## Original Article

# Race and IQ in the postgenomic age: The microcephaly case

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**Abstract** A convergence of contextual factors, technological platforms and research frameworks in the genomics of the human brain and cognition has generated a new postgenomic model for the study of race and IQ. Centered on the case study of Bruce T. Lahn's 2005 claims about the genomic basis of racial differences in brain size and IQ, this article maps the disciplinary terrain of this research, analyzes its central claims and examines the rigor of critical debate within the genomics community about new race and IQ research. New postgenomic race and IQ research, while displaying some continuities with previous eras of racial science, also differs in important ways, both contextual and conceptual. In particular, this new research draws on methods and hypotheses that are widely accepted across many fields of the contemporary molecular genetic sciences. This has implications for the forms of critical engagement that science studies scholars might pursue.

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Since the completion of the sequencing of the human genome in 2001, science studies, race/ethnicity and genetics scholars have undertaken extensive discussions about the implications of new genomic research for conceptions of human difference.[1] Absent from these discussions, however, has been the classically charged issue of the genetics of racial differences in intelligence.[2] In this article, I suggest that the postgenomic age – a term specifying shifts both temporal and technical – may initiate a new era of scientific claims about the genetics of racial differences in IQ. Research in an emerging field that some are calling 'evolutionary cognitive genetics' is producing renewed and challenging claims about human genetic variation and IQ. While some methodological issues with this research are familiar, these new

1 See, for example, Fujimura *et al* (2008); Koenig *et al* (2008); and Whitmarsh and Jones (2010).

2 'IQ' refers to a measure of general intelligence arrived at by cognitive tests and in relation to statistical measures of age-appropriate performance on those tests. In this article, I use the terms 'IQ' and 'intelligence' interchangeably. For more general claims not specific to IQ, I use phrases such as 'cognitive differences' or 'cognitive performance'.

 

claims about the genetics of intelligence emanate from a different context and conceptual framework than that of previous eras – one rooted in evolutionary biology, neuroscience and the molecular biosciences, as well as the distinctive discursive and institutional climate of the postgenomic biosciences. These features, as well as the social and ethical implications of this research, require the attention and close scrutiny of scholars of genetics, science studies and race/ethnicity.

This article proceeds in four parts. I begin with a brief overview of how postgenomics – what *The Economist* magazine recently called 'Biology 2.0' (2010) – is changing the race and IQ landscape and producing a new research platform for claims about biologically based racial differences in intelligence that I will refer to, following Bates *et al* (2008, p. 690), as 'evolutionary cognitive genetics'. Second, I characterize the central claims, hypotheses and theoretical frameworks of the intersecting fields of evolutionary cognitive genetics. Third, I critically examine University of Chicago geneticist Bruce T. Lahn's 2005 claims that variants of two genes known to be associated with microcephaly, or small brain size, are more common among Eurasian populations than African ones, and may explain group-based differences in intelligence. Finally, I analyze the broader context and reception of the claims about microcephaly gene variants within human genetics, assessing the rigor of the genetics community's critical response to Lahn's research.

I argue that the microcephaly case reveals a lack of community standards around central claims and practices in brain genomics and studies of race and intelligence. Moreover, I argue that discursive and disciplinary shifts render new genetic research on race and IQ more resistant to oppositional methodological and ideological critique than were its predecessors. Although researchers express awareness of the sensitive nature of genetic research on IQ, there is a need for critical discussion – what I call 'transformative conversations' – around the questions asked, accepted methodologies, structuring assumptions and descriptive and representational discourses used in genomics research areas relevant to race and IQ.

## Race and IQ in the Postgenomic Age

Claims about the genetic basis of racial differences in IQ have in the past been hampered by well-known methodological and political problems. Methodologically, defenders of race and IQ research have never unequivocally demonstrated the validity of IQ tests as culturally independent measures of innate intelligence, persuasively disambiguated genetic and environmental (for example, socioeconomic) factors in intelligence, or provided evidence of any gene or genetic mechanism biochemically implicated in differences in IQ among racial and ethnic groups (Gould, [1981] 1996). In part as a result of these methodological and empirical weaknesses, studies of the genetics of race and IQ have found themselves isolated from the molecular sciences, including genomics and molecular neuroscience, unable to find mainstream sources of research funding and, in general, pegged as ideological, fringe or not 'real' science (Snyderman and Rothman, 1988; Panofsky, forthcoming).

We are now, arguably, in a 'postgenomic' age, which I define as the period after the completion of the sequencing of the human genome and in which whole genome technologies are a shared platform for biological research across many fields and social arenas.

The term 'postgenomic' specifies not just contemporary genome research, but, more broadly, any biological research after the completion of the major genome projects that employs genomic technologies and draws upon genomic knowledge. 'Whole genome' technologies include: human genome databases and biobanks; microarray chips for assessing the expression of hundreds of thousands of genes in human tissue at once and over time; rapid, inexpensive next-generation genome sequencing technologies; bioinformatic and computational advances in genome-wide association studies; and low-overhead mail-order mass sequencing and genome analysis facilities. The falling cost and increasing speed of whole genome technologies is exceeding expectations, and a variety of private and public projects of global scale are now generating massive databases of genomic information (Biology 2.0: A Special Report on the Human Genome, 2010).

Today, as biomedical scientists work in a transdisciplinary research environment framed in part by the interests and agendas of commercial pharmaceutical, biotechnology and direct-to-consumer genetics enterprises, they are driven to apply human genomic data and technologies to locate variation in the human genome that may be marketable as a biomarker for disease, forensics, ancestry or human enhancement (Kahn, 2008b; Clarke *et al*, 2009). Racial or biogeographical ancestry is among the principal tools for probing the human genome for differences (Fullwiley, 2008; Kahn, 2008a; Bliss, 2009; Fujimura and Rajagopalan, 2011). As such, human genomic data and technologies are poised to proliferate scientific claims of biological differences between human populations, often racially defined.

In the pre-genomics era of race and IQ research, claims about the genetic basis of IQ primarily emanated from the field of psychology and its subfields of studies of intelligence and of behavioral genetics (particularly twin studies) (Plomin and Rende, 1991; Panofsky, forthcoming). Genomics now provides a shared technological platform allowing heightened collaboration and rapid translation between these areas and previously dispersed and far-flung fields. Specifically, the postgenomic era of race and IQ research has drawn into its orbit the fields of *neuroscience*, particularly genetic studies of brain development, structure and function; *human evolution*, with a focus on the genomic evolution of the human brain; and *population genetics*, the study of the dynamics of human population genetic diversity (Figure 1). These developments are evidenced in the emergence of the new field of evolutionary cognitive genetics.

## Evolutionary Cognitive Genetics

A new and highly interdisciplinary research area, evolutionary cognitive genetics is best characterized as a convergence of research trends in genomics and the brain sciences, represented by an informal and diverse constellation of brain genomics, human population genetics and molecular anthropology researchers, rather than as an intimate, self-conscious and institutionally settled field. Three nodes of trending interest characterize investigations in evolutionary cognitive genetics: (i) efforts to map the human 'brain genome' and discover genes associated with mental and brain disorders; (ii) studies of recent positive selection in the human genome; and (iii) comparative genomics models of the evolution of human cognition. In the following, I describe the central sources, claims and theoretical frameworks of each of these research areas.

