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# Theoretical Perspectives on Age Differences in Brain Activation: HAROLD, PASA, CRUNCH—How Do They STAC Up?

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## Summary and Keywords

Cognitive neuroimaging studies often report that older adults display more activation of neural networks relative to younger adults, referred to as *overactivation*. Greater or more widespread activity frequently involves bilateral recruitment of both cerebral hemispheres, especially the frontal cortex. In many reports, overactivation has been associated with superior cognitive performance, suggesting that this activity may reflect compensatory processes that offset age-related decline and maintain behavior. Several theories have been proposed to account for age differences in brain activation, including the Hemispheric Asymmetry Reduction in Older Adults (HAROLD) model, the Posterior-Anterior Shift in Aging (PASA) theory, the Compensation-Related Utilization of Neural Circuits Hypothesis (CRUNCH), and the Scaffolding Theory of Aging and Cognition (STAC and STAC-r). Each model has a different explanatory scope with regard to compensatory processes, and each has been highly influential in the field. HAROLD contrasts the general pattern of bilateral prefrontal activation in older adults with that of more unilateral activation in younger adults. PASA describes both anterior (e.g., frontal) overactivation and posterior (e.g., occipital) underactivation in older adults relative to younger adults. CRUNCH emphasizes that the level or extent of brain activity can change in response to the level of task demand at any age. Finally, STAC and STAC-r take the broadest perspective to incorporate individual differences in brain structure, the capacity to implement functional scaffolding, and life-course neural enrichment and depletion factors to predict cognition and cognitive change across the lifespan. Extant empirical work has documented that compensatory overactivation can be observed in regions beyond the prefrontal cortex, that variations in task difficulty influence the degree of brain activation, and that younger adults can show compensatory overactivation under high mental demands. Additional research utilizing experimental designs (e.g., transcranial magnetic stimulation), longitudinal assessments, greater regional precision, both verbal and nonverbal material, and measures of individual difference factors will continue to refine our understanding of age-related activation differences and adjudicate among these various accounts of neurocognitive aging.

Keywords: aging, functional magnetic resonance imaging (fMRI), compensation, HAROLD, PASA, CRUNCH, STAC-r and positron emission tomography (PET)

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On average, increasing age brings general declines in such fluid cognitive abilities as response speed, memory, and problem

solving, along with systematic brain shrinkage (e.g., Fjell & Walhovd, 2010; Park et al., 2002; Raz, Ghisletta, Rodrigue, Kennedy, & Lindenberger, 2010). Yet, in many instances, measures of brain function during cognitive tasks reveal greater brain activity in older adults relative to younger adults. A handful of groundbreaking positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies found somewhat paradoxical evidence for such age-related overactivation (e.g., Cabeza et al., 2004; Grady et al., 1994; Reuter-Lorenz et al., 2000), often in the presence of equivalent task performance and involving bilateral activation of both cerebral hemispheres, especially the frontal cortex (e.g., Cabeza, 2002; Reuter-Lorenz et al., 2000). These observations were revolutionary because older adults' behavioral and structural deficits relative to younger adults (e.g., lower accuracy, slower response time, cortical thinning, brain atrophy) led many researchers to expect that older adults would show less neural activity than younger adults when performing cognitive tasks. A frequent interpretation of these counterintuitive results is that greater brain activation during cognitive tasks reflects potential compensatory processes in older adults.

Nonetheless, it is important to note that overactivation has not been universally observed in functional imaging studies of aging. In some reports, older adults, often classified as 60 years of age and older, exhibit less brain activation than their younger counterparts, for example during memory encoding (e.g., Grady et al., 1995; Logan, Sanders, Snyder, Morris, & Buckner, 2002) and sensory processing (e.g., Grady et al., 1994), or in association with poor performance (e.g., Jonides, Marshuetz, Smith, Reuter-Lorenz, & Koeppe, 2000; Nagel et al., 2009). As such, cognitive neuroscientists have been posed with a complex range of results, prompting finer-grained questions about when and why older adults would show greater brain activation than their younger counterparts.

In response to this challenge, several models have been proposed (see also Fabiani, 2012; Greenwood, 2007). This article focuses on four major cognitive neuroscience theories that remain highly influential: the Hemispheric Asymmetry Reduction in Older Adults (HAROLD) model, the Posterior-Anterior Shift in Aging (PASA) model, the Compensation-Related Utilization of Neural Circuits Hypothesis (CRUNCH), and the Scaffolding Theory of Aging and Cognition (STAC; STAC-r). Many ideas postulated in these models overlap—especially the ideas of compensatory recruitment of neural networks to buttress performance and greater reliance on frontal brain regions for compensation. In this review, the propositions of each theory are summarized and evaluated based on how they fare, given recent empirical evidence. Importantly, these models are complementary, with different explanatory scopes, and none have yet been falsified.

The main theories of neurocognitive aging focus on compensatory (i.e., beneficial, counteracting) processes in older adults to assist cognitive performance, with less emphasis on decline. One important exception is the related theoretical notion of dedifferentiation. This notion originated from the finding of increased correlations among disparate behavioral measures with increasing age (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1997). In the neuroscience domain, the term *dedifferentiation* refers to greater similarity or reduced distinctiveness of neural responses, such that brain activation patterns are less specific to a particular type of input or mental state. Dedifferentiation has been best documented in the visual modality, where older adults show a breakdown of the expected domain-specific activation in response to various categories of stimuli in ventral visual pathways (e.g., faces or houses; Park et al., 2012; see also Carp, Gmeindl, & Reuter-Lorenz, 2010). Dedifferentiation is also observed in older adults during associative encoding of memoranda, with less dedifferentiation (i.e., greater specificity) correlated with better memory (Saverino et al., 2016; see also Grady, 2012).

