A Novel Ecological Account of Prefrontal Cortex Functional Development

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Abstract

In this paper, we argue that prefrontal cortex ontogenetic functional development is best understood through an ecological lens. We first begin by reviewing evidence supporting the existing consensus that PFC structural and functional development is protracted based on maturational constraints. We then examine recent findings from neuroimaging studies in infants, early life stress research, and connectomics that support the novel hypothesis that PFC functional development is driven by reciprocal processes of neural adaptation and niche construction. We discuss implications and predictions of this model for redefining the construct of executive functions and for informing typical and atypical child development. This ecological account of PFC functional development moves beyond *descriptions* of development that are characteristic of existing frameworks, and provides novel insights into the *mechanisms* of developmental change, including its catalysts and influences.

Keywords: prefrontal cortex, functional brain development, executive functions, adaptation

The PFC is perhaps the most elaborated and highly interconnected neocortical region in humans, and is necessary for complex thought and action characteristic of higher-level cognition (Badre, 2008; Badre & D'Esposito, 2009; Badre & Wagner, 2004; Koechlin, 2016; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier, Noelle, Braver, Cohen, & O'Reilly, 2005). The PFC has several unique characteristics. It is domain-general; through direct and indirect connections, it integrates and processes signals from almost every other neural region in the brain (Duncan & Owen, 2000; Fedorenko, Duncan, & Kanwisher, 2013). In addition, the PFC develops in the absence of direct input from sensory registers (Cahalane, Charvet, & Finlay, 2012). This is in contrast to more domain-specific neural regions that receive direct, stable sensory input, such as primary visual cortex. The PFC remains plastic at least through late adolescence (Diamond, 2002; Giedd et al., 1999; Gogtay et al., 2004) and possibly throughout the lifespan (Anguera et al., 2013; Lee, Ratnarajah, Tuan, Chen, & Qiu, 2015; Li et al., 2014; Lövdén et al., 2010), providing increased opportunities for the changes in the internal and external environment to shape PFC development.

The model of prefrontal cortex (PFC) functional development proposed here is inspired by ecological explanations for developmental change in cognition and behavior (Gibson, E. J., Pick, 2000; Rovee-Collier & Cuevas, 2009; Schneirla, 1957; Spear, 1984; Turkewitz & Kenny, 1982) and by a recent application of these ideas to brain development and risk for developmental psychopathology (M. H. Johnson, Jones, & Gliga, 2015). Ecological accounts consider infants, children, and adults to be different organisms who occupy different ecological niches, each of which carries its own unique demands and challenges. Ecological approaches emphasize that organisms from all species have evolved to be adapted to their unique niches at each point in development, since optimal development of phenotype depends on adaptation to *all*

environments, rather than adaptation only to the *final* environment (Lehrman, 1953). In this ecological view, infants and children have different sets of problems to solve for learning and behavior (Rovee-Collier & Cuevas, 2009). Thus, we may be limiting our understanding of ontogenetic brain development if we measure developmental change as relative only to the adult state.

We begin by examining the existing literature on the structural and functional development of the PFC. We will argue that these accounts are highly apt descriptions, but that they offer little mechanistic insight into how the system is developing, its catalysts, and its influences. We will then consider recent evidence that points to the hypothesis that *adaptation,* and not *maturation,* best describes the process of PFC developmental change. Throughout, we highlight novel predictions raised by this account of PFC development, and will examine implications of this ecological model for redefining executive functions and for informing typical and atypical developmental trajectories.

PFC: The State of the Art

The human prefrontal cortex is a collection of interconnected neocortical regions that send and receive projections from nearly all primary sensory and motor systems, as well as many subcortical regions in the brain (Gilbert & Li, 2013; E. K. Miller & Cohen, 2001). The PFC is anatomically defined as the projection zone of the mediodorsal nucleus of the thalamus in both primates and non-primates (Fuster, 2008). Within the PFC, there are a number of subregions that are delineated based on anatomical connections and granular structure (Barbas & García-Cabezas, 2016). These include the orbitofrontal PFC, ventrolateral PFC, dorsolateral PFC,

rostrolateral, and medial PFC (Badre & D'Esposito, 2007, 2009; Bunge & Zelazo, 2006; Crone & Steinbeis, 2017; Koechlin, 2016).

PFC subregions are separately critical for supporting flexible and goal-directed control over cognition and action, emotion, and social behaviors across time and contexts (Badre, 2008; Cohen, Braver, & Brown, 2002; Kolb et al., 2012; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier et al., 2005). Collectively, these are referred to as *executive functions*. Different regions of the PFC appear to be specialized for different executive functions. For example, lateral portions of the PFC (including dlPFC) are important for goal-directed thought or action (Badre, 2008; Badre, Kayser, & D'Esposito, 2010; Barch et al., 1997; Bunge, 2003; Curtis & D'Esposito, 2003), whereas dorsomedial portions of the PFC are involved in decision-making under risk and uncertainty (Hadland, 2003; Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006; Rushworth, Walton, Kennerley, & Bannerman, 2004). Ventromedial PFC (vmPFC, including the orbitofrontal cortex) is implicated in decision making relevant to maximizing benefit and minimizing cost based on stimulus-reward relationships (Fellows & Farah, 2003; Hornak et al., 2004; Tsuchida, Doll, & Fellows, 2010). The functional specialization of the various subregions of PFC is due in part to differences in the input to each subregion from connections with other neural regions (Duncan & Owen, 2000; Fedorenko et al., 2013; E. K. Miller & Cohen, 2001).

Prenatal PFC Structural Development

During prenatal brain development, the primary structural features of PFC, and its anatomical connections with other regions, are formed by two major patterns of maturation, which in turn have significant implications for the subsequent function. First, during prenatal brain development, the cortex is populated with neurons in an anterior-to-posterior or "front-to-

back" gradient, such that cell migration ceases in the anterior rostrolateral cortex first, and then progresses caudomedially towards more posterior regions (Cahalane et al., 2012; Finlay & Uchiyama, 2015, 2017). This gradient of neurogenesis results in frontal neurons becoming differentiated *earlier* than neurons in more posterior regions. It also occurs opposite of the gradient of thalamic innervation, where thalamic input innervates sensory regions prior to anterior association regions (Cahalane et al., 2012; Finlay & Uchiyama, 2017). These opposing patterns, of thalamic innervation and relative to cortical neurogenesis, are hypothesized to bias the PFC for its association role. That is, this anterior-to-posterior neurogenesis in PFC occurs in the absence of sensory input from thalamic registers and is therefore primarily shaped by the PFC's intrinsic cortical activity (Cahalane et al., 2012; M. H. Johnson et al., 2015).

Prenatal brain development is simultaneously characterized by an "inside-out" gradient with respect to the formation of cortical laminar structure. The laminar structure of the subregions of PFC varies. There are agranular and dysgranular regions that have fewer than six layers, such as the orbitofrontal (pOFC), anterior cingulate cortex (ACC), and vmPFC. There are also eulaminate regions that have six well-defined and delineated layers, such as the dlPFC (Barbas & García-Cabezas, 2016). This layered structure is formed during prenatal development in an inside-out pattern, where deeper layers are formed prior to upper layers (Sidman & Rakic, 1973).

The variance in laminar structure across the subregions of PFC is thought to be driven by differences in prenatal developmental timing of neural migration (Barbas & García-Cabezas, 2016; Dombrowski, Hilgetag, & Barbas, 2001). Specifically, agranular and dysgranular regions with fewer than six layers have a shorter developmental period relative to eulaminate regions. These differences in developmental timing are thought to arise from decreased neuronal density

in the upper layers of agranular and dysgranular regions relative to eulaminate regions (Barbas & Garcia-Cabezas, 2016).

The systematic variations in laminar structure across the subregions of PFC bias their subsequent functions and connectivity with other regions (Barbas & Garcia-Cabezas, 2016). For example, regions with fewer or less complex layers, such as the anterior cingulate cortex (ACC) and vmPFC, primarily have feedforward and feedback connections with subcortical structures including the amygdala, hippocampus, and hypothalamus. Consistent with their patterns of connectivity, these subregions of PFC primarily process "internal" environmental information, such as emotions, rewards, motives, and drives (Barbas & Garcia-Cabezas, 2016). In contrast, eulaminate regions with six well-defined layers, such as the dlPFC, have feedforward and feedback connections from other eulaminate sensory regions, such as the primary sensory cortices, in addition to the agranular and dysgranular subregions of PFC. These patterns of connectivity thus make dlPFC well-placed to process information from sensory cortices as well as internal emotional and motivational information.