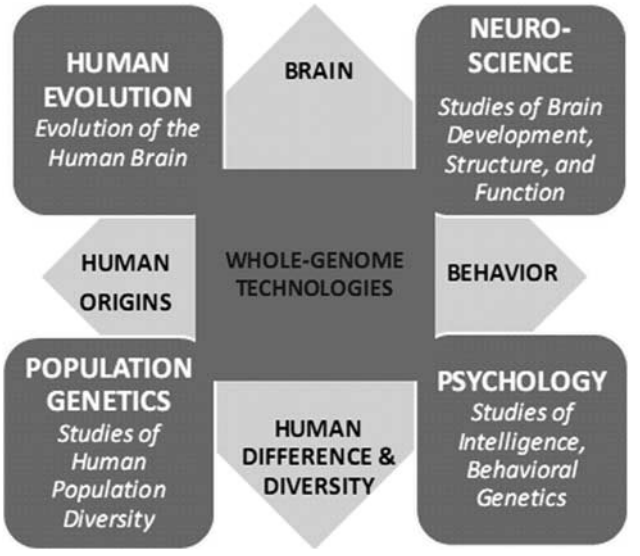


**Figure 1:** The disciplinary terrain of postgenomic race and IQ research.
*Source*: Author's illustration.

## 'Brain genes'

Neurogeneticists are currently in the process of identifying the genes and gene families that express in the human brain, and medical researchers are racing to discover genes implicated in mental and intellectual disorders. Prominent research streams include studies on attention deficit hyperactivity disorder, depression, Alzheimer's and other degenerative mental disorders, severe mental retardation, and developmental brain disorders such as autism.[3] The number of genes associated with such disorders is already large and will certainly grow in the future as whole genome technologies and human genomic databases develop further. These studies are providing a new and massive data pool for investigating the genetics of intelligence.[4] As Robert Plomin, author of *Behavioral Genetics* (2008), the leading textbook in the field, writes, '[We predict that] when genes are found for common disorders such as mild mental retardation or learning disabilities, the same genes will be associated with variation throughout the normal distribution of intelligence, including the high end of the distribution' (Plomin *et al*, 2006, p. 515).

Although behavioral geneticists stress the unlikelihood that any single gene variant will have a large effect on behavioral phenotype, the intensive study of the genomics of cognitive, mental and brain disorders will surely yield examples of genes with variants of different frequency in different populations, portending a wide-open pathway for genetic claims

3 For example, the Autism Genome Project has become a generative location for genes that many assume will also be implicated in intelligence (Abrahams and Geschwind, 2008; 'GenomeWeb', 2010).

4 Researchers believe that cortical thickness, thickness of the corpus callosum, and grey and white matter volumes of the total cerebrum are relevant predictors of intelligence, all features that have been shown to be influenced by genes (Deary *et al*, 2010). Any gene influencing these features is, in theory, a candidate 'intelligence gene'.

about racial or ethnic differences in brain and cognition. In addition to *ASPM* and microcephalin, at least 30 'brain' genes, including *CDK5RAP*, *CENPJ*, *ADCYAP1*, *AHI1*, *SHH*, *SRPX2*, *MAOA*, *BRCA1*, *COMT*, *ADRA2A*, *BDNF*, *CHRM2*, *DRD2*, *GRM3*, *HTR2A*, *SLC6A4*, *NCSTN*, *HLA*, *DRB1*, *IGF2R*, *CTSD*, *CBS*, *MSX1*, *SSADH*, *APOE*, *ACE*, *MTHFR*, *AHI1*, *GLUD2* and *FOXP2*, are current targets for research on human brain evolution, the genetics of intelligence and human population variation (Dorus *et al*, 2004; Plomin *et al*, 2006; Bates *et al*, 2008; Vallender *et al*, 2008). Some even suggest that such research may lead to drugs tailored for African Americans to correct genetic deficits in IQ. As intelligence researchers Earl Hunt and Jerry Carlson write, 'In 2005, the Food and Drug Administration licensed a drug, BiDil, targeted specifically for the treatment of heart disease in African Americans. In principle, similar race-specific treatments could be developed for individuals at risk for cognitive conditions, including low intelligence … There is no reason to regard such research as either impossible or undesirable' (2007, p. 197).[5]

## Recent positive selection

Positive selection is defined as evolutionary selection for an allele (gene variant) because it contributes to fitness (the ability to produce viable and fertile offspring). The study of ongoing and recent evolution of human populations through positive selective pressure on human gene variants is a new area of intense interest in human genetics, enabled by expanding global genomic databases and bioinformatic infrastructure. As Sabeti *et al* write, 'The advent of whole-genome sequencing and increasingly complete surveys of genetic variation represent a turning point in the study of positive selection in humans' (2006, p. 1614).[6]

Recent positive selection in human racial and ethnic populations is a provocative subject that has now begun to receive broader public uptake and circulation. As *The New York Times* science writer Nicholas Wade reported in a 2010 article titled 'Adventures in Very Recent Evolution',

> Many have assumed that humans ceased to evolve in the distant past, perhaps when people first learned to protect themselves against cold, famine and other harsh agents of natural selection. But in the last few years, biologists peering into the human genome sequences now available from around the world have found increasing evidence of natural selection at work in the last few thousand years, leading many to assume that human evolution is still in progress. (Wade, 2010)

5 *Perspectives on Psychological Science* published three accompanying commentaries on Hunt and Carlson (2007). Although two present some broad criticisms of research on racial differences in IQ, the commentaries praise Hunt and Carlson's article as offering 'sensible guidelines for the conduct of research on group differences in intelligence' (Brody, 2007) and as 'a major work of … high caliber', 'sensible, well-written, and balanced' (Sternberg and Grigorenko, 2007). None of the commentaries, nor any of the dozen or more scholarly publications that have since cited Hunt and Carlson (Google Scholar, 29 July 2011), remark on their striking claim that molecular research on group differences in intelligence could lead to BiDil-like therapies for race-based cognitive deficits. To my knowledge, however, no one is taking serious steps toward developing such a drug at this time.

6 See Proctor (2003) for a disciplinary and intellectual history of what Proctor calls 'the recent emphasis on recency' (p. 227) in the fields of archaeology, paleontology and molecular anthropology.

Examples of research on recent positive selection for gene variants in human racial and ethnic groups include the 2010 claims that Tibetans possess different variants of the genes controlling oxygen capacity, allowing them to flourish at high altitudes (Simonson *et al*, 2010; Yi *et al*, 2010), and that southern Chinese carry variants that function to help the kidneys more efficiently rid the body of alcohol's toxins (Peng *et al*, 2010). These genes, the authors claimed, cause the so-called 'Asian flush reaction'. According to Wade, searches for similar examples of recent positive selection unique to particular racial or ethnic groups are underway in Eskimos, Bolivians and other populations living in extreme environments.[7]

Applied to brain and intelligence genes, and against the backdrop of human variation research on genetic differences among racial and ethnic groups, research on recent positive selection in human populations has potentially explosive implications. A long-standing hypothesis of the evolution of modern human behavior, attributed to anthropologist Richard Klein, holds that although *anatomically* modern humans arose in Africa, *behaviorally* modern humans arose in Europe, through a genetic-neurological change undetectable by fossils (Klein, 1989, pp. 343, 358–359, 397). There is a conviction among many brain and behavioral genetics researchers that human genes involved in intelligence likely continued to evolve among modern humans after the migration from Africa and that populations with different genetic ancestries may have distinctive intellectual strengths as a result of this ongoing process.

Genetic anthropologist Henry Harpending and colleague Gregory Cochran write in their 2009 book *The 10,000 Year Explosion: How Civilization Accelerated Human Evolution* that, 'The obvious between-population differences that we knew of a few years ago were only the tip of the iceberg' (Cochran and Harpending, 2009, p. 20); 'The most interesting genetic changes are surely those that change minds rather than bodies' (ibid, p. 54). Citing the microcephalin gene as an example, Harpending and Cochran carve a vision of a bold new science of the evolution of modern human behavioral differences. At the center of their argument is the notion that genetic changes caused the 'explosion' in European, Middle Eastern and Asian cultures after their exit from Africa. 'Obviously, something important, some genetic change, occurred in Africa that allowed moderns to expand out of Africa and supplant archaic species' (ibid, p. 31), they assert. This genetic change, they argue, must have yielded enhanced intelligence for those who carried the favored alleles.

