Austin Journal of Neuropsychiatry and Cognitive Science

Open Access

Austin Publishing Group

Review Article

HIV Neurocognitive Impairment: Perspectives on Neurocognitive Reserve and Behavioral Remediation/ Compensation Strategies

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Received: October 08, 2014; **Accepted:** February 18, 2015; **Published:** February 20, 2015

Abstract

Pharmaceutical advancements in combination Antiretroviral Therapy (cART) has led to effective interruption in the replication cycle of HIV; this in turn has resulted in a better prognosis and perhaps even a normal lifespan. Yet, even in the post-cART era, the brain remains a reservoir for HIV as many of these medications do not easily cross the blood brain barrier. As a result, a biological cascade of complex interactions between HIV and the brain produces neurotoxic molecules that disrupt neuronal functioning. Overtime, these biological processes deplete neurocognitive reserve and produce neurocognitive impairment ranging from mild neurocognitive impairment to HIV-associated dementia. This brief article articulates a few practical and experimental strategies in which adults with HIV can use to remediate or compensate for neurocognitive impairments.

Keywords: Aging; Compensation; Remediation; Neurocognitive Prescription; Neuroinflammation

Introduction

The replication cycle of HIV within CD4+ lymphocyte cells consists of seven stages: Fusion, reverse transcription, integration, transcription, translation, budding, and maturation [1]. Basically, during this process, HIV enters the cell (Fusion), uses one of its three enzymes (Reverse Transcriptase) to cut HIV RNA strands into smaller pieces so it can merge with the nucleus and the DNA of the host cell (Integration). Once in the nucleus, copies of HIV can be created producing more RNA molecules of HIV within the intracellular fluid, which are then put back together again (Transcription) to create strands of HIV RNA. From this RNA, other HIV proteins are produced (Translation) which are assembled (Budding) before leaving the cell and taking part of the CD4+ lymphocyte's membrane with it (Maturation) as it enters the intercellular fluid and infects more cells [1]. Fortunately, our understanding of this replication cycle has led to the development of numerous pharmaceutical advancements (e.g., nucleoside reverse transcriptase inhibitors) that interrupt key stages during this process. By doing so, this has resulted in keeping the virus from overwhelming the immune system, allowing people the opportunity to either protect their immune system from collapsing or help people to reconstitute their immune system. Likewise, this has led to better overall health and the opportunity to age with HIV. In fact, although controversial, some studies suggest that thanks to cART, adults with HIV are projected to have a normal lifespan comparable to those without HIV [2-5].

cART has not only improved prognosis with this disease, it also has resulted in neurocognitive benefits. Before cART, the prevalence of HIV-associated Dementia (HAD) has been ~16% [6]; in a post-cART era, the prevalence has dropped dramatically to 2% [7,8]. Unfortunately, the brain is an HIV reservoir where the virus can hide from cART. Even though some cART medications can pass through the blood brain barrier well (e.g., Zidovudine, Nevirapine, Indinavir), others are less effective in doing so (e.g., Tenofovir, Ritonavir, Enfuvirtide), which means HIV can still exert a detrimental influence on brain health and cognition. Using Frascati criteria of classifying neurocognitive impairment, Heaton and colleagues [9] found that 52% of adults with HIV in the United States had observable neurocognitive impairment ranging from Asymptomatic Neurocognitive Impairment to HAD. Likewise, Bonnet and colleagues [10] found that 56% of adults with HIV in France had observable neurocognitive impairment ranging from Asymptomatic Neurocognitive Impairment to HAD. Although the severity of such HIV-Associated Neurocognitive Disorders (also known as HAND) has been reduced, the neurocognitive profile has changed and the prevalence remains high.

The explanations for the continued impact of HIV on neurocognitive performance, neurobiology, and brain health are complex and varied; in general, the mechanisms can be explained as follows. HIV crosses the blood brain barrier via infected monocytes and macrophages (referred to as the Trojan horse hypothesis) [11]. While in the brain, HIV continues to replicate. Although HIV does not infect neurons, it does infect glial cells which support the health and functioning of neurons. As HIV replicates and glial cells die, a cascade reaction occurs whereby neurotoxic molecules are released (i.e., Tat protein, gp120, gp41) in the brain; nearby cells exhibit an immune response and secrete a number of pro-inflammatory molecules (i.e., IL-1β, IL-6, TNF-α, quinolinic acid, platelet activating factor, eicosanoids) which over time can damage neurons, especially their synaptodendritic connections, and hinder interneuron communication in both gray matter, subcortical structures, and white matter tracks [12]. The extent to such neuronal damage can eventually compromise "neurocognitive reserve."

