

LETTERS TO THE EDITOR

Neuropsychiatric alterations and neurocognitive performance in HIV/AIDS: A response to C. Bungener and R. Jouvent

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The findings reported in the Letter to the Editor authored by Bungener and Jouvent are consistent with results we have presented here (Castellon et al., 2000) and again underscore the importance of considering the potentially multifactorial nature of depression in many neurologic diseases/disorders. We have suggested, although the idea is hardly a new one, that depression in HIV/AIDS can be secondary to any of multiple potential etiologies. For example, it may be a direct central nervous system (CNS) consequence of infection (i.e., neurochemical and/or neuropathological changes), a result of increased exposure to social, medical, and financial stressors secondary to living with HIV, a reaction to multiple losses (e.g., bereavement, loss of instrumental capacity), or be an admixture of multiple etiological factors. The phenomenology of this disruption of mood, motivation, and affect may differ as a function of etiology/pathophysiology. We believe that a prominent amotivation/apathy syndrome may be a more pure manifestation of the CNS effects of HIV-1 infection than is the more heterogeneous construct of depression and therefore more closely associated with other putative measures of CNS integrity (e.g., neurocognitive performance).

The ERP study by Bungener and Jouvent (1996) mentioned in their Letter to the Editor reports an association between the dimension of depression they label “emotional deficit” and reduced P300 amplitude. They conclude that the emotional deficit dimension may be an early marker of “subclinical manifestations of HIV” and associated with higher risk for developing cognitive impairment. Their construct of “emotional deficit” bears similarity to what we have been calling apathy in prior work (Castellon et al., 1998, 2000) and we have drawn similar conclusions based on its association with cognitive performance. Similarly, the “loss of control” dimension of Bungener and Jouvent

includes the construct of irritability, which we believe may also be a potential indicator of HIV-associated CNS involvement based on its association with subtle cognitive compromise. While our findings and those from the laboratory of Bungener and Jouvent converge, further study is needed, including the use of more direct markers of CNS function (e.g., neuroimaging or CSF viral load). Further empirical scrutiny of these constructs may be particularly indicated given the findings of a recent study by Rabkin et al. (2000), which failed to find an association between apathy and neurocognitive performance in a sample of HIV-infected subjects.

Another important point raised by Bungener and Jouvent is that syndromal depression need not be (and often is not) present in patients experiencing prominent apathy or irritability. As we point out in our original article, the overlap between depression, apathy, and irritability is considerable (these findings are similar to those reported by Rabkin et al.) but these constructs are not synonymous.

Finally, taking a more “dimensional approach” using a factor analysis of a depression rating scale with a different sample of HIV+ patients, we found that a factor containing several items reflective of apathy and/or amotivation was more strongly associated with neurocognitive performance than factors representing self-reproach or somatic disturbance (Castellon et al., 1999). These findings, along with those of our original article and those alluded to by Bungener and Jouvent emphasize the importance of focusing on the sometimes subtle and oft-understudied neuropsychiatric aspects of neurological disease. Many times these symptoms or signs are not captured by traditional measures of depression.

REFERENCES

- Bungener, C., Le Houezec, J., Pierson, A., & Jouvent, R. (1996). Cognitive and emotional deficits in early stages of HIV-infection: An event-related potentials study. *Progress in Neuro-psychopharmacology and Biological Psychiatry*, 20, 1303–1314.

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- Castellon, S.A., Hinkin, C.H., & Myers, H.F. (2000). Neuropsychiatric disturbance is associated with executive dysfunction in HIV-1 infection. *Journal of the International Neuropsychological Society*, 6, 336–347.
- Castellon, S.A., Hardy, D., Stenquist, P., Hinkin, C.H., van Gorp, W.G., Moore, L., & Satz, P. (1999). Apathy and nonapathy items on the Beck Depression Inventory: Their neurocognitive correlates in HIV-1 infection. *Journal of the International Neuropsychological Society*, 5, 154.
- Castellon, S.A., Hinkin, C.H., Wood, S., & Yarema, K. (1998). Apathy, depression and cognitive performance in HIV-1 infection. *Journal of Neuropsychiatry and Clinical Neurosciences*, 10, 320–329.
- Rabkin, J.G., Ferrando, S.J., van Gorp, W.G., Rieppi, R., McElhiney, M., & Sewell, M. (2000). Relationships among apathy, depression, and cognitive impairment in HIV/AIDS. *Journal of Neuropsychiatry and Clinical Neurosciences*, 12, 451–457.