The idea that there may be 'brain genes' continuing to evolve in human populations, however speculative and racially tinged, is a compelling narrative, appealing to many, as well as a provocative media-ready hypothesis. A 2005 *Science* news article reporting on Lahn's findings was headlined, 'Evolution: Are human brains still evolving? Brain genes show signs of selection' (Balter, 2005). The article was accompanied by an image of a muscular, nude Caucasian male, head shorn to exaggerate the size of the cranium, posed in profile with chin resting on fist as 'The Thinker', and captioned, 'Big thinker? Certain forms of two brain genes may confer a selective advantage'. The consistent, if often implicit, message of genetic

7 It should be noted that such claims frequently present, as scientific fact, a simplistic and uncritical picture of present-day racial and ethnic groups as reflecting known population genetic substructures with distinctive, unbroken histories. This picture of the genetic basis of race and ethnicity is at best an idealization of human population variation and, at worst, a crude concretization of human folk racial conceptions in genetic terms.

claims about recent positive selection and intelligence is that this research will reveal gene variants that explain why or how European and Asian peoples are more evolved, more 'complex' or 'higher', or somehow more specialized for certain tasks than other populations.

## Molecular evolution of the human brain, cognition and culture

A third guiding and motivating hypothesis of work in the emerging field of evolutionary cognitive genetics holds that clues to the evolutionary history of the human brain, cognition and culture may be found in studying genetic differences in the brain and in intelligence among contemporary humans. Two streams of research converge within this area of study: inter-species comparative genomics of the brain, and 'big brain' theories of the evolution of human cognition.

The ability to compare the genomes of modern humans, primate species (such as the chimpanzee) and recent human cousins (such as the Neanderthal) is presently reinvigorating evolutionary studies of the origins of human cognition.[8] As Vallender *et al* write,

> Decades of research have made important strides in identifying anatomical and physiological substrates underlying the unique features of the human brain. By contrast, it has become possible only very recently to examine the genetic basis of human brain evolution. Through comparative genomics, tantalizing insights regarding human brain evolution have emerged. (Vallender *et al*, 2008, p. 637)

Contemporary studies of the genetics of human intelligence invoke these comparative genomic studies. Brain genes or gene variants identified as unique to humans through these comparative genomic studies are considered candidate genes for exploring human variation in cognition and intelligence. As Gilbert *et al* (2005) write:

> … the search for the genetic basis of human brain evolution is not only relevant to understanding the emergence of *H. sapiens* as a species, but might also shed light on the differences in brain phenotype – such as brain size and organization, cognitive abilities, personality traits and perhaps even psychiatric conditions – among individual humans. (Gilbert *et al*, 2005, pp. 581–582)

This comparative genomic framework for understanding the origin and evolution of the human brain and cognition holds that the genetic changes correlating with cognitive differences between different species (interspecies comparisons) are valid hypotheses for locating the biological basis of cognitive-intellectual differences among contemporary human populations and individuals (intraspecies comparisons) – and vice versa. Lahn has

8 For example, the publication of the 2010 draft sequence of the Neanderthal genome has reinvigorated debate over whether Neanderthals and Eurasians exchanged genetic material (Green *et al*, 2010). Evidence in favor of this hypothesis is seen as bolstering claims about unique gene variants and recent positive selection in non-African populations. In his article, 'Genomics Refutes an Exclusively African Origin of Humans' (Eswaran *et al*, 2005), Harpending and coauthors advance the idea that infusions of genes from other hominids occurred after humans left Africa, and that these genes may be implicated in racial and/or cultural differences between modern humans. Indeed, Lahn has suggested that the advantageous microcephaly gene variants may have originated from introgression with Neanderthals or another hominid species (Evans *et al*, 2006; Thornton and Woods, 2009).

published a four-stage 'scheme for studying human evolution' using this method:

1. The first stage requires large-scale comparisons of genes across several strategically selected species.
2. In the second stage, outlier genes … such as those with highly elevated rates of evolution in primates are compared over a much wider range of taxa that includes both non-primate and primate species.
3. In the third stage, genes showing the most interesting evolutionary patterns are subject to polymorphism studies in humans.
4. In the final stage … polymorphisms identified in humans, especially those associated with signatures of positive selection, are subject to association studies to examine whether they correlate with phenotypic differences among individual humans. In the case of brain-related genes, phenotypes such as brain size and morphology, cognitive abilities, personality traits and even psychiatric conditions can be investigated. (Gilbert *et al*, 2005, pp. 585–586)

Buttressing comparative brain genomics research is a particular theory of the evolution of the human brain. The *encephalization hypothesis* predicts that the higher the ratio of brain size to body size, when looking at primate species, the more advanced, complex or human-like one may expect the primate to be. According to this widely held hypothesis, enlargements and structural developments in the brain were the principal drivers of the evolution of primate species from simple mammals to complex primates with rich cultural and social practices. On this theory, intellectual advances – language, tool-making and complex social systems – distinguish modern humans from their ancestors, and these advances came about in part due to enlargement of the human brain (Klein, 1989). Applying this interspecies hypothesis about the genetics of brain and cognition to intraspecies comparison among humans leads to the notion that differences between the races may be explained, like the hierarchy of primates and mammals, by differences in the genetics of brain development.

The recent book *Big Brain: The Origins and Future of Human Intelligence* (2008), by UC-Irvine psychiatrist Gary Lynch and Dartmouth computational brain scientist Richard Granger, explicitly connects comparative evolutionary genomics of the brain in humans, primates and mammals to a genetic determinist model of intelligence and to a theory of the genetic basis of contemporary variation among groups and individuals. Lynch and Granger suggest that genomic and evolutionary studies of the brain lead inevitably to the conclusion that there will be 'group differences' in intellectual ability. They argue that just as we observe different characteristic behaviors among primate species, we should expect that people with different racial ancestries, and therefore different brain genetics, will have different cognitive or behavioral qualities:

> … Different groups of people have different mixtures of genetic features. Slight gene changes can give rise to differences in brain path connectivity. Differences in brain paths can affect the ease with which certain behavioral functions may be performed. The implication is clear: innate brain connectivity differences can lead to individual and group differences, with disparate talents arising from various connectivity patterns. (Lynch and Granger, 2008, pp. 124–125)

Many researchers are now suggesting that genes involved in the evolutionary ancestry of the human brain may have, in recent human history, diverged in different directions, accounting for variation in cognition and culture among modern humans (Klein, 1989; Gilbert *et al*, 2005; Cox *et al*, 2006). This coupling of evolutionary models of the differences between primate brains with research on genetic variation in the modern human brain represents a powerful new framework for studies of genetic differences in intellectual ability among races and ethnicities. Investigations into recent positive selection for microcephaly gene variants show this framework in action.

## The Microcephaly Case

Microcephaly is a rare genetic disorder of reduced prenatal brain growth. In 2005, Lahn, a human genetics professor at the University of Chicago, reported in the journal *Science* that *variants* of two genes associated with microcephaly, *ASPM* and microcephalin, were extremely common in Eurasian populations and rare in sub-Saharan Africans (Evans *et al*, 2005; Mekel-Bobrov *et al*, 2005).[9] Lahn and coauthors, some of whom subsequently distanced themselves from the papers' claims, argued that these gene variants had rapidly arisen and proliferated among Eurasian populations after migration out of Africa and during the period in which humans began to migrate across the globe and establish the first large urban and agricultural settlements. Lahn hypothesized that the gene variants had spread quickly among these populations because, in contrast to microcephaly variants at those loci that decrease brain size, the favored variants increased the brain size, and hence intelligence, of those who carried them.

Provocatively, Lahn further suggested that the widespread frequency of these gene variants in non-African populations may help to explain the civilizational and cultural success of Eurasian populations, including the spread of domestication and the development of cities and written language. Lahn and coauthors wrote of the gene *ASPM*, for example, that 'the age of haplogroup D and its geographic distribution across Eurasia roughly coincide with two important events in the cultural evolution of Eurasia – namely the emergence and spread of domestication from the Middle East ~10,000 years ago and the rapid increase in population associated with the development of cities and written language 5,000 to 6,000 years ago around the Middle East' (Mekel-Bobrov *et al*, 2005). Of microcephalin, they wrote, 'We note that the age of haplogroup D coincides with the introduction of anatomically modern humans into Europe about 40,000 years ago, as well as the dramatic shift in the archeological record indicative of modern human behavior, such as art and the use of symbolism (that is, the "Upper Paleolithic revolution")' (Evans *et al*, 2005).