Postnatal PFC Structural Development

The existing understanding of PFC functional development has been primarily influenced by findings on PFC's *postnatal* structural development. The existing consensus is two-fold: (1) PFC postnatal structural maturation has a protracted course (e.g., Diamond, 2002; Fuster, 2002; Giedd et al., 1999; Gogtay et al., 2004; Shaw et al., 2008); (2) This structural development then supports associated executive functions development as well as individual differences in executive functions efficiency (e.g., Diamond, 2002; Fuster, 2002; Kharitonova, Martin, Gabrieli, & Sheridan, 2013; Paus, 2005; Sheridan, Kharitonova, Martin, Chatterjee, & Gabrieli, 2014). This view is further supported by evidence from twin studies that executive functions are

99% heritable (Friedman et al., 2008). However, these estimates of genetic heritability are likely inflated due to shared environments among twins. For instance, twins in low socioeconomic environments have much lower estimates of cognitive heritability (Harden, Turkheimer, & Loehlin, 2007; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

The existing framework is shaped by findings that PFC postnatal structural maturation is greatly protracted in comparison to other cortical and subcortical neural regions. During the first few years of life, the brain rapidly quadruples in size, reaching 90% of its adult volume by 6 years of age (Courchesne et al., 2000; Knickmeyer et al., 2008). During this period of rapid early development, PFC expands over twice as much as other cortical regions (Hill et al., 2010). This period is characterized by large increases in synaptogenesis and neurogenesis, followed by periods of neuronal pruning and synaptic death to accommodate the early increases in neural and synaptic formation (Stiles & Jernigan, 2010). Some findings suggest that these rates of neural and synaptic formation and elimination occur heterochronously across different cortical regions (Rakic, 2002). During early gestational development, synaptogenesis and neurogenesis are fairly evenly distributed across cortical regions, and neuronal density within PFC is similar to other cortical regions (Shankle, Rafii, Landing, & Fallon, 1999). However, some studies suggest that soon thereafter, rates of neural and synaptic formation across different cortical regions quickly change, with PFC being one of the latest regions to reach its peak. For example, work analyzing post-mortem human brains by Huttenlocher & Dabholkar (1997) found that synaptogenesis peaked at approximately 3 months of age in sensorimotor cortex, whereas synaptogenesis occurred much more slowly in PFC, not reaching its peak until around 3.5 years of age (Huttenlocher & Dabholkar, 1997). That the number of synapses in PFC appears to peak early in childhood does not necessarily indicate that new synapses are no longer forming. Rather, it may

reflect that the rate of synaptic elimination exceeds the rate of synaptogenesis (Petanjek et al., 2011; Selemon, 2013). However, note that several other studies of non-human primate brains found no differences in synaptogenesis across cortical regions (Bourgeois, Goldman-Rakic, & Rakic, 1994; Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986; Zecevic, 1998; Zecevic, Bourgeois, & Rakic, 1989). These discrepancies have been suggested to reflect methodological confounds between primate and human data (Goldman-Rakic, Bourgeois, & Rakic, 1997).

Similarly, PET studies with human infants have shown that PFC has a lag of up to 8 months in reaching peak levels of glucose metabolism – a measure of neural activity (Chugani & Phelps, 1986). This is in contrast to temporal, parietal, and occipital cortices, where peak levels of glucose metabolism are observed soon after birth (Chugani & Phelps, 1986). Another useful index of brain development is cortical thickness. Measuring cortical thickness provides a composite index of overall maturity that includes neurons, synapses, axons, dendrites, and glia. During prenatal development, cortical thickness increases linearly across the entire brain as a function of time (Rabinowicz, de Courten-Myers, Petetot, Xi, & de los Reyes, 1996). During postnatal development, however, overall cortical thickness of PFC follows an inverted U-shaped trajectory, increasing throughout childhood, peaking in adolescence, and then slowly decreasing and stabilizing in early adulthood (Shaw et al., 2006). Protracted changes in white and grey matter volume within PFC also occur well into adolescence (Giedd et al., 1999; Gogtay et al., 2004; Reiss, Abrams, Singer, Ross, & Denckla, 1996). These increases are more protracted in PFC compared to other regions of the cortex, where increases are primarily observed up to around 6-9 years of age (Courchesne et al., 2000). We note that these agranular and dysgranular

PFC subregions seem to also structurally develop, based on cortical thickness, postnatally earlier than eulaminate PFC subregions (Shaw et al., 2008).

Recent work also shows protracted changes in myelination of pathways between PFC and other neural regions. Maturation of white matter tracks is found to broadly follow a posterior-to-anterior gradient, such that sensory and motor regions myelinate first and cortical association regions including PFC myelinate last (Deoni et al., 2011; Deoni, Dean, O'Muircheartaigh, Dirks, & Jerskey, 2012). While changes in myelination are important as it is assumed that they result in an increase the efficiency of neural communication, undermyelinated pathways are also capable of transmitting signals, albeit less efficiently (M. H. Johnson & De Haan, 2015).

PFC Functional Development

The existing framework of PFC structural and functional development has primarily linked PFC postnatal structural development to age-related changes in executive functions (Diamond, 2002; M. H. Johnson, 1990; M. H. Johnson, Posner, & Rothbart, 1994; Luna & Sweeney, 2004; Stuss, 1992). The argument that structural maturation drives cognitive development is particularly compelling since the functional developmental course of executive functions closely follows the developmental course of PFC maturation (Amso, Haas, McShane, & Badre, 2014; M. C. Davidson, Amso, Anderson, & Diamond, 2006; Snyder & Munakata, 2010; Wendelken, Munakata, Baym, Souza, & Bunge, 2012; Zelazo et al., 2003). The close parallels between PFC maturation and improvements in executive functions have therefore led many to propose that structural maturation of PFC allows new or more advanced abilities to "come online".

In human infants, one of the earliest and most comprehensive lines of work relating PFC maturation to changes in executive functions comes from work using Piaget's A-not-B task

(Piaget, 1952). In standard versions of this task, infants watch as an experimenter hides a desirable object in one of two possible locations, and infants are allowed to reach to search for the object after a brief delay. During the task, the object is typically hidden in the same location (A) for multiple trials before it is reversed and hidden in the alternate location (B). The A-not-B error occurs when infants reach to the previously correct location (A) rather than the new location (B) on these reversal trials. Piaget was the first to observe that infants younger than 7 months tend to make this perseverative A-not-B error (Piaget, 1952). That is, infants younger than 7 months of age typically reach to the location where the object was hidden on the immediately preceding trial, rather than to the new location.

Between 7.5-9-months, infants begin to reach to the new location on reversal trials at successively longer delays ranging from 1-5 seconds (Diamond, 1985, 2001). However, as the delay between hiding and searching is incrementally increased, infants continue to make the A-not-B error until around 12 months of age (Diamond, 1985). Infant monkeys show similar developmental progressions on this task as human infants (Diamond & Goldman-Rakic, 1989). Anatomical work with monkeys provides direct evidence that behavioral improvements parallels maturation of dorsolateral PFC (dlPFC), suggesting that structural maturation of PFC supports cognitive changes (Diamond & Goldman-Rakic, 1989). This idea is further supported by evidence that adult monkeys with dlPFC lesions also show the A-not-B error when there is a delay between hiding and searching (Diamond & Goldman-Rakic, 1989). In human infants, evidence that improvements on the A-not-B task relate to PFC development is found from work using electroencephalography (EEG) and near-infrared spectroscopy (NIRS) to record infants' brain activity during cognitive testing. For example, frontal EEG responses in 7-12-month-old infants correlate with behavioral performance in the A-not-B task (Bell & Fox, 1992; Fox &

Bell, 1990). A longitudinal NIRS study with 4-12-month-old infants also indicates that PFC blood oxygenation levels correlate with behavioral performance in this task (Baird et al., 2002).

