

## DIALOGUE

# Reply to West

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West argues that the frontal aging hypothesis remains a useful construct, largely on the basis of evidence that cognitive functions dependent on frontal regions decline at an earlier age than those independent of frontal regions. This point was considered in my review, which found only sparse evidence in its favor. Few investigations have been aimed at determining the age at which decline begins for a set of functions. West cites one behavioral study (Shimamura & Jurica, 1994) and one PET study (Shaw et al., 1984). Given the size of the literature from which these studies are drawn, a finding of age effects in one "frontal" memory task with mean age 61 but not in one "nonfrontal" memory task can only suggest that age-related functional decline is more rapid in the frontal-dependent task. This finding must be viewed in the context of a broader range of functions. Several large scale studies aimed at this question have examined age-related declines on a broad range of functions and concluded verbal abilities are preserved until late in life while visuospatial abilities decline throughout adulthood (Arenberg, 1978; Eisdorfer et al., 1959; Koss et al., 1991). Shaw et al. (1984) must also be considered in the context of other studies of resting cerebral blood flow and metabolism which I reviewed which did not find selectively reduced prefrontal blood flow (Azari et al., 1992; Martin et al., 1991). Indeed, it is difficult either to prove or to disprove the somewhat vaguely-conceived frontal aging hypothesis using the current literature.

West argues against my conclusion that age-related change in activation patterns within prefrontal cortex is not consistent with the frontal aging hypothesis, claiming that result speaks more of a need to refine the theory than to jettison it. While I agree that the frontal aging hypothesis could be made more specific, I would make two further points. First, a theory of brain aging such as suggested by West claiming selective decline in one or another of the cytoarchitectural regions of the frontal lobes may be limited in explanatory power. Second, evidence of decreased posterior brain activation in aging (Grady et al., 1994) coupled with evidence of age-related decline in posterior-dependent functions (reviewed in Greenwood, 2000), emphasizes the importance of accounting for interlobe processes when formulating models of brain aging.

Rather than refining the frontal aging hypothesis, I would argue for a model of brain aging that considers age-related changes in interactions between brain regions rather than a selective decline of one region. As I argued in my review, the complex results on imaging studies of age-related changes in frontal activation, as well as the general weakness of the case from studies of volumetric loss and neuropsychology all serve to emphasize the need for both a more detailed and a more sophisticated theory of brain aging. On this point, West and I appear to agree.

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