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Childhood cognitive development as a skill

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Theories view childhood development as being either driven by structural maturation of the brain or being driven by skill-learning. It is hypothesized here that working memory (WM) development during childhood is partly driven by training effects in the environment, and that similar neural mechanisms underlie training-induced plasticity and childhood development. In particular, the functional connectivity of a fronto-parietal network is suggested to be associated with WM capacity. The striatum, dopamine receptor D2 (DRD2) activity, and corticostriatal white-matter tracts, on the other hand, seem to be more important for plasticity and change of WM capacity during both training and development. In this view, the development of WM capacity during childhood partly involves the same mechanisms as skill-learning.

Background and hypothesis

Theories about child development have been plentiful during the 20th century, but few have integrated neuroscience into these theories. In the relatively new field of developmental cognitive neuroscience, three general theories on development can be distinguished: a maturational view, a skill-learning view, and interactive specialization [1–4].

According to the maturational view, development is driven by genetically pre-programmed, structural maturation of the brain. The skill-learning view emphasizes environmental influences as the driving force of development. Interactive specialization also views the environment as a driving force, but emphasizes that cortical areas change their functional characteristics as a result of the interaction.

These developmental theories are not mutually exclusive, and the proposed processes can coincide or occur at different stages of development. For example, prenatal myelination could provide the necessary structural maturation of connections between language areas, exposure to language in early childhood leads to specialization of the function of language areas in the left hemisphere, and skill-learning could drive reading improvements in later childhood and adolescence [5]. Another example of skill-learning is the suggestion that mechanisms of perceptual training are the same as those for childhood development of face perception [6].

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For executive functions – including WM, inhibition, shifting, and reasoning – the role of training has been unclear and many developmental neuroimaging studies have defaulted to a maturation interpretation. However, a range of different WM training paradigms have now demonstrated that WM capacity is significantly increased by training, including paradigms focusing on visuospatial WM [7–10], dual *n*-back tasks [11], updating [12,13], manipulation [14], and complex WM tasks [15] (reviewed in [16]). The implications of these findings for theories of cognitive development have not yet been explored.

WM training has ignited research on the associated neural plasticity using a range of methods including receptor studies in mice, neurophysiology in monkeys, and human studies using genetics, electroencephalopathy (EEG), functional magnetic resonance imaging (fMRI), and positron emission tomography (PET). This review will explore the hypothesis that the neural mechanism underlying training-induced plasticity could provide a model for childhood development. It is hypothesized that:

i The development of WM capacity during childhood depends partly on environmental influence, in other words, training through cognitive challenges in everyday life and education.
ii The neural mechanisms that underlie training-induced plasticity over weeks of WM training are to a large extent the same as those underlying environmental effects over years.
iii Increased WM capacity is caused by strengthened functional connectivity within and between frontal and parietal cortical regions.
iv The neural networks underlying plasticity can be partly differentiated from those of capacity, where striatum, DRD2 activity, and frontostriatal white-matter connections are more important to plasticity (i.e., potential for change; Figure 1).

The distinction between maturation and skill-learning is not equivalent to the distinction between nature and nurture in its most simplistic form because there are genetic predispositions to higher or lower plasticity [17–20] as well as gene–environment interactions where there is genetic predisposition for choosing a particular environment.

This article will first summarize evidence in support of the role of connectivity for WM capacity in both training and development (Hypothesis 3). The role of the striatum and dopamine for plasticity is then reviewed (Hypothesis 4). The

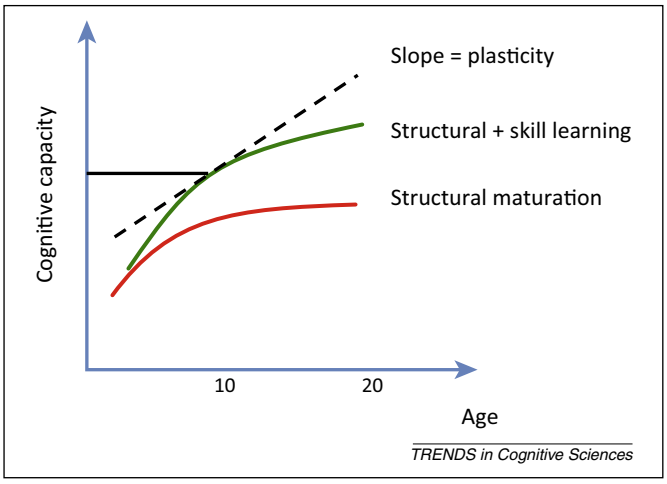

Figure 1. Illustration of how development of working memory (WM) capacity can be determined by structural brain maturation, skill-learning, or both. The height of the curve reflects the current cognitive capacity that can be differentiated from the slope (i.e., the change over time), which reflects the plasticity. Capacity here is assumed to be a quantifiable measure of a cognitive ability. WM capacity can be measured as the maximum amount of information that can be stored, for example, during a spatial-span test. This might correspond to what in earlier developmental theories has been termed 'mental capacity' [59]. Plasticity refers to modification of neural structures, which can be indirectly observed by changes in behavior. According to the hypothesis of the current paper, current capacity is associated with functional connectivity of the cortex. Changes in cortical connectivity are facilitated by the striatum, dopamine receptor D2 (DRD2)-mediated neurotransmission, and corticostriatal connectivity, which are strongly related to the slope of WM development.