Data support the linkage between PFC maturation and executive functions development into late childhood and adolescence. For example, a large cross-sectional study of 3-25-year-olds who were tested on an extensive battery of cognitive tasks showed that tasks that are dependent on more posterior brain regions, such as recognition memory, tend to stabilize around 8 years of age (Luciana, 2003; Luciana & Nelson, 1998, 2002). However, tasks that recruit prefrontal cortical regions show more protracted development, not nearing adult levels of performance until around 12 years of age. Maturation of white matter tracts within frontoparietal networks also correlates with improvements in executive functions. For example, in children ages 8-18 years, maturation of white matter tracts, as measured by diffusion tensor imaging (DTI), is associated with increased working memory capacity (Olesen, Nagy, Westerberg, & Klingberg, 2003), as well as with increased task-dependent functional activation within frontal and parietal brain regions (Klingberg, Forssberg, & Westerberg, 2002).

Taken together, this very brief review of an extended body of evidence provides support for a structural maturation account of PFC functional development. However, by necessity, these studies reflect inferences drawn based on correlations between structure and function, most often across different studies and populations, and no evidence of directional causation. Further, the existing framework has primarily examined developmental changes in PFC functional development using a model of function that was developed based on adult data. Thus, the existing account, of the causes of PFC developmental change, offers little in the way of mechanistic predictions other than the assumption that functions “come online” at certain ages. Moreover, these viewpoints consider PFC functional development largely with a spotlight on

frontostriatal and frontoparietal change, using a construct of executive functions that is defined relative to an adult state (Dosenbach et al., 2007). However, PFC is a widely interconnected region, sending and receiving inputs from auditory, emotion, memory, and motor areas as well. The narrow spotlight constrains a full ontogenetic understanding of PFC functional development by limiting explanatory power to descriptions of how PFC functions become more "adult like", rather than considering the *mechanisms* driving developmental change.

PFC: Revising Assumptions

Recent behavioral, neuroanatomical, and electrophysiological research provides novel insights into the functional development of the PFC. Specifically, the PFC is functionally active and involved in organizing complex behaviors from the first months of life, that its rate of development is not deterministic but can be impacted and even accelerated by extreme experience, and that executive functions reflect changes in whole brain connectivity above and beyond simple PFC structural maturation. We review each of these findings in turn.

PFC is Active and Implicated in Behavior as Early as Birth

Research examining resting-state brain activation in infants using fMRI shows that infants have PFC activity in frontoparietal networks from birth (Doria et al., 2010). This indicates that PFC is active even before the emergence of classic executive functions later in infancy. Evidence from near-infrared spectroscopy (NIRS) and positron emission tomography (PET) studies also shows that infants frequently have PFC activation during cognitive tasks, even in cases where adults *do not* have PFC activation. For example, one study used NIRS to examine newborn infants' PFC activation when listening to adult-directed compared to infant-directed speech (Saito et al., 2007). The authors found that infants showed strong cortical activation over

anterior PFC when listening to infant-directed speech, but not when listening to adult-directed speech. Other work showed that 3-month-olds have activation in the dorsolateral PFC when listening to forward speech, but not backward speech (Dehaene-Lambertz, Dehaene, & Hertz-Pannier, 2002). This finding was evident only when infants were awake and attentive and not when they were asleep. These studies suggest that PFC is involved in processing linguistic input that is relevant to infants during early life.

PFC might also help process social information in young infants. For example, work by Grossmann and colleagues (2008) used NIRS to test whether PFC is implicated in early social cognition in young infants. During this study, 4-month-olds were shown videos where an actor either established mutual gaze with the infants, or averted their gaze away from infants. Infants had increased right fronto-polar cortical activation only when viewing videos where the actor established mutual gaze, and not when the actor averted their gaze. Similar work shows that 5-month-olds also have strong dorsal PFC activation when attempting to coordinate their attention with others (Grossmann & Johnson, 2010; Grossmann, Lloyd-Fox, & Johnson, 2013). As infants get older, the specificity of PFC activation to social information may increase. For example, 9-12-month-olds have increased anterior orbritofrontal activation only when they view their mother smiling and not when they view strangers smiling (Minagawa-Kawai et al., 2009). These studies suggest that PFC may play an important role in processing socially relevant information in infants' environments.

PFC also appears to be implicated when infants plan motor or oculomotor actions, or when they view actions of others. For example, work by Csibra, Tucker, and Johnson, (2001) examined 4-month-old infants' frontal ERP responses while viewing sequences of predictable stimuli presentations. When making proactive (anticipatory) eye movements, infants had positive

eye movement related potentials over frontal regions. Importantly, however, they did not have these frontal potentials when making reactive eye movements, which suggests that PFC may be involved in planning oculomotor actions from very early in life (Richards, 2000). Other work using NIRS shows that 5-month-olds also have increased inferior frontal cortical activation when viewing videos of human motor actions, but not when viewing other forms of motor actions, such as mechanical, non-human actions (Lloyd-Fox, Blasi, Everdell, Elwell, & Johnson, 2011).

A body of research also points to a role for PFC in regulating and processing emotional responses (Ahmed, Bittencourt-Hewitt, & Sebastian, 2015; Gilmartin, Balderston, & Helmstetter, 2014; Ochsner & Gross, 2008) as early as infancy. For instance, work using EEG has found that left frontal asymmetry, as measured by increased activation in left relative to right frontal regions, relates to emotional regulation and temperament in infancy (R. J. Davidson & Fox, 1982, 1989; Dawson, Panagiotides, Klinger, & Hill, 1992; Diaz & Bell, 2012; Fox, 1991, 1994; Fox & Davidson, 1987). Frontal asymmetry is also modulated by environmental factors. For example, infants of depressed mothers typically have opposite patterns of frontal asymmetry relative to typical controls (e.g., right frontal asymmetry, rather than left frontal asymmetry), which parallels findings from inhibited infants and from chronically depressed adults (Field, Fox, Pickens, & Nawrocki, 1995; Jones, Field, & Almeida, 2009; Jones, Field, Fox, Lundy, & Davalos, 1997; Lusby, Goodman, Bell, & Newport, 2014).

Given evidence for architectural constraints on the prenatal organization of the PFC, as well as constraints on the computational properties of neurons in the human brain (e.g., Elman et al., 1996), it would seem unlikely that the PFC and its subregions are performing fundamentally different tasks in infants than in children or adults. We propose that each PFC subregion is likely performing similar computations across the lifespan. The domain generality of PFC regions

affords opportunities for those computations to be applied to information that is relevant and available to the organism in their current developmental state.

In support of this hypothesis, recent work suggests that dlPFC may be involved in constructing abstract rules that guide learning and behavior during infancy across both linguistic and visual domains (Werchan, Collins, Frank, & Amso, 2015, 2016). In this research, 8-month-old infants were presented with simple visual stimulus-response pairings that could be learned as individual associations or as a set of hierarchical rule structures, a learning mechanism that involves PFC and its dopamine-innervated connections with striatum (Collins, Cavanagh, & Frank, 2014; Collins & Frank, 2013). Results suggested that infants spontaneously constructed hierarchical rules when learning the simple visual stimulus-response associations, which then supported generalization in novel contexts (Werchan et al., 2015). Additional results suggested that infants also used this learning mechanism to help structure different spoken labels for the same objects into hierarchical rule structures, akin to learning in a bilingual environment. Recent work using NIRS provided evidence that this learning mechanism is related to right dlPFC activation in 8-month-old infants (Werchan et al., 2016). Taken together, this body of evidence suggests the possibility that PFC, and dlPFC in this example, may be involved in creating rule structures that help organize linguistic, social, emotional, and oculomotor learning and action as early as in infancy.

Connectivity, not PFC structural maturation *per se*, may be key to executive functions development

Methodological and statistical advances, resting state fMRI combined with functional connectivity analyses, have furthered our understanding of the structural and functional changes that occur during human brain development. Recent data from these methods show that changes

in PFC *connectivity*, rather than solely structural maturation of PFC, may support changes in executive functions.

Connectomics is a recent addition to the field of functional magnetic resonance imaging (fMRI) research that examines the functional coupling of brain regions into networks, as measured by correlated activation of brain regions (Sporns, 2013). One benefit of connectomics research for understanding PFC development is that it examines how PFC changes and develops *within the context of the entire brain*, rather than as a singular region. Examining changes within the context of a large interconnected system is especially important for understanding PFC development, since PFC has extensive connections with other neural regions.

