

Comment on Greenwood (2007): Functional Plasticity in Cognitive Aging

Timothy A. Salthouse
University of Virginia

In this commentary on P. M. Greenwood's *Functional Plasticity in Cognitive Aging: Review and Hypothesis* (2007), the author raises a number of questions stimulated by the article. Although it may be premature to expect answers to those questions, the author argues that they ultimately need to be addressed and answered before Greenwood's speculations can be considered true hypotheses rather than a conceptual framework.

Keywords: cognitive aging, conceptual framework, plasticity

The primary goal of Greenwood's (2007) article is to account for the puzzling paradox that, compared with young adults, older adults have been found to exhibit increased functional neuroimaging activation in the same brain regions that exhibit the greatest amount of age-related atrophy. In somewhat oversimplified terms, her suggestion is that neurobiological degeneration in specific brain regions results in deficits in cognitive performance, and these trigger changes in strategy that are associated with functional reorganization. She then suggests that one manifestation of this functional reorganization is increased neural activation in the brain region undergoing degeneration. I believe that these speculations are intriguing, and my commentary is primarily composed of questions stimulated by the article.

I will begin with a few general comments and then turn my focus to more specific issues. First, it may be useful to distinguish between a framework and a hypothesis because the former is a set of related assumptions that guide research, whereas the latter is associated with a specific prediction. Hypotheses can therefore be falsified by empirical results, whereas frameworks simply become less useful as inconsistent findings accumulate. Although she refers to her speculations as a hypothesis, I suggest that Greenwood is actually proposing a framework rather than a hypothesis because many critical details necessary for specific predictions are missing.

Second, if the hypothesis (framework) is intended to account for the aging of cognition, then it is important to specify exactly what occurs with age and why. The speculations would still be interesting if the proposed linkages were found to exist at all ages, but in that case they would not necessarily be specific to aging. It is possible that the patterns of relations are qualitatively different at different ages; another possibility is that people of different ages merely differ in the prevalence of the triggering conditions, in which case there might be nothing in the speculations that is intrinsic to aging. A related issue concerns the suggestion that "functional plasticity alters the course of cognitive aging" (p. 659). This statement seems to imply that the relation between age and cognitive functioning would be different if functional plasticity did not occur, but this might not be the case if "functional plasticity"

is possible at all ages and is associated with increases in the level of performance without altering the relation between age and performance.

The remainder of this commentary consists of questions about the major relations in the hypothesis (framework). The first postulated relation is a linkage between atrophy and performance deficits. Among the questions related to this linkage are the following:

Does atrophy refer to only a reduction in brain volume, or does it apply to a variety of possible determinants of volumes, including cortical thinning, dendritic regression, and so on? Brain volume, and even regional brain volume, is a very crude variable because there could be many functional changes prior to, or possibly independent of, any volume changes. Furthermore, not all of the factors that affect volume are involved in cognitive functioning, and therefore it is important to know which neurobiological changes are considered critical in triggering the other postulated relations.

Is it the absolute volume that is important in the link with cognitive deficits and plasticity, or is it reduction over time that is critical? If it is absolute volume, then do the other relations in the hypothesis (framework) apply to any case in which brain volumes might differ? If so, could these cases perhaps include contrasts of female with male brains, since the former tend to be smaller?

Does the time course of the atrophy matter? That is, would the consequences be the same with a sudden dramatic decline in regional "brain integrity" such as that associated with a stroke and with slow gradual decline that takes place over a period of decades?

The "apparently weak relationship between regional atrophy and cognitive performance in old age" (p. 667) is interpreted as consistent with the assumption that other factors (such as plasticity) are operative at older ages. However, there is little evidence for this differential relation, and it is not clear how a failure to find a significant difference in volume–cognitive performance relations across age groups with comparisons of adequate power would lead to changes in the hypothesis (framework).

The second postulated relation links performance deficits to strategy changes as well as to functional plasticity in the atrophied region and in the parietal and prefrontal cortices. Some of the questions about this relation are the same as those raised about the first relation. For example, is it merely low performance or also a decrease in performance over time that triggers the new strategy or

Correspondence concerning this article should be addressed to Timothy A. Salthouse, Department of Psychology, University of Virginia, Charlottesville, VA 22904-4400. E-mail: salthouse@virginia.edu

the functional plasticity? Is the time course important, with the same consequence for a sudden deficit as for one that occurs gradually over decades?

What is it about a low level of performance that instigates these changes—is it a conscious and deliberate attempt to overcome the performance deficit, or is it an automatic and unconscious adaptation?

How can the existence of a strategy be determined? Strategies can be considered optional methods of performing a given task, but if other methods of performing the task are no longer possible because of deterioration of the neurobiological substrate, should the only available mode of performance still be considered a strategy? Is it necessary to establish that other modes of performance are within the capability of the individual before using the term strategy?

Perhaps the most important question in the framework is the following: If the strategy (or functional plasticity) is within the capability of the individual and is beneficial, then why was it not used at earlier periods in life or by anyone who is not performing at the maximum for any reason? One would normally expect that higher functioning individuals would be better able to develop and implement novel strategies, and thus it is surprising that individuals experiencing decline would be capable of formulating and using new strategies. Is it because increased age is associated with better utilization of cognitive or neural resources, and if so, what is the evidence for this?

What if high-density longitudinal assessments of performance and strategy (or plasticity) were available—would a failure to find a lead-lag relation among the constructs, with the presence of a deficit preceding the use of a strategy, be damaging to the hypothesis?

Similar questions can be raised about functional plasticity and its relations to reorganization and activation. More details are desirable about how plasticity (recruitment, task activation) contributes to improved cognitive performance, which is presumably necessary if the deficits are at least partially compensated as a consequence of the plasticity.

In conclusion, I believe that this article contains many intriguing speculations, but as is almost always the case with new ideas, much remains to be done to elaborate the proposed linkages and convert a somewhat vague framework into a set of specific hypotheses with explicit falsifiable predictions. However, the mere fact that the article has stimulated many questions that might not otherwise be asked can be considered evidence that it has made a contribution to scientific progress.

Reference

Greenwood, P. M. (2007). Functional plasticity in cognitive aging: Review and hypothesis. *Neuropsychology*, *21*, 657–673.

Received January 16, 2007

Accepted July 17, 2007 ■