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REVIEW ARTICLE

The Role and Sources of Individual Differences in Critical-Analytic Thinking: a Capsule Overview

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Abstract Critical-analytic thinking is typically conceived as a meta-construct that arises at the junction of a problem state (i.e., a situation that requires analysis that challenges previous assumptions) and an individual (i.e., an entity with the capacity to exercise critical-analytic thinking). With regard to the latter, there is a substantial body of research focusing on developmental and educational prerequisites for critical-analytic thinking. A less studied aspect of critical-analytic thinking pertains to individual differences, particularly in the set of foundational or componential cognitive skills that embody this construct. The bottom line here is whether, all else being equal (i.e., the same situation and the same developmental/educational stage), there is variation in whether, when, and how people think critically/analytically. We argue that there is unequivocal evidence for both the existence and importance of individual differences in critical-analytic thinking. This review focuses on theoretical and empirical evidence, identifying the cognitive processes that serve as the sources of these individual differences and capturing these processes' differential contributions to both the critical and analytic components of this construct.

Keywords Critical analytic thinking · Neurocognitive mechanisms · Fluid reasoning · Executive function · Genetic factors · Environmental factors

For better or for worse, people differ. The number of dimensions on which they differ is endless; one such dimension is the way they think. Here we are interested in surveying both the presentation and sources of individual differences in critical-analytic thinking. Whereas

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both professionals and laypeople have a sense about what critical-analytic thinking is, the construct is ill-defined and this has been a source of constant debate. Although the debate has swung from a notion of the skills and capacities necessary for critical-analytic thinking to one of disposition toward truth-seeking (Burbules and Berk 1999), the former provides for analysis and the measurement a set of requisite cognitive processes to unpack, with respect to variability in performance in situations and on tasks requiring critical-analytic thinking. As critical-analytic thinking occurs so naturally for some, yet so painfully arduous for others, it is likely that this type of thinking embeds a "vast variety of cognitive skills" (Lipman 1988) that must be orchestrated together, both developmentally as a capacity, and concurrently, as an act. Thus, both the skills and the way they are combined generate a source of individual differences. The intent of this review is to provide an understanding of how variations in several core neurocognitive systems, stemming from genetic and environmental sources, lead to differences in the capacity for and competency in critical-analytic thinking.

Critical-analytic thinking involves the ability to overcome one's own biases by decontextualizing an argument (the critical component) and evaluating it rationally (the analytic component), rather than looking to prior beliefs and attitudes (Macpherson and Stanovich 2007). For the purposes of this review, we view critical-analytic thinking, narrowly defined,[1] as a set of generalized "fluid" abilities that rely on domain-specific "crystallized" knowledge and are exercised under stringent cognitive control in order to evaluate the validity of arguments, particularly those which stand in contrast with one's own beliefs or biases. The literature is rife with constructs to describe the inherent capacities of critical-analytic thinking. However, while many focus on those skills most ontologically related to critical-analytic thinking in general, we must take into account the realm of lower level skills and abilities from which it emerges. For instance, the formation of reasoning bias emerges from an automatic system, which stands in contrast to the rational, controlled cognitive system that is needed to override bias (Tversky and Kahneman 1981). This latter system is referred to as executive function (EF), which is just one of several multifaceted systems that enable us to think critically. Therefore, accounting for the sources of variability at the EF level, we will, at least partially, apportion the variability in critical-analytic thinking.

In our framework, we argue that the foundations of critical-analytic thinking are rooted in a core set of neurocognitive capacities, that is, a set of cognitive processes that have unique underlying neural components. This thesis is in accord with a "neuroconstructivist" account of cognitive development in which the dynamic interactions of genes, the brain, and environment over time bring about the multitude of cognitive functions (Karmiloff-Smith 2009). From this perspective, the higher-order cognitive capacity of critical-analytic thinking is a product of the successful development of multiple component systems (see Fig. 1). Although not an exhaustive set, this review focuses on three prospective components that have been identified as components of higher-order problem solving and reasoning:

1. The *fluid reasoning* system encompasses the analytic component of critical-analytic thinking in terms of reasoning inductively and deductively and relating distal concepts for the purpose of drawing valid inferences (Ferrer et al. 2009).
2. The *executive function* system regulates our cognitive processes enabling us to consider multiple pieces of information and inhibit biased responses which are critical to higher-order reasoning, problem solving, and decision making (Diamond 2013; Kane and Engle 2002).

[1] Critical-analytic thinking also involves weighing the reliability of evidence, engaging values, making judgments, and other cognitive and motivational components that are not going to be discussed here.

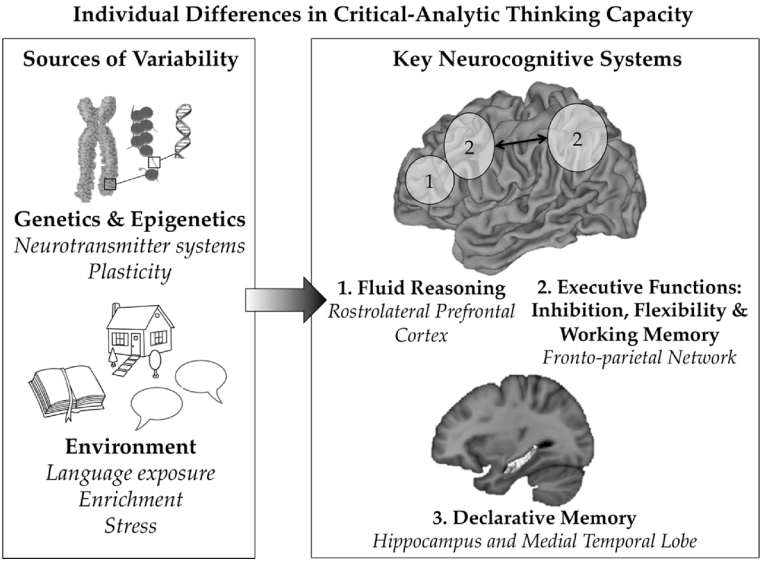

Fig. 1 A consideration of how sources of variability, genetics and epigenetics, as well as environment conditions are related to individual variability within key neurocognitive systems (i.e., fluid reasoning, declarative memory, and executive functions) associated with critical-analytic thinking capacity

3. The *declarative memory* (also referred to as declarative knowledge) system enables the storage and retrieval of prior (crystalized) knowledge including abstract semantic concepts and episodic memories that are critical to reasoning and decision making processes (see Thomas et al. 2008).

