Increased susceptibility to bacterial superinfection as a consequence of innate antiviral responses

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The reason why severe localized or systemic virus infections enhance and aggravate bacterial superinfection is poorly understood. Here we show that virus-induced IFN type I caused apoptosis in bone marrow granulocytes, drastically reduced granulocyte infiltrates at the site of bacterial superinfection, caused up to 1,000-fold higher bacterial titers in solid organs, and increased disease susceptibility. The finding that the innate antiviral immune response reduces the antibacterial granulocyte defense offers an explanation for enhanced susceptibility to bacterial superinfection during viral disease.

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