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Cognitive systems and the changing brain

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The notion of *cognitive system* is widely used in explanations in cognitive psychology and neuroscience. Traditional approaches define cognitive systems in an agent-relative way, that is, via top-down functional decomposition that assumes a cognitive agent as starting point. The extended cognition movement challenged that approach by questioning the primacy of the notion of cognitive agent. In response, [Adams, F., and K. Aizawa. 2001. *The Bounds of Cognition*. Oxford, UK: Wiley-Blackwell.] suggested that to have a clear understanding of what a cognitive system is we may need to solve "the demarcation challenge": the problem of identifying a reliable way to determine which mechanisms that are causally responsible for the production of a certain cognitive process constitute a cognitive system responsible for such process and which ones do not. Recently, [Rupert, R. 2009. *Cognitive Systems and the Extended Mind*. Oxford: Oxford University Press.] offered a solution based on the idea that the mechanisms that constitute a cognitive system are *integrated* in a particular sense. In this paper I critically review Rupert's solution and argue against it. Additionally, I argue that a successful account of cognitive system must accommodate the fact that the neural mechanisms causally responsible for the production of a cognitive process are diachronically dynamic and yet functionally stable. At the end, I offer a suggestion as to how to accommodate this diachronic dynamicity without losing functional stability. I conclude by drawing some implications for the discussion on cognitive ontologies.

Keywords: cognition; demarcation; extended mind; stability; ontology

1. Introduction

The notion of *cognitive system* is widely used in explanations in cognitive psychology and neuroscience. Traditionally, this notion has been agent-relative. Specifically, it has been assumed that any distinct functional component that processes information and that constitutes, and causally contributes to, the operations of a cognitive agent is a cognitive system (Bermudez 2010). According to this "top-down" approach, a cognitive agent is decomposable into different cognitive systems. In turn, each of these systems is decomposable into computationally more tractable cognitive sub-systems, until eventually the decomposition bottoms out at the level in which the information processing of the ultimate system is so simple that it cannot be broken further down. The idea that agents are so decomposable has been defended by philosophers (Dennett 1978; Lycan 1987) and cognitive scientists (Simon 1969; Marr 1976; Shallice 1988). Indeed, it has been

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suggested that functional decomposition – that is, the process of explaining cognitive faculties via breaking them down into its component systems – is the psychologists' preeminent scientific goal (Cummins 2000).

However, arguments from partisans of the extended cognition hypothesis (e.g., Clark and Chalmers 1998) have shown that the physical limits of cognitive agents are questionable, thus providing a shaky foundation for a definition of cognitive system. Without a clear understanding of where the boundaries of a cognitive agent are, it is difficult to determine which mechanisms compose it and which do not. As a result, philosophers are starting to support a different approach to understanding cognitive systems: one that does not take the notion of cognitive agent as basic. In essence, this "bottom-up" approach consists in finding necessary and sufficient conditions for a certain component to be a constitutive member of a cognitive system and/or for a set of components to constitute a cognitive system – as opposed to either a non-cognitive system or a non-system. The quest for finding such necessary and sufficient conditions has been dubbed "the demarcation challenge" (Rupert 2009; see also Adams and Aizawa 2001): the problem of identifying a reliable way to determine which of the mechanisms causally responsible for the production of a cognitive process constitute the cognitive system responsible for such process, and which ones do not.

A proposal to meet this challenge has been put forth by Rupert (2009, 2010, 2011). He suggests that cognitive systems are constituted by those mechanisms, capacities, abilities, external resources, etc. (mechanisms, for short) that differentially contribute to a specific task and that are *integrated*. Which mechanisms are integrated into a cognitive system depends, according to Rupert, on their conditional probability to causally contribute to the target cognitive task relative to any other causally contributing mechanisms (Rupert 2009). And such conditional probability, Rupert suggests, is likely determined by the relative frequency with which such mechanism co-contribute to bringing about the relevant task.

In this paper I argue that Rupert's notion of integration is inadequate to capture the nature of cognitive systems. Specifically, I suggest that, at least in is current formulation, it cannot capture the fact that the mechanisms that causally contribute to bringing about a task change over time. I shall argue that a successful account of cognitive system for cognitive neuroscience needs to be sensitive to the fact that neural mechanisms change over time – they exhibit *diachronic dynamicity* – without necessarily changing their functional profile – they exhibit *functional stability.* To that end, in Section 2, I reconstruct Rupert's notion of integration. Next, in Section 3, I offer a thought-experiment to show how one can manipulate the frequency of engagement of causally responsible mechanisms to end up with precisely the kinds of cognitive systems Rupert's notion of integration wanted to exclude. After evaluating some possible responses on Rupert's behalf, I introduce, in Section 4, evidence from the cognitive neuroscience of aging suggesting that the apparently far-fetched structure of the thought-experiment may actually reflect the crude reality of aging brains. More precisely, I argue that, as we age, the frequency with which certain brain mechanisms are involved in bringing about a cognitive task vary in ways that cannot be captured by Rupert's notion of integration. This leads me to suggest that if we want to keep using the notion of cognitive system to refer to mechanisms that underlie stable functional processes, we may need to replace Rupert's notion of integration. In Section 5 I suggest a possible strategy for such replacement based upon recent work in network

neuroscience, before concluding by drawing some implications for cognitive ontologies, in Section 6.

2. Integration

To meet the demarcation challenge, Rupert (2009) begins by noting that not everything that has ever causally contributed to bringing about a cognitive task is a proper mechanism of the cognitive system for that task and, thus, not every set of mechanisms causally responsible for bringing about a certain cognitive task constitutes a cognitive system. For a set of causally responsible mechanisms to constitute a cognitive system they need to be *integrated*. Despite the importance of the notion of integration, however, Rupert only spends a page and a half in spelling it out. In part, I think, because the gist of it can be succinctly put. In his review of Rupert's book, for instance, Klein (2010) summarizes the strategy of determining whether a set of mechanisms is integrated as a two-step procedure. The first step consists in ranking the set of all the mechanisms (internal and external) that have ever contributed to bringing about a cognitive task based upon their frequency of co-occurrence, and finding "a suitable gap" below which every set is removed. The second step consists in registering the frequency of occurrence of each mechanism in the remaining set and, as before, finding a "suitable gap, and throw[ing] out all the mechanisms that fall below the gap" (Klein 2010, 254). That's it. The members of this resultant set are integrated and, thus, constitute a cognitive system.

The devil is in the details, however, and to fully assess the notion of integration it is important to look at it formally: For any given subject, S, at any given time, t_n, there is a set of mechanisms, $M_n = \{m_1, m_2, m_3, \ldots, m_n\}$, causally contributing to a particular cognitive task, T, brought about by S. Two steps are now required to determine which M_n is S's cognitive system for T. First, we list all the M_n that, through time, have brought about T, and select only those that frequently co-occurred. For example, suppose that, through time, six different mechanisms $\{m_1, m_2, m_3, m_4, m_5, m_6\}$ have distinctively causally co-contributed to T. Thus, our list may look like this:

$$\begin{aligned}
&S'sT, \text{ at } t_1, \text{ was brought about by } M_1 = \{m_1, m_2, m_3, m_4, m_5\}\\
&S'tT, \text{ at } t_2, \text{ was brought about by } M_2 = \{m_1, m_2, m_3, m_6\}\\
&S'tT, \text{ at } t_3, \text{ was brought about by } M_3 = \{m_1, m_2, m_3, m_4\}\\
&S'tT, \text{ at } t_4, \text{ was brought about by } M_4 = \{m_2, m_3, m_5\}\\
&\ldots\\
&S'tT, \text{ at } t_m, \text{ was brought about by } M_m = \{m_1, m_2, m_3\}.
\end{aligned} \tag{1}$$