It is important to note that, just as the brain is a dynamic system that functions as an interactive network, these constructs (i.e., fluid reasoning, EF, and declarative memory) are highly interactive and interdependent as well. Although we refer to these processes as distinctive elements of cognition, the extant literature has shown that they are not truly independent from one another. In addition to these three central componential systems, other systems play a supportive role. These systems include motivation and reward, which we will discuss further, and language.[2]

In sum, our search for individual differences in the ability to think critically and analytically will rest on the interactions of fluid reasoning, EF, and declarative memory, and the biological systems that undergird these capacities. To provide an overview of this composition, we will highlight the core elements and facets of fluid reasoning, EF, and declarative memory and describe how each interacts with the others to play a role in the process of critical-analytic thinking. We will then provide some insight into the neural systems involved in these capacities. Following the discussion of neural systems, we will introduce evidence for how genetic variation leads to differences in capacities of fluid reasoning, EF, and declarative knowledge. Then, we will discuss the role of environment and experience in these capacities and how enhancements to critical-analytic thinking may occur through intervention. Finally,

[2] The neurocognitive system for language is complex and dynamic as there are subcomponents of production and reception for phonology, syntax, and semantics. Many structures overlap with those described including frontal and temporal-parietal regions predominantly in the left hemisphere.

we will conclude by talking about the developmental emergence of critical-analytic thinking, and how it is constrained by the genome and modified by the environment.

Neurocognitive Systems Involved in Critical-Analytic Thinking

Fluid Reasoning

Fluid reasoning (which is referred to in the class of fluid abilities) is the ability to solve novel problems and to operate logically enabling the induction of new knowledge and the mapping of familiar knowledge to novel situations (Cattell 1963, 1971). The ability to reason fluidly about a novel problem or concept arguably entails analogical mapping to a known problem, concept, or task-set (Hofstadter 2001). From our perspective, relational reasoning is a type of fluid reasoning that enables the induction of new knowledge or the mapping of familiar knowledge to novel situations through the relational mapping between sets of features of the source and target information (Holyoak 2012). The measurement of fluid abilities has typically been achieved using matrix reasoning tasks such as Raven's Progressive Matrices (Raven et al. 1998) or relational reasoning tasks (Bunge and Wright 2007). Fluid abilities are globally correlated with a variety of academic skills including reading comprehension and mathematics (Snow et al. 1984). A neurobiological model of fluid reasoning has been proposed (Bunge and Wright 2007) involving a network of brain regions: the left fronto-parietal system (including the left dorsolateral prefrontal cortex, DLPFC, and inferior parietal lobule, IPL) as well as the anterior pole of the frontal cortex known as the rostrolateral prefrontal cortex (RLPFC), which is thought to play a role in setting and executing complex abstract rules (Badre et al. 2005; Koechlin and Summerfield 2007). These regions are ubiquitously activated across verbal and nonverbal analogy tasks (Wright et al. 2007) and relational reasoning tasks (Wendelken and Bunge 2010), as well as matrix reasoning tasks (Crone et al. 2009), yet the functions they subserve may be distinct.

Whereas the entire fronto-parietal network, including RLPFC, shows increased activation for complex relative to simple matrix reasoning tasks (Kroger et al. 2002; Lee et al. 2006), only the RLPFC increases in activity as a function of the number of dimensions required in the matrix reasoning tasks (Crone et al. 2009; Kroger et al. 2002). It has been suggested (Bunge and Wright 2007) that while the fronto-parietal network and hippocampal complex are involved in the maintenance and retrieval of information in fluid reasoning tasks, it is the RLPFC that is responsible for abstracting across source and target information (Green et al. 2006). On the other hand, all types of reasoning tasks may not engage the same brain regions. In a meta-analysis of 28 studies of reasoning (Prado et al. 2011), it was found that the studies consistently implicated the left fronto-parietal network as subserving reasoning, along with the basal ganglia, a reward system structure, which has been shown to be engaged in many types of executive function and learning tasks. Variation in other cortical regions between studies was explained by the utilization of differential types of reasoning (categorical, causal, and relational/fluid) in the experimental tasks.

Individual differences in fluid reasoning in adults are correlated with the structure and function of the RLPFC, and the fronto-parietal network more broadly (Gray et al. 2003; Lee et al. 2006). As we will discuss, the fronto-parietal network also supports EF, which is consonant with the finding that across individuals, fluid reasoning and EF share substantial variance (Kane and Engle 2002). Over the course of development, individual differences in fluid reasoning are related to the trajectory of brain maturation (Shaw et al. 2008). A slower developmental trajectory of cortical thickness, particularly in superior frontal cortex, has been

linked with superior intelligence. Based on these findings, it has been suggested that superior reasoning may arise from delayed cortical thinning (reductions in gray matter that naturally occur with age see Gogtay et al., 2004) in RLPFC and the fronto-parietal network (Shaw et al. 2008). In short, variations in developmental trajectories of brain structures in general result in differential ability to problem solve and reason, fundamentally impacting the capacity for critical-analytic thinking.

Executive Functions

Executive functioning (EF) is a multidimensional construct of the generalized (also in the class of fluid) abilities (Diamond 2013) necessarily for effortful imputing of top-down control over mental operations. Thus, EF is an umbrella term used to describe an assemblage of cognitive control processes (Miyake et al. 2000) that allow humans to organize behavior, i.e., formulate goals and attain them in a flexible manner. There are multiple accounts of what processes constitute EF. Here we will focus on three which appear in most, if not all, theoretical accounts of EF[3]: (a) the capacity to inhibit prepotent or automatic thoughts and to monitor and resolve conflicting information (inhibition); (b) the capacity to hold, monitor, and update information in mind (working memory); and (c) the ability to flexibly deploy different sets of rules or contextual constraints (cognitive flexibility). They are often measured using different behavioral tasks and are thought to be subserved by separable neural networks.