Citation: Vance DE, Nicholas N and Cody SL. HIV Neurocognitive Impairment: Perspectives on Neurocognitive Reserve and Behavioral Remediation/Compensation Strategies. Austin J Neuropsychiatry & Cogn Sci. 2015;1(1): 1002.

Neurocognitive Reserve

Neurocognitive reserve refers to a theoretical explanation of how the brain can continue to function in lieu of damage and insults to it [13]. Simplistically, the stronger, healthier, and more sophisticated the synaptodendritic connections between neurons, the stronger one's neurocognitive reserve is considered to be. Thus, as one neural path is compromised, an adjacent one can be used to convey the neuronal information so that neurocognitive functioning can continue. The more access to adjacent neuronal pathways, the easier the brain can compensate for damage caused by HIVor other neurological insults [13].

Furthermore, the amount of neurocognitive reserve varies from person-to-person. There are several reasons for such variation. Some people may innately/genetically have better developed and robust brains. Yet, neurocognitive reserve is also the result of a mechanism broadly referred to as neuroplasticity, more specifically, positive neuroplasticity and negative neuroplasticity. Positive neuroplasticity refers to the complex bio-mechanisms whereby the connections and sophistication between neurons are strengthened due to active use, learning, and environmental demands. Likewise, negative neuroplasticity refers to the complex bio-mechanisms whereby the connections and sophistication between neurons atrophy due to disuse and lack of environmental demands dependent on their use [14].

These mechanisms are eloquently observed in the classic enriched environmental paradigm whereby genetically similar rats are randomly placed to live in one of three cages (i.e., enriched, standard, and impoverished) and biobehavioraly observed over time [13,15-17]. The enriched cage consists of several other rats and toys to explore; so in this environment, the rats have plenty of interesting things to do. The standard cage only consists of three rats to a cage and no toys to explore. The impoverished cage consists of a single rat in a cage and no toys to explore; so in this environment, rats placed in this cage have nothing interesting to do. Using this paradigm, rats living in the enriched cage have more synaptodendritic connections, larger/heavier brains, more brain-derived neurotrophic factor, and perform better in maze tasks (i.e., a proxy of neurocognitive functioning) while rats in the impoverished cage experienced the opposite effect. Thus, rats in the enriched cage display positive neuroplasticity and more neurocognitive reserve, while rats in the impoverished cage display negative neuroplasticity and less neurocognitive reserve.

Similar positive and negative neuroplasticity mechanisms have been observed in human studies showing that exposure to neurocognitively enriching environments produces effects on brain health that build neurocognitive reserve and delay the effects of pathologies producing neurocognitive impairments. For example, in a sample of 181 older adults, Stine-Morrow and colleagues [18] found the experimental group (n = 107) who attended 20 weekly social meetings and engaged in team problem-solving experienced better neurocognitive functioning compared to the control group (n = 74) who did not attend any social meetings. Findings suggest that social engagement and neurocognitively enriching activities contribute to better neurocognitive functioning and may be neuroprotective against neurocognitive impairments related to HIV-Associated Neurocognitive Disorders [18]. Although beyond the scope of this brief article, such effects are observed in the London Taxi Driver Study [19], the Older Juggler Study [20], and several Alzheimer's studies that show those with more education or a specialized skill (i.e., musical instrument) experience a delayed on-set of the neurocognitive symptoms [21-24].

In a related cross-sectional study focusing on 139 adults with HIV (M_{age} = 48.7 years), Fazeli and colleagues [25] examined the prevalence of neurocognitive functioning on HAND and its relationship to three factors related to neuroplasticity: 1) employment status as a measure of mental activity, 2) engagement in physical activity, and 3) social engagement. These researchers found as these adults engaged in more of these activities (i.e., factors related to an enriched environment and positive neuroplasticity), these adults were less likely to exhibit observable neurocognitive impairment, which is reflective of having greater neurocognitive reserve [25]. Albeit, even with greater amounts of neurocognitive reserve, HIV, other neurological insults (i.e., transient ischemic attack), poor health status (i.e., hepatitis C), and a host of other factors that contribute to negative neuroplasticity (i.e., poverty, substance abuse, depression/anxiety/stress, lack of education, poor healthcare), such reserve can still be compromised resulting in neurocognitive impairment [26]. For that reason, neurocognitive remediation and compensation strategies are needed.