Dedifferentiation is considered an adverse consequence of aging, such that more of it is associated with poorer behavioral outcomes (e.g., Park, Carp, Hebrank, Park, & Polk, 2010) Importantly, dedifferentiation and compensation are potentially

opposing interpretations of more widespread or nonspecific patterns of brain activity in older adults relative to younger adults, and distinguishing between these interpretations requires consideration of the nature of the process or task eliciting the activity pattern, the specific brain regions in which the pattern is observed, associations between activity and behavioral performance, and other variables. Critically, evidence for both compensation and dedifferentiation can be found in different brain regions within the same individuals (Carp et al., 2010; Du, Buchsbaum, Grady, & Alain, 2016), which is consistent with the general proposal that overactivity can provide compensation that offsets the adverse effects of neural decline in the aging brain, including dedifferentiation or less efficient neural circuitry.

Thus, we acknowledge that dedifferentiation also contributes to age differences in brain activation, but here, we focus on compensatory accounts of age-related neural activation differences. We review the tenets of four prominent models next. To assist with these model comparisons, the key features of HAROLD, PASA, CRUNCH, STAC, and STAC-r are summarized in Table 1. We also include Figure 1, which depicts the explanatory scope of each model.

Table 1. Summary of Model Considerations for Each Theory of Age-Related Neural Compensation

| **Model Considerations** | **HAROLD** | **PASA** | **CRUNCH** | **STAC** | **STAC-r** |
|---|---|---|---|---|---|
| Compensation | ✓ | ✓ | ✓ | ✓ | ✓ |
| Frontal recruitment | ✓ | ✓ | ✓ | ✓ | ✓ |
| Dedifferentiation | ✓ | ✓ | ✓ | ✓ | ✓ |
| Predicts brain overactivation | ✓ | ✓ | ✓ | ✓ | ✓ |
| Predicts brain underactivation | | ✓ | ✓ | ✓ | ✓ |
| Predicts cognition | | | ✓ | ✓ | ✓ |
| Specific to older adults | ✓ | ✓ | | | |
| Relevant to all ages | | | ✓ | ✓ | ✓ |
| Task-demand dependent | | | ✓ | | |
| Within-individual predictions | | | ✓ | | ✓ |
| Regional specificity | ✓ | ✓ | | | |
| Bilateral activation | ✓ | | ✓ | ✓ | ✓ |
| Brain structure | | | ✓ | ✓ | ✓ |
| Modulate with training and experience | | | ✓ | ✓ | ✓ |
| Individual difference factors | | | ✓ | ✓ | ✓ |
| Longitudinal predictions | | | | | ✓ |
| Life-course experiences | | | | | ✓ |

*Note*. Blank cells indicate that a particular model characteristic is not a primary component of the model in question.

# Summary of the Theories

## HAROLD

In 2002, Roberto Cabeza proposed the HAROLD theory (Cabeza, 2002). According to this model, older adults show less lateralized prefrontal activity than younger adults while performing the same cognitive task. That is, younger adults show largely unilateral prefrontal activation patterns, whereas older adults show bilateral activation of the prefrontal cortex. This so-called asymmetry reduction was proposed to reflect either compensatory processes or dedifferentiation. Cabeza notes that the two processes need not be mutually exclusive, in that it is possible for dedifferentiation to trigger compensatory responses (see also Reuter-Lorenz, 2002; Reuter-Lorenz, Stanczak, & Miller, 1999). For example, older adults may bring more domain-general processes to bear on cognitive operations that younger adults' brains can mediate with domain-specific processes in the event that domain-specific processes are compromised by dedifferentiation or other age-related adverse processes (e.g., Carp et al., 2010). Nonetheless, the HAROLD model also offers the possibility that dedifferentiation could be an age-related by-product that does not necessarily serve a compensatory function.

The HAROLD model was based on several empirical studies that documented bilateral prefrontal recruitment in older adults during tasks that correspond to a range of cognitive domains, including episodic memory, working memory, semantic memory, visual perception, and inhibitory control. For instance, during recall of paired associates, older adults exhibited bilateral prefrontal activation, whereas younger adults exhibited right-lateralized activation (Cabeza et al., 1997). Moreover, older adults showed bilateral prefrontal recruitment during both verbal and spatial working memory tasks, whereas younger adults had left-lateralized activity during verbal working memory and right-lateralized prefrontal activity during spatial working memory (Reuter-Lorenz et al., 2000).

As originally proposed, HAROLD was solely applied to older adults (i.e., as specified within the name of the model). Moreover, the locus of asymmetry reduction was restricted to the prefrontal cortex. These features distinguish the model from CRUNCH, STAC, and STAC-r, which allow compensatory bilateral overactivation in regions beyond the prefrontal cortex, and at all ages of the lifespan, and relax the assumption that compensatory activity is necessarily bilateral. In 2012, the HAROLD model was revised to encompass a broader understanding of age-related compensation that loosens these original restrictions and distinguishes between successful compensation and unsuccessful compensation (Cabeza & Dennis, 2012). Specifically, successful compensation occurs when, in response to a discrepancy between task demands and typically used neural resources, the recruitment of additional neural reserve resources is associated with better cognitive performance. Compensation is unsuccessful, however, when this recruitment of additional neural resources is not associated with cognitive performance or is associated with worse cognitive performance.

