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Invited Review

Heritability and the evolution of cognitive traits

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A critical question in the study of the evolution of cognition and the brain concerns the extent to which variation in cognitive processes and associated neural mechanisms is adaptive and shaped by natural selection. In order to be available to selection, cognitive traits and their neural architecture must show heritable variation within a population, yet heritability of cognitive and neural traits is not often investigated in the field of behavioral ecology. In this commentary, we outline existing research pertaining to the relative influences of genes and environment in cognitive and underlying neural trait variation, as well as what is known of their heritable genetic architecture by focusing on several cognitive traits that have received much attention in behavioral ecology. It is important to demonstrate that cognitive traits can respond to selection, and we advocate for an increased emphasis on investigating trait heritability for enhancing our understanding of the ecological, genetic and neurobiological mechanisms that have shaped interspecific and intraspecific variation in cognitive traits.

Key words: **adaptation, brain, cognition, evolution, heritability, natural selection.**

INTRODUCTION

The evolution of cognition and the brain draws attention from multiple biological disciplines including animal behavior, evolution, ecology, neuroscience, and cognitive science (Burgess 2008; Van Overwalle 2009; Pravosudov and Roth 2013; Woodgate et al. 2014). Recently, there has been an increased focus on the role of natural selection in shaping cognitive abilities and brain morphology, as well as on causes and consequences of individual variation in cognition within populations (Rowe and Healy 2014). Such focus has been especially pronounced in the field of behavioral ecology, and has coincided with an increased interest in the evolution of mechanisms underlying behavior, including physiology, neurobiology and genetics. In the case of cognition, connecting phenotypic variation in cognitive traits to specific neural mechanisms has been particularly fruitful in advancing our knowledge of how the environment might affect the evolution of such traits and the brain (e.g., Sherry 2006; Pravosudov and Roth 2013).

Behavioral ecology as a field is concerned with understanding whether and how behavioral traits are adapted to specific environments, and is focused on behavioral adaptations arising via the process of natural selection. Most recently, many behavioral ecologists have undertaken the challenge to quantify individual variation in cognitive traits, defined here as brain and neural processing characteristics allowing for the acquisition, retention, and use of information (sensu Dukas 2004; Shettleworth 2010). This is most often associated with an attempt to understand whether and how natural selection can act on these traits to generate adaptive phenotypic divergence (Cole et al. 2012; Hopkins et al. 2014; Quinn et al. 2014; Rowe and Healy 2014; Thornton et al. 2014). Natural selection acting on specific cognitive traits should produce changes in associated brain regions resulting in the evolution of both cognitive traits and their neural architecture. For example, selection on spatial memory associated with food caching has been hypothesized to produce changes in the size and structure of the hippocampus, the primary brain region involved in spatial memory in food-caching species (e.g., Sherry 2006). More importantly, neural mechanisms may trade-off with or constrain the evolution of cognition, as any evolutionary changes in cognition must be accompanied by some associated changes in either brain architecture or physiology. For these reasons, documenting simultaneous changes in both cognitive traits and brain morphology is an important task for behavioral ecologists interested in the evolution of cognition. Yet, in spite of many advances in understanding of how the environment shapes variation in cognition and the brain, direct evidence for the role of natural selection in driving the evolution of cognitive traits and their neural mechanisms remains elusive.

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Natural selection can result in adaptive changes when specific traits are 1) variable, 2) heritable, and 3) when variation in these heritable traits is associated with fitness consequences (Darwin 1871). Heritability estimates act as a measure of the relative additive genetic effects in generating a population range of phenotypes (Falconer and Mackay 1996). In other words, heritability estimates provide a measure of the relative contribution of genetic and environmental factors in generating phenotypic variation. As both cognition and the brain are known to be plastic, and plasticity in general acts as a buffer against environmental variation potentially minimizing and slowing down evolutionary processes (West-Eberhard 1989; Agrawal 2001), understanding the relative contributions of plasticity and heritability of specific cognitive and neural traits is of paramount importance. In traits under differential selection, heritable variation is necessary for evolutionary divergence, both at a population level and during the speciation process, whereas plasticity serves as a buffer for such divergence. Hence, heritability estimates are a necessary component in understanding the mechanisms allowing animals to adapt to changing conditions. In other words, we cannot know the fitness value of cognitive and neural traits without also knowing the extent to which these traits will be inherited. Similarly, full understanding of plasticity cannot be achieved without measure of heritability, as plasticity and heritability represent 2 sides of the same coin. Yet, heritability estimates are largely missing from studies of adaptive cognitive variation in nonmodel systems (Thornton and Lukas 2012).

Recently, there has been a push toward investigating and identifying the genetic architecture of cognitive and neural traits (e.g., Rogers et al. 2008; Deary et al. 2009; Forstmeier et al. 2009; Knowles et al. 2014; Pravosudov et al. 2013; Benyamin et al. 2014; Soria-Carrasco et al. 2014). Identifying genetic variation underlying variation in cognition and the brain would indeed aid our understanding of evolutionary processes shaping cognitive variation. However, due to the myriad difficulties in identifying genes associated with specific cognitive traits based on both currently available technology and the inherent complexity of the genetic architecture underlying cognitive traits (see Soria-Carrasco et al. 2014), we argue that a parallel effort devoted to measuring trait heritability would bring our field further toward understanding the adaptive nature and evolution of these traits than would a continued focus on their specific genetic architecture. Heritability estimates can provide tenable measure of the ability of traits to respond to selection.

In this commentary, we focus on several major cognitive traits that have received much attention in the field of behavioral ecology. Our purpose is not to provide exhaustive information on heritability of all cognitive traits, but rather to illustrate the importance of studying heritability by using several well-studied traits as exemplars. We provide a brief review connecting specific cognitive and neural traits with what is known about heritability and genetic structure of these, and we discuss the role of heritability estimates in advancing our understanding of cognitive trait evolution.

GENERAL COGNITION AND THE BRAIN

Cognition can be defined as the neuronal processes concerned with the acquisition, retention and use of information (Dukas 2004). Because cognitive processes determine behavioral decisions, they can give rise to behavioral variation, through which natural selection can act on a given population as long as such variation is heritable. The evolution of large brain size and associated increases in cognitive capacity has been of great interest in the cognitive and evolutionary sciences since Darwin (1871) proposed a connection between brain size and cognitive function (Rushton and Ankney 2009; Sol 2009). Because the brain is a major information-processing organ, increases in brain size as a whole are often (though somewhat controversially; see Healy and Rowe 2007 for discussion) assumed to have evolved as a result of selection for greater cognitive capacity. This has given rise to several adaptive hypotheses explaining the evolution of overall brain size including the social brain hypothesis (Dunbar 1998), the cognitive buffer hypothesis (Sol 2009) and other environmental/ecological hypotheses (Clutton-Brock and Harvey 1980; Mace et al. 1980; Potts 1998).

