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Critical roles of the meq-clustered micrornas of marek's disease virus in oncogenesis

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Marek's diseasevirus(MDV)induces rapid-onset T-cell lymphomain its natural host and is regarded as anideal model for investigating viral microRNAs in tumorigenesis. To investigate the roles of the Meq-clustered miRNAsin MDVoncogenesis,we have constructed a series of mutants by bacterial artificial chromosome mutagenesis in GX0101, avvMDVstrain, and demonstrate that the Meq-clustered miRNAsare not essential forreplication of MDV and have no effects on the early cytolytic or latent phases of the developing disease. However, compared toGX0101, mortality of birds infected with GXΔmiR-M2,GXΔmiR-M3, GXΔmiR-M4,GXΔmiR-M5, GXΔmiR-M9,GXΔmiR-M12andGXΔMeq-miRswere reduced from 100% to 18%, 30%, 18%,48%, 24%, 14%and 4%,and gross tumor incidences reduced from 28% to 8%,4%, 4%, 12%, 8%, 0%and 2%, respectively, suggesting thatin MDV oncogenesis, thesemiRNAs possibly play different roles, so miR-M12 may be a more important regulator, followed by miR-M2, miR-M3,miR-M4 and miR-M9 while miR-M5 may be lesssignificant. Furthermore, we have identified thelatent transforming growth factor beta (TGF-β) binding protein 1 (LTBP1) as a critical hosttarget of miR-M4-5p, aviralanalogofmiR-155. We found thatdown-regulation of LTBP1 expression by miR-M4-5p led to a significant decrease of the secretion and activation of TGF-β1, with suppression of TGF-β signaling and a significant activation ofthe expression ofc-Myc, a well-known oncogene which is critical for virus-induced tumorigenesis. Our findings reveal anovel and important mechanism to understand howmiR-M4-5p contributes to MDV-induced oncogenesis.

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