Behavioral Remediation and Compensation Strategies

Few attempts have been successful in improving neurocognitive functioning in adults with HIV. Obviously, cART is at the most recommended strategy in showing some neurocognitive improvements by reducing the amount of HIV in the body which is known to cause neuroinflammation [13]. Yet, some studies also suggest that cART itself can be neurotoxic and cause calcium homeostasis dysregulation, amyloidosis, inhibition of microglial phagocytosis, and mitochondrial damage [27]. In fact, one study showed that 167 adults with HIV who were medically stable (i.e., good immune response as exhibited by a CD4+ > 350 cells/mm^{3}and low Viral Load (VL) as exhibited by VL < 55,000 copies/mL), and taken off of cART actually experienced progressive neurocognitive improvements over a 96-week period [28]. Yet, despite this controversy, it is still recommended that all people be placed on cART to avoid immune dysfunction and reduce systemic inflammation [28] which can have obvious neurocognitive benefits.

Pharmacological studies have examined the use of psychostimulants (i.e., methylphenidate, modafinil) with some limited improvement in attention and speed of processing [29-31]. Yet others such as memantine (blocking NMDA/glutamate receptors) have been attempted in adults with HIV but with limited effects. The use of donepezil, a cholinesterase inhibitor used in treating Alzheimer's disease, is not recommended at this time since it works on increasing acetylcholine in the brain [32]. In HIV, a depletion of dopamine is more likely than a decrease in acetylcholine [33]; therefore, Alzheimer's-type drugs are not recommended [32]. Furthermore, selegiline (an MAO-inhibitor with antioxidant properties), as delivered in a 24-hour transdermal patch was also attempted in 128 adults with HIV in three groups comparing a 3 mg dose, a 6 mg dose, and placebo over a 24-week

period; unfortunately, although it was well tolerated, neurocognitive improvement was effective as placebo [34]. In adults with HIV who are already susceptible to polypharmacy which contributes to kidney and liver burden [35], behavioral approaches which are not reliant on medications to improve cognition are particularly appealing for this population.

In this brief review, two remediation strategies and a few practical compensation strategies are also suggested. The remediation strategies are speed of processing training and cognitive prescriptions. In a pre-post two-group experimental design, Vance and colleagues [36] randomly assigned middle-aged (40+ years) and older adults to either a no-contact control group or to a speed of processing training group. Those in the training group received ~10 hours of computerized neurocognitive exercises specifically designed to improve visual attention and speed of processing. From baseline to posttest approximately 5-6 weeks later, those in the speed of processing group improved on a computerized measure of visual speed of processing (i.e., Useful Field of View Test); this also translated to improved performance on a measure of everyday functioning (i.e., Timed Instrumental Activities of Daily Living Test).

Another remediation technique has also been suggested by Vance and colleagues [37] called Cognitive Prescriptions. In this proposed intervention, several areas known to influence brain health are targeted for behavioral modification; these include physical activity, mental activity, nutrition, sleep hygiene, substance use, and mood support. The basic premise undergirding this approach is "That which is good for the body is also good for the brain." The intervention consists of a patient dialoguing one-on-one with a coach who uses motivational interviewing skills to develop tailored, individualized/intrinsic goals in each of these areas. For example, the patient might express a desire to go for afternoon walks again like she use to years ago but simply got out of the habit. Thus, the coach would suggest going for a walk twice a week but the patient expresses that the goal should be four times a week; not wanting to set the patient up for immediate failure of the goal, the coach negotiates the goal to three times a week. Certainly, if the goal is consistently reached, this patient-coach dyad can discuss increasing the goal or switching the goal entirely to a more rigorous activity such as jogging or hiking. Of course, this is all patient-centered by focusing on the wants, needs, and motivation of the patient, with the coach serving as an external support and guide in setting and completing the goals. Although no formal testing of this holistic approach is in the literature, separate components of this approach have been observed in the literature where mood support, nutrition, physical exercise, and mental exercise have improved neurocognitive functioning [25,38].