## PASA

The PASA model (Davis, Dennis, Daselaar, Fleck, & Cabeza, 2008; Dennis & Cabeza, 2008) emphasizes a pair of observations in older adults—less activation of posterior (e.g., occipital) brain regions, along with greater activation of anterior (e.g., frontal) brain regions relative to younger adults. The observation that this pattern can be found even when performance on a given task is equivalent between both age groups may lead some to predict that older adults experience more subjective task difficulty, which drives activation differences across age groups. However, this task difficulty explanation is not favored by the creators of

PASA because activation differences occurred even when both older and younger adults reported comparable subjective confidence. The PASA model also proposes that the frontal overactivation is compensatory, in that it reflects an attempt by older adults to maximize their behavioral performance and offset occipitotemporal sensory deficits. As such, the model derives from evidence that age-related frontal overactivation is often positively correlated with performance and negatively correlated with occipital activity (e.g., Davis et al., 2008).

Many neuroimaging studies report this PASA pattern (see Cabeza & Dennis, 2012; Dennis & Cabeza, 2008). For example, Grady et al. (1994) noted that older adults exhibited more activation in anterior brain regions (e.g., the prefrontal cortex) and less activation in occipitotemporal regions than younger adults during a visual perception task, and speculated that this pattern reflected compensation. Gutchess et al. (2005) demonstrated that compared to younger adults, older adults overactivated frontal regions and underactivated medial temporal lobe regions during memory encoding. Those older adults who exhibited the lowest activation of posterior regions tended to have greater frontal activation, again suggesting the possibility of a compensatory mechanism. Moreover, Cabeza et al. (2004) documented this posterior-to-anterior shift in activation in visual attention, working memory, and episodic memory-retrieval tasks, emphasizing that the effect is not restricted to a particular cognitive domain.

The PASA model is unique in that it outlines two effects (i.e., both a decrease and an increase in activation), whereas the other theories reviewed here focus primarily on overactivation (although CRUNCH also considers underactivation at very high task demands). Furthermore, PASA has also been used to describe deactivation of the default mode network, in that older adults can have reduced deactivation of posterior midline default regions (e.g., precuneus) yet increased deactivation of default regions along the anterior midline (e.g., the medial prefrontal cortex; Davis et al., 2008). One of the major distinguishing features of PASA is that it does not specify a bilateral pattern of activity—solely a shift from posterior activation to anterior activation. Thus, the model is more specific about regional localization (i.e., frontal lobe, although compensatory activity in the parietal lobe is also considered) but less specific about laterality. PASA is also exclusively proposed as an aging model, whereas CRUNCH and STAC encompass the lifespan.

## CRUNCH

The CRUNCH model posits that age-related overactivation is compensatory and varies with the level of task demand (Reuter-Lorenz & Cappell, 2008; Reuter-Lorenz & Lustig, 2005; Reuter-Lorenz & Mikels, 2006). In general, older adults are more likely to show greater activation than younger adults at relatively low levels of task demand, even when performance is age-equivalent. As task demand increases, older adults reach their capacity for compensatory recruitment, and additional compensation is not possible. Thus, during the most difficult tasks, activation levels may fall below those of younger adults. The inflection point along the task demand axis where activity reaches its peak level and begins to fall off is referred to as the *crunch point*. This point is specific to a particular individual and, in principle, could be shifted by exercise, cognitive training, neural damage, sleep deprivation, or genetic predisposition (Lustig, Shah, Seidler, & Reuter-Lorenz, 2009). Group-level crunch points are obtained when aggregating data across participants.

Importantly, the CRUNCH pattern is proposed to occur at any age, when the recruitment of additional neural circuitry provides computational resources needed to meet task demands (Banich, 1998; Reuter-Lorenz et al., 1999). Therefore, even younger adults can show compensatory neural activation when task demands are sufficiently high. One key method to test the CRUNCH model is to parametrically vary the task demands and observe the resulting differences in brain activation. For instance, Cappell, Gmeindl, and Reuter-Lorenz (2010) varied the set size (i.e., memory load) of a working memory task and found different

patterns of activity depending on the task demands. At low loads (i.e., before the group-level crunch point), age-related overactivation was observed in the right dorsolateral prefrontal cortex (DLPFC), with equivalent behavioral performance between both age groups. At high loads, (i.e., beyond the crunch point), older adults showed lower DLPFC activity than younger adults, as well as poorer performance (see also Mattay et al., 2006).

The CRUNCH model also posits that regions exhibiting overactivation could be compensating for processing inefficiency within that region or elsewhere in the brain. Such inefficiency may be due to structural degradation, faulty input, or increased neural noise (e.g., dedifferentiation or default mode network dysregulation). Thus, CRUNCH differs from HAROLD by focusing specifically on compensation and why compensation might occur (i.e., degraded brain structures/functions, as a means to assist performance with increasing task difficulty). It also allows compensatory activity to occur anywhere in the brain—for instance, parietal cortex (Berlingeri, Danelli, Bottini, Sberna, & Paulesu, 2013; Huang, Polk, Goh, & Park, 2012), as well as bilateral prefrontal cortex—at any age. Nonetheless, the defining feature of CRUNCH is that the extent and functional impact of compensation-related activity vary with the level of task demand and the availability of resources to meet that demand. In this regard, CRUNCH uniquely makes within-person predictions that can include both underactivation and overactivation as functions of task demand and resource supply, unlike HAROLD and PASA, which were proposed based on comparisons between participants.

