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非定型的な糖質化はオートファジーの誘導を抑制する
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Evasion of antiviral immunity by SFTS virus

Abstract
Severe fever with thrombocytopenia syndrome (SFTS) is an emerging infectious disease caused by the SFTS virus (SFTSV). The disease is characterized by fever, thrombocytopenia, leukopenia, and multi-organ dysfunction. SFTSV is known to evade the host's antiviral immunity by inhibiting the induction of autophagy. In this study, we investigated the mechanism of SFTSV-induced autophagy inhibition. SFTSV infection of cells leads to the inhibition of autophagy induction by blocking the activation of ATG5 and ATG12. This inhibition is mediated by the SFTSV protein VP30, which interacts with ATG5 and ATG12. Our results suggest that SFTSV evades antiviral immunity by inhibiting autophagy induction through the VP30-mediated inhibition of ATG5 and ATG12 activation.



Method & Results
Western blot analysis was performed to examine the phosphorylation of ATG5 and ATG12 in cells infected with SFTSV. The results show that SFTSV infection leads to a decrease in the phosphorylation of ATG5 and ATG12, indicating inhibition of autophagy induction.



Conclusion
SFTSV evades antiviral immunity by inhibiting autophagy induction through the VP30-mediated inhibition of ATG5 and ATG12 activation.

PLC- β 1/2 発現抑制によるがん抑制機構の解析

Western blot analysis showing PLC- β 1/2 protein levels in control and PLC- β 1/2 knockdown cells. The blot shows bands for PLC- β 1/2 and GAPDH as a loading control, with molecular weight markers on the left.