Even among those researchers who agree with the adaptive hypotheses for the evolution of the brain and cognition, there is debate whether the brain evolves as a whole or through a mosaic process (Barton and Harvey 2000; Dunbar and Shultz 2007). Similarly, there is ongoing debate whether cognitive traits evolve together (Deaner et al. 2006; Lefebvre and Sol 2008) as a cognitive complex, referred to as general cognitive ability or Spearman's *g* (sensu Rushton and Ankney 2009; Reader et al. 2011), or whether specific cognitive traits evolve independently alongside specific brain regions controlling these traits (e.g., spatial memory and the hippocampus, Sherry 2006; Pravosudov and Roth 2013). Various cognitive traits are positively correlated in both human and nonhuman animals (Reader and Laland 2002; Deaner et al. 2006; Rushton and Ankney 2009), and general cognitive ability does seem somewhat related to brain size (Rushton and Ankney 2009) in humans, rats, and primates (Plomin 2001; Posthuma et al. 2002; Reader et al. 2011), though some argue that total neuron number is a much better predictor of cognitive abilities then is the overall brain size (Herculano-Houzel 2011).

In the last few decades, the field of behavioral ecology has seen a resurgence of research associating the total brain with some aspects of species behavior, cognition, life-history, ecology, and the environment. Many of these studies use a comparative phylogenetic approach to associate brain size with traits such as innovation (Reader and Laland 2002; Lefebvre et al. 2004; Reader et al. 2011), social learning (Reader et al. 2011), tool use (Reader and Laland 2002; Lefebvre et al. 2004; Reader et al. 2011), extractive foraging (Reader et al. 2011), deception (Reader et al. 2011), sociality (Dunbar 1998; Dunbar and Shultz 2007), and diet (Clutton-Brock and Harvey 1980; Dunbar and Shultz 2007). Although these traits may confer fitness advantages, only a handful of studies have directly connected brain size or general cognition with fitness. For example, Sol et al. (2007) used a comparative approach to show that avian species with big brains survive better as adults. However, in guppies (*Poecilia reticulata)*, larger brain size resulting from artificial selection experiments was associated with reduced gut size and, most importantly, with reduced offspring production (Kotrschal et al. 2013). These data would seem to indicate conflicting tradeoffs between brain size and fitness, and serve as evidence that further study is needed to quantify the fitness value of a large brain and greater general cognitive ability, in order to understand their evolutionary implications, which remain elusive in part because we do not know the extent to which cognitive traits are inherited, and/or inherited together across generations.

Heritability of general cognition

General cognitive ability (*g*) is one of the best-studied cognitive traits, and a diversity of evidence from humans under a variety of conditions indicates a strong heritable component. *g* represents a suite of correlated cognitive abilities and is generally evaluated

using a battery of cognitive tasks. Heritability of *g* is studied extensively in human populations (reviewed in Plomin and Spinath 2002; Toga and Thompson 2005; Deary et al. 2009; Table 1), and increases with age (Deary et al. 2009, but see Finkel et al. 1998), suggesting that the relative effect of environment on *g* decreases across developmental time. A theory for human intelligence that is based strictly on environmental influence would predict the opposite trend (Toga and Thompson 2005). Finally, a recent study reported high heritability of both intelligence and other behavioral traits contributing to individual variation in educational achievement in humans (Krapohl et al. 2014). If intelligence is also heritable in other animal species, this knowledge would allow for the formulation of more specific hypotheses explaining how animals cope with various types of environmental change, for example in response to climate change.

In nonhuman animals, heritability of *g* has been examined across only a very small, phylogenetically disparate suite of species, including rodents and primates (e.g., mice *Mus musculus*; chimpanzees *Pan trogolodytes*; Table 1). A recent meta-analysis (Reader et al. 2011) revealed evidence for independent evolution of *g* across diverse primate lineages, but data pertaining to heritability of *g* per se are largely lacking in nonhuman lineages.

Heritability of whole brain size

Heritability of brain morphology itself is important in understanding the evolution of cognition due to known associations between various brain structures and cognitive abilities, and known tradeoffs between neural tissue and other physiological and behavioral traits (e.g., expensive tissue hypothesis; Kuzawa et al. 2014). Heritability of brain morphology has been studied extensively through the use of magnetic resonance imaging (MRI) in vivo, particularly for human twin subjects (reviewed in Kremen et al. 2010; 2013; see Table 1). Entire brain volume, for example, is highly heritable in humans (Table 1), as are measures of volume for many cortical regions-of-interest (see references in Kremen et al. 2010; Table 1).

To date, there is relatively little information available pertaining to heritability of brain structures in nonhuman animals, though some data are available for nonhuman primates (Table 1). Among these, heritability estimates for brain volume tend to be quite high, however, we note that measures of neural tissue volume alone as a measure of brain morphology should be taken with some caution. Volume can change as a result of changes in neuron soma size, neuron number, or glia, and measures are highly subjective based on tissue handling procedures, which may vary between labs and preparations (Pravosudov and Roth 2013). Likewise, due to the difficulty in measuring cognitive and/or neural trait heritability in wild populations, heritability estimates largely derive from single replicate studies of small captive research colonies (e.g., Cheverud et al. 1990; Rogers et al. 2007; Fears et al. 2009). As such, interpretation should be limited due to lack of replication, and existing heritability estimates likely represent ceiling values. These caveats further highlight the need for specific attention to measuring heritability in wild populations, which might be actively under selection. Measuring brain size heritability in wild populations represents a significant challenge to the field, as the logistics of measuring traits in the wild and tracking individuals through reproduction can be daunting. There is, however, technology available which may be adapted to facilitate such research, including but not limited to radio frequency identification tracking technology, which can be used to assess various spatial and problem-solving abilities in wild animals, and have been used in this way for birds (e.g., Morand-Ferron and Quinn 2015; Morand-Ferron et al. 2015; Aplin et al. 2015). Such methods can be adapted to track survivorship and reproductive success, particularly for year-round resident species (sensu Johnson et al. 2013). Using MRI would allow repeatable measurements of brain volume in wild animals and generating pedigrees of individuals with known brain volume would provide a basis for estimating its heritability.

