



Title	INHIBITION OF AUJESZKY'S DISEASE VIRUS REPLICATION BY A CHIMERIC TRANS-GENE PRODUCT REPRESSING TRANSCRIPTION OF THE IMMEDIATE-EARLY GENE
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INHIBITION OF AUJESZKY'S DISEASE VIRUS REPLICATION BY  
A CHIMERIC *TRANS*-GENE PRODUCT REPRESSING TRANSCRIPTION  
OF THE IMMEDIATE-EARLY GENE

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To establish a strategy for intracellular immunization against Aujeszky's disease virus (ADV), various *trans*-genes were constructed and analyzed for their inhibitory effect on transcription of the immediate-early (IE) gene and viral replication.

*Trans*-genes encoding tail-truncated Vmw65 of herpes simplex virus type 1 lacking the transcriptional activation domain, the DNA binding domain of ADV IE protein (IE180) and their fusion protein were constructed. The inhibitory activity of each *trans*-gene product on transcription under the control of the ADV IE promoter was determined by the chloramphenicol acetyltransferase (CAT) assay. In a transient expression assay, CAT expression in the cells transfected with the chimeric *trans*-gene encoding the fusion protein was one-third of that in the control cells. The inhibitory effect was similar to that in the cells transfected with the ADV IE gene encoding IE180, which confers autoregulation. It was found by a CAT assay using a reporter plasmid containing the ADV promoter and enhancer that expression of the chimeric gene reduced the CAT product. On the other hand, no inhibitory effect was observed in the cells transfected with the *trans*-genes encoding tail-truncated Vmw65 or the DNA binding domain of IE180, even in the case of cotransfection with both genes.

Stable cell lines of HeLa cells transfected with the chimeric gene encoding the fusion protein were established. In these cell lines synthesis of IE mRNA was inhibited upon virus infection. Virus growth was 100-fold less in the cell lines than in the control cells. These observations suggest that the inhibition of ADV replication in the transfected cell lines was due to interference with transcription of the ADV IE gene by the fusion protein.