The transcriptional transactivation function of HBx protein is important for its augmentation role in hepatitis B virus replication[1].

Tang H*, Delgermaa L, Huang F*, Oishi N, Liu L*, He L*, Zhao L*, Murakami S.

The roles of HBx in regulating HBV transcription and replication were examined with a transient transfection system in HepG2 using wild-type or HBx-minus HBV genome constructs, and a series of mutation HBx expression plasmids. HBx has augmentation effects on HBV transcription and replication as HBV mutant genome with defective X gene led to decreased levels of 3.5-Kb HBV RNA and HBV replication intermediates, and these decreases can be complemented *in trans* by ectopic expression of HBx. The truncation mutant HBx-D1 (aa. 51-154) containing the coactivation domain is enough for this function. By alanine scan mutagenesis strategy, the regions between aa 52 - 65 and aa 88 -154 are important for the augmentation function of HBx in HBV replication. With reporter gene analysis, the transactivation and coactivation activities of HBx are well coincide with its augmentation function in HBV transcription and replication. Therefore HBx has important role in stimulating HBV transcription and replication, and the coactivation function of HBx may be critical for the augmentation effect on HBV replication. (*The project is based on the international collaboration between Sichuan Univ. and Kanazawa Univ,)

Reference 1: Tang H, et al., (2005) J. Virol., 79: 5548-5556.

Figure illustrates quantitative analysis of the 3.5-kb HBV RNA and HBV DNA replication intermediates of 1.2 genome-unit of wild and defective in X-ORF in the absence or in the presence of varying amount of the HBx-expression plasmid.