Mutation of the first ATG ORF-3 of Chiken Anemia Virus into ACG completely abolish the apoptin production

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Abstrak

To test effect one point mutation one the first initiation codon of Chiken Anemia Virus (CAV) open reading frame-3 (ORF-3), an opoptin knocked out expressor plasmid pCLS-VP3(-) and a CAV opoptin knocket out plasmid pCAV/Ap(-) were constructed. In both plasmids, the first ATG in COS-1 cells tranfectedwith pCLS-VP3(-) using western blotting and immunofluorescence type. After released from pCAV/Ap(-), the complete genome of CAV/Ap(-) was ligated to form the replicative form. the oppotin prodective was completely abolished in MDCC-MSB1 cells transfected with replicative form of CAV/Ap(-). The opoptin production was fully regained after a reverse mutation into CAV/Ap(-)RM. These data shows the first evidence that mutation of the first ATG of ORF-3 into ACG could completely abolish the production of apoptin