Once we have this list, we look at the history of co-contribution of every possible arrangement of m_n in each M_n that have brought about T, and we assign a probability for each m_n in bringing about T. In other words, we look at the number of times each possible arrangement of m_n appears in each M_n, and we rank their frequency of appearance as subsets of the M_n on the list. Notice that the assigned probability for each m_n will be conditional to every other m_n, as well as every other subset of m_n within each M_n. So, for instance, we may end up with

a ranked-ordered list of probability assignments that might look like this:

$$\begin{aligned} &P(m_1|m_2) = 0.9 \\ &P(m_1|m_3) = 0.8 \\ &P(m_2|m_3, m_4) = 0.7 \\ &\ldots. \\ &P(m_4|m_2, m_3) = 0.4 \\ &P(m_5|m_3, m_4, m_6) = 0.1. \end{aligned} \tag{2}$$

The next step is to identify the set of m_n which, given the frequency of co-contribution, is more likely to bring about *T*. To that end, Rupert suggests that once we have ranked every probability for every m_n in bringing about *T* given any other m_{m-n}, and every other subset of m_n, we find the set of those m_n whose combined probability is above.5 (unless there is another, more obvious cut off). This identifies the set of m_n whose history of co-contribution in bringing about *T* is way above chance.[1] For instance, in our example, that set could be $M_T = \{m_1, m_2, m_3\}$. For, let's say, $P(T \mid M_T) = .78$ since $[(P(m_1 \mid m_2), P(m_2 \mid m_3), \ldots P(m_3 \mid m_1 \& m_2)] = .78$. That's it! M_T forms an integrated set, and it can be identified with the cognitive system for *T*, as opposed to mere ancillary resources used in the process of bringing about *T*.

Following Rupert (2009, 2011), we can use this strategy to mark off mechanisms that may causally contribute to bringing about *T* at some point, but that should not be considered proper parts of the cognitive system for *T* insofar as they are not *integral* to it – that is, they are not part of the set of integrated mechanisms that, after following the procedure described above, are found to reliably bring about *T*. To illustrate, consider an adapted version of the fictional characters, Inga and Otto, introduced by Clark and Chalmers (1998) influential paper on the extended mind. One day, Inga hears about a new exhibition in the MoMA and decides to go. She thinks for a second and remembers that the museum is on 53rd St. Let's assume that, when retrieving this semantic information, she employs the following neural mechanisms $\{m_1, m_2, m_3, m_6\}$. Suppose further that we want to know which of these neural mechanisms constitute the cognitive system for retrieving semantic information – such as the MoMA's address – versus those that were merely employed as ancillary resources in that particular occasion.[2] Following Rupert's strategy, we should first list all the mechanisms ever involved in retrieving semantic information in accordance with (1) above. Next, we assign and rank-list the probabilities, as in (2), in order to identify the set of integrated mechanisms casually responsible for retrieving semantic information. For simplicity's sake, let's say that in the case of Inga such set, M_T, is in fact $\{m_1, m_2, m_3\}$. As such, m_6, which happened to have been causally involved in retrieving the address of the MoMA upon Inga's hearing about the new exhibition, was employed by her brain merely as a resource, and should not to be considered part of the cognitive system for retrieval of semantic information.

Now consider Otto. He, too, hears about the new exhibition, and he, too, thinks for a second where the MoMA is located. Unfortunately, Otto is starting to show signs of Alzheimer's disease, and is having a hard time remembering semantic information. For that reason, he keeps a notebook with handy facts written down, such as the address of the MoMA. Thus, if we were to look at the mechanisms engaged by Otto in remembering that the MoMA is on 53rd St. upon hearing about the new exhibition, we would find, say, a set comprised by brain regions $\{m_1, m_2, m_3, m_6\}$ plus the notebook, m_7. Originally,

Clark and Chalmers (1998) alluded to a functional parity principle to argue that since "for Otto, his notebook plays the role usually played by a biological memory", then it should be accepted that the notebook, however extra-cranial, was nonetheless part of the cognitive processing whereby Otto remembered the MoMA's address. Should we go the extra mile, then, and conclude that the notebook, m_7, is part of Otto's cognitive system for semantic information retrieval? Rupert thinks that we need not, for if we follow his strategy, we will end up finding that m_7 is *not* included in the final set of integrated mechanisms, M_T, constituting the cognitive system for retrieving semantic information. Despite the fact that now Otto uses the notebook to store semantic facts, the history of co-contribution of brain regions plus notebook does not meet the probability cut off necessary to be included in M_T. When Otto heard the news about the new exhibit, he merely used the notebook, m_7, as an ancillary external resource and in no way should it be considered part of the integrated system supporting semantic retrieval.

Unsurprisingly, partisans of the extended cognition hypothesis have recently offered counter-arguments against Rupert's attack (Wilson and Foglia 2017). However, I am not interested in engaging in this dialectic. Instead, my interest lies in assessing whether or not integration is a good criterion to determine which mechanisms, causally involved in bringing about a certain task, constitute the cognitive system responsible for bringing about such task. To anticipate: my assessment is negative; integration turns out not to be a good criterion for marking off sets of mechanisms that constitute cognitive systems from those that do not. And the reason, I suggest, is straightforward: integration has a hard time accommodating *diachronic dynamicity*, that is, changes in co-contribution of mechanisms for a particular task over time.

3. Maude and the dripping faucet

I plan to initially motivate this claim by way of a thought-experiment, still in the spirit of the extended cognition hypothesis. Meet Maude, a 5-year-old girl who absolutely loves mental arithmetic – specially multiplication. Suppose we want to find out which of the mechanisms engaged during Maude's arithmetical operation of multiplication are integrated so as to constitute the cognitive system responsible for such cognitive task. Now consider also this interesting fact: every time Maude mentally multiplies digits, she taps her index finger against a surface, like a table or a book. Moreover, she cannot mentally multiply unless she's tapping her finger. She's done it since she first started multiplying, just a few months ago. Now, for simplicity's sake, let's suppose that we compile a list of those mechanisms that more frequently have co-contributed to bringing about Maude's mental operation of multiplication, as in (1), and end up with a set of six mechanisms, $\{m_1, m_2, m_3, m_4, m_5, m_6\}$. Next, suppose we assign and rank-order the appropriate probabilities for each m_n, as in (2), and end up with the candidate set $M_T = \{m_1, m_2, m_6\}$. Since, say, $P(T \mid M_T) = .65$ is above the appropriate stipulated cut off, then we conclude that, given the frequency of co-contribution of mechanisms m_1, m_2 and m_6 in bringing about the task of mental multiplication, T, the set of integrated mechanisms engaged in bringing about T, M_T, constitutes Maude's cognitive system for T – a cognitive system that, as it happens, includes her index finger as an integral part.

Calamity strikes, though, and Maude suffers a severe accident that forces her to wear a cast over her upper body, effectively immobilizing her hands – and thus her fingers – for over a year. To top it off, poor Maude has to spend that year in the hospital. Maude is saddened by this fact because she won't get to tap her finger, and thus would not be able to do mental multiplication. Thankfully, though, soon she realizes that there is a dripping faucet

in the sink of her hospital room, and that the sound of the dripping drops perfectly coincides with the tapping of her index finger for the purposes of mental multiplication. So she starts employing the dripping faucet in lieu of her index finger to mentally multiply digits. Her days of mathematically deprived medical confinement are finally over.

By the end of her hospital days, Maude has spent much more time mentally multiplying with the dripping faucet than with her index finger. Indeed, were we to list again all the mechanisms that ever contributed to Maude's mental multiplication, assign to each one a frequency-based probability of co-contribution given every other mechanism in the initial set, rank-order the resultant probabilities, and find a natural cut off to "throw out all the mechanisms that fall below that gap" (Klein 2010, 254), we would end up with an integrated set, M_{T^*}, that would *not* include m_6 as before the accident, but that instead would include m_7, the dripping faucet. Indeed, suppose that by now $P(T \mid M_T) = .41$, where $M_T = \{m_1, m_2, m_6\}$, whereas $P(T \mid M_{T^*}) = .78$, were $M_{T^*} = \{m_1, m_2, m_7\}$, which is not only higher than the former, but also higher than the cut off of.5, and then the probability of T given M_T prior to Maude's accident. Does this mean, then, that the dripping faucet is part of Maude's cognitive system for mental multiplication?