relations of this research to theories of development (Hypotheses 1 and 2) are discussed in the concluding section.

WM development would, according to this hypothesis, have many similarities with training of motor and other skills, including the gradual improvement with repetitive training and the reliance on the interaction between striatum and cortex. In this view, development of WM capacity during childhood partly involves the same mechanisms as skill-learning.

Connectivity and capacity in WM development

Neurophysiological data and computational models suggest that the strength of neuronal connectivity and firing rate during the memory delay are important for determining cognitive capacity (Box 1). Several neural mechanisms are known to take place during childhood that could provide a basis for development of cognitive capacity, including: (i) pruning of synapses, that is thought to provide a finer resolution of representation; (ii) myelination of axons, which could influence axonal conduction; (iii) strengthening of connectivity within functional areas or local networks; (iv) strengthening of connectivity between functional areas (e.g., fronto-parietal connections); and (v) changes in expression of genes. In a biologically realistic simulation, the first four of these neural mechanisms were implemented in different neural networks [21], but only the strengthening of synaptic connectivity (neural mechanisms 3 and 4) led to higher delay firing rates and better resistance to distractors. Predictions from this model were also consistent with inter-individual differences in brain activity during a WM task. An increase of BOLD activity with increase in WM capacity during childhood is consistent with most developmental studies (e.g., [22–25]).

Box 1. Neurophysiology of WM

During a WM task, information is thought to be maintained during a delay by persistent activity in prefrontal and posterior areas, including the posterior parietal cortex [60,61]. An important aspect is that the firing rate of neurons during the delay is associated with the accuracy of the memory [41,61].

In addition to areas coding the sensory information, it is likely that WM activity includes a top-down signal from more anterior prefrontal regions (e.g., area 46) that could enhance maintenance or prioritize representations in more posterior frontal areas (e.g., areas 6 or 8) as well as parietal and sensory areas [62–64]. The mechanisms for visuospatial WM are largely identical to those underlying spatially selective control of attention [65].

The concept of sustained neuronal activity as a basis for memory retention has also been described using neuronal network models [41,66]. In these models, information is stored in the activity of a network by recurrent excitatory loops. The models have explored potential hypotheses for mechanisms that could provide enhanced and more stable storage. The models are consistent with neurophysiological data in suggesting that stronger inter-areal connectivity and higher firing rates during the delay are associated with better maintenance [41,63].

These data are consistent with electrophysiological data from young and adult macaque monkeys, where it was found that adult monkeys had stronger functional connectivity within prefrontal areas and a higher firing rate during performance of a WM task [26] (Figure 2A,B).

Increased functional connectivity is thus a prime candidate for producing the observed increases in firing rate and WM improvements during development. Fair and colleagues specifically analyzed changes in connectivity using resting-state data in children and young adults [27,28] (Figure 2C). The principal finding was a weakening of local networks (i.e., between different areas within the same lobe) but strengthening of long-range (>60 mm) connections. The strength of connectivity in functional networks measured as modularity during the resting state is also strongly associated with WM capacity [29].

Connectivity and capacity in WM training

The neurophysiological changes associated with improvement of WM during several weeks of WM training in monkeys include more neurons being activated during both cue and delay periods, and a higher firing rate during the memory delay [30] (Figure 2D). Increased firing rate and more neurons exhibiting delay activity are consistent with the increase in frontal and parietal BOLD signal that has been found in some studies of WM training in humans [14,31,32], although many studies also find decreased cortical activation [33,34]. It should be emphasized that translation of firing rate in a particular population of cells into their BOLD signal is not straightforward. The reasons for these inconsistencies might include differences in the amount of training time, amount of transfer to non-trained tasks (i.e., how much capacity is increased), as well as subtle differences in behavior during scanning, including inspection time and response time.

The effect of WM training specifically on fronto-parietal connectivity has been explored in humans with transcranial magnetic stimulation (TMS) [35]. In this study, functional connectivity was evaluated by activating the parietal cortex using TMS and then recording the elicited electrical