Lahn's research on microcephaly gene variants illustrates how genomic methodologies and the transdisciplinary practices of the contemporary biosciences are yielding genetic findings about the evolution of human cognition with implications for our understanding of racial variation in IQ. In the following, I analyze the methods and empirical claims of *ASPM* and

9 I focus my discussion here on Lahn, the primary author of the papers and the principal intellectual architect and defender of their claims. Two coauthors, genetic anthropologist Sarah Tishkoff, then of the University of Maryland, and population geneticist Richard Hudson of the University of Chicago, were not students or postdocs in Lahn's lab. These external coauthors consulted on the elements of the papers having to do with the signature of positive selection. They distanced themselves from the papers after publication, claiming that they had not signed off on the papers' conclusions about the role of the gene variants in the evolution of modern human behavior, language and culture (Richardson, 2010).

microcephalin research. As I will show, although many of the specific claims asserted in Lahn's original papers were subsequently debunked, their fundamental assumptions and larger explanatory frameworks remain uncontested and widely accepted.

## *ASPM* and microcephalin: Genes for big brains

The leading researcher on the clinical genetics of microcephaly and discoverer of the *ASPM* and microcephalin genes, Cambridge, UK, geneticist Geoffrey Woods, has argued since the late 1990s that microcephaly presents a model for the origin and evolution of the human brain and cognition (for example, Cox *et al*, 2006). Woods's claim is now foundational in the literature (Bond *et al*, 2002; Zhang, 2003; Evans *et al*, 2004a, b; Wang and Su, 2004; Ponting, 2006). A widely cited 2001 review on the 'Molecular genetics of human microcephaly', for example, begins by asserting that, 'Identifying … microcephaly genes promises to elucidate important causes of mental retardation as well as the normal development and evolution of the human brain' (Mochida and Walsh, 2001, p. 151). Lahn, following the lead of the field, asserted in his 2005 papers that *ASPM* and microcephalin were validated genes 'determining' and 'controlling' human brain size.

The force of acceptance of a theoretical framework for approaching the genetics of human intellectual differences may be assessed by the ease with which it is accepted despite the lack of original empirical studies – and ample contradictory evidence. In fact, there was no evidence of an association between the alleles and either IQ or brain size. Based on what was known about the actual role of the microcephaly gene loci in brain development in 2005, it was not appropriate to describe *ASPM* and microcephalin as genes controlling human brain size, or even as 'brain genes'. The genes are not localized in expression or function to the brain, nor specifically to brain development, but are ubiquitous throughout the body.[10] Their principal known function is in mitosis (cell division).[11] The hypothesized reason that problems with the *ASPM* and microcephalin genes may lead to small brains is that early brain growth is contingent on rapid cell division of the neural stem cells; if this process is disrupted or asymmetric in some way, the brain will never grow to full size (Kouprina *et al*, 2004, p. 659; Ponting and Jackson, 2005, p. 246).

The further assertion that larger brain size leads to higher intelligence was also not grounded in empirical evidence. Studies of the association between brain size and intelligence have a long history, made notorious by Stephen J. Gould's reanalysis of the empirical and interpretational slippages of the nineteenth century racial craniometry of Samuel Morton and Paul Broca (Gould, [1981] 1996). Despite much effort, scientists have never demonstrated an association between brain size and normal variation in intelligence in humans.[12]

10 Microcephalin is expressed in the fetal brain, but as Jackson *et al* (2002) report, 'A similar level of expression is also present in fetal liver and kidney, and transcripts are detectable at low levels in a range of other fetal tissues, as well as in a number of adult tissues' (p. 139).

11 *ASPM* and microcephalin are involved in spindle pole organization and orientation during mitosis, including related processes, such as DNA damage repair, chromosome condensation, microtubule dynamics, centrosome maturation and cohesion, and centriole biogenesis (Bond *et al*, 2002).

12 The lack of correlation between brain size and IQ is demonstrated by the so-called 'paradox of sex'. Though women's brains are on average 100 grams lighter than males when corrected for body size, men and women show negligible or no differences in IQ. Some continue to claim, however, that there is a small correlation between brain size and IQ, and that Africans have, on average, smaller brains (Rushton and Ankney, 2009).

Unsurprisingly, Lahn's postulated association between *ASPM* and microcephalin, brain size and intelligence in different racial/ethnic groups was refuted by subsequent studies, including one by Lahn's lab. Several groups raced to test Lahn's hypothesis, using a variety of methods ranging from IQ tests to brain imaging studies to assess possible correlations between the gene variants and intelligence in different racial and ethnic populations. Six such studies have appeared in print since Lahn's 2005 *Science* papers; none have found a correlation between the gene variants and either brain size or any measure of intelligence (Dobson-Stone *et al*, 2007; Mekel-Bobrov *et al*, 2007; Rushton *et al*, 2007; Timpson *et al*, 2007; Bates *et al*, 2008; Maghirang-Rodriguez *et al*, 2009).

In this case, the assumption that a gene that, if lesioned, causes cognitive deficit, may be expected to, if normal or enhanced, lead to enhanced intelligence, cohered what would otherwise be a wholly unpublishable speculative hypothesis. While the hypothesized correlation was in this case disproven, discussion of Lahn's claims has focused on their empirical disconfirmation, not on their underlying assumptions. That a gene implicated in brain development, function or disorder is a viable target for genetic research on human intelligence is, it seems, accepted as a plausible claim, sufficient for publication, in work at the intersection of brain genomics, human population genetics and studies of human intelligence. Yet, as the microcephaly case shows, association with brain development, cognitive impairment or psychological disorders is not sufficient to establish a gene as a candidate determinant of normal variation in intelligence and cognition. This points to a need for critical discussion among genomicists, and brain and behavior researchers of the relevant disciplines, to clarify the assumptions, aims and ethics of this emerging research.

## Recent positive selection in non-Africans

Lahn and coauthors further claimed that microcephaly gene variants *ASPM* and microcephalin had undergone positive selection among subgroups of modern humans since migration out of Africa. Modeling and validating recent positive selection in humans is a controversial area of human evolutionary population genetics (Akey *et al*, 2004; Sabeti *et al*, 2006; Akey, 2009). Methodologies are still nascent and often vigorously contested. Efforts to demonstrate recent positive selection in human populations are, as a group of leading medical and population geneticists warned in 2006, 'fraught with methodological challenges' (Sabeti *et al*, 2006). To argue that an allele has undergone recent positive selection, one must show a sudden historical increase in the variant's frequency in a particular population and one must tie that increase in frequency to fitness advantages conferred by the allele with respect to selective forces in the environment. It is extremely difficult to distinguish changes in allele frequency resulting from demographic history – migration, bottlenecks, founder effects and random drift, for example – and changes resulting from selective forces. Moreover, it is challenging to empirically validate a hypothesis that a gene variant specifically enhanced the fitness of those who carried it in the distant past.

There are well-founded cases of recent positive selection in humans, such as lactase persistence. Most modern humans are not able to proficiently digest dairy after infancy, but some individuals who carry variants of the genes for lactase, the enzyme that breaks down dairy proteins, are able to persist in drawing nutritional value from dairy products into adulthood. Half a century of genetic research has confirmed several examples of selection for these gene variants in particular populations of human within the last 10 000 years. In

**Table 1:** Comparison of evidence for recent positive selection for lactase persistence genes and for microcephalin/*ASPM* genes

| *Measure of positive selection in last 10 000 years* | *Microcephalin/ASPM haplotype D* | *Lactase persistence genes* |
|---|---|---|
| Large frequency differences among groups by Fst, Pexcess and long-range haplotype measures | No – modest and controversial signature of positive selection by some measures | Yes – strong signature of positive selection by all methods and measures |
| Confers validated phenotypic advantage | No | Yes – nutritional |
| Correlation of allele with phenotype using *in vitro* and lab studies | No | Yes – well-validated metabolic pathway |
| Selective story rooted in historical, geographical and/or demographic evidence | No – loose selective story not well-grounded in history, geography or demography | Yes – dairy farming settings; arose independently in these contexts |
| Specific estimate of time of selective sweep | No – large error bars of tens of thousands of years | Yes – localized to 7000–10 000 years ago |
| Validated in large, diverse population samples | No | Yes |

*Source*: Compiled by author.

northern Europeans, selection occurred after colonization of Europe and in the setting of dairy farming (Bersaglieri *et al*, 2004). Today, approximately 90 per cent of Swedes and Danes carry these lactase persistence gene variants. In a case of convergent selection, different lactase persistence gene variants independently arose in East African populations that subsisted on dairy-based agriculture within the same time frame as northern Europeans. Today, approximately 90 per cent of Tutsi carry these gene variants. East Asians, in contrast, rarely carry lactase persistence gene variants (Tishkoff *et al*, 2007).