Suppose we say "yes" and agree that the dripping faucet is an integral part of Maude's cognitive system for mental multiplication. This answer, however, leads to a counterintuitive prediction: once Maude's hand heals, and she leaves the hospital room – and, thereby, the dripping faucet – she should not be able to mentally multiply. After all, she would be missing an integral component of the cognitive system that allows her to perform such a mental operation. But, presumably, this prediction is incorrect, for we should *not* expect her to have any trouble at all performing mental multiplications, as her index finger is now healed. Yet, her index finger – m_6 – is no longer part of the integrated set of mechanisms that, due to their frequency of co-occurrence, is supposed to form Maude's cognitive system for mental multiplication.

Alternatively we may answer "no", and reject the claim that the dripping faucet is part of Maude's cognitive system for mental multiplication. But on which basis should we support this alternative? After all, it was Rupert's notion of integration that got us into this situation to begin with. One possibility is to deny that the dripping faucet is part of Maude's cognitive system because, actually, Maude's finger was never part of her cognitive system for mental multiplication. This strategy may help, but is problematic: after all, Maude was never able to perform mental multiplications without the tapping of her finger. Since up to the point in which she entered the hospital she was able to perform mental multiplication, and her finger was a necessary component of the causal explanation of this cognitive ability, failing to consider the finger as part of the cognitive system underlying Maude's arithmetical operation will harm rather than help Rupert's view.

Another possibility is to claim that neither the dripping faucet nor the tapping finger were ever part of Maude's cognitive system for mental multiplication. Instead, what should be included is some component that both the index finger and the dripping faucet share. But what might this mutual component be? We could, for instance, identify that the particular rhythm and frequency of the tapping of Maude's index finger and the drops of the dripping faucet affect the hair cells in Maude's cochlea in the exact same way, suggesting that it is the organ of Corti – or maybe the whole inner ear – that should be included in the set of integrated mechanisms that constitute Maude's cognitive system for mental multiplication, rather than her index finger or the dripping faucet. Unfortunately, this strategy seems to be motivated by the agent-relative, cranium-centric top-down approach Rupert's notion was supposed to be an alternative to, for it suggests that

even if something outside of our brain appears to be integrated, it shouldn't really be included because, well, it isn't part of the agent, the brain, or the organism.

Partisans of the extended cognition hypothesis may want to continue with the exercise of finding something common to the dripping faucet and the tapping finger outside of Maude's cranium – air vibrations, perhaps? – and we would surely end up in a discussion about the precise way to demarcate the boundaries of a cognitive system, a challenge that Weiskopf aptly calls "the Goldilocks problem" (Weiskopf 2010). However, my intention in what follows is not to offer a solution to the Goldilocks problem, but rather to highlight a feature of the logical structure of my thought-experiment: the fact that Rupert's notion of integration has a hard time accommodating changes over time in the frequency of co-contribution of mechanisms that, allegedly, should be considered integral parts of cognitive systems for particular tasks. And this, I will argue, is worrisome because such dynamic changes, far from being proprietary of fanciful thought-experiments, may actually be the norm for our aging brains.

4. HAROLD, PASA, and the changing brain

Brains change over time. Many of those changes are now well documented. For instance, there is a substantial evidence demonstrating that, as we age, our brains undergo significant volumetric reduction on a number of structures, particularly the caudate, cerebellum, hippocampus and prefrontal cortex (Raz 2005), as well as considerable thinning of the cortical mantle (Salat et al. 2004). Aging also affects white matter integrity. For example, relative to younger adults, older adults show marked increases in white matter hyperintensities (WMHs) – abnormal signals from structural MRI scans associated with myelin degradation, microvascular disease and fiber loss, among other causes – particularly in prefrontal regions (Wen and Sachdev 2004). Convergent results have been reported using diffusion tensor imaging (DTI), an imaging technique that capitalizes on neural tracts' anisotropy: a structural feature of fibers that makes water molecules move along, rather than across, the axis of the fiber. Myelin degradation reduces anisotropy in neural fibers, which in turn decreases the speed and probability of water molecule diffusion. Using fractional anisotropy to quantify these reductions a number of researchers have demonstrated loss of white matter integrity as a function of age, particularly in the prefrontal cortex (Madden et al. 2004, 2007; Davis et al. 2009). Finally, extant evidence also suggests significant age-based changes in the dopaminergic circuit, including loss of dopaminergic receptors, and reduction in receptor density and transporter availability, both in the striatum and connected regions in the prefrontal cortex (Volkow et al. 2000; for recent reviews, Park and Reuter-Lorenz 2009; Cabeza and Dennis 2013; Grady 2012).

Critically, many of these changes seem to be correlated with reductions in performance in a number of cognitive tasks. For instance, using voxel-based morphometry and stereology to measure gray matter density, Gong and collaborators (2005) reported a significant correlation between reduction in gray matter volume in prefrontal cortex and lower scores in measures of fluid intelligence as a function of age. Likewise, Gunning-Dixon and Raz (2003) reported significant correlations between increments of WMHs and errors in the Wisconsin Card Sorting Test (WCST), a neuropsychological test geared toward measuring executive function. Comparable correlations have been reported for changes in the dopaminergic circuit. For instance, Volkow and colleagues (1998) report a significant negative correlation between availability of D2 dopamine receptors and errors in the WCST and the Stroop task in a large sample of participants from 24 to 86 years of age. Moreover, Backman and colleagues (2000) reported that a large portion of

age-related variance in processing speed and episodic memory deficits in older adults is accounted for by availability of dopamine receptor bindings. And this is only the tip of the iceberg. As I write these lines, more and more findings are reported about the way in which structural and functional changes in the brain affect cognitive performance (Cabeza, Nyberg, and Park 2017).

In general, brain changes that are correlated with differences in task performance are experimentally identified in one of two ways: via between-group cross-sectional studies comparing two different cohorts – one of younger, and one of older adults – or via within-group longitudinal studies comparing the same cohort at two different times. Either way, both kinds of studies employ as dependent variables some kind of neurobiological measure (e.g., WMHs for DTI, or volumetric reduction of brain tissue for stereology and voxel-based morphometry) as well as task performance, in an attempt to find not only a significant effect given the independent variable – age-group, for cross-sectional studies, or age, for the longitudinal ones – but also a significant correlation between task performance and one or more neurobiological measures. However, with the advent of neuroimaging and other non-invasive techniques, we can now afford to look at age-based brain changes *in the absence* of task performance differences. By employing as dependent variables brain-based changes – such as variations in blood oxygenation level dependent (BOLD) signal, as measured by fMRI, or metabolic activity indexed by regional glucose uptake, as measured by Positron Emission Tomography (PET) scans – researchers can now explore age-dependent changes in the engagement of different brain regions as a function of age even if there is no correlated change in task performance. And it is the results of this line of research that, I believe, strongly suggest a parallel between the Maude thought-experiment and the reality of our aging brains.