Despite the associated challenges, it is essential that scientists interested in understanding variation in cognitive traits and the fitness consequences of such variation undertake to move their studies outside of a laboratory setting. Most studies of cognitive traits in a laboratory setting lack an explicit link between abstract cognitive trait assays and a naturally occurring behavior (Morand-Ferron and Quinn 2015; Morand-Ferron et al. 2015). Such a link is essential in interpreting data linking cognitive traits scores to fitness—indeed, even fewer studies posit specific selective implications of variation in such traits, and least of all with careful consideration of myriad noncognitive behavioral components of cognitive trait assays (Rowe and Healy 2014; Morand-Ferron and Quinn 2015; Morand-Ferron et al. 2015). Future research should explicitly test predictions associated with cognitive trait variation in a wild population, for example, by testing for fitness consequences of variation in problem solving or spatial learning tasks among species and populations with varying ecology. Likewise, field-based studies provide data essential for an intraspecific comparative approach, which would yield important advances in our understanding of cognitive and neural trait plasticity, and their adaptive value (Eifert et al. 2015). Without knowing the heritability of these traits, any hypothesis about the evolution of brain size would remain speculative.

SPATIAL MEMORY AND THE HIPPOCAMPUS

Spatial memory has received much attention in behavioral ecology because of its association with food caching behavior (Krebs et al. 1989; Sherry et al. 1989; Sherry 2006; Pravosudov and Roth 2013). Scatter-hoarding animals store numerous food items across many different locations during the fall, and then retrieve their caches as food becomes unavailable throughout the winter. Several facets of spatial memory, including size (i.e., the number of locations remembered), longevity (i.e., the duration that locations can be remembered), and accuracy (i.e., the relative number of correct locations remembered), appear necessary for the retrieval of food caches (e.g., Sherry 2006; Pravosudov and Roth 2013). Because success of cache retrieval may affect winter survival and hence fitness, food caching has been hypothesized to impose selection pressure on spatial memory and the hippocampus, a brain region known to be involved in spatial memory (Sherry 2006; Pravosudov and Roth 2013). Although the hippocampus is not the only brain region involved in spatial memory processing, the evidence for its involvement is overwhelming, and so it is a likely target of selection, providing a neural mechanism for spatial memory (e.g., Sherry 2006).

The adaptive specialization hypothesis predicts that food-caching species have better spatial memory and a larger hippocampus relative to noncaching species, as a result of natural selection (Sherry 2006). Multispecies comparisons have provided somewhat mixed results, but on the whole have supported the adaptive specialization hypothesis by showing that food-caching species generally have a larger hippocampus and are better performers in at least some spatial memory tasks (see Pravosudov and Roth 2013 for a complete review). Similarly, this hypothesis can explain variation within

Table 1

Summary of known heritability estimates for the brain and cognitive traits discussed in text.

Taxon	Common name	Trait	Source	Heritability Est.	Notes	Reference
Cognitive traits						
Homo sapiens	Human	General cognitive ability *g*		0.50	All ages	Bouchard and McGue (1981)
Homo sapiens	Human	General cognitive ability *g*		0.30	Children	McCartney et al. (1990)
Homo sapiens	Human	General cognitive ability *g*		0.48	Verbal, Age 5	Hoekstra et al. (2007)
Homo sapiens	Human	General cognitive ability *g*		0.64	Nonverbal, Age 5	Hoekstra et al. (2007)
Homo sapiens	Human	General cognitive ability *g*		0.26	Age 5	Bartels et al. (2002)
Homo sapiens	Human	General cognitive ability *g*		0.39	Age 7	Bartels et al. (2002)
Homo sapiens	Human	General cognitive ability *g*		0.41	Age 9, Significant linear increase with age	Haworth et al. (2010)
Homo sapiens	Human	General cognitive ability *g*		0.54	Age 10	Bartels et al. (2002)
Homo sapiens	Human	General cognitive ability *g*		0.64	Age 12	Bartels et al. (2002)
Homo sapiens	Human	General cognitive ability *g*		0.55	Adolescents, significant linear increase with age	Haworth et al. (2010)
Homo sapiens	Human	General cognitive ability *g*		0.83	Full scale IQ, Ages 7–17	Edmonds et al. (2008)
Homo sapiens	Human	General cognitive ability *g*		0.85	Verbal IQ, Ages 7–18	Edmonds et al. (2008)
Homo sapiens	Human	General cognitive ability *g*		0.73	Performance IQ, Ages 7–19	Edmonds et al. (2008)
Homo sapiens	Human	General cognitive ability *g*		0.45	Inspection time, Ages 7–20	Edmonds et al. (2008)
Homo sapiens	Human	General cognitive ability *g*		0.66	Age 17, significant linear increase with age	Haworth et al. (2010)
Homo sapiens	Human	General cognitive ability *g*		0.84	Verbal, Age 18	Hoekstra et al. (2007)
Homo sapiens	Human	General cognitive ability *g*		0.74	Nonverbal, Age 18	Hoekstra et al. (2007)
Homo sapiens	Human	General cognitive ability *g*		0.81	Ages 27–65	Finkel et al. (1995)
Homo sapiens	Human	General cognitive ability *g*		0.77	Adult twins reared apart	Johnson et al. (2007)
Homo sapiens	Human	General cognitive ability *g*		0.64–0.74	Adult twins reared apart	Bouchard et al. (1990)
Homo sapiens	Human	General cognitive ability *g*		0.86	Adult twins, correlated with gray-matter volume	Posthuma et al. (2002)
Homo sapiens	Human	General cognitive ability *g*		0.62	Ages >80	McClearn et al. (1997)
Homo sapiens	Human	General cognitive ability *g*		0.53	Ages >80	McClearn et al. (1997)
Homo sapiens	Human	General cognitive ability *g*		0.54	Ages 65–85	Finkel et al. (1995)
Homo sapiens	Human	Nine psychological domains associated with GCSE scores		0.35–0.58		Krapohl et al. (2014)
Homo sapiens	Human	Academic achievement		0.58	As General Certificate of Secondary Education (GCSE) score	Shakeshaft et al. (2013)
Homo sapiens	Human	Working memory		0.67	Adult twins	Posthuma et al. (2002)
Homo sapiens	Human	Verbal ability		0.55	Ages >80, twins	McClearn et al. (1997)
Homo sapiens	Human	Spatial ability		0.32	Ages >80, twins	McClearn et al. (1997)
Homo sapiens	Human	Speed processing		0.62	Ages >80, twins	McClearn et al. 1997
Homo sapiens	Human	Memory		0.52	Ages >80, twins	McClearn et al. (1997)
Homo sapiens	Human	Face-specific recognition ability		0.39		Zhu et al. (2010)
Pan trogolodytes	Chimpanzee	General intelligence *g*	Captive reared and Wild-caught	0.53		Hopkins et al. (2014)
Pan trogolodytes	Chimpanzee	Communication	Captive reared and Wild-caught	0.34		Hopkins et al. (2014)
Pan trogolodytes	Chimpanzee	Spatial memory	Captive reared and Wild-caught	0.54		Hopkins et al. (2014)
Macaca mulatta	Rhesus macaque	Conditioned fear	Captive-reared	0.38	As freezing duration. Correlated with metabolic activity of amygdala and bed nucleus of stria terminalis	Rogers et al. (2008)
Macaca mulatta	Rhesus macaque	Anxious temperament	Unknown	0.36		Oler et al. (2010)
Macaca mulatta	Rhesus macaque	Orienting to intruder (vigilance)	Captive-reared	0.91		Rogers et al. 2008
Sus domesticus	Domestic pig	Avoidance learning	Captive-reared	0.45	Based on 2 breeds, both acquisition and extinction.	Willham et al. (1964), but see Wahlsten (1972)
Rattus norvegicus	Brown rat	Avoidance learning	Laboratory strain	0.56	Selected for high and low learners. Calculated using data from Bignami (1965)	Wahlsten (1972)
Rattus norvegicus (Long Evans)	Brown rat	Conditioned fear	Laboratory strain	0.36	As freezing duration postextinction	Shumake et al. (2014)