Inhibition Inhibitory control is central to the regulation of one's thoughts, emotions, and impulses. As we discussed, critical-analytic thinking arguably entails effortful control and a meta-awareness of one's own biases and beliefs (Stanovich and Stanovich 2010). These metacognitive skills are considered synonymous with the monitoring and inhibitory functions of EF (Fernandez-Duque et al. 2000). It is this aspect of EF that enables attention to be driven by our intentions or drawn by the salient information in the environment (Posner and DiGirolamo 1998). That we can monitor our own thoughts and biases and consider alternative choices, particularly potential errors in our thoughts and action, places EF at the forefront of our ability to think and act rationally. Failures or difficulties in applying our inhibitory control lead to impulsivity and, on a clinical scale, to Attention Deficit Hyperactivity Disorders (Aron et al. 2007). Developmentally, inhibitory control does not peak until well into adolescence, often in the later stages (Casey et al. 2005; Luna et al. 2004), which is consistent with the development of critical-analytic thinking skills.

Working Memory The role of working memory (WM) to maintain a limited set of information, to manipulate and update that information, and eventually store it away underlies the set of fluid abilities and makes reasoning and crystallization possible (Engle et al. 1999; Sprenger et al. 2013). WM (as measured by complex span tasks) accounts for the largest amount of variance in fluid intelligence tasks (Engle and Kane 2004) such as the Raven's Progressive Matrices (RPM). Clearly, the relation between fluid reasoning and WM suggests that these abilities share underlying cognitive processes. Whereas many consider WM to be similar to the short term memory store, it is clear that the construct of WM, which is measured in tasks requiring the maintenance of information in the face of distraction or other cognitive load, loads far more strongly on fluid reasoning tasks compared to simple short term memory tasks (Engle and Kane 2004). Thus, the underlying relation between WM and fluid reasoning lies

[3] These accounts can refer to these processes using different labels; nevertheless, these are essentially the same processes.

not in the capacity to hold a number of memoranda in mind, but to do so in lieu of interfering information. In short, the EF of cognitive control and inhibition are an inherent part of WM (at least as it is measured). It was presumed that both WM and fluid reasoning were fairly stable constructs and not malleable beyond the peak of maturation (Kane and Engle 2002), that is, they are fixed capacities. However, a spate of cognitive training studies has put doubt to these notions.

Cognitive Flexibility The third aspect of EF is the capacity to flexibly switch between different cognitive states or perspectives (Diamond 2013). For instance, cognitive flexibility provides us with the ability to change points of view spatially such as in Piaget's "Three Mountains" task or to switch between sets of rules as in the Wisconsin Card Sorting Task (Milner 1964), in which one must sort cards based on a changing set of rules. This ability to switch perspective is thought to underlie our capacity to understand the thoughts and perspectives of others, or exercise theory of mind, which is necessary for critical-analytic thinking. In concert with inhibitory control and WM, flexibility allows us to hold in mind a set of ideas that are foreign to our own way of thinking, suspend our own biases and beliefs, and update our model or perspective by taking on a different viewpoint. Thus, flexibility is essential to critical-analytic thinking.

Overlapping Neural Correlates of EF The cortical circuits associated with EF have been generally referred to as the Cognitive Control Network (CCN), which consists of several separable subsystems including the fronto-parietal network bilaterally (D'Esposito 2007; McNab et al. 2008; Olesen et al. 2004; Smith and Jonides 1998), the cingulo-opercular network, and the cerebellum and striatal system (Dosenbach et al. 2007; McNab and Klingberg 2008).

In the case of WM, the fronto-parietal circuit has long been assumed to be the locus of WM (Goldman and Rosvold 1970; Goldman-Rakic 1995; Jacobsen 1935). Functional neuroimaging studies in humans have consistently cited its role in the maintenance of information over time (Curtis and D'Esposito 2003; D'Esposito 2007). Both the inferior parietal lobe (IPL) and the dorsolateral PFC (DLPFC) exhibit sensitivity to load in WM tasks (Leung et al. 2002; Rypma and D'Esposito 1999; Vogel and Machizawa 2004). However, there has been some debate as to the role of the PFC in maintaining as opposed to monitoring/controlling the stored contents of WM (D'Esposito 2007). Recent frameworks suggest that the anterior portions of the PFC in conjunction with anterior cingulate (ACC) and insula regions form a cingulo-opercular network that is responsible for the control of cognition and task-demands (Dosenbach et al. 2007). As opposed to the fronto-parietal network, which is argued to maintain stimulus information, the cingulo-opercular network maintains task goals/rules, monitors for errors, and provides feedback (Dosenbach et al. 2007).

It is thought that response inhibition is substantiated via the recruitment of the fronto-striatal circuits (Aron et al. 2007), a network of connections between the prefrontal cortex and the striatal system (the brain's reward network). This system constitutes a bidirectional system of initiation of behavior and impulse control with regions in the medial and lateral prefrontal cortex. Deficits in impulse control are associated with low activity in the right PFC (Aron et al. 2004) and diminished PFC connections with the striatal system (Aron et al. 2007), suggesting the importance of the PFC's role in mediating and stopping impulsive behaviors. Moreover, deficits in the striatal system due to conditions like Parkinson's disease restrict functioning of the PFC and result in poor performance on inhibitory tasks (Middleton and Strick 2001). In sum, individual differences in inhibitory control are connected to the fronto-striatal system. Such variations are associated with weaker reasoning skills specifically on tasks in which biases in beliefs are prevalent (Handley et al. 2004).

It is also important to mention the relevance of motivation both for the manifestation and the development of critical-analytic thinking as well as the fundamental fluid skills necessary for reasoning and problem solving. In general, the brain's reward circuitry's enhancements—related to EF in general and WM in particular—are thought to engage the fronto-parietal networks, which are presumed to be driven by dopaminergic reward structures in the striatal system including the basal ganglia and caudate, as well as thalamic nuclei (Diamond 2007; Levy et al. 1997). The circuitry from the striatal system to the PFC exhibits the same trajectory described throughout this section where the peak of development is tied to the maturation of the white matter fiber connections connecting the PFC to the rest of cortex. Thus, the ability to sustain attention and inhibit interfering information is driven, in part, by the brain's reward system suggesting that a disposition toward higher-order cognition begins with motivation.

