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Radicicol誘發聚合肌動蛋白促進腦神經壞死病毒感染細胞

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腦神經壞死病毒是引發海水魚腦神經壞死病的病源體，其高發病性及高死亡率，造成養殖業嚴重經濟損失。Radicicol為HSP90蛋白的特異性抑制劑，可抑制病毒的複製。本研究發現高濃度的radicicol(15μM)，雖可抑制腦神經壞死病毒的複製，但在低濃度radicicol(0.1 μM)反而可促進病毒的複製。為了釐清此現象，我們發現0.1 μM radicicol處理細胞，促進病毒附著於細胞及細胞胞飲作用。若抑制劑阻斷胞飲酸化作用時，可延緩radicicol促進作用。而胞飲作用通常與細胞膜上肌動蛋白的聚合作用有關，radicicol可誘發細胞膜肌動蛋白聚合，當添加肌動蛋白聚合作用仰制劑時，亦可延緩radicicol促進作用。本研究藉由化學抑制劑的阻礙作用得知，radicicol誘發細胞膜肌動蛋白聚合作用，促進細胞胞飲而增強對病毒感染力。

關鍵字：radicicol，細胞胞飲，肌動蛋白，腦神經壞死病毒
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F-actin induced with radicicol enhances NNV infection in fish cell line

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Nervous necrosis virus (NNV) belongs to Betanodaviruses, members of the family nodaviridae are the causal agent of viral nervous necrosis (VNN) in many species of marine fish. NNV infected to larval and juvenile marine fish worldwide causing severe morbidity and mortality and significant economic loss in aquaculture. Radicicol, Hsp90-specific inhibitor, suppresses nodaviruse replication in infected cells. In the study, we found high concentration of radicicol (15μM) could inhibit NNV replication in GF-1 fish cell line, but we also found that low concentration (0.1 μM) of Radicicol enhanced NNV replication. Because the entry of NNV into cells depends on the endocytic pathway, adding 0.1 μM radicicol in medium could improve the attachment and entry of NNV to GF-1 cells . Treatment of radicicol-inducecd GF-1 cells with the endosomal acidification inhibitors potently attenuated the accumulation of protein A and protein α. Cells treated with radicicol induced actin stress fiber formation. The disruption of actin microfilaments by cytochalasin D significantly decreased radicicol-inducecd NNV replication. Our data indicated that 0.1 μM radicicol induced stress fiber F-actin formation, which enhance viral endocytosis and infection.

Key words: radicicol, endocytosis, F-actin, betanodaviruse
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