## STAC

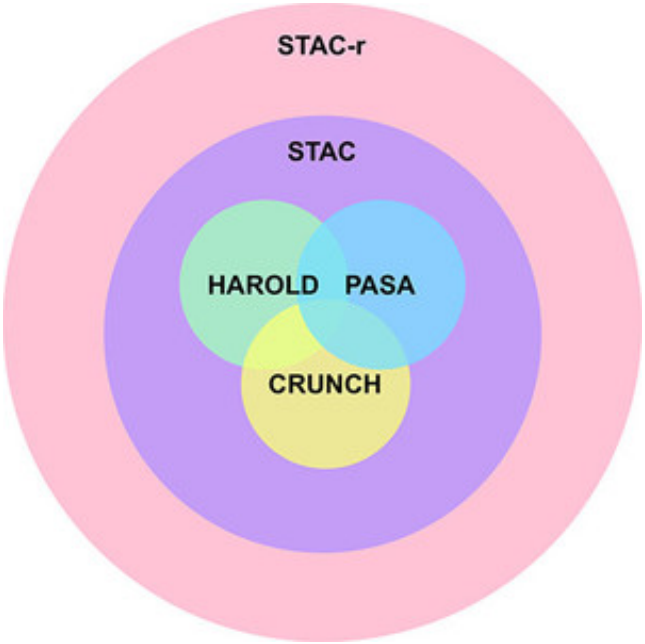
Relative to the aforementioned models, STAC takes a broader scope, predicting overall cognitive function while also specifically considering brain structure, as opposed to focusing principally on brain function (Park & Reuter-Lorenz, 2009). The theory posits that older adults recruit compensatory neural *scaffolding* as an adaptive response to declining brain structure and brain function. Those older adults with less neural degeneration, greater ability to recruit compensatory scaffolds, or both will have better cognitive performance than older adults who are unable to use scaffolding to compensate for their neural declines. Although the theory is formulated to compare extreme groups of younger and older adults, the authors also note that the model can be applied to cross-sectional comparisons across the adult lifespan.

The concept of scaffolding is adopted from theories of child development whereby existing cognitive abilities support the acquisition of new ones, and from skill acquisition, where early stages of skill development are supported by broader networks that become honed and specialized with increasing expertise (Park & Reuter-Lorenz, 2009). In the context of older age, scaffolding entails the recruitment of additional, complementary neural resources to support specialized circuitry to preserve cognitive performance. Scaffolding may occur in any brain region, although the prefrontal cortex is a frequent locus because its neural circuits are more flexibly configured and domain-general (e.g., Badre, 2008; Miller, Freedman, & Wallis, 2002), and thus they are particularly well suited to provide compensation. Somewhat consistent with the PASA model, STAC suggests age-related underactivation may cooccur in more specialized brain areas (e.g., visual regions), whereas overactivation is more likely in regions better suited for higher-order, flexible processing (e.g., the prefrontal and parietal cortices).

Unlike the previous theories, the STAC model explicitly incorporates structural factors by highlighting the fact that preserved brain structure will support cognitive stability via both the absence of neural decline and greater capacity to utilize scaffolding if neural declines emerge. Thus, the STAC model also incorporates the concept of brain maintenance (Nyberg, Lovden, Riklund, Lindenberger, & Backman, 2012). *Brain maintenance* posits that individuals with a relative lack of brain pathology (i.e., a more youth-like brain structure) will exhibit successful aging and better cognition. According to the STAC model, brain maintenance

would reduce the need for compensatory scaffolding, but compensatory scaffolding may still be used to support performance in the face of sufficient neural challenge. For example, older adults showing activation patterns similar to younger adults during memory encoding—that is, low functional activity deviation during encoding (FADE)—did not show compensatory prefrontal activity and had spared recollective memory (Duzel, Schutze, Yonelinas, & Heinze, 2011). On the whole, the STAC theory emphasizes that an interplay between neural preservation, neural challenges, and compensatory scaffolding determines cognitive function.

## STAC-r




*Click to view larger*

*Figure 1.* Depiction of the explanatory scope of the HAROLD, PASA, CRUNCH, STAC, and STAC-r models. STAC-r has the broadest scope, incorporating longitudinal changes and life-course neural enrichment and depletion factors, in addition to the influences of brain structure and brain function on compensatory scaffolding and cognition, which were modeled cross-sectionally by the original STAC model. HAROLD, PASA, and CRUNCH explain portions of the functional brain changes discussed within STAC and STAC-r. All the models converge on the idea that age-related differences in neural activation can be compensatory to support cognitive performance.

The STAC-r (the *r* stands for "revised") model builds on the STAC model to take a more comprehensive perspective that extends beyond cross-sectional age comparisons (Reuter-Lorenz & Park, 2014). STAC-r incorporates all aspects of STAC while also including longitudinal change in cognition and life-course experiences, which can serve as neural enrichment (e.g., education, physical fitness) or neural depletion (e.g., depression, vascular disease) factors. These factors are posited to influence brain structure, brain function, and the potential for compensatory scaffolding, which collectively predict the level of cognition and rate of cognitive change. Unlike the concept of *cognitive reserve* (e.g., Barulli & Stern, 2013; Stern, 2002), which focuses on how enriching lifestyles (i.e., most commonly high education or occupation complexity) counteract the cognitive impact of brain pathology, the STAC-r model proposes that these enriching and depleting factors can directly influence brain structure, brain function, and compensatory scaffolding throughout the lifespan, even in the absence of brain pathology.

According to STAC-r, the influence of life-course factors, brain structure, brain function, compensatory scaffolding, and one's baseline cognitive level are all proposed to influence the rate of change in cognition over time, making STAC-r the only model that specifically considers changes in brain structure and function over time. In sum, STAC-r takes the broadest scope to consider how life-course and neural factors contribute to functional activation differences, varying levels of cognitive performance, and longitudinal trajectories of cognitive change.

# Summary of Recent Empirical Evidence

Since these neurocognitive aging theories were originally proposed, cognitive neuroscientists have been actively testing tenets

of these models to understand the functional significance of overactivation and the potential for compensation in the aging brain. Studies have been specifically designed to determine the boundary conditions for observing overactivation in specific brain regions and to identify individual difference variables that might predict characteristic patterns of activation. This section reviews a sampling of recent studies and meta-analyses, chosen based on their great relevance to the four models of neurocognitive aging that are the focus of this review.