Specifically, I have in mind two striking, well-supported and consistent results from the cognitive neuroscience of aging. The first finding is the observation that tasks that tend to recruit hemispherically localized regions of the prefrontal cortex in younger adults show a reduction in hemispheric asymmetry in older adults. Thus, if a particular task, e.g. semantic-recall, recruits mostly right regions in the prefrontal cortex in young adults, the same task would recruit the same regions but bilaterally in older adults, despite the fact that there is no difference in task performance (Cabeza et al. 1997). This pattern of age-based change in brain activity has been dubbed the "Hemispheric Asymmetry Reduction in Older Adults" or HAROLD, and it was initially documented for both fMRI and PET studies involving perceptual, working and episodic memory tasks (Cabeza 2002). More recently, however, this pattern of asymmetric reduction in brain activity has been documented on a large number of other tasks, including decision-making (Lee et al. 2011), semantic processing (Wierenga et al. 2008), inhibitory control (Nielson, Langenecker, and Garavan 2002), language (Persson et al. 2007; Tyler et al. 2010), and conceptual priming (Bergerbest et al. 2009), among others (for a review, see Cabeza and Dennis 2012).

The second finding is known as the "Posterior-Anterior Shift with Aging" or PASA. Initially observed by Grady and collaborators (1994) during a perceptual task, PASA refers to the phenomenon that older adults tend to increase activity in anterior regions while decreasing activity in posterior regions relative to younger adults. Importantly, in their study, Grady et al. (1994) found no differences in perceptual accuracy between younger and older adults, even though they did find a difference in posterior versus anterior recruitment between groups. Soon after, Madden and collaborators (1997) corroborated this age-based pattern of shifting activation with a different cohort and a different perceptual task, also matched for performance. Ever since, PASA has been identified in a large number of tasks, including visual perception (Davis et al. 2008), attention (Solbakk et al.

2008), working memory (Cabeza et al. 2004), problem solving (Nagahama et al. 1997), and episodic memory encoding (Gutchess et al. 2005; Dennis, Daselaar, and Cabeza 2007) and retrieval (Cabeza et al. 2000; Davis et al. 2008). These studies – among others (Cabeza and Dennis 2012; Grady 2012) – consistently find that older adults show increased anterior activity, particularly in the prefrontal cortex, and decreased posterior activity, mainly in the occipital cortex, relative to younger adults when performing the same task at comparable levels.

Why HAROLD and PASA occur is, unsurprisingly, still a matter of debate. An early interpretation of these findings framed them in terms of compensatory mechanisms. Specifically, it was suggested that these age-based shifts in brain activation are a biological strategy to recruit extra-neural resources to keep cognitive and behavioral performance unaltered (Cabeza et al. 2002). However, a number of subsequent findings showed that, for certain tasks, such shifts were still present although they did not seem to yield advantages in performance (Zarahn et al. 2007). As a result, recent accounts propose somewhat more nuanced explanations of both HAROLD and PASA. It has been suggested, for instance, that these compensatory patterns may depend on the cognitive demands of the task, since perceptual processing tends to be easier for younger than older adults. A compatible account makes use of the notion of "de-differentiation", which refers to the fact that certain cortical areas appear to become less selective to stimuli over time (Logan et al. 2002). Both the asymmetric reduction and the posterior-anterior shift may just be the brain's strategy to compensate for such gradual reduction in selectivity. But regardless of what the actual explanation is, for the present purposes what matters is not *why* such age-based shifts in brain activity occur, but simply that they *do* happen. And it matters, I argue, because these natural age-based dynamic changes in the mechanisms involved in bringing about a certain task, invite us to question the notion of integration as a good criterion for individuating cognitive systems, and for reasons similar to those uncovered by the thought-experiment of Maude and the dripping faucet.

To elaborate, let us assume a simpler model of the findings just reviewed in the context of Rupert's strategy for integration, as discussed above (Sections 2 and 3). Consider Harold – an experimental subject in an idealized longitudinal neuroimaging study comprising two phases. Phase one occurs when Harold is in his late 20s. During this phase, Harold is asked to perform a particular task, *T*, every two months, for five years, while undergoing fMRI and PET scanning. The purpose of this phase is to identify the mechanisms most frequently engaged by Harold during the performance of task *T*. The results of every scanning session can be seen as giving us a list of brain regions likely engaged during the performance of *T*, and the list could presumably be arranged in the same manner as (1), above. We can next statistically compare the activation maps across all the studies listed in (1) and come up with a probability distribution for each region of activation given any other activation, very much in the spirit of (2), above. And suppose, finally, that we rank-order all such probabilities and come up with a rather conservative threshold to calculate the probability of any such regions to be jointly active in bringing about *T* – which may give us a set, M_T, of (let's say) four different brain regions $\{m_1, m_2, m_3, m_4\}$ (Figure 1). Following Rupert's strategy for integration, then, M_T would be the set of integrated mechanisms constituting the cognitive system that brings about *T*.[3]

Time passes and Harold begins the second phase of the experiment. This second phase is just like the first phase except that it occurs when Harold is in his 60s. Again, Harold is asked to perform *T*, with the same frequency, and under the same scanning circumstances. The resultant neuroimaging data is listed, rank-ordered and statistically thresholded in the exact same way as during phase one. Behaviorally, there are no differences in Harold's

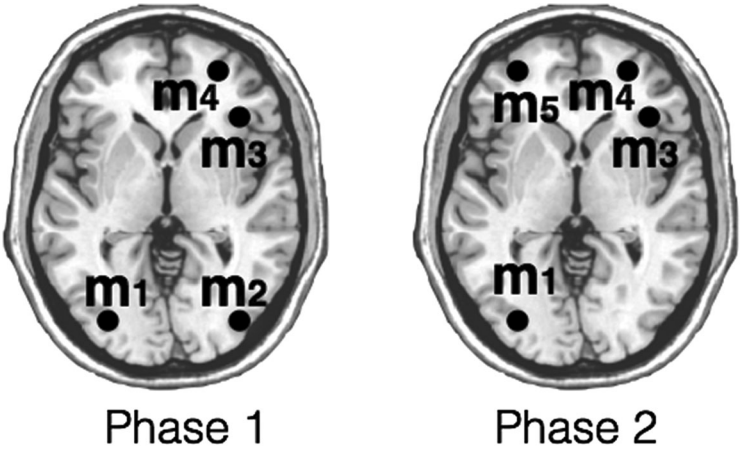

Figure 1. M_T (Phase 1) and M_{T^*} (Phase 2) in Harold's imagined longitudinal study.

performance of T between phases 1 and 2. However, the neuroimaging data now conforms to the findings of HAROLD and PASA and the final set of engaged brain mechanisms, M_{T^*}, looks somewhat different than M_T, as it now includes brain structures $\{m_1, m_3, m_4, m_5\}$ (Figure 1). But, then, which set of integrated mechanisms constitutes the cognitive system responsible for bringing about T in Harold – M_{T^*} or M_T?

Notice that Harold and Maude's situations are structurally similar. In both cases we generate a set of integrated mechanisms over two distinct temporal windows – pre-accident versus post-accident in Maude's case; younger versus older in Harold's – while controlling for differences in task performance. Moreover, in both cases the generated sets in the first and second temporal windows include some overlapping but also some unique components. Yet, we don't seem to have a principled reason to prefer the sets generated on either of the temporal windows as constituting the cognitive system for the relevant task. How shall we proceed, then? Before offering an alternative strategy, let's first explore three possible objections to Harold's case.

