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HIV: Current Research

Short Communication

Morphine's Interaction with HIV Infection: Its Role in HIV-Associated Neurocognitive Disorder

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DESCRIPTION

With over 33 million individuals infected with HIV, the HIV epidemic continues to be a serious public health issue and concern in the United States and around the world. Since injection drug users are more likely to experience HIV-related neurocognitive dysfunctions than HIV-infected people who do not use drugs, the prevalence of drug usage among HIV-infected people is a significant problem that is rising quickly. The brain is one of the main targets for HIV and many recreational drugs. Opiate misuse may increase the chance of contracting HIV, developing neurological malfunction, and developing AIDS, according to evidence. There is a paucity of knowledge on morphine's role as a cofactor in the neuropathogenesis of HIV. This study provides an overview of the findings that aid in comprehending the usage of morphine with neurological impairment in HIV infection. According to studies, morphine increases the risk of contracting HIV-1 by inhibiting IL-8, downregulating chemokines, and upregulating HIV coreceptors in the opposite direction. Additionally, morphine upregulates cAMP response element-binding protein while activating MAPK signaling (CREB). New therapeutic approaches to combat HIV-1 infection in the opiate-using HIV-infected population may be developed with a better understanding of the function of morphine in HIV infection and the mechanisms *via* which morphine exerts its effects.

With over 33 million individuals worldwide living with HIV, the HIV epidemic continues to be the most serious public health issue panic. The term HIV-associated neurocognitive disorder (HAND) refers to a variety of neurological and neurocognitive diseases that HIV-1-infected patients experience as the disease progresses. Other infections or the use of illegal drugs have been connected to severe neuropathological alterations that result in noticeably increased neurocognitive dysfunctions. Recent research suggests that one of the main factors contributing to HIV transmission in the USA is the use of illegal drugs [1].

Due to increased hazardous sexual behaviour and sharing contaminated needles, injection drug users are more likely to contract HIV, develop neurological abnormalities, and contract other opportunistic illnesses. Given that these people are living longer in the post-HAART period, HIV infection and opiate drug addiction have received more attention recently. However, the related neurological abnormalities continue to be among the majority of clinical diseases seen in HIV-infected patients [2].

Heroin is the most abused narcotic among the primary class of addictive medicines known as opioids. The favoured substance of investigation has been morphine since heroin is converted to morphine in the brain. The immune system has been shown to be significantly harmed by morphine due to the modulation of a range of cell functions, including those of phagocytes, T cells, and dendritic cells [3].

Opioids are referred to as cofactors for HIV infection because they work synergistically with HIV viral proteins to cause increased immunosuppression. Particular brain areas, like the striatum and hippocampus, have been linked to higher viral titers in HIV-infected patients and express opioid receptors at high levels. Opiate medicines alter not only the neuronal response to HIV directly but also the levels of endogenous opioid peptides, which in turn affect the central nervous system's operations.

Although there are various types of opioid receptors, the most potent opioids, including morphine and opioid agonists like naloxone, bind to the opioid receptor. The evidence from our lab and other labs is compiled in this work to support the idea that morphine increases HIV-1 infectivity and contributes to the neuropathogenesis of HIV-associated neurocognitive disorder [4]. Chronic opiate drug misuse considerably raises viral titers and has an impact on CD4 T cells in HIV-infected individuals. The considerable loss of T cells, which worsens the clinical state of HIV-infected patients, has been attributed to apoptosis.

It has been discovered that the HIV virus and several of the proteins encoded by the HIV genome, such as gp120, Tat, Nef, Vpr, Vpu, and HIV protease, exhibit pro- and/or antiapoptotic properties. In human peripheral blood mononuclear cells (PBMCs) that are HIV-infected for an extended period of time, Peterson and their colleagues have demonstrated that morphine

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promotes HIV-1 replication [5].

For the first time, our experiments have shown that morphine treatment of PBMCs significantly increases apoptosis. Compared to the corresponding controls, DNA fragmentation was clearly visible in morphine-treated cells. Overall, our study indicated that morphine can decrease immune function when HIV is present, possibly by activating apoptosis either on its own or in conjunction with other factors that contribute to the pathogenesis of HIV infection. In a different investigation, Zheng and Gendelman found that HIV-1 gp120/anti-gp120 or morphine (3 M) alone did not significantly induce apoptosis in newly isolated human PBMCs [6].

CONCLUSION

According to these findings, many cell types, including PBMCs and CNS cells, are more susceptible to HIV-1 replication and infection when drugs of abuse like morphine are present. Modifying chemokines and HIV-1 coreceptors may be one of the mechanisms underlying these effects. Opioids' capacity to change how diverse central nervous system cells express chemokines and chemokine receptors may dramatically increase HIV's capacity to infect the brain. When combined with HIV infection, morphine also affects the status of both pro- and antiapoptotic molecules, increasing the incidence of apoptosis. These actions are mediated by multiple signaling systems.

Overall, viral binding and cellular factors may both increase HIV infection in the brain through enhancing the expression of chemokines or HIV coreceptors. Together, these research offer crucial knowledge on the molecular basis of morphine use, HIV infection, and HIV pathogenesis, which may aid in the creation of fresh anti-HIV tactics that target chemokines and coreceptors.

REFERENCES

1. Antinori A, Arendt G, Becker JT, Brew BJ, Byrd DA, Cherner M, et al . Updated research nosology for HIV-associated neurocognitive disorders. Neurology. 2007;69(18):1789-1799.
2. Goodkin K, Wilkie FL, Concha M, Hinkin CH, Symes S, Baldewicz TT, et al. Aging and neuro-AIDS conditions and the changing spectrum of HIV-1-associated morbidity and mortality. J Clin Epidemiol. 2001;54(12):35-43.
3. McArthur JC, Hoover DR, Bacellar H, Miller EN, Cohen BA, Becker JT, et al. Dementia in AIDS patients: incidence and risk factors. Neurology. 199 ;43(11):2245.
4. Mintz M. Clinical comparison of adult and pediatric Neuro AIDS. Adv Neuroimmunol. 1994;4(3):207-221.
5. Robertson KR, Hall CD. Human immunodeficiency virus-related cognitive impairment and the acquired immunodeficiency syndrome dementia complex. Semin Neurol . 1992 ;12(01): 18-27.
6. Zheng J, Gendelman HE. The HIV-1 associated dementia complex: a metabolic encephalopathy fueled by viral replication in mononuclear phagocytes. Curr Opin Neurol . 1997 ;10(4):319-325.