Table 1 *Continued*

Taxon	Common name	Trait	Source	Heritability Est.	Notes	Reference
Mus musculus	House mouse	General cognitive ability *g*	Laboratory strain	0.40		Galsworthy et al. (2005)
Mus musculus	House mouse	Ability to learn	Laboratory strain	0.37	Acquisition of conditioned freezing	Radcliffe et al. (2000)
Mus musculus	House mouse	Ability to learn	Laboratory strain	0.35	Selection for high and low learners.	Schaefer (1968)
Mus musculus	House mouse	Ability to learn	Laboratory strain	0.30		Tyler and McClearn (1970)
Mus musculus	House mouse	Ability to learn	Laboratory strain	0.40		Oliverio et al. (1972)
Mus musculus	House mouse	Conditioned fear	Laboratory strain	0.21		Henderson (1968)
Mus musculus	House mouse	Avoidance learning	Laboratory strain	0.48–0.50		Oliverio (1971), Oliverio et al. (1972)
Taeniopygia guttata	Zebra finch	Maximum frequency	Captive colony	0.96	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Maximum frequency	Captive colony	0.56	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Peak frequency	Captive colony	0.25	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Peak frequency	Captive colony	−0.18	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Phrase length	Captive colony	0.02	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Phrase length	Captive colony	0.03	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Syllable number	Captive colony	0.31	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Syllable number	Captive colony	0.23	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Proportion unique syllables	Captive colony	0.35	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Proportion unique syllables	Captive colony	0.47	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Various acoustic measures of call	Captive colony	0.39–0.64	Female call	Forstmeier et al. (2009)
Taeniopygia guttata	Zebra finch	Various acoustic measures of call	Captive colony	0.11–0.28	Male call	Forstmeier et al. (2009)
Taeniopygia guttata	Zebra finch	Various acoustic measures of song	Captive colony	0.10–0.28	Male song	Forstmeier et al. (2009)
Apis mellifera capensis	Cape honey bee	Ability to learn	Captive colony	0.39–0.54	Under a variety of learning tasks	Brandes (1988, 1991), Brandes et al. (1988)
Poecilia reticulata	Guppy	Algal-foraging ability	Captive reared	0.57–0.66		Karino et al. (2005)
Girardinus falcatus	Goldbelly topminnow	Lateralization of detour behavior	Artificial Selection	0.56–0.60	As pooled from 2 experiments	Bisazza et al. (2000)
Drosophila melanogaster	Fruit fly	Ability to learn	Laboratory strain	0.08	Known tradeoff with larval competitive ability	Lofdahl et al. (1992)
Neural traits						
Homo sapiens	Human	Total brain volume		0.94	Same data set as [a]Wright et al. (2002)	Bartley et al. (1997)
Homo sapiens	Human	Total brain volume		0.66	Same data set as Bartley et al. (1997)	[a]Wright et al. (2002)
Homo sapiens	Human	Total brain volume		0.80	Children and adolescents Ages >12	Pennington et al. (2000)
Homo sapiens	Human	Total brain volume		0.94	Age 9	Peper et al. (2009)
Homo sapiens	Human	Cerebrum volume		0.61–0.65		[a]Geschwind et al. (2002)
Homo sapiens	Human	Cerebrum volume		0.88		[a]Schmitt et al. (2007)
Homo sapiens	Human	Hemisphere volume		0.60–0.66		[a]Geschwind et al. (2002)
Homo sapiens	Human	Cerebellum volume		0.88	Age 9	Peper et al. (2009)
Homo sapiens	Human	Cerebellum volume		0.66–0.67	Young-middle aged	[a]Wright et al. (2002)
Homo sapiens	Human	Cerebellum volume		0.81	Young-middle aged	Posthuma et al. (2000)
Homo sapiens	Human	Cerebellum volume		0.55		[a]Schmitt et al. (2007)
Homo sapiens	Human	Frontal lobe volume		0.51–0.66	Based on 2 hypothesis-testing stats approaches, study also contains MLE models	[a]Geschwind et al. (2002)
Homo sapiens	Human	Occipital lobe volume		0.49–0.69		[a]Geschwind et al. (2002)
Homo sapiens	Human	Parietal lobe volume		0.47–0.52		[a]Geschwind et al. (2002)
Homo sapiens	Human	Temporal lobe volume		0.42–0.61		[a]Geschwind et al. (2002)
Homo sapiens	Human	Thalamus volume		0.72		[a]Schmitt et al. (2007)
Homo sapiens	Human	Basal ganglia volume		0.81		[a]Schmitt et al. (2007)
Homo sapiens	Human	Caudate nucleus volume		0.80		[a]Wallace et al. (2006)
Homo sapiens	Human	Corpus Callosum area		0.78	Data from Pfefferbaum et al. (2000)	Sullivan et al. (2001)
Homo sapiens	Human	Temporal horn volume		0.63–0.65	Ages 68–78, Male	Sullivan et al. (2001)
Homo sapiens	Human	Hippocampus volume		0.40	Ages 68–78, Male	Sullivan et al. (2001)
Homo sapiens	Human	Hippocampus volume		0.66–0.71	Young-middle aged	[a]Wright et al. (2002)
Homo sapiens	Human	Hippocampus volume		0.29–0.40	Ages 68–78, Male	Sullivan et al. (2001)
Homo sapiens	Human	Whole-brain gray matter volume		0.82	Adult twins, correlated with *g*	Posthuma et al. (2002)