First, one may say that the actual cognitive system in Harold is the intersection of M_T and M_{T^*}, so neither m_2 nor m_5 are part of the cognitive system for T. But then the same reply used for Maude's case is available here. By hypothesis, the pre-accident Maude's index finger was part of her cognitive system for multiplication. Ditto for Harold. As revealed by the results of Phase 1, m_2 is part of the cognitive system for T. And, presumably, just as pre-accident Maude wouldn't have been able to multiply without her index finger, young Harold wouldn't have been able to T without m_2.[4]

Second, it may be objected that since Harold used M_T up until he was 60, then the mechanisms in M_T are de facto more frequently co-active than those of M_{T^*}. Thus, the cognitive system for T must be M_T, not M_{T^*}. There are at least two possible replies. First, this objection makes being part of a cognitive system dependent on the contingent fact that humans tend to live for less than a century. But what if Harold gets to live to be 130? Now M_{T^*} would have been more frequently used than M_T. It just doesn't seem appropriate to adjudicate matters of metaphysics of cognition on the basis of shifting life expectancies. Second, even if we don't bank on Harold's longevity, he may still use M_{T^*} more frequently than M_T. After all, brains do not only change when we are old: they also change dramatically when we are young. So far, I've avoided talking

about brain changes during childhood and adolescence, mainly because we know less about brain activity shifts during early development than during aging. However, extant evidence in the cognitive neuroscience of brain development also suggests that a number of brain structures engaged during specific tasks vary as a function of both increased maturity and learning (Goddings and Giedd 2014). Given the possibility that M_T may actually be the set of integrated mechanisms of Harold's *mature* brain, and that up until his early 20s the set of neural structures engaged during T looked significantly different, it may well be that, on average, Harold actually uses M_T less frequently that both pre-M_T mechanisms and M_{T^*} during his lifespan.

Finally, a third objection is to say that the task young Harold was doing is different from old Harold's task. As such, there is no problem in talking about two different systems as they are really bringing about two different tasks. The problem with this objection is that it relativizes precisely the one parameter we kept constant: the task. What would be the motivation to claim that what appears to be exactly the same task at two different times are actually two different tasks? One possibility, sometimes employed in the aging literature, is to suggest that even though both young and old adults reach similar levels of performance, they do so by deploying different cognitive *strategies*. Under this view, tasks are determined by a combination of behavioral performance and cognitive strategies. This response might work for certain tasks, specifically for those for which we have independent evidence – for example, introspective reports, reaction times, behavioral responses – to the effect that older adults are, in fact, deploying a different strategy. Unfortunately, often times researchers have no more reason to postulate the deployment of different strategies than a recorded difference in brain activity between younger and older participants. Without further evidence, attributing a difference in brain activity between the two groups to an alleged difference in cognitive strategy is unwarranted. Moreover, often times brain differences between younger and older adults are found for tasks for which it makes little sense to talk about distinct cognitive strategies, such as Stroop (Zurrón et al. 2014) or Rapid Serial Visual Presentation tasks (Quigley, Andersen, and Müller 2012). Are there really different strategies for completing these fast and automatic tasks? I doubt it. Finally, even if we admit that, for whatever reason, young and old Harold are performing different tasks, then we may face the opposite problem: we may end up proliferating tasks beyond necessity. Suppose we collected neuroimaging data on Harold through his whole life, and we analyze it at four different age-windows: teens, twenties, forties, and sixties. It turns out, each temporal window produces somewhat overlapping but distinct integrated systems: M_{T+}, M_T, $M_{T'}$, and M_{T^*}. Shall we say that each of these sets brings about a different task, despite the fact that T does not change across Harold's lifespan, and despite the fact that it yields the same behavioral performance across all four time windows? Conditionalizing tasks to integrated systems seems to get the order of explanation backwards.

Let's take stock. In Section 2 I formally reconstructed Rupert's notion of integration. In Section 3, I offered a thought-experiment to motivate the claim that Rupert's notion of integration fails to capture what I called *diachronic dynamicity*: changes in co-contribution of causal mechanisms for a particular task over time. Then, in Section 4, I argued that the structure of the thought-experiment mirrors what may actually occur with our aging brains. As we age, a number of neural mechanisms exhibit diachronic dynamicity without concomitant changes in task performance – that is, they also exhibit task or *functional stability*. Can we find an alternative way to understand integration and, thus, to underwrite the identification of cognitive systems, such that we can have both diachronic dynamicity and functional stability? I suggest one approach next.

5. Toward an account of dynamic cognitive systems

The foregoing discussion suggests at least two modifications to improve upon Rupert's integration strategy to identify cognitive systems. First, we need to be able to parametrize time independently of frequency of engagement. Second, we need a way to identify the same cognitive system across subjects – both diachronically and synchronically – rather than solely in a single individual. There may be several strategies to achieve these goals. Here, I briefly describe what I take to be a promising approach to understanding cognitive systems in a way that allows us to accommodate their diachronic dynamicity while retaining functional stability. Since this proposal builds heavily on recent topological formalisms from cognitive network neuroscience, it may be helpful to review some basic concepts.

Brains can be seen as hierarchical networks of basic elements, or *nodes*, and their pairwise connections, or *edges*. Being hierarchical, nodes can be identified at the micro-level of individual neurons, the meso-level of individual voxels, or the macro-level of brain regions. Cognitive network neuroscience deals with networks at the meso- and macro-levels, leveraging data from different neuroimaging techniques, such as fMRI, DTI, EEG, MEG, etc (Bullmore and Sporns 2009). Defining both nodes and edges depends on the type of data and the research question. For MRI-based networks, nodes are typically either single-voxels or multiple-voxels anatomically (Achard et al. 2006) or functionally (Power et al. 2011) defined (see Stanley et al. 2013, for a critical discussion). In turn, edges can be structural – as measured by white matter connections between brain regions – or functional – as measured by statistical relationships between brain regions, e.g., correlation, coherence or phase synchrony (Muldoon and Bassett 2016). To explore their properties, researchers model brain networks as graphs and employ topological statistics (Bullmore and Sporns 2009; Newman 2010; Rubinov and Sporns 2010) to draw inferences. Some of these statistics measure the extent to which a given node is interconnected with other nodes during a given state. The *Clustering coefficient*, for instance, quantifies the number of triplets a node has relative to all possible triplets it could have (Bullmore and Sporns 2009). *Local efficiency* quantifies how connected the neighbors of a node are among themselves. Finally, *modularity* identifies subsets of nodes that are more densely interconnected among themselves than they are to the rest of the network (Newman 2010; Stanley and De Brigard 2016).

Although to date most analyses of neuroimaging data in terms of networks have been conducted on resting state data, there has recently been a move to analyze task-dependent data as well. This approach has yielded a number of interesting findings, many of which are relevant for identifying the neural underpinnings of cognitive systems. For instance, studies with motor tasks revealed that nodes in the primary motor cortex exhibit great degrees of connectedness and high clustering coefficients. However, when the motor task is only prepared but not executed, clustering is higher in the premotor cortex. Likewise, it has been shown that changes in clustering and modularity predict differences in performance on working memory tasks as a function of cognitive load. When cognitive load is low, nodes in the default mode network become more densely and consistently interconnected, whereas this occurs for regions in the dorsolateral prefrontal and posterior parietal cortices when cognitive load is high (Stanley et al. 2014). These, and similar task-specific analyses, are slowly converging on the observation that certain tasks, geared to index specific cognitive operations (e.g., attention, working memory, language comprehension, etc.), tend to have identifiable functional topological properties wherein subsets of nodes that are involved in the task become densely and consistently interconnected amongst each other (Medaglia, Lynall, and Bassett 2015).

However, these studies characterize functional brain networks underlying particular cognitive tasks during a very short window of time (e.g., 8 minutes), and thus cannot describe the temporal dimension we require to capture our cognitive system's diachronic dynamicity. Fortunately, there are at least two recent developments in network neuroscience that could potentially help us to capture change through long temporal windows while keeping functional stability. One strategy is to make use of recently developed algorithms, such as *scale inclusivity* (Steen et al. 2011), which allows to quantify the extent to which community structures vary across networks, both cross-sectionally and longitudinally, by evaluating how consistently each individual node is classified in the same community across networks. To see how this may work, consider again our idealized longitudinal study with Harold. The first step would involve modeling data at different consecutive time-points, t_1, t_2, ... t_n, as adjacency matrices that can then be rendered into a topological space as functional networks, whereby community structures can be found at each time-point. One could, for instance, employ Newman and Girvan (2004) Q metric of modularity to find such community structures. The next step consists in evaluating the consistency of the modules between networks at two different times, say, t_1 and t_2. Statistical analyses could reveal the degree of correlation across modules between these two networks. Finally, this step could be iterated n-times to cover all temporal comparisons. After correcting for multiple comparisons, we would end up with a functional network whereby each node represents a probability of being included in the same module every time Harold did T during his life. Moreover, the same process can be conducted across subjects, to identify consistent modules not only within- but also between-individuals through time. Although, to the best of my knowledge, this approach hasn't been employed to explore longitudinal data, a number of researchers have used it to look at network evolution at different task-stages (Moussa et al. 2011; Meunier et al. 2014; Stanley et al. 2014). Looking at developmental and aging data from longitudinal samples would be a promising future direction.

