Iron is essential for the growth and survival of the cells in our body as well as the pathogens attacking them. As such, cells have developed complex mechanisms of both regulating their iron stores and withholding iron from microbial invaders. In particular, lung epithelial cells are a target for fungal infection because of constant exposure to airborne pathogens. Upon fungal infection in the airway, an innate immune response is initiated to combat the pathogen. The ensuing struggle is a battle for iron, with the host triumphing if it can deprive the fungus of enough iron and the fungus winning if it can overcome the iron deficiency induced by the host’s immune proteins. We present a logical model of iron metabolism in lung epithelial cells exposed to proinflammatory cytokines and the fungi Aspergillus fumigatus and Alternaria alternata. It makes predictions about the way in which lung epithelial cells sequester excess extracellular iron, along with how internal iron is stored and released from the cell. Additionally, it allows for the testing of conditions that are experimentally intractable, a process beneficial to many fields, as novel interactions and relationships can be explored without laboratory experimentation. (Received August 21, 2011)