Declarative Memory

Historically, fluid reasoning and executive functions (fluid abilities) were defined by what they are not, which is a practiced set of routines or crystallized knowledge (Cattell 1963). However, fluid and crystallized abilities are generally well correlated (Engle et al. 1999), and they have been hypothesized to be interdependent: Fluid abilities enable the acquisition of crystallized knowledge (Cattell 1971), and crystallized knowledge supports fluid reasoning by allowing analogical mapping from a known concept or task-set to a novel problem (Hofstadter 2001; Holyoak 2012). As stated in the Common Ground Claims (see Alexander this issue), prior knowledge is a fundamental component to critical-analytic thinking, and the neural systems that enable the access and retrieval of that knowledge are central to explaining variations in this higher-order capacity.

From our perspective, the construct of crystallized knowledge is consistent with the notions of long-term memory, specifically the processes of declarative memory, which encompass semantic knowledge and episodic experiences. Nondeclarative or proceduralized knowledge also falls under the umbrella of crystallized knowledge; however, for the purposes of our discussion of critical-analytic thinking, we will focus on the declarative memory that is invoked in top-down reasoning.[4] This declarative memory system provides explicit, verbalizable knowledge of events and concepts that have been learned (Jacoby et al. 2010; Schacter and Addis 2007). As we will detail, the storage of crystallized or declarative knowledge is likely to occur in a domain-specific fashion in the cortex such that information is organized according to modality.

A wealth of neurological evidence suggests that the hippocampus and the surrounding region of the medial temporal lobes (MTLs) are responsible for the consolidation of declarative memory (Squire 2009). These findings come from both animal models and, more famously, patients such as HM, who had medial temporal lobe lesions (Scoville and Milner 1957) and displayed an inability to acquire and retain new declarative information. Damage to these structures leaves prior semantic and autobiographical memory intact and impairs recent memory (Squire and Bayley 2007), suggesting that the role of the medial temporal region provides a transient architecture for indexing representations stored across other modalities in the cortex.

More importantly, the circuitry extending from the medial temporal lobes is responsible not only for the recollection of old memories but also for the projection of future or unexperienced events (Schacter and Addis 2007). That is, the role of the MTL region is the reconstruction of information from other cortical regions responsible for processing the color, smells, and

[4] Certain aspects of non-declarative memory may play a role in critical thinking, particularly when it comes to biases and emotional valence that are attached to concepts or beliefs. That is, the non-declarative memory system is akin to the automatic conditioned response system that sets biases and beliefs and is tied to our reward system.

sounds of our experiences. As such, the creation of dreams and simulations of future events occur because the MTL can uniquely construct events using knowledge of the past. Specific evidence that the hippocampus and MTL are involved in fluid reasoning has been shown in tasks of relational reasoning and making transitive inferences (Greene et al. 2006; Wendelken and Bunge 2010). Thus, this system would be ideally involved in a fluid process, such as critical-analytic thinking, in which one must create or construct new knowledge from episodes and concepts stored in memory.

Summary

To summarize, critical-analytic thinking is a higher-order cognitive skill that emerges from a set of lower-level interacting neurocognitive systems: fluid reasoning, EF, and crystalized knowledge/memory. Sources of variation in critical-analytic thinking between individuals putatively arise from deviations in the functioning of these psychological processes and their underlying cortical structures. Such variations are seen both at a specific neurocognitive level, that is, damage to a specific structure/function. However, from a neuroconstructivist perspective, dynamic variation in the neurocognitive system impacts cognition on a broader scale. For example, variations in the connective fibers between the frontal and parietal cortex are correlated with processing speed and fluid reasoning (Ferrer et al. 2013), as well as WM (Nagy et al. 2004). Moreover, variations in the connective tissue between the prefrontal and the hippocampal region are predictive of memory retrieval processing (Wendelken et al. 2014). Thus, it is not simply specific cortical structures that enable the higher-order processes of critical-analytic thinking, it is the global neurocognitive architecture working together as a well-tuned orchestra to enable information to be maintained, comprehended, and evaluated, while prior assumptions and biases are inhibited. As we will discuss next, the sources of neurocognitive variation lie in the interaction of our genetic code and the environment surrounding the individual.

Sources of Individual Differences in Neurocognitive Systems *Involved in Critical-Analytic Thinking*

Having outlined the componential view of critical-analytic thinking and summarized the neurocognitive systems substantiating these components, we will now outline two major clusters of variation that are essential for understanding where intraindividual and interindividual differences in critical-analytic thinking originate. Following the established tradition, we review the corresponding literature focusing on the genome- and the experience- (i.e., environment) based sources of this variation (see Fig. 1). Yet, as stressed in the "Conclusion" section, we are completely aware of the fact that this separation is artificial and exercised, as an illustration of constructive reductionism, to appreciate the variety of sources of causal influences on the formation and manifestation of critical-analytic thinking, rather than to delineate how these factors really act or co-act.

The Genome-Based Sources of Individual Differences in Critical-Analytic Thinking

Our survey of the literature has not located a single genetically informed study of critical-analytic thinking. Yet, the componential approach to critical-analytic thinking that we have adapted in this review permits expanding the parameters of the literature review to include

genetically informed studies of fluid reasoning, EF, and declarative knowledge. Such studies are in abundance and the corresponding literature is substantial. Given the parameters of this review, here we highlight only selected aspects of this literature. These highlights are structured so that we first summarize the observations pertaining to the heritability and familiality of fluid reasoning, EF, and crystallized knowledge and, second, those observations pertaining to studies of the variability in the genome structure and function as it is associated with the variability in the components of critical-analytic thinking.