Research in this area demonstrates the existence of highly connected "hub" regions that play an important role in global information integration between different regions of the brain (van den Heuvel & Sporns, 2013). Interestingly, hub regions in the human connectome change across development. In early postnatal life, the first functional hubs are found in unimodal cortical areas, including auditory, visual, and sensorimotor cortices (Fransson, Åden, Blennow, & Lagercrantz, 2011). The location of these hubs shifts throughout development, eventually settling to hubs in the posterior cingulate, insula, and other heteromodal cortices by adulthood (Fransson et al., 2011).

These shifts are thought to partially reflect changes from segregation to integration with respect to regional functional connectivity. During early infancy a "small-world" architecture dominates functional connectivity across the entire brain, with increased short-range connections within cortical regions and decreased long-range connections between cortical regions. This pattern reflects segregation. The overall number of connections remains constant across development; however, the number and strength of long-range connections between different

cortical regions increases while the quantity of short-range connections within regions decreases (Fair et al., 2007; Gao et al., 2011; Supekar, Musen, & Menon, 2009). This pattern reflects regional integration and relates to executive functions in tasks assessing reasoning ability. For example, shifts in functional connectivity from short-range intracortical connections within PFC to long-range frontoparietal connections correlates with age-related improvements in relational reasoning abilities during the transition from childhood to adolescence (Wendelken, Ferrer, Whitaker, & Bunge, 2016). Similarly, developmental improvements in inhibitory control during an antisaccade task correlate with increases in long-range functional connections between multiple regions of the PFC (including the ACC, medial frontal gyrus, inferior frontal gyrus, and frontal eye fields) and subcortical and parietal regions, which is thought to reflect increased top-down modulatory control over behavior (Hwang, Velanova, & Luna, 2010). However, note that the magnitude of shifts from short- to long-range functional connections may have been exaggerated due to increased motion artifacts in younger children relative to older children and adults (see Power, Barnes, Snyder, Schlaggar, & Petersen, 2012).

It is notable that changes also occur at different rates for anatomical compared to functional connections. Nonhuman primate work has shown that structural constraints, in the form of feedforward and feedback cortical connections, are in place prenatally (Goldman-Rakic, 1987). Anatomical connections within the frontoparietal network are adult-like by around 9 months of postnatal life (Conel, 1939); however, functional connectivity between these regions only *begins* to be evident between 6-9 months of age (Fransson et al., 2007) and develops into young adulthood, which coincides with increases in myelination (Giedd et al., 1999; Klingberg et al., 2002).

Simultaneous increases in functional connectivity and myelination are thought to occur, at least in part, in response to experience. A principle idea in the postnatal neural development of cortex is that there is a minimum of genetic instruction necessary for organized developmental change, and that developmental change instead occurs largely in response to experience (Finlay & Uchiyama, 2017; Kolb et al., 2012). There is, however, strong evidence that cortical areas are hierarchically organized along a rostro-caudal gradient (e.g., Van Essen & Maunsell, 1983). In this context and given its location, PFC necessarily computes over inputs from sensory, motor, and basal forebrain regions (Amso & Scerif, 2015; Finlay & Uchiyama, 2015; Gilbert & Li, 2013).

Experience-based changes in the mammalian cortex are thought to relate to changes in connectivity (Caroni, Donato, & Muller, 2012; Holtmaat & Svoboda, 2009), which can be improved via changes in axonal myelination, as well as synaptic plasticity via dendritic remodeling and synaptic pruning. For instance, in recent work, C. M. Johnson and colleagues (2016) examined changes in PFC dendritic spine formation and elimination in response to experience using two-photon imaging and a rule learning task in a rodent model. They found that animals in the rule training group had greater OFC to dmPFC bouton turnover. At the same time, the total bouton density did not differ between the groups and boutons gained were not lost after rule reversal training. These data represent a change in synaptic structure in PFC in response to learning a novel rule structure. They argue that there is not a net pruning of dendritic spines in response to rule reversal learning, but that experience instead results in persistent changes to PFC synaptic structures (Hofer, Mrsic-Flogel, Bonhoeffer, & Hübener, 2009; Holtmaat, Wilbrecht, Knott, Welker, & Svoboda, 2006; Muñoz-Cuevas, Athilingam, Piscopo, & Wilbrecht, 2013; Yang, Pan, & Gan, 2009).

As such, changes in functional connectivity that occur in response to environmental experience may be relevant to functional PFC development. Connectivity thus becomes an important piece in the ecological account, which considers PFC development as a process of experience-dependent adaptation.

Early life adversity modulates PFC development

A third line of work shows that, rather than PFC having a strict maturational course, early childhood adversity, variations in socioeconomic status, and early life deprivation can modulate the development of PFC and its functional connections with other neural regions thereby impacting PFC function (Brito & Noble, 2014; Farah et al., 2006, 2008; Hackman, Farah, & Meaney, 2010; Hackman, Gallop, Evans, & Farah, 2015; McLaughlin et al., 2014; Noble et al., 2015). For instance, a number of studies have found that adversity experienced early in childhood results in delayed cortical development, relative to those who do not experience adversity, as measured by delayed gray matter maturation and decreased cortical thickness within PFC (Hair, Hanson, Wolfe, & Pollak, 2015; Mackey et al., 2015; McLaughlin et al., 2014; Noble et al., 2015), as well as significant reductions in white matter tracts between PFC and other cortical and subcortical regions (Eluvathingal, 2006; Hanson et al., 2013; Hanson, Knodt, Brigidi, & Hariri, 2015; Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012). These structural changes are paralleled by worse performance on classic measures of executive functions (see Brito & Noble, 2014; Hackman & Farah, 2009; S. B. Johnson, Riis, & Noble, 2016, for review), potentially reflecting adaptation to environments with little enrichment or challenges for learning and behavior. This idea is also supported by evidence showing that the most effective interventions for children at risk for poor executive functions development tend to be those that increase overall environmental enrichment and that progressively increase the demand placed on

PFC processing (Diamond & Lee, 2011). Thus, these studies highlight that PFC structural maturation is not deterministic, but is greatly impacted by the environment and experience, potentially as an adaptive response to the characteristics of an individual's environmental constraints.

Additional evidence for the role of adverse early environments in modulating PFC development comes from work examining early life stress in the form of maternal deprivation. This work has found that early life stress impacts the functional development of circuitry linking ventromedial PFC (vmPFC) and amygdala (for a review, see Callaghan & Tottenham, 2016). In typical development, maturation of vmPFC-amygdala circuitry occurs during the transition from childhood to adolescence, and it is also associated with improvements in emotional regulation (Callaghan, Sullivan, Howell, & Tottenham, 2014). For example, typically developing children have immature patterns of vmPFC-amygdala connectivity (e.g., positive coupling between vmPFC and amygdala activity) during emotional regulation tasks (Gee, Humphreys, et al., 2013). This positive coupling mirrors behavioral patterns of fear responses, such as increased (but developmentally normative) levels of separation anxiety. During the transition to adolescence, vmPFC-amygdala connectivity begins to mature to a more adult-like pattern, such that adolescents have negative coupling between vmPFC and amygdala (Gee, Humphreys, et al., 2013). These changes also parallel decreases in reported levels of separation anxiety.

Recent work examining the impact of early life stress on the timing of vmPFC-amygdala circuitry maturation provides support for the idea that maturation may reflect ecological adaptation. For example, rodent models indicate that early life adversity in the form of maternal deprivation *accelerates* the development of vmPFC-amygdala circuitry, such that adult-like states are reached at an earlier age than in typical development (for a review, see Callaghan &

Tottenham, 2016). This accelerated development is modulated by elevated cortisol levels (Callaghan & Richardson, 2012, 2014), and it also relates to the early emergence of adult-like fear learning that is supported by this circuitry (Callaghan & Richardson, 2011). Similar findings have also been observed in humans. For example, work by Gee, Gabard-Durnam, and colleagues (2013) found that previously institutionalized children who were maternally deprived during early childhood had accelerated maturation of vmPFC-amygdala circuitry. Specifically, maternally-deprived children exhibited positive coupling between vmPFC and amygdala activity when viewing emotional faces earlier in development than typical comparisons. This accelerated development was also paralleled by decreases in developmentally normative levels of separation anxiety, which suggests that accelerated maturation of vmPFC-amygdala circuitry may be an adaptive response to environments where extreme maternal separation is experienced. Similar work also shows that history of maternal separation influences exploration versus exploitation strategy use in children (Humphreys et al., 2015), providing further support that atypical developmental trajectories associated with early life stress might reflect adaptations to adverse early environments.