The second, compatible approach comes from recent developments in multilayer networks, where a different strategy to parametrize time has been developed. In essence, this strategy allows researchers to quantify changes in clustering and modularity between networks at different time-points by building "temporal networks". To do so, they first create static networks at different points in time, just as in the aforementioned approach. However, instead of looking at specific nodes within similar partitions of the network, they take a more holistic view whereby they find clusters or network communities that are similarly active across networks across time. The resultant adjacency matrix is in essence a probability matrix that treats each element as the probability that any two nodes are assigned to the same network community across all subjects and across all times. If this dynamic clustering yields a modular architecture, such modules are named *module allegiances.* To date, only a handful of studies have made use of multilayer dynamic networks to analyze neuroimaging data across different time-points. The best demonstration I know of was reported by Bassett and collaborators (2015). In this study, Bassett et al. (2015) looked at the evolution of neural networks engaged during learning a simple sequence-production task. There were two time windows they were interested in: the temporal dynamics of the networks during the training session, and the changes in the network from novice to expert. Their results revealed one network community with two strong modular alliances across both time windows: one comprising regions in the primary and secondary sensorimotor areas, and another comprising the primary visual cortex.

Although Bassett and colleagues (2015) were interested in finding modular allegiances as a function of a change in behavior (i.e., learning), we can build upon this idea by keeping

task performance fixed. If so, the suggestion I would like to put forth is that cognitive systems in the brain can be identified with modular allegiances revealed either by scale inclusivity or by temporal-dynamic network analyses on neural data. This strategy has the advantage of allowing us to see cognitive systems as integrated sets of mechanisms whereby integration is not defined by frequency of use, but rather by degrees of connectedness and clustering between groups of nodes forming modules across time. Thus, from this perspective, the answer as to whether regions m_2 and m_5 are or not part of Harold's cognitive system is going to depend on whether or not such nodes end up being part of the modular alliance that underlies *T*. Moreover, we may actually find out that the cognitive system Harold deploys to perform *T* actually includes *both* m_2 and m_5, even if their degrees of connectedness to the rest of the network vary as a function of time. Finally, this approach could also help us in the quest to meet the demarcation challenge, as it could provide useful and quantifiable criteria to delimit which nodes are or are not part of the modular alliance associated with a particular task across time.

6. Conclusion: implications for cognitive ontologies

In the current paper I critically evaluated Rupert's notion of integration to identify cognitive systems in the brain. I did so by noting that a notion of integration that relies so heavily on frequency of use may fail to capture a natural property of brains: they change over time, and so does the causal engagement of neural regions during specific tasks even when there is no difference in performance. Brains are diachronically dynamic but functionally stable. Additionally, I suggested a couple of strategies from cognitive network neuroscience to possibly preserve Rupert's important insight regarding the need of integration, but without making it dependent exclusively on frequency. Now I want to conclude by linking this discussion to the overall project of understanding the brain's cognitive ontology.

I began this paper by contrasting two ways of thinking about cognitive systems: a "top-down" and a "bottom-up" approach. Both approaches mirror our strategies to successfully map brain structures to cognitive processes. On the top-down approach, one starts with a taxonomy of cognitive processes and goes down to the brain to see what fits what. Conversely, the bottom-up approach begins with neural data and tries to find structure within them, usually by employing some kind of dimensionality reduction analyses (i.e., ICA, factor analysis, hierarchical clustering, etc.). Unfortunately, both approaches face difficulties. On the one hand, the first approach risks being too chauvinistic, as it forces brain categories to fit our psychological taxonomies, which not only vary as the discipline evolves (Poldrack 2010; Anderson 2015) but are also unlikely to have made it into our vernacular to help us carve the brain's nature at its joints (De Brigard 2006, 2015). On the other hand, the second approach risks being too liberal, not only because different dimensionality reduction analyses can yield different solutions (Van deer Maaten et al. 2009), but also because one may end up reifying over mathematical constructs (i.e., common factors, latent variables, etc.) that in reality may not correspond to actual individual brain processes (McCaffrey and Machery 2016).

The solution probably lies somewhere in the middle. First we may have to reach an agreement as to which tasks index which cognitive processes, and then use data driven statistical analyses to identify structures that would allow us to map tasks to regions. This process would likely be cyclical, and both sides should inform one another. Nevertheless, as I have tried to argue in the current paper, no mapping from brain to mind – from neurons to cognition – would be complete without taking into account the fact that brains change

through time. The mechanisms supporting our cognitive operations are dynamic, and this fact may not easily be captured by a cognitive ontology that only looks at static neural data.

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Notes

1. Since, presumably, most cognitive tasks would have engaged many m_n at some point or another, actual chance is probably way below .5.
2. Notice that there is a complicated issue here having to do with how are we to individuate cognitive tasks. I am assuming that remembering the address of the MoMA is an instance of the larger cognitive task of retrieving semantic information. As such, the cognitive system that supports Inga's retrieval of the MoMA's address would be the cognitive system that supports retrieval of semantic information, maybe even the cognitive system for semantic memory, rather than a cognitive system for remembering addresses or museum locations. However, as we will see, this turns out to be a huge assumption, likely not determinable in the bottom-up way of Rupert's proposal.
3. Here I am over-simplifying, of course, by equating brain *region* to brain *mechanism*. At best, a region is a proxy for a mechanism, but not identical to it. However, for the sake of the argument, I am going to overlook the important yet difficult issue of what is the precise relationship between a brain region and a brain mechanism.
4. I'm not implying that fMRI studies alone can tell us which brain regions or mechanisms are necessary for bringing about a certain task. To infer necessity we need much more than neuroimaging; we probably would need experiments involving neuro-interventions, neurospsychological testing, neural and behavioral dissociations, etc. But, as mentioned before (endnote 3), for the sake of the argument I am using these hypothetical BOLD signal changes as idealized measures of a mechanism's engagement during a task.

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References

Achard, S., R. Salvador, B. Whitcher, J. Suckling, and E. Bullmore. 2006. "A Resilient, Low-frequency, Small-world Human Brain Functional Network with Highly Connected Association Cortical Hubs." *Journal of Neuroscience* 26 (1): 63–72.

Adams, F., and K. Aizawa. 2001. *The Bounds of Cognition*. Oxford: Wiley-Blackwell.

Anderson, M. L. 2015. "Mining the Brain for a New Taxonomy of the Mind." *Philosophy Compass* 10 (1): 68–77.

Backman, L., N. Ginovart, R. A. Dixon, T. B. R. Wahlin, A. Wahlin, C. Halldin, and L. Farde. 2000. "Age-related Cognitive Deficits Mediated by Changes in the Striatal Dopamine System." *American Journal of Psychiatry* 157 (4): 635–637.

Bassett, D. S., M. Yang, N. F. Wymbs, and S. T. Grafton. 2015. "Learning-induced Autonomy of Sensorimotor Systems." *Nature Neuroscience* 18 (5): 744–751.

Bergerbest, D., J. D. E. Gabrieli, S. Whitfield-Gabrieli, H. Kim, G. T. Stebbins, D. A. Bennett, and D. A. Fleischman. 2009. "Age-associated Reduction of Asymmetry in Prefrontal Cortex Function and Preservation of Conceptual Repetition Priming." *Neuroimage* 45: 237–246.