Heritability and Familiality Heritability and familiality studies of cognitive abilities (including fluid reasoning and crystallized knowledge) have consistently fashioned substantial estimates of heritability, that is, the percent of the variance in the abilities which can be attributed to variation in the genome (Deary et al. 2009). Although there is a certain amount of fluctuation in these estimates for various indicators of fluid reasoning and crystallized knowledge, with the latter being more heritable than the former (Kan et al. 2013), the field has converged, after a number of meta-analyses, on the estimate of 0.5 for general cognitive ability, which is a higher-order index derived from both fluid and crystallized capacities (Plomin et al. 2013a). Although there is not unanimous enthusiasm regarding the value of heritability and familiality studies to the field (Charney 2012; Nisbett et al. 2012; Richardson 2013), there is still a massive related research effort to further differentiate and stratify the obtained estimates throughout the life span (e.g., Haworth et al. 2010; van Soelen et al. 2011) and in the context of different moderators (e.g., Molenaar et al. 2013). Yet, the epicenter of the related research has moved away from the question of whether the genome influences cognitive abilities to the question of how it actually exerts this influence.

As WM is closely related to general cognitive ability (Engle et al. 1999), it is, perhaps, of no surprise that the heritability estimates of WM are also significant and substantial (Ando et al. 2001; Karlsgodt et al. 2010; Luciano et al. 2001). Moreover, it has been shown that the phenotypic correlation between general cognitive ability and WM is almost entirely (95.5 %) attributed to shared genetic variance (Goldberg et al. 2013).

Although the number of quantitative genetic studies of EF is substantially smaller compared to the studies of fluid reasoning, crystallized knowledge, or general cognitive ability, there are some and they have generated a range of heritability estimates, indicating from negligible to substantial genetic influences (Anokhin et al. 2003; Friedman et al. 2008; Groot et al. 2004; Kuntsi et al. 2006; Polderman et al. 2006; Stins et al. 2005). Although the discussion of the different reasons for such a variation in heritability estimates (from methodological to conceptual and reflective of the psychological texture of a studied indicator) is highly important, such a discussion is outside the scope of this article.

Structural Variation in the Genome It might appear that such a consistent picture pointing to high heritability estimates for the cognitive processes featured in this review should pave a straight road to the discovery of the specific locations in the genome, whose structural variation underlines these high heritability estimates. Led by this general assumption, researchers have applied two general approaches—ahypothetical and hypothesis-driven, searching for these structural variation.

The first, ahypothetical approach, is represented, largely, by two methodologies. The first, known as genomewide association studies (GWASs), is aimed at gauging associations between a trait and a large number (now—up to 5,000,000) of DNA markers (typically single-nucleotide polymorphisms, SNPs) distributed throughout the genome. A number of GWASs of general cognitive ability have been carried out (Davies et al. 2011; Davis et al. 2010), although their results did not meet the expectations generated by the heritability estimates. The

great gap between the amount of variance explained by the DNA markers identified so far in GWASs (e.g., the largest effect identified accounted for less than .005 of the variance) and the stated heritability estimates is referred to as the "missing heritability problem" (Maher 2008). Grappling with this problem, the field has considered multiple possible reasons, such as the incorrect (overestimated) heritability coefficients (Albert and Haley 2013), the need to consider epistatic effects (Zuk et al. 2012), the possible missed contribution of rare DNA variants (Cirulli and Goldstein 2010), and the "incorrectness" of the very GWAS approach to complex traits (Visscher et al. 2012).

It is the latter consideration that resulted in the development of the approach called genomewide complex-trait analysis (GCTA), which can be used to estimate genetic variance accounted for by all the SNPs that have been genotyped in any sample, comprised of either relatives or single individuals (Yang et al. 2011a, b). The special appeal of the GCTA is that it can utilize the data from completed GWASs, assuming that the resulting sample size is large and the number of SNPs is large. The main difference between GWAs and GCTAs is that the latter do not identify specific DNA markers associated with traits. What it does is capitalize on chance similarity across hundreds of thousands of SNPs to predict phenotypic similarity for each pair of individuals (as compared within and across pairs) in a sample of related and/or unrelated people. A limitation of GCTA is that it estimates only additive genetic effects, i.e., only those effects that are caused by the independent effects of alleles, which add up in their effect on a trait; nonadditive, i.e., interactive, genetic effects are not considered. Regardless of this shortcoming, the utilization of GCTA in studies of cognitive ability has resulted in much less discrepant heritability estimates—0.40 and 0.51 for crystallized and fluid intelligence, respectively (Davies et al. 2011, n=3,500)—and a range of estimates for general cognitive ability—0.47 (Chabris et al. 2012, n=10,000), 0.48 (Deary et al. 2012, n=2,000), 0.66 (Plomin et al. 2013b, n=7,308), and 0.22–0.46 (Benyamin et al. 2014, n=17,989).

Thus, at this point, based on the results of the GWA and GCTA studies, it is rather clear that the genome's contribution to individual differences in critical-analytic thinking is quite important. The leading hypothesis today is that many genes (and, correspondingly, polymorphisms in these genes) contribute to the emergence of individual differences in critical-analytic thinking, but the field has still only a very approximate understanding of what these genes and the contributing mechanisms are.

Yet another approach to understanding the importance of the structural variation in the genome with regard to its association with the variation in componential processes of critical-analytic thinking is the approach known as research of so-called gene candidates. In such studies, hypotheses formulated a priori with regard to what genes might be relevant for what particular cognitive process and the structural variation in these particular genes are investigated. There is a considerable controversy with regard to the pluses and minuses of GWA and GCTA approaches as compared to candidate gene studies for understanding the genetic bases of complex behavior (Abbott 2008). From our point of view, they are complementary in generating both important data and insight into the mechanisms by which the genome substantiates the diversity found in human behavior in general and critical-analytic thinking and related processes in particular.

There are multiple ways in which such hypotheses might be generated. First, as indicated, the field has currently identified and characterized specific neurocognitive systems that localize the componential processes of critical-analytic thinking in specific areas of the brain. Thus, what researchers do is investigate the transcriptional mechanisms involved in human brain development in those specific areas and identify the genes that are expressed in the relevant areas of the brain at developmental windows that are critical for the emergence of critical-analytic thinking. Currently, there are public resources available to support the

generation of such hypotheses. One such resource is the BrainSpan atlas (http://www.brainspan.org/), which provides both genome (exome array) and transcriptome (RNA sequencing) data profiling for up to 16 cortical and subcortical structures across the full course of human brain development.