Cases such as the lactase gene stand as the central pillar of evolutionary cognitive genetics claims. If it can be so in the case of lactase, evolutionary cognitive geneticists ask, why not in the case of so-called brain or cognition genes?[13] Yet there are critical differences between a case such as positive selection for lactase gene variants and positive selection for brain, behavior, cognition or intelligence gene variants among human populations. As summarized in Table 1, the case for recent positive selection for Lahn's microcephaly gene variants is not comparable to that of lactase persistence genes. Lactase persistence genes show strong statistical signatures of positive selection by all available methods and measures. The nutritional advantage of the gene variants and their role in the relevant metabolic pathways has been well-validated and the gene variant has been correlated with lactase persistence in large-scale and diverse epidemiological studies, and *in vitro*, human *in vivo* and laboratory animal studies. The selective story for how these lactase persistence alleles arose is strongly

13 In numerous interviews and publications, Lahn has cited lactose intolerance and skin pigmentation as examples demonstrating the plausibility of recent positive selection for human traits – including cognitive traits. As he wrote in a 2009 *Nature* article, skin pigmentation and lactase persistence show that recent positive selection can produce 'differences among groups … so substantial that the trait displays an inter-group difference that is non-trivial compared with the variance within groups, and the extreme end of a trait may be significantly over-represented in a group' (Lahn and Ebenstein, 2009, p. 728).

rooted in historical, geographical and demographic evidence: lactase gene variants arose in dairy farming settings, even evolving independently in different regions of the world where this context was present. The time of the selective sweep for these lactase persistence genes has been tightly located to 7000–10 000 years ago (see, for example, Akey *et al*, 2004; Bersaglieri *et al*, 2004; Sabeti *et al*, 2006; Tishkoff *et al*, 2007).

In contrast, the two genes at the center of Lahn's 2005 claims showed only modest signatures of positive selection by some measures, and according to many critics, did not adequately rule out alternative demographic scenarios (Balter, 2006; Currat *et al*, 2006; Mekel-Bobrov and Lahn, 2006; Mekel-Bobrov *et al*, 2007; Yu *et al*, 2007; Bates *et al*, 2008).[14] More egregiously, as we saw above, Lahn did not demonstrate that these gene variants confer a validated phenotypic advantage, nor did he conduct epidemiological or laboratory studies to explore the hypothesis that the genes conferred cognitive advantage via enhanced brain size. Further, Lahn's story about why and how the genes were positively selected in non-African populations was not well grounded in history, geography or demography, but relied on loose and sweeping generalizations about human cultural history (Nielsen, 2009). Lahn's estimates of the time at which the gene variants swept to frequency in Eurasian populations had large error bars of tens of thousands of years, also calling into question his selective story. Despite widespread comparisons of the microcephaly variants to lactase persistence, then, Lahn's claims did not, by any measure, substantiate a case of recent positive selection for functional human allele variants comparable to the lactase gene.

The dramatic gap between Lahn's claims about the *ASPM* and microcephalin variants and much better documented cases such as lactase persistence should have been clear, but was not illuminated either in a scientific context or in the media coverage of Lahn's claims. Although claims about recent positive selection unique to different racial and ethnic groups are proliferating, standards regarding evidence of recent positive selection are not well established. 'Brain genes' are uncritically arrayed as peers alongside simple, well-studied changes in response to clear environmental factors, such as skin pigmentation and lactase persistence. Sabeti *et al* predict that 'many more examples [of genes that show signatures of recent positive selection] are likely to be found in the coming years' (p. 1620), warning that

> The field is expanding rapidly, as evidenced by the continual flood of papers claiming new regions as candidates for selection and reporting new methods for detecting selection. It will be a challenge to interpret this new information. (Sabeti *et al*, 2006, p. 1620)

In this wide-open field of play, we may realistically expect many assertions of race or ethnic population-based positive selection for genes putatively related to intelligence, the brain or cognition to be advanced in coming years. The microcephaly case points to the clear need for fundamental discussion among researchers of research standards in studies of recent positive selection in human brain- and behavior-related traits.

14 Lahn's data on population difference in frequency of the *ASPM* and microcephalin gene variants was based on resequencing of the gene loci using a panel of 89 samples (90 in the case of *ASPM*) from the Coriell Institute. The panel included small samples from geographically disparate global populations, labeled as follows: nine sub-Saharan Africans, seven North Africans, nine Iberians, seven Basques, nine Russians, nine Middle Easterners, nine South Asians, eight Chinese (nine for the *ASPM* locus), one Japanese, eight Southeast Asians, six Pacific Islanders and seven Andeans (online supplementary material for Evans *et al*, 2005; Mekel-Bobrov *et al*, 2005, available through www.sciencemag.org).

## Linking brain evolution to group variation in human intelligence

Most provocatively, Lahn's 2005 papers suggested that the intellectual boost provided by new *ASPM* and microcephalin gene variants may explain cultural developments such as domestication, cities and written language that occurred in the past 10 000–50 000 years as humans migrated outward from Africa into Eurasia. Lahn and coworkers appealed to the encephalization hypothesis, discussed above, in developing their argument that *ASPM* and microcephalin gene variants may be implicated in higher intelligence in humans. Perhaps, they argued, the 'normal' functioning gene variant accounts for humans' larger brain size, whereas the microcephaly version of the gene corresponds, atavistically, to an ancestral stage of primate brain evolution. In imagery strikingly similar to figures in nineteenth century craniometry texts, Lahn and colleagues pictured primate skulls alongside normal human brains and microcephalic human brains, to suggest that microcephaly represents an earlier stage in the evolutionary expansion of the primate brain (Figure 2; Dorus *et al*, 2004; Ponting and Jackson, 2005).[15]

Using the paradigm of inter-species differences to reason about variation among humans has a rich history in human biology, and this is not the first time that microcephaly has been invoked to fuel arguments about the evolution of the human brain and of racial differences in intelligence and cognition. Speculation that microcephaly is an 'atavism', or reversion to an ancestral evolutionary state, and that finding the genes for it could unlock the code of the evolution of the human brain, and cognitive differences among the races, reaches back to the earliest studies of microcephaly in the mid-nineteenth century.[16]

In the second half of the nineteenth century, a famous sibling pair of Central American microcephalics was exhibited in the freak shows, anthropological societies and royal courts of North America and Western Europe. As cultural historian Nigel Rothfels (1996) documents, the pair were described as 'representatives of a lost race of Aztecs' (p. 159), providing evidence of the 'missing link' (p. 162) between humans and their ape ancestors. Leading scientists took note. In his 1867 treatise, 'About the Microcephalics, or Ape-People', the renowned German anatomist Carl Vogt suggested that microcephaly was the reappearance of an ape-sized cranium in modern humans and that, as such, 'the "Aztecs" might somehow represent "one of the milestones" of human evolution" ' (Vogt, 1869 [English trans.]; Rothfels, 1996, p. 166). The pathologist Rudolf Virchow, one of the foremost biomedical scientists in Germany and a member of the Berlin Anthropological Society, measured the siblings' skulls 28 ways and compared them to apes. Disputing Vogt, Virchow argued that microcephaly was merely a pathology of arrested brain development. Virchow's account was largely accepted by the late 1880s, according to Rothfels (p. 168; Virchow, 1877). Microcephalics, however, continued to be described in racialized terms, and as closer to apes, for at least half a century. A 1922 article in *The Journal of Heredity*, for example, referred to microcephalics as 'pinheaded, anthropoid, simian, theroid, pithecoid, atavistic, foxy, apish, mimics, etc.' (Berstein, 1922, p. 30), and described one microcephalic, 'Freddy', as an ' "Aztec" like youth' (p. 31). Similarly, the 1924 book *The Mongol in our Midst* (Crookshank, 1924) described 'mongolism', now known as Down Syndrome, as a

15 For example, microcephaly has been described in the contemporary literature as an 'atavistic brain size reduction' (Bates *et al*, 2008, p. 690), 'atavistic – a "throwback" disorder' (Gilbert *et al*, 2005, p. 581), and 'an atavistic disorder' (Jackson *et al*, 2002, p. 136; Wang and Su, 2004, p. 1131).