Bermudez, J. 2010. *Cognitive Science: An Introduction to the Science of the Mind*. Cambridge: Cambridge University Press.

Bullmore, E. T., and O. Sporns. 2009. "Complex Brain Networks: Graph Theoretical Analysis of Structural and Functional Systems." *Nature Reviews Neuroscience* 10 (3): 186–198.

Cabeza, R. 2002. "Hemispheric Asymmetry Reduction in Old Adults: The HAROLD Model." *Psychology and Aging* 17: 85–100.

Cabeza, R., N. D. Anderson, S. Houle, J. A. Mangels, and L. Nyberg. 2000. "Age-related Differences in Neural Activity during Item and Temporal-order Memory Retrieval: A Positron Emission Tomography Study." *Journal of Cognitive Neuroscience* 12: 1–47.

Cabeza, R., N. D. Anderson, J. K. Locantore, and A. R. McIntosh. 2002. "Aging Gracefully: Compensatory Brain Activity in High-performing Older Adults." *Neuroimage* 17 (3): 1394–1402.

Cabeza, R., S. M. Daselaar, F. Dolcos, S. E. Prince, M. Budde, and L. Nyberg. 2004. "Task-independent and Task-specific Age Effects on Brain Activity During Working Memory, Visual Attention and Episodic Retrieval." *Cerebral Cortex* 14: 364–375.

Cabeza, R., and N. A. Dennis. 2013. "Frontal Lobes and Aging: Deterioration and Compensation." In *Principles of Frontal Lobe Function*. 2nd ed., edited by D. T. Stuss, and R. T. Knight. New York: Oxford University Press.

Cabeza, R., C. L. Grady, L. Nyberg, A. R. McIntosh, E. Tulving, Shitij Kapur, Janine M. Jennings, Sylvain Houle, and Fergus I. M. Craik. 1997. "Age-related Differences in Neural Activity During Memory Encoding and Retrieval: A Positron Emission Tomography Study." *Journal of Neuroscience* 17: 391–400.

Cabeza, R., L. Nyberg, and D. Park. 2017. *Cognitive Neuroscience of Aging*. Oxford: Oxford University Press.

Clark, A., and D. J. Chalmers. 1998. "The Extended Mind." *Analysis* 58: 7–19.

Cummins, R. C. 2000. "'How Does It Work' Versus 'What Are the Laws?': Two Conceptions of Psychological Explanation." In *Explanation and Cognition*, edited by F. Keil, and Robert A. Wilson, 117–145. Cambridge, MA: MIT Press.

Davis, S. W., N. A. Dennis, N. G. Buchler, L. E. White, D. J. Madden, and R. Cabeza. 2009. "Assessing the Effects of Age on Long White Matter Tracts Using Diffusion Tensor Tractography." *Neuroimage* 46: 530–541.

Davis, S. W., N. A. Dennis, S. M. Daselaar, M. S. Fleck, and R. Cabeza. 2008. "Que PASA? The Posterior-anterior Shift in Aging." *Cerebral Cortex* 18: 1201–1209.

De Brigard, F. 2006. "Capas limítrofes y dominios de evidencia en ciencia cognitiva." *Universitas Philosophica* 46: 53–77.

De Brigard, F. 2015. "What Was I Thinking? Dennett's *Content and Consciousness* and the Reality of Propositional Attitudes." In *Content and Consciousness Revisited*, edited by C. M. Muñoz-Suárez, and F. De Brigard, 49–71. New York: Springer.

Dennett, D. C. 1978. *Brainstorms*. Cambridge, MA: MIT Press.

Dennis, N. A., S. Daselaar, and R. Cabeza. 2007. "Effects of Aging on Transient and Sustained Successful Memory Encoding Activity." *Neurobiology of Aging* 28: 1749–1758.

Goddings, A. L., and J. N. Giedd. 2014. "Structural Brain Development during Childhood and Adolescence." In *The Cognitive Neurosciences*. 5th ed., edited by M. Gazzaniga, and G. R. Mangun, 15–22. Cambridge, MA: MIT Press.

Gong, Q. Y., V. Sluming, A. Mayes, S. Keller, T. Barrick, E. Cezayirli, and N. Roberts. 2005. "Voxel-based Morphometry and Stereology Provide Convergent Evidence of the Importance of Medial Prefrontal Cortex for Fluid Intelligence in Healthy Adults." *Neuroimage* 25: 1175–1186.

Grady, C. 2012. "The Cognitive Neuroscience of Ageing." *Nature Reviews Neuroscience* 13: 491–505.

Grady, C. L., J. M. Maisog, B. Horwitz, L. G. Ungerleider, M. J. Mentis, J. A. Salerno, P. Pietrini, E. Wagner, and J. V. Haxby. 1994. "Age-related Changes in Cortical Blood Flow Activation during Visual Processing of Faces and Location." *Journal of Neuroscience* 14: 1450–1462.

Gunning-Dixon, F. M., and N. Raz. 2003. "Neuroanatomical Correlates of Selected Executive Functions in Middle-aged and Older Adults: A Prospective MRI Study." *Neuropsychologia* 41: 1929–1941.

Gutchess, A. H., R. C. Welsh, T. Hedden, A. Bangert, M. Minear, L. L. Liu, and Denise C. Park. (2005). "Aging and the Neural Correlates of Successful Picture Encoding: Frontal Activations Compensate for Decreased Medial-temporal Activity." *Journal of Cognitive Neuroscience* 17: 84–96.

Klein, C. 2010. "Critical Notice: 'Cognitive Systems and the Extended Mind by Robert Rupert'." *The Journal of Mind and Behavior* 31 (3&4): 253–264.

Lee, Y., C. L. Grady, C. Habak, H. R. Wilson, and M. Moscovitch. 2011. "Face Processing Changes in Normal Aging Revealed by fMRI Adaptation." *Journal of Cognitive Neuroscience* 23: 3433–3447.

Logan, J. M., A. L. Sanders, A. Z. Snyder, J. C. Morris, and R. L. Buckner. 2002. "Under-recruitment and Nonselective Recruitment: Dissociable Neural Mechanisms Associated With Aging." *Neuron* 33: 827–840.

Lycan, W. G. 1987. *Consciousness*. Cambridge, MA: MIT Press.

Madden, D. J., J. Spaniol, W. L. Whiting, B. Bucur, J. M. Provenzale, R. Cabeza, Leonard E. White, and Scott A. Huettel. 2007. "Adult Age Differences in the Functional Neuroanatomy of Visual Attention: A Combined fMRI and DTI Study." *Neurobiology of Aging* 28: 459–476.

Madden, D. J., T. G. Turkington, J. M. Provenzale, T. C. Hawk, J. M. Hoffman, and R. Edward Coleman. 1997. "Selective and Divided Visual Attention: Age-related Changes in Regional Cerebral Blood Flow Measured by H2(15)O PET." *Human Brain Mapping* 5: 389–409.

Madden, D. J., W. L. Whiting, S. A. Huettel, L. E. White, J. R. MacFall, and James M. Provenzale. 2004. "Diffusion Tensor Imaging of Adult Age Differences in Cerebral White Matter: Relation to Response Time." *Neuroimage* 21: 1174–1181.

Marr, D. 1976. "Early Processing of Visual Information." *Philosophical Transactions of the Royal Society B: Biological Sciences* 275: 483–519.

McCaffrey, Joseph B., and Edouard Machery. 2016. "The Reification Objection to Bottom-up Cognitive Ontology Revision." *Behavioral and Brain Sciences* 39. doi:10.1017/S0140525X15001594, e125.

Medaglia, J. D., M.-E. Lynall, and D. S. Bassett. 2015. "Cognitive Network Neuroscience." *Journal of Cognitive Neuroscience* 27 (8): 1471–1491.