Second, there is a well-developed line of investigation in which the attempts to find genes associated with cognitive abilities emerge directly from investigating the variation in specific genes known to be involved in brain function (Payton 2009). However reasonable this approach is to generating hypotheses, it has been difficult to replicate reported associations between such candidate genes and general cognitive ability (Chabris et al. 2012). One of the points of criticism for these attempts is that the phenotype of general cognitive ability is not sufficiently differentiated and that researchers should look for associations with much more specific componential processes that are known to be predictive of the complex behavior in question, e.g., critical-analytic thinking (Miyake and Friedman 2012) and functionally associated with specific genes (Barnes et al. 2011; Need et al. 2009).

The central assumption underlying this approach is that there are certain proteins that are either known or hypothesized to be relevant either to the anatomy or to the function of a particular brain region (e.g., the brain region just described). Although all humans have these proteins, they are observed to differ among people on a variety of parameters (e.g., precise amino acid content, spatial folding, and thermostability), which, in turn, are related to differences in the structure of the genes coding for these proteins. Thus, alternative versions of the genes of interest (so-called genetic or allelic, if only a specific locus of the genes is considered, variants) could be associated with qualitative and quantitative differences in EF. In other words, it appears that the morphology of the anatomical regions central to the manifestation of critical-analytic thinking and the structures to which these regions connect are highly influenced genetically (Winterer and Goldman 2003). Similarly, it has been demonstrated that physiological parameters of the function of these regions (e.g., different parameters of neurotransmission) are also highly controlled by the genome. Therefore, as specific roles of the genome's components (e.g., genes) become better understood, it is possible to identify, a priori, gene candidates, whose allelic variation may be relevant to fluid reasoning, EF, and declarative knowledge.

Here, due to the space constraints, we provide only selective illustrations of this approach. For example, although there is still both a lot of data that need to be systematized and interpreted comprehensively and a high need for ongoing research, there are certain points of consensus with regard to specific anatomical and functional networks that are central to particular facets (or assembled processes) of EF (Barnes et al. 2011). Thus, in our illustrations, we will focus on EF. Again, anatomically, the task is to superimpose the brain map of the neurocognitive maps discussed above, on the data from the BrainSpan atlas or any other comparable databases. Functionally, the task is to consider the structural variability in the genes coding for major neurotransmitters such as dopamine, serotonin, and noradrenaline, which have particular patterns of performance in the neurocognitive systems of interest.

To illustrate, consider the research on the role of dopaminergic genes in substantiating the cognitive function of inhibition. Specifically, dopamine beta hydroxylase (DβH) is a catalytic enzyme that aids in the conversion of dopamine to noradrenaline within noradrenergic neurons (Levin et al. 1960). It is coded by the DβH gene (*DBH*), which has a number of functional polymorphisms that determine plasma DβH activity. Specifically, the functional 1021 cytosine (C)/thymine (T) single-nucleotide polymorphism (SNP) in the 5′ flanking region of the *DBH* gene has been shown to account for 35–52 % of plasma DβH activity, with the T allele being associated with lower DβH activity and, correspondingly, decreased dopamine-to-noradrenaline conversion (Zabetian et al. 2001). It has been demonstrated that having more

copies of the T allele is associated with poorer performance (i.e., more errors of commission) on a sustained attention test (Greene et al. 2009). Indicators of sustained attention have also been associated with various alleles in dopamine (*DAT1/SLC6A3*, *DRD2*, *DRD4*) and serotonin (*5-HTT/SLC6A4*) genes. Indicators of response inhibition have also thought to be associated with the variation in noradrenaline and dopamine signaling systems, but specifics of these associations are somewhat different, pointing, in particular, to the variation in the *DRD4* and *DAT1/SLC6A3* (Congdon et al. 2008) and *COMT* (Congdon et al. 2009) genes. Although serotonin neuromodulation does not appear to be pivotal to response inhibition, there are studies connecting variation in this facet of EF and variation in the genes involved in the turnover of serotonin in the corresponding brain circuits, e.g., the variation in the *TPH2* gene (Baehne et al. 2009).

Functional Variation in the Genome Although the structural variation in the genome appears to be of importance to understanding of sources of individual differences in critical-analytic thinking, it is also highly important to consider the so-called functional variation (i.e., the variation that arises from differences in transcripts and, subsequently, proteins that are generated from the same sequence of DNA). As of today, these mechanisms are substantially less studied in the context of understanding individual differences in cognition. Thus, here we only mention them briefly, in anticipation that research in the immediate future will generate data for our appreciation of their importance.

The distance between the structure of DNA and the resulting protein can be gauged in the genome in a number of different ways (Richardson 2013). Specifically, different gene transcripts (messenger RNAs, mRNAs) can be generated by a mechanism that is known as exon reshuffling, in which sections of the gene are unavailable for transcription in different combinations (i.e., alternatively spliced). One result of exon reshuffling is the diversity of resulting proteins that can be produced from the same gene, with potentially widely different functions. It is estimated that at least 74 % of human gene products can be alternatively spliced (Johnson et al. 2003). Of note is that mRNAs themselves have additional functions besides being protein templates. These functions are diverse and include, among others, the capacity to change the efficiency of transcription and translation in response to external stimulation (Kong and Lasko 2012).

Moreover, in addition to mRNAs, there are now multiple other RNA transcripts (so-called noncoding RNAs) that are present in abundance, not to be translated into proteins, but having multiple regulatory functions, such as developmental control, activation of hormone receptors, modulation of promoters, and silencing genes, acting as co-activators of transcription (Ørom et al. 2010). Although the specifics of these functions are just being understood, the gist of their role is to connect the internal and external cellular environments (Kim et al. 2009; Krol et al. 2010; Valadkhan and Nilsen 2010).

Finally, the last few years of research into the etiology of complex human behavior have been marked by increasing interest in studies of epigenetics—a set of processes that are able to change both the spatial configuration and the chemistry of the DNA sequence, determining the specifics of the utilization of the genome in response to environmental influences (Meaney 2010). In particular, these mechanisms are hypothesized to be responsive to a wide variety of external stimuli from in utero exposure to maternal stress, malnourishment, and environmental toxicants (Bollati and Baccarelli 2010) to experiences that modulate the ability of neurons to synthesize and respond to neurotransmitters or to remodel themselves by modifying dendrites or myelinating axons (Knudsen 2004; Sng and Meaney 2009). In other words, epigenetics play a critical role in neuroplasticity. In the next section, we will consider how experiences and exposures "get under the skin" to shape brain structure and function at every level, from synapse to system.

