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A tumor necrosis factor receptor-like protein encoded by Singapore grouper iridovirus modulates cell proliferation, apoptosis and viral replication

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It has been demonstrated that tumor necrosis factor receptor (TNFR) homologues encoded by viruses are usually involved in virus immune evasion by regulating the host immune response or mediating apoptotic cell death. Here, a novel TNFR-like protein encoded by Singapore grouper iridovirus (SGIV VP51) was cloned and characterized. Amino acid analysis showed that VP51 contained three cysteine-rich domains (CRDs) and a transmembrane domain at its C terminus. The expression of VP51 *in vitro* enhanced cell proliferation and affected cell cycle progression via altering the G1/S transition. Furthermore, VP51 overexpression improved cell viability during SGIV infection via inhibiting virus-induced apoptosis, evidenced by the reduction of apoptotic bodies and the decrease of caspase-3 activation. In addition, overexpression of VP51 increased viral titer and the expression of viral structural protein gene MCP and cell proliferation promoting gene ICP-18. In contrast, the expression of the viral apoptosis inducing gene, LITAF, was significantly decreased. Although all three CRDs were essential for the action of VP51, CRD2 and CRD3 exerted more crucial roles on virus induced apoptosis, viral gene transcription and virus production, while CRD1 was more crucial for cell proliferation. Together, SGIV TNFR like products not only affected cell cycle progression and enhanced cell growth by increasing the expression of the virus encoded cell proliferation gene but also inhibited virus induced apoptotic cell death by decreasing the expression of the viral apoptosis inducing gene. Our results provided new insights into understanding the underlying mechanism by which iridovirus regulated the apoptotic pathway to complete its life cycle.

Biography

Yepin Yu has completed his Bachelor degree from Xiamen University and currently pursuing Doctorate in Marine Biology in South China Sea Institute of Oceanology, Chinese Academy of Sciences under the Prof. Qin, the finder of Singapore Grouper Iridovirus (SGIV). His academic interests include viral immune evasion and virus-host reaction. He has published 3 papers in reputed journals.

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