16 For a history of speculations about the relationship between head size, race, class and intellectual ability before the nineteenth century, see Goodey (2005).

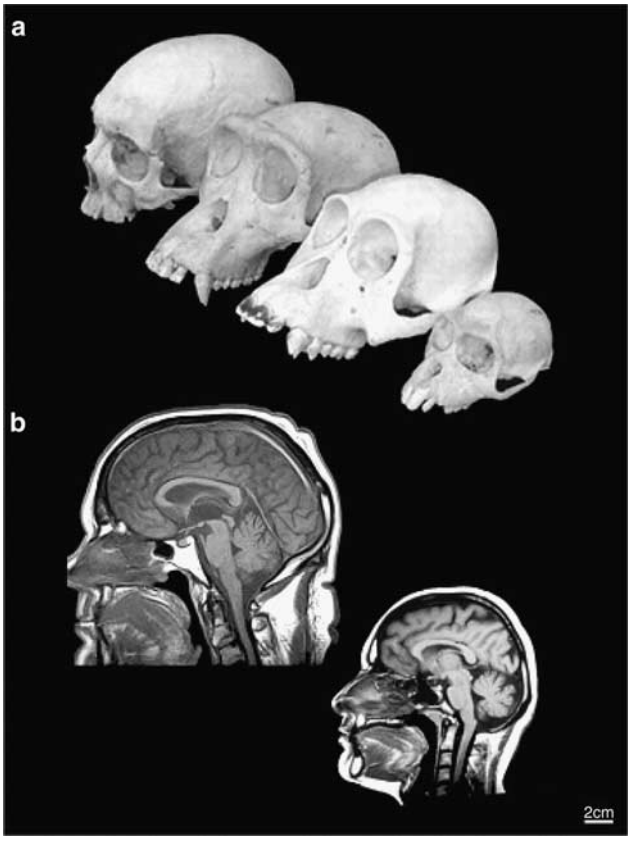


**Figure 2:** 'Comparison of the evolutionary expansion of the primate brain with the reduction of brain size seen in microcephaly'. Reprinted from *Current Opinion in Genetics and Development*, Copyright (2005), with permission from Elsevier (Ponting and Jackson, 2005, p. 243).

'Mongolian atavism' to the orangutan type – and microcephaly as a 'negroid atavism' to the gorilla type (see also Down, 1866; Ireland, 1877; Miller, 1996).[17]

17 The use of the term 'atavism' by contemporary brain genomicists in the microcephaly case is striking not only because of this painful history, but also because today 'atavism' is not widely accepted as a term with any precise technical meaning in medicine or evolutionary biology. As the anthropologist Ashley Montagu wrote in 1945, 'as a concept to account for the appearance of certain physical characters or forms of behavior, "atavism" belongs in the Academy of Discarded Curiosities' (p. 133). Although today developmental biologists continue to study anomalous human characteristics such as small tails, webbed toes or extra nipples, these are understood primarily as evidence of 'the developmental plasticity that exists within embryos and the relative ease with which development can be switched from one program to another' (Hall, 1984, p. 118), rather than as direct and compelling evidence of the organism's evolutionary past. In any case, given what is empirically known about the etiology and pathology of microcephaly, there would seem to be little reason to conceive of microcephaly as an atavism. The resemblance in size (though not structure) of the microcephalic brain to a lower primate brain is, of course, not sufficient evidence of atavism. Moreover, all evidence indicates that microcephaly is not the result of aberrant expression of normally muted ancestral developmental processes, but a disorder caused by a loss-of-function mutation in a gene essential to early cell division in neurogenesis.

Lahn's claims about race, microcephaly genes and intelligence recall, if unwittingly, these long-standing racialist and racist narratives about cognitive and cultural differences among the races, now in the language of genomics. These include: claims that descendents of peoples who migrated out of Africa are more 'evolved' than descendents of African populations due to novel environments and genetic events; claims that there are visible, stable and statistically significant biometric differences between African brains and skulls and those of other populations; and applications of the explanatory structure used to explain cognitive differences among primate species (brain size) to cognitive and cultural differences among human populations, defined racially.

Whereas some features of Lahn's claims – such as the notion of microcephaly as an atavism, and the focus on brain size as a key to variation in human intelligence – may ultimately prove unique to the microcephaly case, the structure of the argument is not. As I have shown, many researchers believe that studies of genetic variation in intelligence among contemporary human populations may provide clues to the evolution of the human brain, cognition and culture. In this way, work on the molecular evolution of the human brain is becoming a generative location, protective veneer and justificatory scaffold for new genomic research on group variation in brain and intelligence genes. The microcephaly case suggests the need for renewed critical discussion among genomicists about the practice of reasoning from human brain evolution to group variation – often elided or read as 'racial difference' – in intelligence.

## Community Standards in Evolutionary Cognitive Genetics

The microcephaly case demonstrates how genomic claims about the brain, human origins and human genetic diversity are now becoming implicated in theories of differences in IQ between races. Genes implicated in human brain evolution, neurological disease and development, and human population diversity may easily become intensive objects of study for correlations with variation in race and IQ. Despite continuities with past eras, the postgenomic era of race and IQ research poses especially challenging, and possibly novel, issues for those concerned about the misuse of racist and racialist conceptions of human difference in the biosciences. Molecular biologists are now placed in a new position with respect to claims about race and IQ. Whereas this research previously occurred outside of molecular genomics, largely in the realm of psychology and heritability studies, there is now real potential that the results of mainstream genomic investigations targeting the brain and mental disorders will be implicated in claims around race and IQ. As this work carries the imprimatur of elite molecular sciences and the appearance of confirmation across a range of fields, it is not considered ideologically fringe in the way that previous claims about the genetics of race and IQ have often been.

Here I examine the critical reception of the 2005 claims about microcephaly gene variants within the human genomics research community. As we have seen, Lahn's 2005 papers were not a random aberration; his claims grew out of a community of inquiry and adhered to the standards of his field; indeed, they were richly rewarded with its highest honors. Lahn's claims were peer-reviewed and published in rare back-to-back papers in the journal *Science*, the leading American scholarly publication in the biosciences. They were accompanied by a glowing feature piece on Lahn's research (Balter, 2005). *Science* honored Lahn's findings

as a 'Breakthrough of the Year' in December of 2005 ('HHMI Research on Evolution in Action Highlighted in Science's "Breakthrough of the Year" ', 2005a). This was followed a year later with a full profile of Lahn in the pages of *Science*, describing him as a leading up-and-coming brain genomicist with 'golden hands' (Balter, 2006, p. 1871) in the lab. Though *Science* later published an exchange of 'technical comments' regarding Lahn's claims (Mekel-Bobrov and Lahn, 2007; Timpson *et al*, 2007; Yu *et al*, 2007), the original papers have not been retracted or amended, and *Science* has not offered an editorial venue for discussion of the papers' implications or a defense of whether they should have been published. In 2006, Lahn received tenure from the University of Chicago. Additionally, Lahn holds the distinction of a Howard Hughes Medical Institute (HHMI) researcher, an honor recognizing America's leading medical scientists that allows unprecedented resources and freedom to pursue research without administrative and grant writing demands. HHMI touted Lahn's findings in press releases ('Human Brain is Still Evolving', 2005b) and continues to fund Lahn today.

