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Neurocognitive correlates of alexithymia in asymptomatic individuals with HIV

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ABSTRACT

Alexithymia, an impairment of affective and cognitive emotional processing, is often associated with human immunodeficiency virus (HIV) and may reflect effects of the virus on brain areas that are also important for multiple cognitive functions, such as the prefrontal and anterior cingulate cortices. We hypothesized that there would be a correlation between extent of alexithymia and cognitive performance associated with these brain areas, including attention, executive function, and visuospatial processing. Thirty-four asymptomatic HIV+ participants and 34 matched healthy HIV− volunteers were administered the Toronto Alexithymia Scale, a series of neuropsychological tests, and measures of apathy, depression, and quality of life (QoL). The HIV+ participants had significantly higher levels of alexithymia, depression and apathy than the HIV− group. The extent of alexithymia and two of its processing components (Difficulty Describing Feelings [DDF] and Externally Oriented Thinking), but not depression, correlated with performance on measures of executive and visuospatial abilities, consistent with dysfunction of the frontostriatal circuits and their cortical projections. Apathy was related to alexithymia and two processing components (Difficulty Identifying Feelings and DDF) but to only one cognitive measure. The higher rate of alexithymia, as well as cognitive dysfunction, in HIV may be a consequence of the infection on the frontostriatal system and its cortical connections. Our findings also demonstrated a dissociation of apathy and alexithymia in HIV, pointing to overlapping but distinct neural substrates within frontostriatal circuits. Alexithymia correlated strongly with QoL ratings, underscoring the importance of assessment and treatment of HIV-associated emotional and cognitive processing deficits.

1. Introduction

Infection with human immunodeficiency virus (HIV) is associated with deficits in cognition and emotion, but the relation between them is not understood. This study explores the association between alexithymia and cognition in individuals with HIV in its early asymptomatic stage.

HIV affects frontostriatal thalamocortical circuits (Everall et al., 1999) early in the course of the disease (Avison et al., 2004; Berger & Nath, 1997; Chang et al., 2001, 2004; Ernst, Chang, Jovicich, Ames, & Arnold, 2002; Gray, 1996). Structural magnetic resonance imaging (MRI) has shown that HIV infection is associated with reduced volumes of frontal cortex, thalamus, hippocampus, and caudate (Jernigan et al., 1993), as well as with tissue loss in frontal and parietal areas (Thompson et al., 2005). Neuroimaging studies of asymptomatic HIV+ patients have documented reduced brain volume (Aylward et al., 1993), cerebral metabolic asymmetry (left or right) in prefrontal and specifically orbitofrontal areas (Pascal et al., 1991), and differences in signal changes in lateral frontal and posterior parietal areas (Castelo, Sherman, Courtney, Melrose, & Stern, 2006).

Neuropsychological studies of cognition in HIV have shown that asymptomatic HIV+ individuals exhibit cognitive deficits consistent with dysfunction of frontostriatal circuits (Bogdanova, Neargarder, & Cronin-Golomb, 2008; Castelo, Courtney, Melrose, & Stern, 2007; Heaton et al., 1995) and parietal cortical areas (Bogdanova & Cronin-Golomb, 2005; Bogdanova et al., 2008; Olesen, Schendan, Amick, & Cronin-Golomb, 2007). Disruption of frontostriatal circuits affects neuropsychological performance in HIV, specifically on tests of frontal-lobe functioning, such as processing speed, working memory, and executive function (Heaton et al., 1995; Paul, Cohen, & Stern, 2003; Reger, Welsh, Razani, Martin, & Boone, 2002). It has been proposed that HIV-associated cognitive deficits are caused by neuropathological changes produced by the HIV infection in the basal ganglia and associated pathways (Grant & Martin, 1994), including the prefrontal cortex and parietal lobes (Ernst, Chang, & Arnold, 2003; Woods, Moore, Weber, & Grant, 2009).

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1.1. Alexithymia and HIV

HIV produces changes in emotion regulation and emotional cognition. In particular, patients experience alexithymia, an impairment of affective and cognitive emotional processing (Fukunishi, Hirabayashi, Matsumoto, Yamanaka, & Fukutake, 1999; Lumley, Neely, & Burger, 2007; Temoshok et al., 2008; Thome, 1990). Alexithymia is a multifaceted construct characterized by: (a) difficulty identifying and distinguishing between feelings and bodily sensations of emotional arousal, (b) difficulty describing feelings, (c) reduced imaginal processes as evidenced by a paucity of fantasies, and (d) a stimulus bound, externally oriented cognitive style (Bagby & Taylor, 1997). These characteristics reflect deficits in the cognitive processing and regulation of emotions, which may contribute to the development and course of several medical and psychiatric disorders. The inability to apply adaptive processes for affect regulation, such as modulating arousal, expressing or suppressing emotions, using social support, tolerating painful emotions, and cognitive assimilation, is thought to be a core factor contributing to conditions such as depression, anxiety, compulsive and addictive behavior, heightened physiological arousal, and physical symptoms (Lumley, Stettner, & Wehmer, 1996; Lumley et al., 2007; Taylor, Bagby, & Parker, 1997).