Instead of improving neurocognitive functioning through remediation strategies, compensation strategies are necessary as well to help patients better met the neurocognitive demands in negotiating the complexities of their environment. One study by Neundorfer and colleague [39] recruited 10 older (M_{age} = 53 years) men and women with HIV who were also experiencing neurocognitive impairment as observed by their physician. These researchers introduced a combination of spaced retrieval method and mnemonics to help these participants accomplish vital everyday goals such as remembering to take their medications as prescribed or attending clinic appointments. The spaced retrieval method consists of recalling discrete units of information over progressively longer periods of time (e.g., 30 second – 1 minute – 2 minutes – 4 minutes – 8 minutes – 16 minutes) up to ~16 minutes when it is then integrated into one's long-term memory. These researchers used this technique to help these participants learn to use their external mnemonics (e.g., lists, calendars, posted reminders) in order to accomplish their everyday goals. In general, these results were favorably and anecdotally well-liked by the participants.

In a recent workshop of the *26th Annual Social Work and HIV/AIDS Conference* in Denver, CO, Nick Nicholas presented a workshop entitled, "Help! I've Lost My Mind! There's an App for that!" In this talk, he presented a litany of low-tech and high-tech mnemonic strategies for adults with HIV experiencing neurocognitive impairment and dementia. In this workshop, he provided some low-tech and high-tech solutions for coping with slower mental processing and memory problems. Some of the low-tech mnemonics suggested included journaling to keep track of daily tasks and events, having friends or a partner remind one about appointments, placing items (e.g., keys, wallet, cellphone) in a basket, and just being organized. Also, making several sets of keys, keeping a second list of daily tasks and events, and keeping a voice recorder to make supplemental lists can help with forgetfulness. With adherence to cART being so important in keeping HIV from replicating, over time it can be easy to forget a dose here and there; for this, he suggested a simple weekly pill box. That way, if individuals are not sure whether they already took their medication for the evening, they simply have to check whether the pill box for that period of time is empty or not.

Meanwhile, many clever high-tech mnemonics were also suggested. For example, Ever note (evernote.com) and Wunderlist (wunderlist.com) are free apps useful for keeping track of lists. iCal, which comes with iPad, is a useful calendar for keeping up with appointments, whether medical or social; thus, this app was suggested to help with planning, an executive functioning ability commonly impaired in those with HIV-associated neurocognitive disorders Sometimes adults with HIV-associated neurocognitive disorders experience a poor sense of time [40]; fortunately, there is an app called 30/30 App that allows one to set a certain amount of time on a task, and then gives an alert when time is up. This can be very useful for time management.

Since neurocognitive impairments are known to influence everyday functioning tasks such as driving [38], the following tips on ways to prevent at-fault crashes may be considered: leave early to allow extra time to arrive at the point of destination, adhere to speed limits, avoid tailgating, signal during lane changes, and pay close attention to surroundings. Post-It notes can be placed on the dashboard as reminders, because it can be frustrating for adults who forget where they are going and their purpose once they arrive at their destination. During these moments, a Global Positioning Satellite (GPS) unit may also be beneficial to facilitate safe travel. Finally, financial management can be an issue for many adults with HIV-associated neurocognitive disorders [41], but the mint app can be used to keep track of bills, credit cards, and bank accounts. Although some adults find the use of such technologies challenging, friends, partners, and caregivers may need to help set up such apps with their

loved one.

Conclusion

cART has improved the prognosis, quality of life, and lifespan for many with HIV; as a result, the number of adults 50 and older with this disease will comprise 50% of all cases in the United States by 2015 [42] and this is expected to rise to 70% by 2020 [43]. With the graying of the epidemic, clinicians, neuropsychologists, social workers, nurses, and allied health professionals must be cognizant of the neurocognitive vulnerability of this growing population and be prepared to address what can and cannot be recommended to those experiencing such neurocognitive impairments, especially as many may also develop age-related neurocognitive declines [44]. Likewise, preventive lifestyle strategies should also be emphasized in early and midlife in order to promote greater neurocognitive reserve which is important as people age and experience age-related neurological insults as well as HIV-related neurological insults along the way.

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Austin J Neuropsychiatry & Cogn Sci - Volume 1 Issue 1 - 2015
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Citation: Vance DE, Nicholas N and Cody SL. HIV Neurocognitive Impairment: Perspectives on Neurocognitive Reserve and Behavioral Remediation/Compensation Strategies. Austin J Neuropsychiatry & Cogn Sci. 2015;1(1): 1002.