Note that whether adaptation results in protracted or accelerated development may depend on whether the driver is "external" (e.g., sensory input) or "internal" (e.g., emotions, motives, and drives). As discussed in the prenatal development section above, vmPFC has fewer cortical layers, matures earlier, and primarily sends and receives connections from subcortical structures, whereas dlPFC has six well-defined layers, matures more slowly, and sends and receives projections from sensory regions as well as from other subregions of PFC (Barbas & Garcia-Cabezas, 2016). Thus, internal drivers, such as heightened amygdala reactivity and stress resulting from maternal deprivation, may lead to accelerated development of vmPFC through

direct feedforward pathways from amygdala and other subcortical structures to vmPFC (Barbas & Garcia-Cabezas, 2016). In contrast, delays in dlPFC development, as often seen as a function of differences in children from varying socioeconomic environments or in children who have experienced early life deprivation, may be mediated through either connectivity with sensory regions or indirectly with subcortical limbic regions (Barbas & Garcia-Cabezas, 2016), which may result in delayed maturation (Hair et al., 2015; Mackey et al., 2015; McLaughlin et al., 2014; Noble et al., 2015). This is supported by recent neuroimaging findings indicating that differences in socioeconomic status in children's home environments appear to influence PFC's cortical thickness and white matter microstructure through limbic regions (Lawson, Duda, Avants, Wu, & Farah, 2013; Ursache & Noble, 2016).

An Ecological Account of PFC and Executive Functions Development

Taken together, recent findings require a reconceptualization of PFC functional development. The PFC is active and implicated in organizing learning and action, particularly in social, linguistic, emotional, and oculomotor domains relevant to infants. In childhood, the PFC adapts to be involved in regulating thought and action control processes. Rather than being on a fixed maturational time-course, PFC development can be delayed or accelerated in response to environmental demands. Finally, executive functions development is linked to PFC connectivity with the rest of the brain, and not to volumetric changes within PFC *per se*.

This literature supports the hypothesis that PFC development may be an *adaptive response* to changes in the organism's internal or external environment. The term *environment* here reflects the integrity of inputs to PFC from the rest of the brain (which are additionally constrained by the physical structure of the changing body), the nature and content of the

knowledge gained, and the opportunities and challenges present in a child's external environment. As noted earlier, PFC serves as a domain general processing system that performs computations across a range of different inputs. While neuroconstructivist viewpoints that incorporate interactive specialization may be better suited for understanding cortical specialization in regions that process specific, stable input, such as visual cortex (M. H. Johnson, 2000, 2011), an ecological approach may be more apt for understanding how domain general systems develop as a function of adaptation to changing multi-modal input that necessitates organization. This account rests on the tenet the PFC performs similar computations across the lifespan, and that these computations are adapted to the unique ecological niche occupied by individuals at each point in development. This is a process-oriented account of PFC and executive functions, which dissociates the cognitive processes supporting executive functions from the specific representations that these processes operate over (e.g., (Duncan, 2001; Duncan & Miller, 2002).

The key premises of the ecological account of PFC functional development are:

(1) The PFC performs the same basic computations across the lifespan, but these computations are continually adapted for novel learning demands relevant to an individual in their unique ecological niche.

(2) PFC functional development will be reciprocally influenced by adaptation to changes in the input to the PFC via feedforward connections, as well as through niche construction via PFC's feedback connections to other neural regions.

(3) Both adaptation and niche construction will be constrained by anatomical proximity of regions in relation to the PFC.

We will expand on each of these key ideas and highlight existing empirical support for them, as well as discuss novel testable predictions arising from them, in the subsequent sections.

PFC Computations and Redefining Executive Functions

A neural computation is defined here as a mathematical means of describing a neural pattern of response relevant to function. Computational models provide support for the idea that many of the broad thoughts and behaviors that PFC supports can be captured by a set of neural computations. For instance, a recent broad view implicates PFC in mounting adaptive responses in uncertain or variable environmental contexts (Koechlin, 2016). In this view, computations in vmPFC are used to learn the expected reward values of stimulus-action pairings, whereas those in lateral portions of the PFC implement more complex and hierarchically nested state-action mappings. A derivative process within lateral PFC, for example, is that it supports the active maintenance of information, which is represented by distributed patterns of neural activity (Badre, 2008; Cohen et al., 2002; D'Esposito & Postle, 2015; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier et al., 2005). This process allows information to be maintained in working memory over time, protecting it from interference from distracting or irrelevant inputs. Recurrent excitatory connections within PFC, as well as the intrinsic bistability of PFC neurons support this computation, which is also modulated by dopamine activity within PFC (Cohen et al., 2002; Constantinidis & Klingberg, 2016; Durstewitz, Seamans, & Sejnowski, 2000; Fallon, Williams-Gray, Barker, Owen, & Hampshire, 2013; Lew & Tseng, 2014; O'Reilly, 2006; Rougier et al., 2005).

A second computation supported by PFC is the adaptive updating of patterns of neural activity by dynamically switching between active maintenance and rapid updating of new

representations (Braver & Cohen, 2000; Chatham & Badre, 2015; Chatham, Frank, & Badre, 2014; Chiew & Braver, 2017; Cohen et al., 2002; Frank, Loughry, & O'Reilly, 2001; O'Reilly, 2006; Rougier et al., 2005). This computational process is thought to be supported by a selective, dopamine-modulated gating mechanism that controls the flow of information into PFC (Braver & Cohen, 2000; Chatham & Badre, 2015; Chatham et al., 2014; Chiew & Braver, 2017; Rougier et al., 2005). VMPFC is involved in switching from exploration to exploitation states, relevant to uncertainty in choice value outcomes (Domenech & Koechlin, 2015; Koechlin, 2016). In particular, data suggest that, together with the vmPFC, the dACC is involved in making the switch from exploration to exploitation policy (Donoso, Collins, & Koechlin, 2014; Kolling, Behrens, Mars, & Rushworth, 2012).

A third PFC computation involves modulating neural processing in other cortical areas that are required for successful task execution (Braver, Paxton, Locke, & Barch, 2009; Buschman & Miller, 2007; Duncan, 2001; Fuster, 2008; Gilbert & Li, 2013; B. T. Miller & D'Esposito, 2005; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier et al., 2005). This process is supported by PFC's extensive interconnectivity with other subcortical and cortical neural regions. As mentioned previously, PFC integrates inputs from other neural regions; however, rather than acting simply as an "integrator" and "transmitter" of these inputs, PFC modulates the flow of activity in subcortical and posterior regions via top-down feedback pathways to align with the goals and values of currently relevant information that is maintained within PFC (Miller & Cohen, 2001; Gilbert & Li, 2013).

Framing PFC's role in learning and behavior as a set of computations is useful in that allows for understanding of how a common underlying process or computation can support a diverse range of functions that are appropriate for humans in each stage of their development.

For example, an ecological account would predict that during early infancy, maintenance and gating may underlie planning of saccadic eye movements to sample information required for learning environmental contingencies. This aligns with findings showing that 4-month-old infants have frontal ERPs during anticipatory but not reactive eye movements during a simple stimulus-response learning task (e.g., Csibra et al., 2001). As infants begin learning to reach and grab objects, these same computations may be used to plan goal-directed reaching for objects, as supported by work indicating that 5-month-old infants have increased frontal activation when viewing human motor actions, but not non-human actions (e.g., Lloyd-Fox et al., 2011). During early childhood, these computations may then be adapted to support learning of novel complex social and moral action rules, mathematical concepts etc., in the environment. In adults, the same PFC region may implement its computations of gating and maintenance of task-relevant information to select complex rules for actions (e.g., planning the necessary actions required for cooking a complex meal, balancing a budget, planning for the future, following a diet).

The ecological account, with its focus on adaptation, also offers a revised definition of executive functions. Executive functions can be defined in this framework as those computations performed by PFC at any point in the lifespan. This redefinition offers the prediction that developmental continuity should be observed in PFC's computations when ecologically appropriate tasks are used at different developmental stages. For example, the PFC is involved in hierarchically organizing and processing language in infancy, recalling the data on infant-directed speech (Saito et al., 2007) and hierarchical learning of languages in context (Werchan et al., 2016). Early language development has been shown to be a strong predictor of later, more classic frontostriatal executive functions tasks in childhood (e.g., (Gooch, Thompson, Nash, Snowling, & Hulme, 2016; Kuhn, Willoughby, Vernon-Feagans, & Blair, 2016; Noble,

McCandliss, & Farah, 2007; Noble, Norman, & Farah, 2005). *If executive functions reflect the computations performed by PFC at any point in the lifespan, then these developmental relationships might reflect the efficacy with which PFC's computations are adapted to different demands across the lifespan.* This framework thus provides a novel mechanistic explanation for the relationship between delayed language acquisition and executive functions delays (e.g., (Figueras, Edwards, & Langdon, 2008; Henry, Messer, & Nash, 2012).