Lahn and coworkers' reasoning in the 2005 papers was, moreover, fully within the accepted assumptions and methodologies of contemporary genomics, population genetics, and brain, cognition and intelligence studies. As demonstrated in the above discussion, the framing assumptions of Lahn's studies are widely shared by genome scientists: first, that there is likely recent positive selection for human gene variants, some of which may be implicated in intelligence; second, that genes associated with mental disorders are candidates for intelligence genes; and, third, that the study of brain gene variation in contemporary humans may help us to better understand the origin and evolution of the human brain.

Despite the problems with Lahn's findings, it remains a commonplace conviction among many human genomicists that, if not the *ASPM* and microcephaly genes, some genes *will* be found that tell the same kind of story. Indeed, there remains intense continuing interest in validating the hypothesis that *ASPM* and microcephalin play a role in race-based cognitive variation, despite the methodological, empirical and conceptual problems with Lahn's claims outlined above. One recent study asserted that the gene variants identified by Lahn are 'associated with cranial volume variation in Chinese' populations (Wang *et al*, 2008). Another argued that the 'two brain size genes, *ASPM* and Microcephalin' are associated with ethnicities with more complex linguistic tonal patterns (Dediu and Ladd, 2007). Others are seeking the elusive link to intelligence by plumbing sex differences, as in a recent study arguing that the link between the *ASPM* and microcephalin variants and larger brain size is valid, but only in men, not in women (Rimol *et al*, 2010). Although these claims run against the tide of evidence disproving an association between the microcephaly gene variants and normal human variation in intelligence, they testify to the confidence, by many, that such an association will be borne out, and to the diverse and widespread interest and uptake of these kinds of findings among those interested in human variation in behavior and cognition.

The microcephaly case, therefore, offers an opportunity to assess the present vigor of critical discourse and the clarity and transparency of community standards with respect to research on brain genes, race and IQ. As we will see, the reception of Lahn's claims followed predictable and polarized patterns: positive responses ranging from neutral statements of the virtues of academic freedom to racist celebration; damage control focused on limiting misinterpretation of the research and public controversy; and oppositional responses, external to the field, critiquing the methodology, ideology and ethics of the research. I shall argue that the responses of genomicists demonstrate insufficient critical reflection about the acceptable methods,

questions and practices in this emerging field of research. I conclude by arguing for the need to generate contexts in which scientists can undertake rigorous critical dialogue about the standards of these research fields – a practice I call 'transformative conversations'.

## Let the chips fall where they may

Lahn's 2005 papers were greeted with earnest interest within the scientific community, science media, popular media and scientific and political blogospheres. As Huntington Willard, the head of the Duke University Institute for Genome Sciences, commented in *Science*, 'The possibility that our brains are continuing to adapt is fascinating and important' (Balter, 2005, p. 1662). Profiles in *Science* and features in *The New York Times* and *The Wall Street Journal* portrayed Lahn's research as a scientific breakthrough of major importance with implications for our understanding of human origins, brain and behavior, and cultural differences.

Ideological conservatives ranging from *National Review* commentators to the white supremacist magazine *American Renaissance* lauded Lahn's findings as definitive and scientifically credible evidence for the intellectual inferiority of African Americans. In a *National Review* piece titled 'The Specter of Difference: What Science is Uncovering, We Will Have to Come to Grips With', John Derbyshire wrote that Lahn's 'bombshell papers' demonstrated that geneticists 'have been lying through their teeth' about the supposed genetic similarity of all races:

> … [I]f different human groups, of different common ancestry, have different frequencies of genes influencing things like, for goodness' sake, brain development, then our cherished national dream of a well-mixed and harmonious meritocracy with all groups equally represented in all niches, at all levels, may be unattainable. (Derbyshire, 2005)

The *American Renaissance* reported that 'the Chicago scientists … found that sub-Saharan blacks were the most distinct of the racial groups they studied, in that they had a markedly lower frequency of both variants. This is consistent with the distinct black African profile of smaller brains and lower IQ' ('Race Realism Takes a Step Forward', 2005).

Lahn gave credibility to these claims. *The Wall Street Journal* profile reported that Lahn 'personally believes it is possible that some populations will have more advantageous intelligence genes than others. And he thinks that "society will have to grapple with some very difficult facts" as scientific data accumulate' (Regalado, 2006). In the *Science* profile, Lahn is quoted as saying, 'You can't deny that people are different at the level of their genes … This is not to deny the role of culture, but there may be a biological basis [for differences] above and beyond culture' (Balter, 2006, p. 1871). Champions of Lahn's research, often themselves veterans of past debates over race and IQ, praised Lahn's 'courage in pursuing his research' (ibid, p. 1872): ' "There is widespread fear of this [research] among scientists," says geneticist Henry Harpending of University of Utah in Salt Lake City, who has suggested evolutionary explanations for high IQ scores in Ashkenazi Jews' (Balter, 2006, p. 1872; Cochran *et al*, 2006).[18]

18 See Lee (2008) for further analysis of how this discourse of 'courage' functions ideologically in recent debates over the biology of racial differences.

Broad consensus by all involved ultimately held that the best response to Lahn's research is to 'let the chips fall where they may'. Geneticist Chris Tyler-Smith of the Sanger Institute said, 'We should treat these genes just like any others' (Balter, 2005, p. 1663). Michael Hammer of the University of Arizona in Tucson said, 'I say go at it, let the chips fall where they may' (Balter, 2006, p. 1872). Finally, James Madara, Dean of the University of Chicago medical school, said he advised Lahn to 'let the chips lie where they may', and as long as the science is solid, 'don't worry about the implications' (Regalado, 2006).

## Damage control

Other respondents were more critical. They focused their efforts on managing the public fall-out of Lahn's claims. Francis Collins of the National Genome Sciences Institute and Altshuler of the Broad Institute took the lead. Collins circulated Lahn's papers in an email to leading genomicists, seeking critical commentary, and was quoted in *The New York Times* saying that he was 'worried about the way in which these papers will be interpreted' (Wade, 2005). Altshuler similarly stated that, 'We have a powerful responsibility to think about how society will interpret [such work]' (Balter, 2006, p. 1871). Lahn and the University of Chicago acknowledged these concerns. Lahn agreed that there is 'a lot of potential for over- and misinterpretation' (Balter, 2005, p. 1663) and, according to *The Wall Street Journal*, the head of media relations at Chicago's medical school 'helped Dr Lahn with talking points about his research' and instructed him to 'Don't be shy about telling people what it doesn't mean' (Regalado, 2006).

This message about avoiding misinterpretations of the research implied that the studies were sound but might be misread by lay interpreters. In tension with this message, Altshuler and Collins also hinted that Lahn's research had basic methodological and empirical problems. Altshuler described Lahn's claims about the relationship between the gene variants and intelligence as 'wild speculation' (Balter, 2006, p. 1871) and asserted that there is ' "no evidence whatsoever that these [genetic variants] have any effect" on differences between people' (ibid, p. 1873). Yet, here, both Altshuler's and Collins's message wavered, suggesting that while Lahn had published a solid study by the standards internal to his field, the fact that his claims related to human race and intelligence required that they meet, as Altshuler stated, 'a higher standard of proof' (ibid, p. 1871). As Collins was quoted in *The Wall Street Journal*, 'This is not the place you want to report a weak association that might or might not stand up' (Regalado, 2006).