A recent meta-analysis by Li et al. (2015) assessed over 100 fMRI cross-sectional studies of healthy aging. The main findings were that relative to younger adults, older adults tended to underactivate the visual network and overactivate the frontoparietal control and default networks (see also Maillet & Rajah, 2014). Of particular relevance for the notion of compensation was the fact that increased frontoparietal activation was observed in studies in which older adults achieved performance similar to younger adults. Thus, this meta-analysis corroborates the relative underactivation of posterior (i.e., visual) regions and overactivation of frontal regions described by PASA, and provides evidence that frontoparietal overactivation may assist cognitive performance in older adults. However, an earlier meta-analysis concluded that overactivation of the right prefrontal cortex was associated with worse performance (Spreng, Wojtowicz, & Grady, 2010). While the inconsistency of these conclusions with respect to overactivation and behavior occurs partly because the specific studies included in each meta-analysis differed by more than 70, it also underscores the importance of considering other factors that influence the degree of neural activation, such as the level of task demands, as in CRUNCH, or the fact that compensation can be either successful or unsuccessful (Cabeza & Dennis, 2012).

Moreover, a previous meta-analysis by Turner and Spreng (2012) emphasized the need to consider the specific cognitive domains under investigation. Their analysis indicated that bilateral prefrontal overactivation was observed in older adults during working memory tasks, whereas unilateral but more widespread overactivation of similar regions (i.e., a “young plus” pattern) was observed in older adults during executive function tests of inhibition. Accordingly, although the major theoretical models are formulated in general terms with regard to the types of tasks they encompass, it ultimately may be necessary to specify the range and types of cognitive operations that pertain to specific model predictions.

Another area of focus of recent empirical work concerns interindividual differences. Interindividual differences (e.g., in cognitive abilities, life-course experiences, genetics, brain structure, cardiorespiratory fitness, psychological health, etc.) are an important source of variability in brain activation and cognitive performance in older adults. With regard to episodic memory, for example, some older adults show substantial performance declines over 15–20 years, whereas others display relatively stable memory (see Nyberg, 2017). Interestingly, those older adults who maintained episodic memory performance over time tended to show preserved hippocampal activation during encoding, whereas those older adults with memory declines tended to show decreases in hippocampal activation (Persson et al., 2012).

Given these variable behavioral profiles and interindividual differences in hippocampal activation change, we might expect similar interindividual variability in the extent and trajectory of compensatory frontal overactivation, yet Persson et al. (2012) found no relationship between prefrontal activity and longitudinal changes in memory, but Pudas et al. (2013) did. For instance, several recent studies have found that individual differences in cognitive reserve proxies can influence observed frontal recruitment. Older adults with higher levels of education exhibited greater frontal activations during a working memory task (Boller, Mellah, Ducharme-Laliberte, & Belleville, 2016), and variability in executive function abilities were also informative of compensatory frontal activation patterns on an episodic memory task (Angel et al., 2016). Nevertheless, greater activity is not always associated with better behavioral performance. In Boller et al. (2016), education was not related to n-back performance,

but in Angel et al. (2016), the degree of overactivation of frontoparietal regions was associated with better recognition memory in those older adults with high executive abilities.

When considering each model's predictions about compensatory activation, it will likely be useful to specify the long-standing and acquired factors that influence interindividual variability (e.g., as in STAC-r). This undertaking will be challenging, however, as some individual differences can be influenced by the nature of the task demand (cf. CRUNCH) and may vary by brain region. One example of this complexity comes from a study that assessed cardiorespiratory fitness via a measure of $VO_2$-max (Hayes, Hayes, Williams, Liu, & Verfaellie, 2017). Older adults who were more fit exhibited less age-related underactivation in certain brain regions (e.g., fusiform gyrus, medial frontal cortex) during face-name associative encoding, yet larger age-related overactivation in other regions (e.g., bilateral prefrontal cortex). Notably, the overactivations were associated with better source memory performance in fitter older adults. As the authors discuss, the reduced age differences in the blood-oxygen-level-dependent (BOLD) signal would be consistent with a brain maintenance account, whereas the larger age differences in the BOLD signal would be consistent with compensatory activation. These results simultaneously demonstrated smaller and larger age differences in activation after considering cardiorespiratory fitness as an individual difference factor, indicating that interindividual variability in a single factor will not necessarily have a uniform impact on activation patterns during a particular task.

Given that this complexity cannot be fully unraveled with cross-sectional data, recent empirical work has also increasingly employed longitudinal fMRI paradigms. In a somewhat surprising finding, Nyberg et al. (2010) reported that, in a sample of adults aged 49–84, cross-sectional analyses of semantic classification performance revealed age-related frontal overactivation, while longitudinal analyses of the same task suggested that frontal activation declines with advancing age. Thus, cross-sectional assessment of age differences in brain activation may amplify activation differences that are not observed longitudinally. The inherent differences in statistical analyses utilized by longitudinal and cross-sectional methodologies (i.e., within-person changes versus between-participant differences) constitute one source of this apparent inconsistency. Nyberg et al. (2010) also note the caveat that people who completed both scans may be higher performing (i.e., still able to meet study criteria for the second assessment, able to partake in extensive testing), contributing to the observed decreases in frontal activation over the 6-year interval. Indeed, those older adults who were not lost to attrition had significantly better episodic memory and higher right frontal activity at the first assessment.