Table 1 *Continued*

Taxon	Common name	Trait	Source	Heritability Est.	Notes	Reference
Homo sapiens	Human	Whole-brain white matter volume		0.87	Adult twins	Posthuma et al. (2002)
Chlorocebus aethiops s.	Vervet monkey	Corpus callosum area	Captive colony	0.89		Fears et al. (2009)
Chlorocebus aethiops s.	Vervet monkey	Cerebellum volume	Captive colony	0.86		Fears et al. (2009)
Chlorocebus aethiops s.	Vervet monkey	Cerebrum volume	Captive colony	0.98		Fears et al. (2009)
Chlorocebus aethiops s.	Vervet monkey	Hippocampal volume	Captive colony	0.95		Fears et al. (2009)
Chlorocebus aethiops s.	Vervet monkey	Hippocampus volume	Captive colony	0.95		Fears et al. (2009)
Chlorocebus aethiops s.	Vervet monkey	Total brain volume	Captive colony	0.99		Fears et al. (2009)
Papio hamadryas	Hamadryas baboon	Total brain volume	Captive colony	0.52	After removal of combined effects of age, sex, and their interaction.	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Total brain volume	Captive colony	0.82	Following spatial normalization	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Total brain volume	Captive colony	0.52–0.82		Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Cerebrum volume	Captive colony	0.73	Following spatial normalization	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Gray matter volume	Captive colony	0.67	Following spatial normalization	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Cerebrum surface area	Captive colony	0.73	Following spatial normalization	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Opercular portion of inferior frontal gyrus volume	Captive colony	>0.70	Analogous to Broca's area in humans	Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Superior parietal structure volume	Captive colony	0.4–0.6		Rogers et al. (2007)
Papio hamadryas	Hamadryas baboon	Superior temporal gyrus volume	Captive colony	>0.70	Analogous to Wernicke's area in humans	Rogers et al. (2007)
Saimiri sciureus	Common squirrel monkey	Hippocampal volume	Captive reared	0.54	No effect of stress	Lyons et al. (2001)
Macaca mulatta	Rhesus macaque	Total cranial volume	Wild, free-ranging	0.60–0.75	As measured from skeletal remains	Cheverud et al. (1990)
Macaca mulatta	Rhesus macaque	Hippocampal glucose metabolic activity	Unknown	0.52	For most predictive voxel associated with anxious temperament	Oler et al. (2010)
Macaca mulatta	Rhesus macaque	Amygdalar glucose metabolic activity	Unknown	0.02	For most predictive voxel associated with anxious temperament	Oler et al. (2010)
Macaca mulatta	Rhesus macaque	Superior temporal sulcus glucose metabolic activity	Unknown	0.46	Associated with anxious temperament OR CONTROL?	Oler et al. (2010)
Macaca radiata	Bonnet macaque	Hippocampal volume	Captive reared	Not significant, value not reported	Stress effect specific to left hemisphere from rearing under variable foraging demand	Jackowski et al. (2011)
Taeniopygia guttata	Zebra finch	Brain mass	Captive colony	0.01	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Brain mass	Captive colony	−0.11	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	Brain mass	Captive colony	0.49	Birds were tutored by genetic parents	Airey, Castillo-Juarez, et al. (2000)
Taeniopygia guttata	Zebra finch	Area X volume	Captive colony	0.23	Birds were tutored by genetic parents	Airey, Castillo-Juarez, et al. (2000)
Taeniopygia guttata	Zebra finch	RA volume	Captive colony	0.2	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	RA volume	Captive colony	0.76	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	RA volume	Captive colony	0.72	Birds were tutored by genetic parents.	Airey, Castillo-Juarez, et al. (2000)
Taeniopygia guttata	Zebra finch	HVC volume	Captive colony	0.01	Based on sibling analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	HVC volume	Captive colony	0.21	Based on father–son analysis	Woodgate et al. (2014)
Taeniopygia guttata	Zebra finch	HVC volume	Captive colony	0.38	Birds were tutored by genetic parents. Correlates with song structure	Airey, Castillo-Juarez, et al. (2000)
Taeniopygia guttata	Zebra finch	lMAN volume	Captive colony	0.18	Birds were tutored by genetic parents.	Airey, Castillo-Juarez, et al. (2000)
Taeniopygia guttata	Zebra finch	nl2ts volume	Captive colony	0.47	Birds were tutored by genetic parents.	Airey, Castillo-Juarez, et al. (2000)
Poecilia reticulata	Guppy	Relative brain mass	Laboratory strain	0.48	Females, as brain mass relative to body length	Kotrschal et al. (2013)
Poecilia reticulata	Guppy	Relative brain mass	Laboratory strain	0.45	Males, as brain mass relative to body length	Kotrschal et al. (2013)

For nonhuman animals, all known data pertaining to the traits examined within this commentary are listed. Metrics listed for humans are limited to neurotypical individuals. For humans, we have also included a sampling of data available for additional cognitive and neural traits.

[a]Reference containing heritability data for many brain regions-of-interest not otherwise listed. For more data, see tables within each reference and in Bouchard and McGue (1981); Lenroot et al. (2009); Peper et al. (2007); Schmitt et al. (2008); Deary et al. (2009); Kremen et al. (2010); Polderman et al. (2015).

species such that populations in harsher climates, with likely higher dependence on food caches for survival, are predicted to have better spatial memory and a larger hippocampus compared with populations in milder environments where such memory ability might not provide higher fitness, yet might carry some physiological costs (Pravosudov and Clayton 2002; Pravosudov and Roth 2013). For example, multipopulation comparisons in 2 species of food-caching chickadees (*Poecile gambeli* and *Poecile atricapillus*) were consistent with the adaptive specialization hypothesis and showed that colder winter climate was associated with higher food caching propensity, better spatial memory performance (accuracy, but not longevity in black-capped chickadees and both accuracy and longevity in mountain chickadees), larger hippocampus volume, larger number and size of hippocampal neurons and higher hippocampal neurogenesis rates (Pravosudov and Clayton 2002; Roth and Pravosudov 2009, Freas et al. 2012; Roth et al. 2012).

The underlying premise for both types of comparison is that both spatial memory (or any of the components of spatial memory including acquisition, retention, and accuracy) and the hippocampus respond to natural selection acting on individual and heritable variation in spatial memory ability, yet direct evidence for this link is still lacking (e.g., Bolhuis and Macphail 2001, but see Hampton et al. 2002). Overall, the existing studies are highly consistent with the idea that variation in both spatial memory and hippocampal morphology have been shaped by natural selection associated with dependence on food caching. To provide direct support for this proposition, it would be necessary to show that 1) fitness consequences of such variation are larger in populations/species with higher dependence on food caches and/or under especially harsh conditions, and 2) individual variation in some components of spatial memory and hippocampus morphology is heritable. As such, this is an example where quantifying trait heritability is a necessary component of direct hypothesis testing.