It is also worth emphasizing how this ecological account differs from other accounts that argue that protracted maturation of PFC may be an adaptation during early childhood. According to these alternative accounts, delayed maturation of PFC may be adaptive during early childhood in that it helps individuals in the species more efficiently learn the natural statistics of the environment, without top-down PFC control imposing constraints on this process (Chrysikou, Novick, Trueswell, & Thompson-Schill, 2011; Chrysikou, Weber, & Thompson-Schill, 2014). This account assumes a PFC protracted developmental course is accurate and assigns evolutionary value to this developmental course (Thompson-Schill, Ramscar, & Chrysikou, 2009). In contrast, we argue against protracted maturation and an evolutionary explanation for this being adaptive. We suggest instead that the PFC is instead *directly adapted* to support learning demands that are relevant to individuals in their unique ecological niche, whether it be learning and generalizing relevant environmental contingencies in infancy and early childhood or organizing and exerting control over complex action and thought in adolescence and adulthood.

Thus, rather than having a protracted developmental course, executive functions may be continually adapting to processing information relevant to developing children. Once the physical structure of the body and environmental demands on learning and behavior begin to stabilize from adolescence to early adulthood, our ecological account makes the novel prediction

that PFC may then stabilize in its development. However, as PFC's connectivity and the structure and capabilities of the physical body begin to destabilize again in ageing, PFC may again show changes and deficits in its functions in comparison to the young adult state (Anguera et al., 2013; Lee et al., 2015; Li et al., 2014; Lövdén et al., 2010).

Adaptation and Niche Construction

In this section, we expand on how the concepts of adaptation and niche construction are relevant to PFC functional development. Niche construction is an approach in evolutionary biology that stresses how organisms adapt to the environment by actively modifying it to best suit their current needs and abilities (Laland, Odling-Smee, & Feldman, 2000). Niche construction and adaptation require balancing a trade-off between sampling the environment – to estimate the current state of the environment – and specialization – to efficiently adapt to the demands of the sampled environment (Frankenhuis & Del Giudice, 2012; Frankenhuis & Panchanathan, 2011; Nepomnaschy & Flinn, 2009). This is not unlike Piaget's ideas of assimilation and accommodation driving developmental change (Piaget, 1952) or the exploration/exploitation computations discussed earlier (Koechlin, 2016).

In the context of brain development, niche construction involves adapting and changing neural pathways such that an organism selects information in line with what they most need and with what they can best process given the abilities and neural architecture available (M. H. Johnson et al., 2015). PFC may orchestrate this process of niche construction through computations that facilitate the construction of rules and norms that guide learning and behavior, as well as by exerting top-down control over activity in other neural regions to align with currently relevant rules, goals, or norms. This aligns with PFC's computation of exerting top-

down modulatory control over posterior neural regions (Braver et al., 2009; Buschman & Miller, 2007; B. T. Miller & D'Esposito, 2005; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier et al., 2005).

In turn, niche construction itself is afforded by changes in the integrity of feedforward inputs into PFC, which reflect the development of physical, perceptual, and cognitive skills and abilities that support novel methods for information sampling and interacting with the environment. Thus, PFC developmental change may be driven by adaptation to changes in information sampling, possibly via feedforward connections, and specialization of functions via its feedback connections.

The idea of adaptation involving a trade-off between sampling and specialization is captured in "dynamic optimization" models of development (Frankenhuis & Panchanathan, 2011). These models suggest that individual differences in timing and plasticity of development occur by balancing sampling and specialization. When sampling indicates that the current state of the environment has changed, PFC must then adapt to specialize for the demands of the new environmental state. Models of switching behavior in PFC have offered a similar conclusion with respect to its online role in stability versus flexibility of function. Successful adaptation to changing environments involves balancing cognitive flexibility, or the ability to adjust behavior to align with changing demands and environments, and cognitive stability, or the ability to maintain behavior in the face of distractors (Armbruster, Ueltzhöffer, Basten, & Fiebach, 2012). Switching between cognitive flexibility and cognitive stability requires organisms to monitor changes in the environment to form and maintain attentional biases towards relevant information, which is supported by dopamine-modulated updating and active maintenance in PFC

(Armbruster et al., 2012; Fallon et al., 2013; Rosa, Dickinson, Apud, Weinberger, & Elvevåg, 2010).

Neural Adaptation in Response to Sampling Changes via Feedforward Connectivity

There are a variety of causes of neural uncertainty that can drive adaptive responses. The developmental state of neural regions with feedforward connections into PFC may constrain the information available for sampling. Specialization in other regions of the brain as well as the development of new connections to PFC could modify the input that PFC processes, requiring PFC to adapt to different quantity and quality inputs (Amso & Scerif, 2015). One hallmark of early cortical development is the differentiation of various cortical areas into specialized regions (M. H. Johnson & Vecera, 1996). This process results in previously combined information processing streams specializing for particular types of information, leading to less overlap and interference between them. This may lead to apparent 'improvements' in PFC processing, since it is now computing over inputs that have a higher signal-to-noise ratio. As such, the ecological account predicts that some apparent developmental improvements in PFC processing may originate from adaptation to developmental changes in neural regions that provide feedforward input to PFC, rather than structural maturation of the PFC *per se*.

Apparent development improvements in PFC processing may additionally reflect myelination of connections between PFC and other posterior neural regions, which may influence the efficiency or strength of the input to PFC. Myelination of neuronal axons plays an important role in establishing and maintaining rapid and efficient neural communication across development (Deoni et al., 2012). During the first year of life, myelination proceeds rapidly in a posterior to anterior gradient with early sensory regions being some of the first to become myelinated (Deoni et al., 2011, 2012), likely reflecting increased experience with sensory input

(Barres & Raff, 1993; Demerens et al., 1996; Stevens, Porta, Haak, Gallo, & Fields, 2002; Wake, Lee, & Fields, 2011). Myelination may affect PFC processing in two ways. First, myelination of feedforward connections to PFC may influence PFC functional development by impacting the quality or signal-to-noise ratio of sensory input to PFC. Second, increased efficiency of interneural communication between PFC and other regions as a result of myelination may increase the quality and integrity of input to PFC, leading to apparent improvements in PFC processing due to changes in the quality of the input, rather than due to PFC maturation *per se*.

Thus far, the discussion has focused on neural-level analysis of PFC adaptation. Here we offer complementary data that suggest that changes in cognitive, behavioral, and motor abilities also alter the availability of information for maintenance, updating, and planning at the level of PFC and executive functions. This point is easily illustrated with data from developmental transitions in locomotor abilities. This body of work indicates that interactions with the environment changes for infants as they develop new or more refined locomotor skills (Karasik, Tamis-Lemonda, & Adolph, 2011). For example, when infants first learn to sit stably on their own, their hands become free to manipulate and use objects. The novel visual information that is generated by the infant's object manipulation supports the development of more advanced abilities such as object memory (Ruff, 1981), object discrimination (Soska, Adolph, & Johnson, 2010), and view-invariant object recognition (James, Jones, Smith, & Swain, 2014). Manipulating objects while sitting also allows infants to bring objects close to their eyes such that the objects dominate their visual field. This creates opportunities for further development, such as learning names of objects that were not as readily available before infants could sit stably (Pereira, Smith, & Yu, 2014; Yu & Smith, 2012). Thus, developing new motor abilities – such as learning to sit stably – creates opportunities for development by changing the information that

infants can sample and process from the world. In relation to PFC functional development, these changes require adaptation at the level of executive functions, as PFC adapts to organize and process the influx of information that was previously unavailable. In other words, the ecological account predicts that as new quality or quantity input becomes available, PFC must adapt its computations of active maintenance (Badre, 2008; D'Esposito & Postle, 2015; E. K. Miller & Cohen, 2001; O'Reilly, 2006; Rougier et al., 2005) and rapid updating (Braver & Cohen, 2000; Chatham & Badre, 2015; Chatham et al., 2014; Chiew & Braver, 2017; Cohen et al., 2002; Frank et al., 2001; O'Reilly, 2006; Rougier et al., 2005) to operate over the new input.

