfactant, and evolving into type I pneumocytes after lung injury. The production of surfactant protein-A (SP- A) in patients with P. carinii pneumonia (PcP) has been found to be higher in type II pneumocytes, and this increase is linked to the organism's burden in the lungs. The secretion of phosphatidylcholine from type III cells is also suppressed upon P infection. P. carinii infection results in a deficiency of phosphatidylglycerol and the loss of surfactant function in patients with PcP infection. Our aim was to identify changes in host cell gene expression that occur in response to PPC infection by comparing gene Expression patterns in Pryorhizan virus (Glasmodiumium tuberculosis) over time. Through mRNA differential display, we observed over-expression of the mitochondrial ATPase 6 gene in P. carinius-infected and mock-Infused cells after PPO infection in rats, as well as over expression of this gene also in FCL (which is expressed through catabolic acid deposition) in type II pneumocytes and Clara cells.

CONCLUSION
We have demonstrated that polyclonal anti-MCM2 antibodies offer dependable staining results consistent with fixed tissues without the need to search for specific antigens. The interpretation of the results is simple because there is a significant difference between normal bronchoepithelium and premalignant lesions. MCM2 is a simple marker that can be used to assess the progression and regression of morphologically abnormal lesions in primary lung cancer prevention studies and early detection of lung carcinoma in screening studies.