Heritability of spatial memory

Data related to heritability of specific modular cognitive traits and their associated brain phenotypes of nonhuman animals are particularly sparse, despite that variation in cognitive traits likely has strong selective implications (Thornton and Lukas 2012). A striking example of this is spatial memory. Human spatial memory is well-studied, including age- and sex-related variation, and appears to have a heritable component (McGee 1979; Linn and Petersen 1985). For example, a recent genome-wide association study (GWAS) has identified 2 quantitative trait loci (QTL) associated with spatial memory (and 2 associated with working memory; Knowles et al. 2014). Likewise, in chimpanzees, spatial memory also appears highly heritable, although it is not clear whether such heritability is mainly due to high heritability of general intelligence and not specific modular spatial ability (Hopkins et al. 2014). Variation in spatial memory ability of food-caching animals has been investigated among a small suite of passerine birds (reviewed in Pravosudov and Roth 2013). There is evidence from hippocampal transcriptome profiles of laboratory-reared black-capped chickadees that individuals from populations under stronger selection due to harsh environmental conditions exhibit differential expression of genes known to be associated with hippocampal function, yet it remains unknown if such differential gene expression is associated with genetic or epigenetic differences (Pravosudov et al. 2013). We yet lack the critical evolutionary connections between individual variation in spatial memory, heritability and individual variation in fitness, as relatively little is known about the genetic architecture and heritability of spatial memory.

Heritability of the hippocampus

The human hippocampus has garnered specific attention due its role in mediating spatial ability. Heritability of specific brain regions in humans is most commonly assessed using monozygotic twins and MRI. In general, values of hippocampal heritability in humans are quite high (Table 1 and references therein). Likewise, gene by endogenous hormone interactions may mediate hippocampal volume, driving changes across development (e.g., testosterone, Panizzon et al. 2012).

In nonhuman animals, a handful of studies have illustrated important connections between hippocampus morphology, heritability, and behavioral responses to stress (Table 1). For example, captive rhesus macaques (*Macaca mulatta*) show significant heritability of hippocampal metabolic activity in regions associated with anxious temperament (Oler et al. 2010). This is a particularly important illustration of the connection between heritable neurophysiology and mechanisms for coping with environmental stressors in the natural world, and this study provides an important piece of evidence for how animals may evolve behavioral and cognitive adaptations in a natural context. As such, these data show directly that both behavior and hippocampus physiology can be affected by the environment. That these traits are of known heritability provides a key to understanding mechanisms by which both memory and hippocampal morphology can evolve in wild populations.

In contrast, in food-caching birds, nutritional stress during early development results in impaired spatial memory performance, reduced hippocampus volume, and a smaller number of hippocampal neurons (Pravosudov et al. 2005). These data connect both memory and hippocampal morphology to the environment, but the explicit heritability estimates for these traits in this system remain unknown despite that they are a key component of understanding the mechanisms driving both population and species variation in these traits, and the adaptive specialization hypothesis assumes that spatial memory ability and hippocampus morphology are at least in part heritable.

The number of hippocampal neurons appears a much better predictor of variation in spatial memory than is hippocampus volume (see Pravosudov and Roth 2013), but it is impossible to study neuron number heritability in live animals. It should be possible, however, to estimate the heritability of different hippocampal traits by sampling either siblings from the same broods, or cross-fostered, and comparing their hippocampus traits with those of their parents. Pedigrees can be generated in either case and should allow for heritability estimates (e.g., Morand-Ferron and Quinn 2015). Comparing heritability of different hippocampal traits (e.g., neuron numbers, volume, neuron soma size) would provide critical information about which mechanisms might evolve more rapidly under differential selection. In addition, heritability of specific spatial memory characteristics, such as accuracy and longevity, would also provide critical information necessary for understanding the evolution of spatial memory.

SONG LEARNING AND HVC

Song in male oscine passerines is a species-specific vocal signal that functions consistently in mate choice and male–male competition (Catchpole and Slater 1995) and is learned at the birds' natal site. The high vocal center (HVC) in the songbird brain has been directly linked to song learning and production. Song is modulated in the HVC in numerous ways: 1) The primary HVC pathway develops

while young birds are actually learning their song (Nordeen and Nordeen 1988); 2) Song is primarily used in the breeding season or spring and as such, there are seasonal changes in the morphology of the HVC such that the size of neurons (Nottebohm 1981) and overall volume (Smith et al. 1997) of the HVC increase in the spring, when males are singing more frequently; 3) There is a positive correlation between HVC volume and song repertoire size or complexity, especially when repertoire size strongly influences female mate choice (Brenowitz et al. 1996; DeVoogd and Sz'ekely 1998; Airey, Buchanan, et al. 2000).

There are numerous strategies used to learn song, and these strategies differ significantly among different species. This type of variation can present challenges in relating the mechanism/function of song with its neural substrate on a larger comparative scale (DeVoogd et al. 1993). DeVoogd et al. (1993) used 34 independent phylogenetic contrasts to infer the evolutionary relationship between HVC and song among 41 species of oscines. The results of this work suggest that the number of songs that songbirds sing is positively associated with the proportion of the forebrain devoted to the HVC (DeVoogd et al. 1993). These findings, alongside known female preference for larger repertoire sizes strongly suggest that sexual selection has shaped much of the variation seen in song complexity and HVC morphology (see Boogert et al. 2011 for review), yet these traits must be heritable in order for this to be true.

Heritability of song learning

The heritability of song learning ability is of particular biological interest because song complexity is guided and constrained by genetic biases despite that song is learned from local males, which may or may not be the genetic fathers (Nelson and Marler 1993; Baptista 1996; Podos et al. 2004). Likewise, a greater understanding of learned versus heritable components of song may further our understanding of the geographic variation in song (Rowell and Servedio 2012), including whether and how selection acting on song and song learning can affect the generation and spread of song dialects across generations and geographic ranges.

Multiple characteristics of avian song and song learning, including the complexity of song, as well as speed and accuracy of song learning (Catchpole 1980; Searcy and Andersson 1986; Gil and Gahr 2002; Riebel 2009), are assumed to be under sexual selection, yet for this to be the case, variation in these traits must be heritable. For example, the hypothesis that sexual selection results in increased song learning accuracy predicts that 1) females prefer males singing more accurate song, and 2) song learning accuracy is heritable. Because birdsong is learned from a tutor at the natal site, heritability of bird song per se would be rather challenging to estimate, however, certain song features are conserved over evolutionary time, and studies of specific song features including note duration, song duration, rhythm, and repertoire size have revealed high genetic heritability based on constraints around the expression of these song features (Baptista 1996, Table 1). At the same time, song learning ability and the HVC show tremendous plasticity and are responsive to environmental variation during development (Buchanan et al. 2013). Heritable variation in avian song learning ability has not yet been adequately explored despite its strong selective implications.