In contrast, Hakun, Zhu, Brown, Johnson, and Gold (2015) reported longitudinal increases in activation in bilateral frontal cortex during task-switching in older adults over a 3.3-year interval. This result is consistent with accounts of increased frontal recruitment with increasing age. However, in this study, longitudinal increases in left ventrolateral prefrontal cortex activity were associated with declining performance (i.e., slowing). One source of the discrepancy between these longitudinal studies may be due to the different cognitive processes involved in the tasks (i.e., semantic classification versus task switching). Another potential source of divergence is the influence of individual differences in cognitive trajectories, perhaps due to differences in underlying brain pathology. Older adults who maintained episodic memory performance over 15–20 years tend to show preserved hippocampal activation over time, whereas older adults with longitudinal episodic memory decline tend to show decreases in hippocampal activation (Persson et al., 2012; see also Nyberg, 2017). The field would benefit from additional longitudinal assessments of neural activation and structural integrity, as well as studies that also consider interindividual variability in task performance, reserve capacity, and attrition.

While longitudinal studies improve upon the explanatory capability of cross-sectional studies, they are often still limited by

correlational designs, in that cognitive performance has been associated with a specific pattern of brain activity. Fortunately, another technique, transcranial magnetic stimulation (TMS), allows researchers to experimentally alter the electrical activity of the brain and observe its causal consequences. One mode of TMS allows researchers to momentarily decrease the excitability of a region of cortex (i.e., create a temporary lesion), whereas another mode can increase the excitability of the cortex. Two of the most influential TMS studies related to theories of age-related compensation, discussed next, are considered classic experiments.

Rossi et al. (2004) demonstrated that applying TMS to either the right or left DLPFC interfered with performance in older adults, but not younger adults, suggesting that bilateral recruitment of the DLPFC was serving a compensatory role in the older sample. Using a different form of stimulation that increases neural excitability, Sole-Padulles et al. (2006) demonstrated that high-frequency, repetitive TMS over the prefrontal cortex could improve memory in older adults by increasing bilateral frontal recruitment. Interindividual variability has also been documented within a TMS study. Manenti, Cotelli, and Miniussi (2011) reported that older adults with better memory had less prefrontal asymmetry (i.e., more bilateral recruitment), as disruptive TMS applied to the DLPFC during memory encoding differentially interfered with performance depending on whether the right or left hemisphere was stimulated only in low-performing older adults. Additional research using similar TMS paradigms to replicate whether disrupting (or facilitating) bilateral activation disturbs (or assists) performance under more variable task and stimulus domains will be highly informative.

In addition to meta-analyses, longitudinal studies, and TMS studies, several recent empirical papers have been useful in further assessing the models and determining whether compensatory recruitment can be found at any age. Höller-Wallscheid, Thier, Pomper, and Lindner (2017) matched the level of subjective working memory task demand in younger and older adults and found that both younger and older adults showed bilateral recruitment of the DLPFC and anterior PFC during verbal, spatial, and object maintenance in working memory when the demands were subjectively high. Hence, these results provide more support for the CRUNCH model, which has specific predictions regarding task demands and also allows overactivation to occur at all ages, whereas HAROLD and PASA do not consider the influence of task demands and discuss activation differences only with respect to older versus younger adults. More evidence for compensatory activation across the lifespan comes from Kwon et al. (2016), who reported that greater anterior prefrontal cortex activation during memory retrieval predicted better accuracy in middle-aged adults. Thus, even in middle-aged and younger adults, recruitment of additional neural circuitry has been shown to support performance, given sufficient task difficultly.

# Additional Sources of Age-Related Variation in the BOLD Signal

While age-related increases in functional activation are frequently observed and attributed to cognitive or neural compensation, it is important to consider other potential mechanistic sources of the activation differences. One area of interest is the influence of cerebrovascular changes with age and their effects on functional activation patterns. Some studies suggest that age differences in BOLD activity may be due to differences in vascular reactivity with aging (e.g., Tsvetanov et al., 2015), while others suggest that there is only a small vascular contribution to age differences in the BOLD signal (e.g., Kannurpatti, Motes, Rypma, & Biswal, 2010). Although vascular differences may influence age differences in BOLD activation and the regional specificity of age-related effects, the evidence for variations in prefrontal activity in response to task difficulty and within younger adults argues that compensatory activation patterns are not solely due to age differences in vasculature responsivity.

In addition, differences in brain structure have been examined as sources of activation variation, as brain structure also varies

with age. For instance, gray matter loss has been proposed to account for age-related overactivation of the left DLPFC and left lateral parietal cortex (Kalpouzos, Persson, & Nyberg, 2012). Moreover, areas of the DLPFC displaying functional overactivation have shown high overlap with regions exhibiting large gray matter reduction (Di, Rypma, & Biswal, 2014; but see also Berlingeri et al., 2010; Maillet & Rajah, 2013). Because these studies are correlational, it cannot be determined if gray matter loss causes activation differences. Nevertheless, not all activation differences are likely to be artifacts of structural differences, as gray matter volume did not account for overactivation in all brain regions (e.g., the dorsomedial prefrontal cortex; Kalpouzos et al., 2012).

Age-related white-matter changes have also been discussed as a potential cause of activation changes. For instance, Daselaar et al. (2015) found that greater white-matter decline was associated with greater success-related activity in older adults. They describe this pattern as "less wiring, more firing" and indicate that the observed functional overactivation may compensate for white-matter decline. Such brain structure differences contributing to brain activation are compatible with the CRUNCH, STAC, and STAC-r models, which consider the influence of brain structure on compensatory activity. Nevertheless, as mentioned previously, such structural changes are unable to account for all differences in neural activation, as variability of activation in response to task difficulty cannot be explained exclusively by brain structure, which does not differ within individuals during a single scanning session.

In addition to changes in gray and white matter, age-related variations in dopamine levels, iron concentration, and amyloid and tau protein accumulation have been considered. Bäckman et al. (2011) report that adjusting for age-related differences in dopamine can dampen age-related differences in brain activation (see also Fischer et al., 2010). However, these effects were pertinent to age-related underactivation of task-relevant brain regions, in that after controlling for dopamine receptor binding potential, the load-dependent underactivation of task-relevant regions was attenuated. It is currently unknown to what extent age-dependent dopamine levels influence overactivation or compensation. More work using dopamine agonists and antagonists may help determine the potential causality of these effects.

