Impaired mouse beta-defensin 4 response to A(H1N1)09 influenza virus challenge results in severe infection in respiratory tract of senescent mice

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Mouse beta-defesin 4 (mBD4) is mainly produced by epithelial cells and should play an important role during influenza infection. Firstly, we showed that mBD4 was quickly increased in LA4 cells at 6 hours after inoculation with A(H1N1)09 virus and remained up-regulated until 24 hours post infection. The induction of mBD4 was positively correlated with initial virus inoculation doses. The time course for the induction of mBD4 was in parallel with the up-regulation of inflammatory cytokine IL-6 and TNF-α. The expression of mBD4 in mouse respiratory tissue was studied and compared between young (6-8 weeks) and aged (72 weeks) mice. IHC staining of formalin fixed mice trachea and lung tissues showed there was stronger expression of mBD4 in epithelial cells lining trachea and bronchioles in aged mice which indicated a higher basal expression of mBD4 in aged mice respiratory tissues. Upon infection with A(H1N1)09 virus, a quick induction of mBD4 in young mice trachea tissue was observed at 12 hours p.i. and maintained at this level until day 4 p.i. However, despite the higher basal level, there was no further induction of mBD4 in aged mice trachea tissues. For the lung tissues, delayed induction of mBD4 was observed in aged mice following A(H1N1)09 infection, but no increase was observed in the young mice lung tissues. Accordingly we also see a lower viral load and cytokine levels in young mice. After giving the recombinant mBD4 protein after infection of A(H1N1)09 in aged mice, we saw a reduced viral load in respiratory tissues.

Biography
Zhu Houshun is currently a PhD student in the Department of Medicine of the University of Hong Kong. His research mainly focuses on the treatment of influenza infection.

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