The ecological approach also makes the prediction that some apparent PFC and executive functions 'deficits', that are observed in a number of neurodevelopmental and learning disorders, may develop as *an adaptive response* to early disruptions in physical, perceptual, and motor abilities (M. H. Johnson et al., 2015). In other words, it predicts that early disruptions in perceptual and motor abilities will change the feedforward input to PFC and ultimately the top-down influence PFC exerts through feedback connections in response. We will return to this idea in the niche construction section below.

This focus on sampling in the ecological account has some similarity with a graded-representations account of PFC function, which suggests that PFC's efficiency is dependent on the strength of the representation it is acting on (Morton & Munakata, 2002). A graded-representations account suggests that maturation of PFC increases the strength of representations that can be actively maintained within PFC, thereby leading to improvements in executive functions. The ecological account makes a similar prediction regarding how the strength of active representations influences PFC function; however, instead of suggesting that PFC maturation *per se* increases infants' ability to maintain active representations, we suggest that

representation strength is influenced by adaptation to the internal and external environment through changes in feedforward connectivity.

Specialization and Niche Construction Through Feedback Connectivity

Simultaneously, PFC is involved in the feedback orchestration of widespread neural reorganization and specialization of posterior cortical regions. This ontogenetic mechanism aligns with a recent phylogenetic description of brain evolution, which suggests that cognitive control may have emerged from the increasing convergence of hierarchically organized input to PFC across evolutionary time (Finlay & Uchiyama, 2015). A primary feature of vertebrate brains is conservation of a hierarchical organization across the whole brain, with more rostral regions typically exerting modulatory control over more caudal regions. Across developmental time, the human brain also becomes increasingly hierarchical to accommodate the increased levels of control that become necessary as other neural regions, skills, and abilities develop (Supekar et al., 2009).

A prevalent hypothesis that has been articulated previously is that PFC may be a key player in this process of hierarchical reorganization (M. H. Johnson, 2000, 2011; M. H. Johnson et al., 2015; Thatcher, 1992). Even though functional connectivity innervates from back to front, cortical neurogenesis occurs in the opposite direction along a rostral to caudal progression across the entire brain, with frontal neurons becoming differentiated before more posterior neurons (Cahalane et al., 2012); it has been suggested that this byproduct of developmental timing may place PFC in an optimal position to facilitate the organization and development of other cortical regions (M. H. Johnson et al., 2015). In other words, the early differentiation of neurons within PFC, combined with the early formation of feedback pathways from PFC to other cortical regions (Conel, 1939; Rakic, 2002), may allow PFC to exert modulatory control over the

development and specialization of posterior neural regions from very early in development. This possibility bears similarity to work indicating that PFC exerts modulatory control over neural activity within posterior regions in adults (Gilbert & Li, 2013; E. K. Miller & Cohen, 2001).

Computational models shed light on how this bears on PFC development. In particular, a knowledge-based cascade correlation model has been used to explain how PFC might orchestrate other neural regions during cognitive tasks (Shultz, Rivest, Egri, Thvierge, & Dandurand, 2007, cited in M. H. Johnson et al., 2015). This model is built using an architecture where one central control network (such as the PFC) orchestrates and recruits previously trained networks when they are required for more complex tasks. This unique architecture helps the model to learn tasks faster and more efficiently than models without a central control network. This is because the central control network in this model can recruit other previously trained networks as units when the "skills" or "knowledge" that are represented by those networks are required for a learning task. In the context of PFC functional development, as tasks become more complex and new computational units are needed, functional changes may be observed as PFC adapts to recruit new networks to support the increased task demands (M. H. Johnson et al., 2015; Shultz et al., 2007). In other words, changes may be seen as PFC learns to select and organize appropriate neural regions for new learning problems.

Importantly, we suggest that adaptation to feedforward changes (sampling) and niche construction through feedback changes (specialization) are tightly and intrinsically coupled (Figure 1). Thus, specialization of feedback pathways may influence adaptation by biasing information sampling to align with current goals and demands for learning and behavior. This may reciprocally modify the input to PFC necessitating adaptation at the level of executive functions. In this view, PFC plays an active role in shaping its own development by biasing

sampling of inputs to align with relevant demands for learning and behavior, which is similar to prior ecological theories of development (e.g., Gibson, E. J., Pick, 2000; M. H. Johnson, 2000, 2011; M. H. Johnson et al., 2015). We suggest that PFC's functions may gradually stabilize as feedforward connections and the requirements for learning and behavior in the external environment stabilize with development.

Constraints on Adaptation and Niche Construction via Anatomical Proximity to PFC

Timing of adaptation and niche construction across ontogenetic development may be constrained by the anatomical proximity of other neural regions to the PFC. The existing maturationist explanation of ontogenetic brain development suggests that functional development is driven by structural changes in the form of grey matter volume reduction and cortical thinning (e.g., Gogtay et al., 2004; Shaw et al., 2008). Yet, these developmental progressions do not directly map to functional changes. For example, heuristics based on these developmental progressions in grey matter reduction suggested that visual cortex matures first (Gogtay et al., 2004); yet many aspects of visual processing continue to improve throughout childhood (Konrad et al., 2005; Rueda et al., 2004), which is difficult to reconcile with structural maturation accounts. Further, due to constraints in early imaging methodologies, these heuristics were based upon studies of children and adolescents, with little data from ages 4 and under when a majority of fundamental brain development and neural organization occurs.

The ecological account offers a more theoretically structured alternative for control over thoughts and actions based on the ideas discussed above, data on the timing of PFC functional connectivity, and structural proximity of domain specific regions to the PFC. The testable prediction offered by this account is that regions with the closest anatomical proximity and

shortest direct projections to the PFC are the first to become specialized for control over action and behavior (Figure 2). In other words, what we sample (and hence specialize) first is based on which regions have the shortest direct connections to the PFC. Thus, regions such as the frontal eye field (FEF) within the frontal lobe may be one of the first regions to specialize, which coincides with the relatively early emergence of oculomotor control (Canfield & Kirkham, 2001), and also relates to PFC activity during saccade planning in 4-month-old infants (Csibra et al., 2001). Similarly, short direct projections between anterior temporal lobe and PFC that support auditory processing and speech perception (Romanski & Goldman-Rakic, 2002; Scott & Johnsrude, 2003) may also specialize relatively early to support language development. This prediction aligns with evidence showing increased PFC activation in infants when hearing infant-directed but not adult-directed speech (Saito et al., 2007), forward but not backward speech (Dehaene-Lambertz et al., 2002), and when organizing words by hierarchical contexts (Werchan et al., 2016). Other areas within the frontal lobe, such as the premotor area, may begin to specialize for control over more complex motor actions soon thereafter. We predict that regions with more distant connections to the PFC, such as the parietal and occipital lobes, may have the most protracted trajectories for specialization. This prediction coincides with findings showing that functions that depend on frontoparietal and fronto-occipital connectivity, such as visual or executive attention, continue to develop into adolescence (Konrad et al., 2005; Rueda et al., 2004). Thus, when PFC functional development is examined relevant only to frontoparietal change, using a construct of executive functions that is defined relative to an adult state (Dosenbach et al., 2007), it may seem erroneously protracted.

This new pattern of functional developmental change is supported by research showing that PFC's functional connectivity broadly shifts from predominately short-range local

connections within the frontal lobe to more long-range connections between the PFC and more distal regions across development (Fair et al., 2007; Gao et al., 2011; Supekar et al., 2009). These shifts are thought to partially reflect changes from segregation to integration with respect to regional functional connectivity, and relate to changes in executive functions. For example, shifts in functional connectivity from short-range intracortical connections within the PFC to long-range frontoparietal connections correlate with improvements in relational reasoning abilities from childhood to adolescence (Wendelken et al., 2016). Similarly, developmental improvements in inhibitory control during an antisaccade task correlate with increases in long-range functional connections between the PFC and subcortical and parietal regions (Hwang et al., 2010). Additionally, other studies show that developmental improvements in cognitive control of eye movements in antisaccade tasks coincide with shifts in functional connectivity from short-range local connections between the dlPFC and FEF in children, to more long-range connections between the dlPFC and visual association cortex in adolescents and adults (Simmonds, 2015). Further, this framework aligns with work indicating anterior to posterior shifts in functional activity for many brain networks (M. H. Johnson & De Haan, 2015). However, note that the magnitude of shifts in short- to long-range functional connectivity in prior reports may have been exaggerated due to age-related differences in motion artifacts (see Power et al., 2012).