Experiential Sources of Individual Differences in Critical-Analytic Thinking

The most straightforward answer to the question of why we observe individual differences in critical-analytic thinking is that individuals differ in their experience with critical-analytic thinking. Practice with critical-analytic thinking over the course of one's education likely improves critical-analytic thinking capacity. On one level, this can be understood by considering the related biological mechanisms: Repeated activation of a brain system leads to the strengthening of that system, through changes to neuron structure and function (Sagi et al. 2012; Zatorre et al. 2012). However, as far as we know, there is no brain system devoted to critical-analytic thinking, per se. Rather, as we have described, critical-analytic thinking arises out of the cooperation of a number of brain systems. These brain systems have different developmental trajectories, suggesting that they are differentially sensitive to the environment over the course of development (Knudsen 2004; Raznahan et al. 2011; Sowell et al. 2004b). Further, different brain systems are sensitive to different kinds of experiences (Fox et al. 2010). We will discuss both broad, unsolicited experiences that may shape critical-analytic thinking over the life span (socioeconomic status, stress, and language exposure), and specific, targeted experiences that may boost the cognitive skills that support critical-analytic thinking (cognitive training).

Socioeconomic Status

Low socioeconomic status (SES) has long been associated with lower academic achievement and attainment (Bradley and Corwyn 2002; McLoyd 1998). Low SES might be related to weakened critical-analytic thinking skills, but to our knowledge, this has not been directly tested. Low SES has been linked to worse performance on measures of componential cognitive skills, including EF (Crook and Evans 2014; Mezzacappa 2004) and language (Noble et al. 2005, 2007). Brain structure differences have also been observed between low and middle SES children and adults, with individuals from lower SES backgrounds demonstrating less cortical gray matter and smaller hippocampi (Jednoróg et al. 2012; Lawson et al. 2013; Luby et al. 2012; Noble et al. 2012). There is also evidence that the language network is less left-lateralized in lower SES children (Raizada et al. 2008).

The causal mechanisms that give rise to SES-related differences in cognition and brain structure are unknown. Low SES is associated with risk for a constellation of negative environmental exposures, such as prenatal drugs, lead, and malnutrition (Adler and Stewart 2010), and negative psychosocial experiences. SES-related differences in the psychosocial environment have been hypothesized to fall along two axes: threat/safety and enrichment/deprivation (Hackman and Farah 2009; Noble et al. 2005).

Stress

Experiencing threat, or the lack of safety, triggers a cascade of biological changes known as the stress response. Frequently described as "flight or fight," the stress response leads to the release of cortisol and adrenaline, which trigger the release of energy stores and a cessation of long-term physiological projects, such as immune responsiveness, growth, and reproduction (McEwen 2008). Moderate stress can be beneficial, making a person more resilient in the long term (Lyons et al. 2010). High levels of stress can be tolerable in the presence of "stress buffers" such as social support and a sense of control over the stress (Chen et al. 2011; Luby et al. 2012; Malecki and Demaray 2006). Unfortunately, chronic, unbuffered, uncontrollable stress (i.e., "toxic stress") can be devastating for brain development (Shonkoff 2012). Low

SES is associated with more stressful life events, more perceived stress, and fewer stress buffers (Baum et al. 1999).

The brain shows an uneven sensitivity to stress based on the distribution of receptors for cortisol. The hippocampus and prefrontal cortex are two of the regions that are most negatively impacted by stress (Joëls et al. 2006; McEwen 2004; Sánchez et al. 2000). Stress-related changes in hippocampal development and function make it more difficult to learn new information. Changes in prefrontal development are thought to impact fluid reasoning and EF. Because the prefrontal cortex develops slowly (Shaw et al. 2008; Sowell et al. 2004a, b), it may be vulnerable to stress well into adulthood.

Language Exposure and Bilingualism

Children from low SES backgrounds, on average, experience dramatically less language. They spend less time reading with an adult, even prior to kindergarten (Adams 1990). Hart and Risley (2003) showed that children from low SES backgrounds have heard on average 30 million fewer words by the age of three than children from higher SES families. They demonstrated that exposure to child-directed speech early in life strongly predicted vocabulary at age three, and academic outcomes through the third grade. Differences in early language exposure are thought to be related to differences in the structure and function of language-sensitive cortical regions observed later in childhood (Noble et al. 2012).

It is not only the case that too little language hinders development, but also that more language exposure, especially exposure to multiple languages, can enhance neurocognitive development (Costa and Sebastián-Gallés 2014). Acquiring multiple languages places strong demands on EF because it involves constantly monitoring the speaker (parent or teacher), the context (classroom or home), and the intention. Children who learn multiple languages need to flexibly switch from one language to another on demand, gradually developing the capacity to select and monitor between competing linguistic representations, which entails the engagement of cognitive control in the maintenance and daily use of the different languages and the inhibition of the interfering information that may arise between the multiple languages or their appropriate contexts. A multilingual environment might therefore shape the development of EF.

The evidence for a "bilingual advantage" in EF is growing (Bialystok et al. 2009). Bilingual speakers have been shown to outperform monolingual speakers on a series of domain-general cognitive control tasks, such as the card sorting task involving dimensional change, the Simon task, and the classic Stroop task involving misleading information. The potential root of this advantage may be in superior inhibition skills (Bialystok et al. 2004; Kovács and Mehler 2009; Martin-Rhee and Bialystok 2008) or in the general ability to monitor for conflict (Costa et al. 2009). Bilinguals exhibit differential neural activation particularly in the bilateral prefrontal cortex, ACC, and striatum (Petitto and Dunbar 2009; Rodríguez-Pujadas et al. 2014). However, there has been much debate over whether these findings are specific to language or domain-general (Hilchey and Klein 2011). The impact of bilingualism on critical-analytic thinking is unknown.

Cognitive Training

Because there is evidence that particular kinds of experiences promote optimal cognitive development, there is reason to be hopeful that it is possible to boost critical-analytic thinking through targeted interventions. One approach is to train underlying componential cognitive skills. A useful analogy is to consider training to run faster. In addition to practicing running, an athlete would lift weights to become stronger, and stretch to improve flexibility. The best

way to improve critical-analytic thinking might be to not only practice the skill holistically, but also to hone its parts.