Heritability of HVC

HVC is a specific brain region that is predicted to be under sexual selection due to its known role in the learning and production of male avian song. Females of many songbird species prefer males with larger and more complex song repertoires. Song repertoire size is correlated with the volume of HVC, therefore this brain structure, as well as other parts of the song learning/production neural pathway, has been assumed to be under sexual selection (Brenowitz et al. 1996; DeVoogd and Sz'ekely 1998; Airey, Buchanan, et al. 2000). Implicitly, then, the volume of HVC is also assumed to show some heritable variation. This was validated in the case of zebra finches (*Taeniopygia guttata castanotia*) by Airey, Castillo-Juarez, et al. (2000), who assessed heritability for various neural components of song learning and production in zebra finches (Table 1), and found moderate heritability even for HVC, the most variable brain region measured within this study (Table 1; Airey, Castillo-Juarez, et al. 2000). A second, comparative measure of proportional response to selection for all traits measured on the same scale (evolvability, sensu Houle 1992) yielded higher values for HVC and robust nucleus of the arcopallium (RA) than for any other traits analyzed (HVC: 16.13%, RA: 14.83%; Airey, Castillo-Juarez, et al. 2000). More recently, however, conflicting data suggest that selection acts on genetic determinants of vocal learning ability (e.g., GxE interactions maintaining additive genetic variation, see Woodgate et al. 2014) rather than on HVC per se, and that heritability for neuroanatomy in bird song circuitry is low (Table 1; Woodgate et al. 2014). There is a clear need for further attention to cognitive trait heritability in order to clarify this, in order to test predictions associated with fitness consequences of variation in song and song learning. If the ability to learn complex songs is heritable, but the HVC volume is not, it would be important to focus on identifying the mechanisms that makes song learning ability heritable.

FEAR RESPONSE AND THE AMYGDALA

The ability to learn fear associations is of particular adaptive importance because these responses have the potential to determine an individual's survival when faced with aversive or dangerous stimuli by integrating fearful experiences from past, present, and future. Learned conditioned fear response in rodents, for example, is often measured based on freezing, defecation, heart rate, and other physiological responses, which are paramount when learning the specific conditions that preface or predict a dangerous situation. In mammals, both conditioned and unconditioned fear responses have been linked to the amygdala, which plays a crucial role in the development and expression of conditioned fear. The direct relationship between the amygdala and fear conditioning is evident by 1) actual increases in amygdala activity while conditioned fear memories are established, which are later tested using the conditioned stimulus alone (Quirk et al. 1995; Rogan et al. 1997); 2) Electrical stimulation of the amygdala, which produces fear responses including alteration in heart rate, blood pressure, respiration, and cessation of behavior or freezing (e.g., Blanchard and Blanchard 1969; Applegate et al. 1983); and 3) Lesions or damage to the amygdala, which attenuate typical fear responses including freezing (Blanchard and Blanchard 1972) and heart rate acceleration (Cohen 1975).

The amygdala also plays an important ecological role in regulating exploratory behavior/neophobia in free-ranging animals, and lesions in the amygdala increase exploratory behavior in open field tests (Grijalva et al. 1990). Similarly, black-capped chickadees from milder climates, which demonstrate higher levels of neophobia relative to individuals from harsher climates, also have larger arcopallium volume, the region of the avian brain homologous to the

amygdala (Roth et al. 2012). Individual variation in fear responses and associated brain regions can have strong selective implications, but aside from open field activity studies in laboratory mice (e.g., Hausheer-Zarmakupi et al. 1996), there is little known about either the extent to which each might be heritable, particularly in wild animals (but see Dias and Ressler 2014), or whether natural selection can generate adaptive variation in fear response and the amygdala.

Heritability of conditioned fear response

Heritable individual variation in fear responses is well documented in laboratory animals, as these differences are commonly selected for in the establishment of mouse and rat strains (Shumake et al. 2014). Several studies have documented differences within and among mouse and rat strains in the ability to learn fear (avoidance learning, fear conditioning) in response to specific conditioned stimuli (Table 1). That this trait is readily responsive to artificial selection speaks directly to its having genetic underpinning, but outside of this context, much less is known about the heritability of fear acquisition. The ability to learn appropriate responses to dangerous stimuli is of obvious ecological importance, therefore further study of the heritability of such responses would provide insight into the evolution of mechanisms for coping with danger in wild populations. Similarly, neophobia may interfere with other cognitive tasks (e.g., problem solving—Kozlovsky et al. 2015), therefore, understanding the mechanisms driving variation in fear response is especially critical. For example, the evolution of some cognitive traits might be constrained if these traits are traded-off against fear responses, as fear responses may evolve independently assuming some degree of heritability.

Heritability of the amygdala

Heritability of amygdalar characteristics has gone largely unstudied despite the ecological importance of phenotypes associated with the amygdala, the prominence of research on the amygdala itself, and the known association between amygdalar traits and heritable psychiatric disorders (Pezawas et al. 2005; Oler et al. 2010). Amygdala volume has been shown to be at least partially genetically determined through the study of human twins suffering from major depressive disorder (Liu et al. 2010). Heritability of amygdala activation, however, has not been studied directly. In general, there is a great paucity of data available describing heritability of amygdala features.

In summary, available evidence, however limited in scope, suggests that overall brain morphology and morphology of different brain regions are highly heritable, yet heritability estimates appear to vary. Cognition and the brain are widely considered plastic, yet both intraspecific and interspecific variation in both are also frequently assumed to be shaped by natural selection (e.g., the adaptive specialization hypothesis, Pravosudov and Clayton 2002; Sherry 2006). Heritability estimates are necessary to further our understanding of the role of natural selection and to directly test evolutionary hypotheses.