Meunier, D., P. Fonlupt, A.-L. Saive, J. Plailly, N. Ravel, and J.-P. Royet. 2014. "Modular Structure of Functional Networks in Olfactory Memory." *NeuroImage* 95: 264–275.

Moussa, M. N., C. D. Vechlekar, J. H. Burdette, M. R. Steen, C. E. Hugenschmidt, and P. J. Laurienti. 2011. "Changes in Cognitive State Alter Human Functional Brain Networks." *Frontiers in Human Neuroscience* 5: 1–15.

Muldoon, S. F., and D. S. Bassett. 2016. "Network and Multilayer Network Approaches to Understanding Human Brain Dynamics." *Philosophy of Science* 83: 710–720.

Nagahama, Y., H. Fukuyama, H. Yamaguchi, Y. Katsumi, Y. Magata, H. Shibasaki, and Jun Kimura. 1997. "Age-related Changes in Cerebral Blood Flow Activation during a Card Sorting Test." *Experimental Brain Research* 114: 571–577.

Newman, M. 2010. *Networks: An Introduction*. New York: Oxford University Press.

Newman, M. E. J., and M. Girvan. 2004. "Finding and Evaluating Community Structure in Networks." *Physical Review E – Statistical, Nonlinear, and Soft Matter Physics* 69 (2 2): 1–15.

Nielson, K. A., S. A. Langenecker, and H. Garavan. 2002. "Differences in the Functional Neuroanatomy of Inhibitory Control across the Adult Lifespan." *Psychology and Aging* 17: 56–71.

Park, D. C., and P. Reuter-Lorenz. 2009. "The Adaptative Brain: Ageing and Neurocognitive Scaffolding." *Annual Review of Psychology* 60: 173–196.

Persson, J., C. Lustig, J. K. Nelson, and P. A. Reuter-Lorenz. 2007. "Age Differences in Deactivation: A Link to Cognitive Control?" *Journal of Cognitive Neuroscience* 19: 1021–1032.

Poldrack, R. A. 2010. "Mapping Mental Function to Brain Structure: How Can Cognitive Neuroimaging Succeed?" *Perspectives on Psychological Science* 5 (6): 753–761.

Power, J. D., A. L. Cohen, S. M. Nelson, G. S. Wig, K. A. Barnes, J. A. Church, A. C. Vogel, et al. 2011. "Functional Network Organization of the Human Brain." *Neuron* 72 (4): 665–678.

Quigley, C., S. K. Andersen, and M. M. Müller. 2012. "Keeping Focused: Sustained Spatial Selective Visual Attention is Maintained in Healthy Old Age." *Brain Research* 1469: 24–34.

Raz, N. 2005. "The Aging Brain Observed in Vivo: Differential Changes and Their Modifiers." In *Cognitive Neuroscience of Aging*, edited by R. Cabeza, L. Nyberg, and D. L. Park, 19–57. New York: Oxford University Press.

Rubinov, M., and O. Sporns. 2010. "Complex Network Measures of Brain Connectivity: Uses and Interpretations." *NeuroImage* 52: 1059–1069.
Rupert, R. 2009. *Cognitive Systems and the Extended Mind*. Oxford: Oxford University Press.
Rupert, R. 2010. "Extended Cognition and the Priority of Cognitive Systems." *Cognitive Systems Research* 11: 343–356.
Rupert, R. 2011. "Cognitive Systems and the Supersized Mind." *Philosophical Studies* 152: 427–436.
Salat, D. H., R. L. Buckner, A. Z. Snyder, D. N. Greve, R. S. Desikan, E. Busa, J. C. Morris, A. M. Dale, and B. Fischl. 2004. "Thinning of the Cerebral Cortex in Aging." *Cerebral Cortex* 14: 721–730.
Shallice, T. 1988. *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.
Simon, H. 1969. *The Sciences of the Artificial*. Cambridge, MA: MIT Press.
Solbakk, A. K., A. G. Fuhrmann, A. J. Furst, L. A. Hale, T. Oga, S. Chetty, Natasha Pickard, and Robert T. Knight. 2008. "Altered Prefrontal Function with Aging: Insights into Age-associated Performance Decline." *Brain Research* 1232: 30–47.
Stanley, M. L., D. Dagenbach, R. G. Lyday, J. H. Burdette, and P. J. Laurienti. 2014. "Changes in Global and Regional Modularity Associated with Increasing Working Memory Load." *Frontiers in Human Neuroscience* 8: 3889. doi:10.3389/fnhum.2014.00954.
Stanley, M. L., and F. De Brigard. 2016. "Modularity in Network Neuroscience and Neural Reuse." *Behavioral and Brain Sciences* 39. doi:10.1017/S0140525X15000673.
Stanley, M. L., M. N. Moussa, B. M. Paolini, R. G. Lyday, J. H. Burdette, and P. J. Laurienti. 2013. "Defining Nodes in Complex Brain Networks." *Frontiers in Computational Neuroscience* 7: 169. doi:10.3389/fncom.2013.00169.
Steen, M., S. Hayasaka, K. Joyce, and P. Laurienti. 2011. "Assessing the Consistency of Community Structure in Complex Networks." *Physical Review E* 84: 016111.
Tyler, L. K., M. A. Shafto, B. Randall, P. Wright, W. D. Marslen-Wilson, and E. A. Stamatakis. 2010. "Preserving Syntactic Processing across the Adult Life Span: The Modulation of the Frontotemporal Language System in the Context of Age-related Atrophy." *Cerebral Cortex* 20: 352–364.
Van deer Maaten, L. J. P., E. O. Postma, and H. J. Van den Herik. 2009. "Dimensionality Reduction: A Comparative Review." Technical Report TiCC TR 2009-005.
Volkow, N. D., R. C. Gur, G. J. Wang, J. S. Fowler, P. J. Moberg, Y. S. Ding, R. Hitzemann, G. Smith, and J. Logan. 1998. "Association between Decline in Brain Dopamine Activity with Age and Cognitive and Motor Impairment in Healthy Individuals." *American Journal of Psychiatry* 155: 1325–1331.
Volkow, N. D., J. Logan, J. S. Fowler, G. J. Wang, R. C. Gur, C. Wong, and Christoph Felder. 2000. "Association between Age-related Decline in Brain Dopamine Activity and Impairment in Frontal and Cingulate Metabolism." *American Journal of Psychiatry* 157: 75–80.
Weiskopf, D. 2010. "The Goldilocks Problem and Extended Cognition." *Cognitive Systems Research* 11: 313–323.
Wen, W., and P. Sachdev. 2004. "The Topography of White Matter Hyperintensities on Brain MRI in Healthy 60- to 64-Year Old Individuals." *NeuroImage* 22 (1): 144–154.
Wierenga, C. E., M. Benjamin, K. Gopinath, W. M. Perlstein, C. M. Leonard, L. J. G. Rothi, Tim Conway, M. Allison Cato, Richard Briggs, and Bruce Crosson. 2008. "Age-related Changes in Word Retrieval: Role of Bilateral Frontal and Subcortical Networks." *Neurobiology of Aging* 29: 436–451.
Wilson, R., and L. Foglia. 2017. "Embodied Cognition." In *Stanford Encyclopedia of Philosophy*, edited by E. Zalta. https://plato.stanford.edu/archives/spr2017/entries/embodied-cognition.
Zarahn, E., B. Rakitin, D. Abela, J. Flynn, and Y. Stern. 2007. "Age-related Changes in Brain Activation during a Delayed Item Recognition Task." *Neurobiology of Aging* 28 (5): 784–798.
Zurrón, M., M. Lindin, S. Galdo-Alvarez, and F. Diaz. 2014. "Age-related Effects on Event-related Brain Potentials in a Congruence/Incongruence Judgment Color-word Stroop Task." *Frontiers in Aging Neuroscience* 6: 1–8.