With regard to amyloid, the presence of amyloid plaques in the brain has been associated with overactivation of default regions and the hippocampus (e.g., Sperling et al., 2009), as well as with reduced activity in the dorsolateral prefrontal cortex and reduced suppression (i.e., overactivation) of default regions, including the superior/medial frontal cortex and lateral temporal cortex. Moreover, Kennedy et al. (2012) found evidence that these amyloid-related differences in activation were associated with poorer processing speed, verbal fluency, and reasoning, whereas no associations with memory were observed in Sperling et al. (2009). Thus, the presence of amyloid in cognitively normal older adults has been related to both increases and decreases in neural activation, but is not always related to behavior. The observed overactivation has been suggested as a potential compensatory mechanism to maintain performance or as an indicator of future memory impairment (Sperling et al., 2009); but the consequences are largely unknown. Finally, with regard to iron accumulation, some studies find an association between elevated iron concentration in the brain and poor cognitive function, but more work is needed to assess this relationship and its influence on brain structure and activation (Daugherty & Raz, 2015). Overall, many biological factors likely influence the degree of brain activation observed in studies of neurocognitive aging, although more work is needed to better understand the degree to which each plays a role. Regardless, compensatory processes remain a highly plausible source of age differences in brain activation.

# How the Theories Stack Up

As can be gleaned by this overview, the extant theories of compensatory activation patterns in older adults are relevant to the literature highlighted here. These theories endeavor to account for patterns of results observed repeatedly and have allowed researchers to discuss, appraise, and advance new ideas about plasticity, individual differences, and multiple determinants of neurocognitive aging. Next, we evaluate each of the models to assess its fit with recent research.

## Evaluating HAROLD

The HAROLD model has effectively characterized a prominent pattern of brain activity differences in older adults. The general observation of age-related bilateral prefrontal recruitment has been observed in countless empirical studies across many cognitive domains (Cabeza, 2002; Cabeza & Dennis, 2012; Dennis & Cabeza, 2008), and this is recognized as a hallmark pattern of aging often associated with compensatory interpretations. As with all theories, however, the model does not pertain to all empirical findings. Notably, HAROLD was originally proposed with regard to reduced lateralization (i.e., asymmetry reduction) in the prefrontal cortex. Yet age-related bilateral activation can also be observed outside the prefrontal cortex, such as in the parietal cortex (e.g., Berlingeri et al., 2013; Höller-Wallscheid et al., 2017; Huang et al., 2012; Li et al., 2015; Maillet & Rajah, 2014). Moreover, the model was originally proposed only for older adults, when extant empirical findings indicate that younger adults can exhibit similar bilateral activations at sufficient task demands (e.g., Cappell et al., 2010; Höller-Wallscheid et al., 2017; Schneider-Garces et al., 2010). Overactivation is not always bilateral in older adults; for example, older adults can exhibit broader swaths of activation of the same general lateralized region relative to younger adults during inhibition (Turner & Spreng, 2012). Because individual differences can influence the degree of bilaterality (e.g., Angel et al., 2016; Duzel et al., 2011), HAROLD may not be an inevitable feature of neural aging.

Overall, it appears that HAROLD may be one possible pattern associated with age-related compensation or age differences in activation generally, but not the only one (see also Berlingeri et al., 2013). In addition, it is largely descriptive in nature, without offering mechanistic explanations for why the HAROLD pattern may be observed. Going forward, the field may also benefit from quantification of the degree of bilateral activation (cf. Berlingeri et al., 2013), identification of the extent to which it occurs in homologous brain regions, and more consistent efforts to relate age differences in activation to performance patterns so as to differentiate successful and unsuccessful compensation (Cabeza & Dennis, 2012) versus activity that indicates dysfunction.

## Evaluating PASA

The PASA model has been influential in its characterization of both increases (e.g., frontal) and decreases (e.g., occipital) in age-related brain activation. Meta-analyses find reduced recruitment of occipitotemporal regions and increased recruitment of frontoparietal regions cross-sectionally, which support the general PASA framework (Li et al., 2015; Maillet & Rajah, 2014; see also Morcom & Johnson, 2015). Further, an anterior shift in electrophysiological indices has been observed in older adults (Alperin, Mott, Rentz, Holcomb, & Daffner, 2014). Nevertheless, causal evidence (e.g., from TMS studies) does not yet exist to support PASA (see also Cabeza & Dennis, 2012). For example, it has not been determined whether older adults with the least occipitotemporal activity will be most disrupted by frontal TMS because they are most affected by the TMS-diminished ability to rely on frontal activity to maintain behavior. Moreover, the PASA pattern was not formulated to consider task difficulty, now known to be an important determinant of activation levels (e.g., Cappell et al., 2010; Höller-Wallscheid et al., 2017; Schneider-

Garces et al., 2010).

Future work should document within-individual occipitotemporal activation levels under varying task demands, as occipital activity may be more stable in contrast to prefrontal activity, which clearly varies with demand, at least in some cognitive domains. Like HAROLD, PASA was also originally proposed only for older adults. While increasing activation with increasing task demands has been documented in younger adults, posterior underactivation may be a phenomenon unique to aging, indicative of age-related pathology. If so, this would be consistent with PASA's status as an aging model. Overall, the PASA model is informative in its incorporation of both overactivation and underactivation, but more work is needed to determine the causal nature of this relationship and how task demands and individual differences influence these effects, as is further documentation about activity-performance relationships.

