THE EXPRESSION OF PPAR-γ RECEPTOR IN ADULT AND NEONATAL LUNGS DURING RESPIRATORY VIRAL INFECTION

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Abstract:
Respiratory viral infection is a significant cause of morbidity and mortality worldwide. Severe illness during a viral lung infection may be due to inflammation as a result of the host immune response, rather than direct damage by the virus itself. Previously, a mouse model of respiratory viral infection demonstrated that there was significantly reduced inflammation in neonatal lungs compared to adults. This occurred even though the viral titer and viral clearance was similar in both adults and neonates. The reduced inflammation in neonates was associated with significantly higher levels of Prostaglandin D2 (PGD2). PGD2 is converted into prostaglandin J2 (PGJ2), which binds to the receptor PPAR-γ. Importantly, studies using PPAR-γ agonists in adult lungs during infection reduced inflammation. In contrast, treatment of neonates with PPAR-γ antagonists increased susceptibility to infection. These studies suggested that the anti-inflammatory effects of PGD2 might be through its conversion to PGJ2 and subsequent binding to the PPAR-γ receptor. To date, the expression of PPAR-γ in adult and neonatal lungs during respiratory viral infection has not been determined. Adult and neonatal C57BL/6 mice were infected with 500 pfu/g body weight Sendai virus (SeV) and lungs were fixed in formalin at various times after infection. Five μm lung sections were stained with an anti-PPAR-γ antibody. Results showed that PPAR-γ was expressed in the airway epithelium of uninfected adults and neonates. This expression decreased in both after viral infection. To analyze PPAR-γ expression in isolated airway epithelial cells, tracheas from uninfected C57BL/6 adult mice were treated with pronase and the epithelial cells were cultured in an air-liquid interface. These cells were infected with SeV (MOI=0.1) and RNA was isolated at 24, 48 and 72 hours post-infection. PPAR-γ mRNA expression was measured by qPCR. The results showed no difference in PPAR-γ expression between uninfected and infected cultured airway epithelial cells. This suggests that viral infection of epithelial cells does not directly affect PPAR-γ mRNA expression. Therefore, the mechanism of reduced PPAR-γ protein expression in vivo will need to be determined.