The construct of alexithymia remains a subject of ongoing scientific debate, which has produced a large body of empirical literature that has introduced several measures. The 20-item Toronto Alexithymia Scale (TAS-20) (Taylor et al., 1997) is the most widely used brief self-report measure of alexithymia that has good validity and reliability (Bagby, Taylor, Quilty, & Parker, 2007; Taylor et al., 1997). Three TAS-20 factors reflect affective and cognitive dimensions of alexithymia and assess its distinct components: Factor 1 – difficulty identifying feelings and distinguishing them from bodily sensations of emotion (DIF); Factor 2 – difficulty describing feelings (DDF); and Factor 3 – externally oriented thinking (EOT), which refers to a pragmatic thinking style without affective involvement. The TAS-20 correlated three-factor model was reported to be acceptably well fitting (Bagby et al., 2007; Taylor et al., 1997). Multiple studies have used the three-factor structure to examine affective and cognitive aspects of alexithymia in clinical and non-clinical populations (Bagby et al., 2007; Bankier, Aigner, & Bach, 2001). The TAS-20 three factors (DIF, DDF, and EOT) are associated with neuroticism, introversion, and low openness, respectively (Lumley et al., 2007). Because these three personality constructs are theoretically independent, the authors argued, the three alexithymia factors will have differential validity. High scores on DIF may be associated with complaints of somatic symptoms, DDF with difficulty connecting with others, and EOT with decreased bodily awareness, which in turn may result in decreased health care use.

Two studies have indicated that the TAS-20 factors may be associated with distinct brain regions. In particular, EOT is thought to depend on different brain structures than do DIF and DDF. One study found that DIF and DDF, but not EOT, were related to right-hemisphere lesions (Spalletta et al., 2001), and a second study reported that EOT, but not DIF and DDF, was associated with the size of the anterior cingulate cortex (ACC) (Paradiso, Vaidya, McCormick, Jones, & Robinson, 2008). These findings may explain why scores on DIF and DDF are reported to have stronger correlations with each other than with EOT scores.

While the exact neural mechanisms of alexithymia remain a subject of ongoing investigation, a current model attributes alexithymia to dysfunctional mechanisms in the frontal cortex (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Gainotti, 1989), specifically, in the ACC and prefrontal cortex (Lane, Ahern, Schwartz, & Kaszniak, 1997). The implication of the ACC and prefrontal cortex in alexithymia has been supported by neuroimaging studies (Borsci et al., 2009; Gundel et al., 2004; Huber et al., 2002; Kano et al., 2003; Karlsson, Naatanen, & Stenman, 2008).

1.2. ACC and emotional cognition

The ACC's role in emotional cognition has been studied extensively in recent years. Since Papez postulated that the ACC is involved in emotion regulation (Papez, 1937), several investigations using cytoarchitectonic, lesion and neuroimaging data have identified two ACC subdivisions: an *affective* (rostral) and a *cognitive* (dorsal) division (Bermond, Vorst, & Moormann, 2006; Bush, Luu, & Posner, 2000; Devinsky, Morrell, & Vogt, 1995; Paus, 2001; Vogt, Finch, & Olson, 1992). The rostral subdivision has reciprocal connections with the amygdala, nucleus accumbens, hypothalamus, anterior insula, hippocampus, and orbitofrontal cortex (Carmichael & Price, 1996; Van Hoesen & Solodkin, 1993). It has been implicated in a variety emotional processing tasks (Bishop, Duncan, Brett, & Lawrence, 2004; Bush et al., 2000; Vuilleumier, Armony, Driver, & Dolan, 2001), but also in cognitive processing, such as conflict monitoring (Milham & Banich, 2005), resolving conflicts between stimulus–response associations when performing two tasks simultaneously (Dreher & Grafman, 2003), and monitoring errors (Kiehl, Liddle, & Hopfinger, 2000; Van Veen & Carter, 2002).

The dorsal subdivision is connected to lateral prefrontal cortex, parietal cortex, and premotor and supplementary motor areas (Van Hoesen & Solodkin, 1993) and plays an important role in attention and executive function, as it is involved in response selection, working memory, motor imagery and selection, detection of mismatch, and establishing changes in processing for new programs (Bush et al., 2000; Paus, 2001). It is also involved in emotional processing, such as attention-demanding tasks involving emotional content (Davis et al., 2005; Phan, Wager, Taylor, & Liberzon, 2002). The two ACC subdivisions are interconnected (Bush et al., 2000; Musil & Olson, 1988a; Musil & Olson, 1988b; Van Hoesen & Solodkin, 1993). Some authors view dissociation between cognitive and emotional processing as a reflection of a cross-modal interaction of the two separable reciprocal systems (Drevets & Raichle, 1998). Another view postulates that the two subdivisions of the ACC support a continuum of processing that blends cognitive and emotional components (Mohanty et al., 2007). Using neuroimaging and behavioral techniques, Mohanty and colleagues provided direct evidence for the differential engagement of both ACC subdivisions in cognitive and emotional function.

With regard to alexithymia, Lane (2000) discussed the possibility of both ACC subdivisions participating in different aspects of emotional processing, with dorsal ACC reflecting direct experience of emotion, and rostral ACC (with its connections to medial prefrontal cortex) participating in cognitive operations based on direct experience of emotion (knowing how one is feeling). The structural connectivity between the rostral and dorsal ACC forms the basis for interaction between direct experience of emotion and the formation of mental representations of that emotional experience. Their reciprocal connections with amygdala (involved in implicit emotional cognition) and prefrontal cortex (involved in explicit emotional cognition) provide additional evidence that both ACC subdivisions have a role in emotionalizing and emotional cognition (Bermond et al., 2006).

A recent neuroimaging study examined the association between alexithymia and gray matter volumes of ACC subregions as a function of age (Paradiso et al., 2008). They reported a significant correlation between right rostral ACC volume and alexithymia ratings on TAS-20, for both total score and Factor 3 (EOT). Older age was correlated with higher alexithymia and with smaller overall right ACC total gray matter volume in rostral and dorsal subregions, and in the left rostral subregion. These findings suggest that