## Evaluating CRUNCH

The key tenet of the CRUNCH model is that activation levels will vary as a function of task demand, but the extent to which activation can increase is limited. Note that the compensation account of increased activity assumes that without this activity, performance would suffer; greater activation indicates the recruitment of additional circuitry to support performance. CRUNCH's emphasis on task-demand dependence distinguishes it from the other models, and alterations in brain activation accompanying modulations of task difficulty have been replicated in many studies (e.g., Berlingeri et al., 2013; Cappell et al., 2010; Höller-Wallscheid et al., 2017; Kennedy et al., 2015; Schneider-Garces et al., 2010). Task performance also figures prominently in CRUNCH, in that increasing activation prior to the crunch point is posited to benefit performance, whereas after the crunch point, a resource ceiling is reached, leading to performance decline.

CRUNCH acknowledges that individual differences (e.g., age, variability in working memory span, brain damage, exercise) may heighten or shift one's crunch point, thereby altering the range of task demands to which the brain can respond. In this respect, CRUNCH aligns well with the notion that a more efficient network will be associated with lower activation at any given level of task demand relative to a less efficient network (e.g., Barulli & Stern, 2013; Dunst et al., 2014). Consequently, more efficient networks can respond to a greater range of task demands. Assuming that networks become less efficient with age, CRUNCH predicts increasing activation with increasing age, along with a narrowing dynamic range, or a reduced ability to modulate brain activity in response to demand. Both predictions were supported in a recent lifespan, cross-sectional study by Kennedy et al. (2015), which showed increasing and more widespread activation from young adulthood to middle age and into older adulthood, coupled with decreased modulation in response to differences in task demands. Lifespan and individual differences of these sorts are not specifically considered in HAROLD or PASA.

CRUNCH, like STAC, also recognizes compensatory activity at any age and in multiple brain regions, rather than restricting it to prefrontal regions (as in HAROLD) or older adults (as in HAROLD and PASA). Nevertheless, one criticism of the model is that it is capable of explaining a wide pattern of results by appealing to differences in task difficulty or participant ability, while these variables are not always measured appropriately. However, the capability to make a wide range of predictions depending on varying task demands and individual differences is arguably also a unique strength of the model. A focus on individual differences appears necessary, given the variability documented in both cross-sectional and longitudinal studies (e.g., see Nyberg, 2017). Henceforth, specifying particular hypotheses or objective ranges for individual differences and task difficulty parameters may assist with the assessment of when specific effects would be predicted.

## Evaluating STAC and STAC-r

The STAC and STAC-r models are unique in the breadth of the constructs that they consider when attempting to predict levels of cognition. Unlike the other models, STAC and STAC-r incorporate the degree of structural challenges, functional deterioration, and capacity for functional scaffolding to forecast cognitive performance. The degree of white- and gray-matter integrity has been noted to influence activation patterns (e.g., Daselaar et al., 2015; Di et al., 2014), indicating that variability in structural brain measures is important to consider. STAC-r also specifically posits that parietal regions are likely candidates for neural scaffolds, as bilateral parietal activity has been associated with better cognitive performance in older adults (e.g., Huang et al., 2012). Individual differences have been shown to inform activation patterns (e.g., Angel et al., 2016; Duzel et al., 2011), indicating the utility of STAC-r's unique considerations of life-course experiences (e.g., depression, socioeconomic status, education) as neural enrichment or depletion factors that influence cognitive level and longitudinal cognitive change. Nevertheless, the fact that STAC-r is a large, comprehensive model with many components makes it challenging to test multiple aspects of the model concurrently. There are practical limitations for any one study to assess brain structure, brain function, cognition, longitudinal changes in these measures, and numerous individual differences factors within the same sample. Hence, the STAC-r model is particularly useful for describing cognitive aging from a "big picture" perspective, but it is challenging to evaluate comprehensively.

# Conclusions

In sum, the HAROLD, PASA, CRUNCH, STAC, and STAC-r models have been highly informative for the characterization of neurocognitive aging. Before the development of these models, the idea of neural compensation in late adulthood had been suggested (for a review, see Grady, 2012) but informally. These models summarize patterns of brain activity that have been observed in numerous neuroimaging studies and provide theoretical guidance in the study of age-related compensation and associated benefits to performance. These theories have inspired a proliferation of studies that have revealed important information about the boundary conditions, regional and domain specificity, task difficulty dependence, individual differences that can influence patterns of brain activation, and most critically, the role of differential activity in determining cognitive capacity. With the introduction of these and other factors to consider, it has become increasingly complex to simultaneously model all the factors relevant to neurocognitive aging. Therefore, guideposts provided by these and related theories are even more valuable for designing and evaluating future studies.

With regard to future research, more longitudinal studies and studies implementing methodology that enables causal conclusions (e.g., TMS) will help determine whether changes in activation are truly compensatory. At present, a large majority of the research is based on younger versus older adult extreme group comparisons and observations of correlations between activation and performance at a single time point, which cannot imply causality or rule out preexisting differences in activation patterns (see also Morcom & Johnson, 2015). Because most studies use verbal tasks, future studies should determine how susceptible the laterality effects are to the involvement of language processing (but see Höller-Wallscheid et al., 2017; Reuter-Lorenz et al., 2000). New parcellation schemes (e.g., Glasser et al., 2016) could help gauge if bilateral activations take place in precisely homologous contralateral regions or exhibit more general bilaterality. Finally, the intentionality or degree of control that an individual has on compensatory processing is also unknown and has clear implications for intervention development. It could be that an increase in activation is a relatively automatic reaction to processing deficits elsewhere (see Bergerbest et al., 2009), but

it is also possible that individuals may be able to voluntarily boost compensatory activity by increasing their effort or utilizing different cognitive strategies. These additional areas for research will help refine these models, painting an ever-clearer picture of the aging mind and brain.

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