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The effect of TPA on promoters expression of HTLV-1 and HIV-1 viruses in lymphocytes and epithelial Cells

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HTLV-1 and HIV are retroviruses involved in different human diseases. However, following infection, these viruses inter into a latent state. Since the infected cells, in the latent state of these viruses, contain low Tax and Tat levels, we hypothesize that these viruses activation must be independent of Tax or Tat. In the present research, we focus on exploring the mechanism of activation of these viruses by TPA, which is one of the stress-inducing agents. Our results showed that HIV-1 and HTLV-I LTRs were successfully activated by TPA. We demonstrated that TPA treatment of Jurkat and MCF-7 cells for more than 24 hr resulted in a sever depletion of PKC isoforms. Therefore, to explore the role of PKC in the effect of TPA on these LTRs, we treated the cells with TPA for different periods of times, and transfected them with HTLV-1 LTR-LUC or HIV-I LTR-LUC. While long exposure to TPA considerably reduced the HIV-1 LTR basal expression, it strongly stimulated the expression of HTLV-I LTR. However, short exposure to TPA stimulated only the HIV LTR. We noted that in Jurkat and MCF-7 cells PKC-α and PKC-Σ inhibited the HTLV-1 LTR expression, whereas PKC-β2 and PKC-η stimulated it, while PKC-α and PKC-Σ stimulated the HIV-1 LTR expression. Thus our data indicate that while the effect of TPA on HIV-1 LTR is strictly dependent on PKC activity, its effect on HTLV-I LTR is exerted via a different pathway that does not require PKC activation but rather seems to be antagonized by the activated PKC.

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