

## Natural Killer Cell (NK Cells) Responses against Viral Infections

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### DESCRIPTION

Natural Killer (NK) cells are large granular lymphocytes that mediate natural defenses against some viral infections and tumor cells. NK cells are essential for resistance to various viral infections such as Epstein-Barr virus, human cytomegalovirus, varicella-zoster virus, and herpes simplex virus. The role played by NK cells during influenza virus infection is complex. Several studies have highlighted the central role of NK cells in controlling Influenza A Viruses (IAV) infection. This is because defective NK cell activity or depletion of NK cells leads to delayed viral clearance and increased morbidity and mortality. However, there are also examples of NK cells exacerbating morbidity and pathology during lethal influenza virus infection in mice, suggesting that NK cells play a dual role during influenza virus infection in mice, providing either beneficial or it suggests that it exerts a harmful function. In addition, Interleukin-22 (IL-22) production by NK and NKT cells plays an important role in repairing epithelial damage caused by IAV. Recent studies have shown that NK cells are also involved in thymic atrophy during IAV infection. NK cells, on the other hand, play an important role in bridging innate and adaptive immune responses to IAV. The extensive and complex role of NK cells in IAV infection requires further in-depth investigation.

Previous studies have shown that host genetic background strongly influences the response to IAV infection. However, the function of NK cells during IAV infection on various host genetic backgrounds remained unclear. NK cells have different roles in IAV infection, depending on the genetic background of the host. To investigate whether NK cells contribute to the genetic resistance or susceptibility of different inbred mouse

strains, researchers used six mice including C57BL/6, BALB/c, C3H, DBA/2, FVB and also 129 mice. We depleted NK cells in an inbred mouse strain. Surprisingly, NK cells played a significant protective role only in the 129 mice upon high-dose IAV infection. Depletion of NK cells significantly increased virus titers in 129 mice, but not in the other five mouse strains. Compared to NK cells from C57BL/6 mice, NK cells from 129 mice were shown to be rapidly activated and accumulated in infected lungs. A rapid and potent response of NK cells efficiently controlled early pulmonary viral replication and conferred survival privilege in 129 mice.

Influenza A viruses, with 18 hemagglutinin subtypes and 11 neuraminidase subtypes, consists of thousands of strains, some of which have been responsible for recent influenza epidemics. The NK cell response to high-dose H3N2 virus infection was also faster in 129 mice than in C57BL/6 mice, although the overall size was much smaller than the PR8 virus and the NK cell response was also affected by virus strains.

### CONCLUSION

Three main factors are involved in determining the variability in severity of influenza virus infection: virus intrinsic virulence, acquired host factors (such as immunity and comorbidities), and intrinsic host susceptibility. This demonstration that host genetic background significantly influences the kinetics and magnitude of NK cell responses during IAV infection and thus related NK cell functions is unique. Further investigation of the molecular mechanisms leading to differential activation of pattern recognition receptors in 129 and C57BL/6 mice may identify novel genetic limiting factors for severe influenza infection.

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