Neural Adaptation and Niche Construction Occur in Response to Shifts in Allostatic Load

Finally, it is necessary to elucidate the hypothesized physiological mechanisms that drive the described PFC changes. In terms of PFC development, the ecological model predicts that increased quantity or quality of multimodal input to the PFC may push the system out of

allostasis, which is the maintenance of stable functioning through physiological adaptations (McEwen & Wingfield, 2003). Allostatic overload occurs when energy demand exceeds current supply, which activates adaptive physiological responses to restore a net positive energy balance (McEwen & Wingfield, 2003). Short term fluctuations in function could arise from changes in allostatic load that occur in response to mismatches between the current input to the PFC and the input that the PFC was previously specialized to process at an earlier state. We suggest that this may activate physiological adaptations to increase processing efficiency, such as by promoting experience-dependent myelination in PFC's connections with other neural regions (e.g., Markham & Greenough, 2004), by promoting accelerated development of functional connectivity through mediators such as cortisol (e.g., Callaghan & Richardson, 2012, 2014) or excitatory and inhibitory neurotransmitters such as glutamate and GABA (e.g., Ghisleni et al., 2015; Hensch et al., 1998), or by promoting synaptic pruning within PFC (e.g., Selemon, 2013). These physiological responses may help PFC adapt to process increased or more complex input, helping restore allostasis or a net positive energy balance with time.

Evidence for this idea comes from the data discussed above showing that early life adversity can modulate PFC development as an adaptive response to adverse early environments. For instance, early life stress in the form of maternal deprivation may create an earlier need for PFC to modulate amygdala reactivity due to the lack of external modulation by a maternal caregiver, which may be signaled by increased cortisol levels. This may then push PFC out of allostasis at an earlier stage in development than typical. Accelerated maturation of vmPFC-amygdala circuitry may then occur as an adaptive response to maternal deprivation, via increased levels of cortisol (Callaghan & Richardson, 2012, 2014), thereby helping vmPFC modulate amygdala reactivity in place of an external caregiver (Callaghan & Richardson, 2011; Callaghan

& Tottenham, 2016; Gee, Gabard-Durnam, et al., 2013). Similarly, increases in environmental enrichment in high relative to low socioeconomic environments may also result in faster physiological adaptations due to an abundance of positive acute stressors.

Based on these ideas, the ecological account of PFC development thus makes the counterintuitive prediction that rapid change in one or more developing domains, e.g. vision, language, motor etc., as happens in infancy through childhood, may 'stress' the system sufficiently to result in apparent transient deficits in PFC's functions. These may appear over moments or days, until PFC has time to adapt to managing the rapid influx of information. This transient period of disorganized behavior would theoretically be followed by rapid improvements in executive functions and the newly developed skill. These apparent deficits may take the form of perseverative errors, poor emotion regulation when a child is overwhelmed with information, or they may be a seeming loss of the ability to perform tasks that children had previously mastered.

This pattern is often evident in task performance and on short time-scales. For example, infants make the A-not-B error will eventually adapt to search in the correct location provided a certain number of B trials (Diamond, 1985). Similar findings are seen in deductive reasoning tasks that require participants to relate or integrate a set of variables to verify an abstract rule. When the complexity of information (e.g., number of variables that must be integrated) is parametrically increased, short-term decrements in performance are observed, as well as increased activity within cognitive control networks as PFC adapts to process the more complex information (Cocchi et al., 2014; Shokri-Kojori, Motes, Rypma, & Krawczyk, 2012). Thus, what have been traditionally interpreted as PFC immaturity may reflect processes of adaptation to novel demands on the organism.

Predictions of an Ecological Model of PFC Development

Some of the key principles and testable predictions of the ecological account of PFC functional development are briefly summarized here. First, the ecological account posits that PFC performs the same computations across the lifespan, but that these computations are adapted for the learning demands and challenges relevant to an individual in their unique ecological niche. Our view does not imply that current standardized tasks used to measure executive functions are not valid. Rather, it suggests that these tasks may only capture executive functions during certain windows of development. Thus, the ecological account may offer a more complete view of PFC and executive functions development by considering how PFC functional development may reflect adaptation of these computations for different purposes relevant to the individual in their ecological niche across their lifespan. For instance, these computations may be adapted for language learning or locomotor development in young infants, or for learning social skills or engaging in pretend play in preschoolers. As the environment begins to require children to exercise abilities inherent in more classic measures of executive functions, such as when children begin formal schooling, these computations may then be adapted for behavioral regulation relevant in this new ecological niche.

Thus, this leads to the first prediction that organized patterns of PFC involvement should be seen in infants, children and adults, but in different tasks, contexts, and activities that are ecologically appropriate to the individual at their point in development. Recent advances in imaging technologies, such as NIRS, combined with advancements in machine learning classification techniques for neural data, may allow for direct tests of this prediction by recording and classifying PFC activation in naturalistic contexts across different age groups and in different tasks. For instance, the ecological account predicts that similar patterns of PFC

activation may be seen during object exploration or while learning to grasp in infants, during pretend play in children, or while driving a car in adults. Moreover, if executive functions reflect the computations performed by PFC at any point in the lifespan, then a second prediction is that developmental continuity might reflect the efficacy with which PFC's computations are adapted to novel demands across the lifespan. This explanation may apply to the observed relationship between delayed language acquisition and executive functions delays (e.g., Figueras et al., 2008; Henry et al., 2012), as well as early language proficiency and executive functions development in children from different SES homes (Noble et al., 2007, 2005). Importantly, the ecological account thus suggests that tasks that are ecologically appropriate for an individual at each point in their development should be used to measure the efficiency of executive functions computations across the lifespan.

Second, the ecological account also posits and predicts that PFC functional development will be reciprocally influenced by changes in feedforward (for adaptation) and feedback connectivity (niche construction). A direct prediction is that early disruptions in perceptual and motor abilities will change the feedforward input to PFC and ultimately the top-down influence PFC exerts through feedback connections in response. While PFC's profuse connectivity, protracted plasticity, and dependence on other neural systems makes it highly adaptable, it also makes PFC highly vulnerable to atypical developmental trajectories due to deviations in species expected environments. Small differences in the experienced environments of typically compared to atypically developing infants and children could create cascading effects that impact PFC development and adaptation of the brain to the environment more generally (Karmiloff-Smith, 2009). This can take the form of altered PFC function from subtle differences in early visual or auditory processing (Baruth, Casanova, Sears, & Sokhadze, 2010), in congenital

blindness or deafness (Figueras et al., 2008; Tadic et al., 2009), or from disrupted thalamic organization, such as in individuals with schizophrenia (Cheng et al., 2015).

Finally, the ecological model, in that it relies on the neurobiology of stress-based adaptation, also makes the prediction that changes in the quality or quantity of input to the PFC will push the system out of allostasis. This leads to the prediction that increased input – whether due to the acquisition of new skills and knowledge over ontogenetic time or due to increased input in a specific task over short time scales – would lead to temporary 'deficits' in PFC function. We noted earlier that these 'deficits' are often observable on short time-scales when the task requires adaptation to a novel demand (e.g., Cocchi et al., 2014; Diamond, 1985; Shokri-Kojori et al., 2012). Future work can test whether deficits may be accompanied by short-term increases in cortisol, reflecting the neurobiological stress response, which may temporarily interfere with the efficiency of neural firing (Arnsten, 2009).

Thus, an ecological model that considers PFC functional development as a process of adaptation to the environment is particularly relevant for understanding mechanisms of typical development, as well as the multiple pathways that might lead to deviations from typical developmental trajectories. A wide and diverse array of neurodevelopmental disorders, including ADHD, autism spectrum disorder, schizophrenia, depression, obsessive compulsive disorder, and anxiety disorders, share a commonality in that they are all frequently associated with atypical PFC functioning. Reconsidering PFC functional development as a process of adaptation and niche construction, and not maturation, provides novel mechanistic insights into the multiple pathways that may lead to these deviations from typical development.

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