Fluid Reasoning Training Very little work has been done to train fluid reasoning directly. One study found that playing games involving relational integration and planning led to improved reasoning in children from low SES backgrounds (Mackey et al. 2011). Another found that preschoolers can improve their reasoning skills through practicing computerized tasks involving pattern recognition (Bergman Nutley et al. 2011). Older adults who participated in reasoning training maintain their improved reasoning skills for at least 10 years, and scored better on overall health and quality of life measures than a control group (Rebok et al. 2014). Fluid reasoning training, in the form of preparing for a reasoning-intensive standardized test, has been linked to increased functional connectivity between the RLPFC and the rest of the fronto-parietal network (Mackey et al. 2013), as well as changes in structural connectivity (Mackey et al. 2012).

EF Training A number of programs have been shown to broadly improve EF (Diamond 2012). Computerized games that target EF have been shown to increase EF (Goldin et al. 2014; Rueda et al. 2005), transfer to fluid reasoning (Karbach and Kray 2009), change brain activity (Rueda et al. 2005), and improve academic outcomes (Goldin et al. 2014). In the classroom, a preschool curriculum based on Vygotskian principles, Tools of the Mind, improves performance on all components of EF (Diamond et al. 2007). Tools of the Mind involves simple instructional strategies such has having children take turns and make plans for imaginative play. Mindfulness meditation, a practice that involves prolonged focused attention, improves EF (Holzel et al. 2011; Jha et al. 2010; Y. Y. Tang et al. 2007) and performance on real-world academic measures, such as the Graduate Record Examination (GRE) (Mrazek et al. 2013). Mindfulness meditation training has robust effects on the brain, including increased fronto-parietal structural and functional connectivity (Jang et al. 2011; Tang et al. 2012; Xue et al. 2012).

WM Training WM training deserves special consideration, separate from the other EFs, because it has been by far the most common target of cognitive training (Melby-Lervåg and Hulme 2013; Morrison and Chein 2011). It has been consistently demonstrated that practicing WM tasks leads to large, robust WM improvements. WM training has been associated with changes in the function and structure of the fronto-parietal network (Buschkuehl et al. 2012; Dahlin et al. 2008). Training has been associated with changes in brain activation during WM tasks (Dahlin et al. 2008; Olesen et al. 2004; Westerberg and Klingberg 2007), changes in the organization of white matter connections between frontal and parietal regions (Takeuchi et al. 2010), and changes in cortical thickness (Takeuchi et al. 2011). Further, WM training changes the dopamine system, including the density of dopamine receptors in the cortex (McNab et al. 2009) and dopamine release in the striatum (Bäckman et al. 2011).

Transfer of Cognitive Training to Academic Skills

The debate surrounding cognitive training is focused on whether training *transfers* to untrained cognitive and academic skills. A handful of studies have shown that WM training transfers to gains in fluid reasoning (Jaeggi et al. 2008, 2011); however, these findings have not replicated well (Harrison et al. 2013; Shipstead et al. 2010; Thompson et al. 2013). WM training was initially found to transfer to reading comprehension in adults (Chein and Morrison 2010; Holmes et al. 2009; Loosli et al. 2012), but these effects have not replicated in either adults

(Thompson et al. 2013) or children (Dunning et al. 2013). Inconsistencies in study design, participant population, and training paradigms make it difficult to resolve the question of whether cognitive training transfers to untrained cognitive skills and academic abilities. What tends to be the case is that the skills that are most improved most closely resemble the conditions of training (Sprenger et al. 2013) even when there are a multitude of training tasks.

Central to this review, the question is whether we expect cognitive training to support improvements in critical-analytic thinking. Because the results surrounding the existing cognitive training tools are so mixed, it is critical that we work to develop new, more effective approaches to boosting cognitive skills. At the same time, we need to acknowledge that critical-analytic thinking is not simply the sum of the underlying cognitive skills that we have discussed here. It involves additional processes and abilities. The optimal method to improve critical-analytic thinking may involve both training the underlying skills and teaching it holistically. Developing better ways to train and teach critical-analytic thinking will involve collaborations between scientists and teachers, the people who have the most experience with instilling critical-analytic thinking skills.

Conclusion

In this brief overview, we have attempted to offer a theoretical perspective of the hierarchical structure of neurobiological systems that substantiate critical-analytic thinking and discuss, although briefly, sources of individual differences in these systems, that, in turn, explain individual differences in critical-analytic thinking. The set of Common Ground Claims (see Alexander, this volume) acknowledges the capacity of an individual, both person- and development-based, to engage in and exercise critical-analytic thinking. Here we dissect this notion of capacity by recognizing the importance of the individual's neurobiological faculty (i.e., neurocognitive systems, substantiating critical-analytic thinking) as they emerge through reciprocal co-action of the genome-based biological endowment and multiple experiences throughout the life span. We assume that the "rise" of cognitive complexity as manifested in critical-analytic thinking is substantiated by the human genome and can be understood, most efficiently, transactionally (Li 2012). In this paradigm, the genome substantiates but does not determine the emergence and constant modification of neurocognitive systems that are embedded in all aspects of human functioning (e.g., motor, sensory, perceptual, cognitive, and moral) and all types of contexts (e.g., cellular, environmental, task, situation, social, and cultural).

Educational psychologists are faced with the question of whether neurobiological evidence is of any use to educators (Bruer 1997; Willingham 2009) and whether it offers hope and insight into the way we learn (Goswami 2009; McCandliss 2010). The pedagogical approaches employed are often determined by the implicit attitudes of educators (not necessarily informed by modern science) regarding how nature or nurture accounts for the differential development of our cognitive capacities. As representatives of an applied science, educational psychologists are the engineers of instructional structures and their advances are fostered by a greater depth of understanding of the role that biology, environment, and pedagogy have on the individual. Although maintaining the appreciation of the holistic nature of critical-analytic thinking, our review, we hope, pointed out not only the sources of individual differences in critical-analytic thinking, but also possible targets for intervention—educational, clinical, and experiential.

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