52

A novel soluble factor (HIF) from CEM T cell line induced by an extract from Pinus parviflora Sieb et Zucc can inhibit the replication of human immunodeficiency virus in vitro.

Patrick K. Lai, W.G. Bradley, Y. Tamura,
A. Tanaka & M. Nonoyama.
Laboratories of virology, Tampa Bay Research Institute,
10900 Roosevelt Boulevard, St. Petersburg, FL 33716.

We showed that an extract (PC6) from cones of P. parvifloria Sieb et Zucc, induced the human T cell line, CEM, to produce a pepsin-sensitive soluble factor(s) that could inhibit the replication of HIV-1 in CEM T cells, in U937 histiocytes, THP-1 monocytes and in mitogen activated human tonsillar mononuclear cells. Indirect immunofluorescence staining and PCR analysis of the PC6-induced CEM cells revealed the absence of known lymphokines/cytokines, except GM-CSF, IL3, $TGF\beta_1$ and TNF-alfa. Recombinant IL3, TNF-alfa and $TGF\beta_1$ did not inhibit HIV-1 replication in CEM cells. Neutalizing antibodies to GM-CSF and TNF-alfa also did not abrogate the anti-HIV-1 impact of the PC6-induced HIV-1 inhibiting factor(s). Thus, the anti-HIV-1 factor (HIF) induced by PC6 may be novel. Molecular seive separation showed that the anti-HIV-1 factor(s) is smaller than 30 kDa. Gel-fitration and ion-exchange chromatography by HPLC and FPLC showed that HIF has a molecular weight of 9,000 to 10,000.

53

Inhibition of HIV Replication by Prostaglandin A.

H. Ankel*., O. Turriziani., G. Antonelli., and F. Dianzani.
Institute of Virology, University of Rome "La Sapienza", 00185 Rome, Italy.

Antiviral effects of prostaglandins of the A series (PGA) on ss(-) RNA, ss(+) RNA and ds DNA viruses have been observed previously (Santoro et al. Science 1980, Ankel et al. J. Gen. Virol. 1985, Benavente et al. J. Gen. Virol. 1984). This report describes the antiviral action of PGA on human immunodeficiency virus (HIV), a member of yet another group. We have investigated the effects of PGA_1, on the HIV-1 laboratory strain III B and on an isolate from an AIDS patient, P-1. Cell cultures of C8166 cells were infected at a moi of approximatively 1, 0.1 and 0.01. One hr after infection, PGA_1 was added and infectious progeny was determined after 24, 48 and 72 hrs, respectively. In all cases 90% reduction of virus yield occurred at 10 μM concentration of the inhibitor and an overall yield reduction of 3-4 orderds of magnitude resulted at the highest non-toxic dose of PGA. Both number and size of HIV-induced syncytia as well as amounts of viral antigen were likewise drastically reduced. PGs of the B and E series had no anti-HIV effects, even at concentration as high as 40 μM. Our results provide new documentation of the broad antiviral spectrum of PGAs.

*Supported by Senior International Fellowship 1 F06 TWO 1519-01 from the Fogarty Center, N.I.H.
This work was supported by a grant from Ministero della Sanita' (Progetto AIDS).