Editorial

Neuropsychological testing has shown that approximately fifty percent of patients presenting HIV, in antiretroviral therapy, are afflicted by mild cognitive impairment (MCI)/HIV dementia, or other types of cognitive impairment as well as in the integrity of motor functioning, which implies that the prevalence was unaltered from the pre- to the potent antiretroviral period, although incidence of MCI had increased and HIV dementia decreased [1-3]. The presence -of neuropsychiatry co-morbidity in the pathophysiology of HIV compounds the suffering and detrimental prognosis of those afflicted with a complex relationship between HIV infection and psychiatric co-morbidity [4,5]. For example, HIV-positive status was significantly and independently associated with worse physical and mental health-related quality- of life and with an increased likelihood of depression [6]. Heroin administration, with or without smoking and cannabis, has emerged as a significantly destructive dimension of the HIV epidemic in Kenya [7]. HIV-infected smokers lose more years of life to tobacco-related disease than HIV. In cognitive testing, it has been observed that HIV-infected smokers exhibited a lower level of performance than HIV-uninfected smokers on tasks that included tests of working memory, processing speed, and intra-individual variability [8]. Furthermore, Among HIV-infected patients who smoked, neurocognitive performance was negatively associated with quality-of-life and depression ratings. A more deleterious overall symptom burden among HIV patients compared with healthy elder control subjects (n=236) with episodes of more frequent agitation, depression, anxiety, apathy, irritability and nighttime behavior disturbances has been evidenced [9]. In a large study of HIV patients, the majority of study-participants identified smoking correctly as contributing a potential cause of various smoking-related illhealth and ill-being conditions and correctly identified constituents in cigarette smoke, although lacking knowledge concerning the effects of nicotine [10]. Taken together, the consensus emerges that individuals’ self-injecting/administering drugs of abuse are confronted by barriers to their healthcare arising from reasons including co-morbidity, and particularly the case among HIV patients [11]. It should be noted that HIV-1 proteins affect novelty-seeking behavior and modulate addiction-related genes in the context of nicotine-dependent behavior [12,13].

Substance-abusing HIV individuals evidence a greater incidence of brain-related disorders [14-17]; furthermore, there is a strong relationship between drug abusers with HIV and their non-adherence to antiretroviral treatment [18,19]. Additionally, HIV-infected and HIV/hepatitis c virus-coinfected patients in opiate replacement therapy require higher methadone dose [20]. Among drug users, the analysis of HIV infection and prevention presents a relationship between drug-use and men-who-have-sex with other men, sexual behavior and sexually-transmitted diseases [21-23]. Illicit drug use among HIV-infected individuals is associated with non-adherence to HIV-medicines with the involvement of social factors modulating drug use and abuse [24-26]. In a study of 875 HIV-positive Japanese male subjects, it was found that 282 participants used addictive drugs (32.2%), with 13.8% administering illicit compounds: amphetamine/methamphetamine 5.4%, dusters/sprays/gas 3.5%, 5-methoxy-N,N-diisopropyltryptamine 1.8% and cannabis 1.0% [27], with marked links between HIV diagnosis and drug usage. In this context, the notion that HIV-infections alters structure and functioning in brain reward systems due to infection-drug susceptibility at cellular and molecular sites [28,29], putatively accompanied by neurotoxic signaling [30]. These alterations, accordingly, appear to render the brain systems involved hypersensitive to the rewarding properties of addictive drugs [31,32]. It appears also that methamphetamine-induced CNS pathogenesis involving neurotoxicity is exacerbated in the HIV condition [33]. It has been shown also, in vitro, that the exposure of human and rat primary hippocampal neurons to cocaine and HIV-1 Tat reduced synergistically mitochondrial membrane potential and ATP production, as well as affecting neuronal autophagy [34]. Finally, administration of compounds with a psychostimulant action, both illicit and therapeutic drugs, increases dopamine influx into macrophages [35]. In this regard, it has been shown that the effects of dopamine, e.g. through dopamine receptor activation such as use of a psychostimulant drug, exert contribute significant influences upon the pathogenesis of HIV, and vice versa [36-38].

In conclusion, the abuse of addictive drugs disorder and the HIV-infection disease condition appear to exert a mutually, reciprocal, destructive pathophysiology that may only potentiate health loss and worsen the prognosis of those afflicted. Rehabilitation and lifestyle-adaptation must be studied more carefully if the patient situation is to be resolved.

References


*Corresponding author: Archer T, Department of Psychology, University of Gothenburg, Box 500, S-405 30 Gothenburg, Sweden. Tel: +46 31 7864694; E-mail: trevor.archer@psy.gu.se

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