ARTIFICIAL SELECTION EXPERIMENTS DIRECTLY DEMONSTRATE COGNITIVE TRAIT HERITABILITY

Artificial selection is a well-established method of demonstrating heritability of behavioral and cognitive traits. Many studies using laboratory strains of fruit flies (*Drosophila melanogaster*), rats, and mice have succeeded in artificially selecting for lines divergent in the ability to learn a variety of associations (Table 1, Fuller and Thompson 1978; Lofdahl et al. 1992; Mery and Kawecki 2002; Shumake et al. 2014 and references therein). In fruit flies, lines selected for enhanced learning abilities incurred a variety of costs, including, for example, a decline in larval competitive ability (Mery and Kawecki 2003), and neurobiological experiments in fruit flies have likewise quantified physiological costs directly associated with the maintenance of long term memory (Plaçais and Preat 2013). Dunlap and Stephens (2014) have demonstrated via experimental evolution approach that the ability of *D. melanogaster* to learn certain associations tracks the reliability of the association itself; in combination, these studies underscore the importance of understanding in detail the roles of cognitive traits in determining fitness. Kotrschal et al. (2013) similarly selected for large and small brains in guppy lineages, and revealed a tradeoff such that females of the larger-brained lineage performed better in a visual-learning task (but see Healy and Rowe 2013), yet produced fewer offspring and had smaller guts. Both the fruit fly and guppy studies, then, not only demonstrate the availability of cognitive traits to selection but also provide evidence of physiological trade-offs associated with enhanced cognitive abilities and their associated neural mechanisms. Indeed, examples of artificial selection on behavior, in general, and on the ability to learn more specifically, are countless, but there are relatively few direct measures of heritability of these traits. Although quite limited, available evidence from artificial selection experiments again suggests that cognitive trait heritability is a key component of understanding cognitive trait variation. Overall, artificial selection provides a powerful tool to study heritability, but this approach is limited to short-lived species that readily breed in a laboratory environment. As a result, artificial selection experiments can provide only limited examples of heritability.

GENETIC UNDERPINNINGS OF COGNITION AND BRAIN MORPHOLOGY

Recently, there has been a push toward identifying particular genes associated with aspects of cognition and brain morphology. Quantitative genetics studies have yielded considerable insight into inheritance patterns for such traits, but this approach is somewhat limited in that it does not allow for insight into particular gene associations. Human twin studies have yielded the largest body of research to date addressing the extent to which cognitive traits have heritable genetic underpinnings (e.g., Blokland et al. 2012 and references therein, see also Table 1). Analogous studies are largely unavailable in nonhuman animals, and existing research has generally failed to address any ecological relevance of the traits in question, or to directly address any fitness effects (but see Sol et al 2007; Cole et al. 2012; Kotrschal et al. 2013). Likewise, nuanced genetic evidence is lacking due to the difficulty of isolating any one or suite of genes in association with a particular cognitive task, because any assay designed to isolate genes associated with a given cognitive trait would require a series of outcomes analogous to all possible alleles in the tested population (Benyamin et al. 2014).

GWAS and QTL mapping have detected associations between specific genes and phenotype including cognitive traits. For example, 2 QTLs on chromosome 17 have recently been identified in association with human spatial memory (Knowles et al. 2014). Likewise, Park et al. (2011) identified a QTL associated with cued immobility, a context-dependent fear response, in a mouse strain. Although these studies are an exciting and potentially fruitful advancement in our understanding of the genetic basis of cognitive trait variation,

studying the genomic basis of cognitive traits yet faces significant hurdles. Many cognitive traits are quantitative in nature, and thus should be expected to evolve as a result of selection on many small effect variants through polygenic adaptation (e.g., Pritchard and Di Rienzo 2010; Pritchard et al. 2010). Cognitive and behavioral traits are characterized by complex gene expression (Flint 2002), and can be difficult to quantify, particularly given that GWAS can yield only correlative data. Even where GWAS studies have revealed significant genetic associations, such associations typically explain only a low fraction of trait variance (Yang et al. 2010), likely as a result of unaccounted for genotypes and/or variants, or environmental or epigenetic effects (Manolio et al. 2009; see discussion in Parker and Palmer 2011). Likewise, adaptation in quantitative traits involves additive effects at many small effect genetic loci (Arnegard et al. 2014; Roesti and Salzburger 2014), and selection on a given phenotype may result in different genetic architectures in different studies and different populations (Barton and Keightley 2002; Weiss 2008; Soria-Carrasco et al. 2014). This means that despite emergence of genetic patterns in association with certain phenotypes, it might be difficult to establish whether specific genetic differences are associated with the same specific phenotypes under selection (e.g., Rockman 2012). Considering the complexity of cognitive traits and their underlying genetic architecture, it might be naïve to expect the same genetic architecture underlying parallel evolution of cognitive traits (e.g., Barton and Keightley 2002; Weiss 2008; Soria-Carrasco et al. 2014). For example, even if harsher environments indeed generated stronger selection pressure on spatial memory and the hippocampus in food caching chickadees, evolutionary changes in different populations experiencing comparable environments might involve different genetic architectures. Thus, behavioral ecologists may be better served by parallel efforts toward quantifying trait heritability in order to garner a better understanding of whether and how a trait may be affected by selection (e.g., Rausher and Delph 2015). This would significantly enhance our understanding of the evolution of and variation in cognition and the brain, both within and across species, particularly in instances where we cannot identify specific genetic differences underlying such variation due either to methodological constraints or to the existence of multiple genotypes underlying the same phenotype under selection.

WHAT IS NEXT?

Available data pertaining to whether and how cognitive traits can respond to selection, however limited, strongly suggest that many cognitive and neural traits are indeed heritable, yet such data are unavailable for most species. Since the level and strength of any evolutionary response depends in part on its heritability, and considering the recent explosion of ecologically motivated studies of cognition and the brain under the adaptive umbrella (e.g., Burgess 2008; Van Overwalle 2009; Pravosudov and Roth 2013; Woodgate et al. 2014), now is the time to focus on these essential elements. Understanding heritability should bring much needed clarity about the role of natural selection as a driver in generating cognitive trait variation. Likewise, heritability estimates would provide essential information furthering our understanding of the ranges of plasticity and of how animals may respond to changing environments. Without knowing the heritability of cognitive traits and their neural mechanisms we cannot definitively test any of the major hypotheses about the evolution of cognition, as most data available at this point could support such hypotheses only indirectly.

Measuring heritability of cognitive and especially neural traits is a challenging task. Yet, we believe that sampling wild and wild-caught animals with known pedigrees should be possible (sensu Rogers et al. 2007; Fears et al. 2009), and cross-fostering experiments might be particularly powerful in using sibling comparisons. Likewise, estimating heritability of individual learning abilities in the wild should be possible, based on repeated measurements, using standardized and comparable methods, of individuals with known pedigrees (see Morand-Ferron and Quinn 2015; Morand-Ferron et al. 2015 for discussion). Finally, MRI can be used to repeatedly measure overall brain volume, allowing estimation of heritability of overall brain size. We believe that a focus on specific and targeted functional brain regions would provide much better resolution, yet it might not still be possible using MRI. In those cases, individuals might have to be sacrificed for detailed analyses of brain morphology.

With a focus on heritability, we can better understand the evolution of cognition and the roots of cognitive trait variation, including their adaptive value, trade-offs, and/or constraints linking cognitive, neural, and physiological traits, and the possible linked effects of selection on these.

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