## Oppositional critique

Science studies scholars work within a tradition of oppositional responses to racial science. Oppositional responses to race and IQ research have, in the past, fallen into three principal categories: methodological critique (the science is poor); ideological critique (the science is politics by other means); and ethical critique (the science is immoral or harmful). These approaches, historically represented best, perhaps, by the work of Lewontin (Lewontin *et al*, 1984) and Gould ([1981] 1996) on the one hand, and mainstream bioethics on the other, have been influential, and they remain valid and essential approaches to analyzing genetic research on race and IQ. The discussion of the methodological deficits of Lahn's research that I have presented in this article follows this well-carved critical path. Yet, these oppositional critiques typically do not lead those committed to the central research questions and

methodologies of their field to open their methods and motivating hypotheses to revision. In the microcephaly case, oppositional responses in any form were received as 'anti-science' or as improperly politically motivated, polarizing and solidifying the positions of those under attack.[19]

As the empirical underpinnings of Lahn's 2005 claims unraveled in the year following their publication, Lahn and his defenders portrayed critics such as Collins and Altshuler as 'PC police' with an ideological agenda that is harmful to science and seeks the suppression of his research. Lahn announced that he was ending his research because 'It's getting too controversial' and because 'intellectual "police" in the US make such questions difficult to pursue' (Regalado, 2006).[20] In *The New York Times*, Lahn stated that objections to his research were 'in part scientifically based and in part due to reluctance to acknowledge that selection could occur in a trait as controversial as brain function' (Wade, 2005). In an interview transcribed on the *Science* Gene Expression blog, Lahn asserted that his work had been held to a higher burden of proof: critics 'with a certain political agenda' who are trying to be sensitive to race and ethnicity, he claimed, had gotten 'science and politics … mixed up … I personally feel, like many other scientists, that science should be separate from politics. In particular, *science should meet the same burden of proof regardless of what political implications it might have*' ('10 Questions for Bruce Lahn', 2006, emphasis added).

Using terms such as 'political correctness', 'thought police' and 'political agenda', and asserting that critics wished to hide controversial facts rather than threaten political sensibilities, defenders of Lahn's research drew on a long-standing rhetoric around identity politics and research on race and IQ in the United States, one that his defenders were already mobilizing in anticipation, even before empirical critiques of Lahn's research appeared. Steven Hsu of the Information Processing Blog, for instance, reported Lahn's research as follows:

> Bruce Lahn is at it again. Earlier work from his lab showed that the microcephalin gene (MCPH1), which plays a role in brain development, has undergone strong selection in the last 40k years, with a new variant allele reaching a frequency of 70 percent in Eurasian populations. Shockingly to our politically correct thought police, the frequency in some other populations is much lower. (Hsu, 2006)

Harpending similarly attributed Lahn's troubles to PC surveillance, saying in *The Wall Street Journal* that, 'I think that Bruce doesn't understand political correctness' (Regalado, 2006). Portrayed as, on the one hand, a deer in the headlights, a white-coated scientist struck down by controversies beyond the lab and his understanding, and, on the other, a courageous champion of apolitical science working against the anti-science political agenda of his critics, Lahn emerged from controversies over his research, including trenchant empirical critique and strong condemnation from leading figures, still the 'intellectually fearless and adventuresome' man with the 'golden hands' (Balter, 2006, p. 1871).

19 Sociologist Aaron Panofsky's (forthcoming) ethnographic analysis of the management of controversy in the field of behavioral genetics makes similar observations about the polarizing effects of external criticism and the ineffectiveness of the field in generating forums for the open discussion of research standards and methods.

20 Despite this 2006 pronouncement, Lahn has not ended his work in the field of evolutionary cognitive genetics (Vallender *et al*, 2008; Lahn and Ebenstein, 2009).

## Transformative conversations

As we enter the postgenomic age, there is a growing consensus among science analysts that the race and genetics debates have hit a wall. As rhetoric scholar Celeste Condit writes, 'The debate about the relationship of race and genetics … finds itself in a stale argumentative stasis' (Condit, 2008, p. 390). The strategies of 'letting the chips fall where they may', 'damage control' and 'oppositional critique' have not ultimately promoted critical dialogical practices, values and community standards within the scientific fields in question.

An alternative approach to new research on brain genes, race and IQ would seek to provide contexts in which scientists might engage in vigorous and open debate about community practices, values and standards. I call this strategy one of 'transformative conversations'. The term references philosopher of science Helen Longino's (1990, 2002) emphasis on the ability of a scientific community to take up 'transformative criticism' as a measure of its ability to produce scientific knowledge. Longino argues that it is intersubjective dialogue and criticism – of the kind represented by practices such as peer review – that bestows science with special claims to the production of trustworthy knowledge. Claims produced by a scientific community, she argues, are more or less worthy of description as 'objective knowledge' depending on the robustness of such critical practices, including the willingness of the community to take up criticism and transform its practices in light of it.

Longino presents four criteria for evaluating the objectivity of knowledge produced by a scientific community:

1. First, there must be recognized avenues for the criticism of evidence, of methods, and of assumptions and reasoning;
2. Second, there must exist shared standards that critics can invoke;
3. Third, the community as a whole must be responsive to such criticism;
4. Fourth, intellectual authority must be shared equally among qualified practitioners. (Longino, 1990, p. 76)

These criteria help to diagnose the discursive gaps in the genomics community's reception of Lahn's recent claims about microcephaly alleles, brain size and group differences in intelligence.

Fundamental questions of methods, community standards and accountability went unanalyzed in the debate over Lahn's research: Should the research have been done? Should it have been published? Was Lahn's research held to 'higher standards' than other research in the field, the same standards, or did it not meet the standards in the field at all? What are the standards of the field, and what should they be? What conditions would be required to produce empirically adequate research on the genetics of race and IQ? When are questions ask-able and/or answerable given the theoretical and empirical constraints of present-day genomics and the study of human behavior? And, for whom and for what purposes is evolutionary cognitive genetics research produced?

Metadebate reflecting on such questions is not wholly absent in the scientific discussions around Lahn's research in 2005 and 2006, but its appearance is sparse and muffled. In an interview, Altshuler provocatively quipped that the problems with Lahn's work were 'easily anticipated' (Balter, 2006, p. 1872), suggesting that its publication in *Science* had been premature. *The Wall Street Journal* article quoted legal scholar and medical ethicist Pilar

Ossorio, stating that Lahn is 'doing damage to the whole field of genetics', and cited sociologist Troy Duster warning, with respect to the Lahn studies, 'that scientists will interpret data in ways that fit their prejudices' (Regalado, 2006). Some scientists also speculated that the Lahn case represented an indictment of the reward structure of the contemporary biosciences. Lahn's papers, they suggested, are an example of the style of scientific claim-making encouraged by major journals like *Science* and the entrepreneurial structure of contemporary academic genomics. Harvard geneticist Richard Lewontin, for instance, speculated that, 'These two papers are particularly egregious examples of going well beyond the data to try to make a splash' (Balter, 2006, p. 1872), and with respect to the controversy surrounding Lahn's research, Lahn's Chicago colleague Martin Kreitman suggested that, 'Bruce is in a hurry to be famous' (Regalado, 2006).

The community of those whose work carries implications for genomic claims about group-based differences in intelligence and cognition must find ways to openly discuss community standards while entertaining diverse critical viewpoints. In theory, such conversations might take place in extended exchanges in the pages of academic journals, at the venue of a major disciplinary conference, or at a special forum assembled by a leading national or international science body. To meet Longino-like criteria of robust critical and transformative dialogue, these conversations demand serious, unbounded reflection on the meta-ethical, social, epistemological and methodological standards within the field of human population genetic and genomic research on the brain. Such conversations are not extraneous to science. They are at once ethical and epistemological. They cannot be adequately undertaken by interested outsiders alone. They are, or should be, at the very heart of the scientific method and the efforts of scientists to produce the most empirically adequate and objective knowledge.

Sensitive new research in evolutionary cognitive genetics and related fields demands that researchers and interested science observers vigorously engage in transformative conversations about the community standards of emerging postgenomic race and IQ research. It is possible, however, that the current structure of postgenomic bioscientific research presents distinctive roadblocks to transformative scientific conversations about the community standards of evolutionary cognitive genetics and its overlapping fields. Displacing the traditional notion of scientific communities as static, bounded and autonomous, the postgenomic biosciences are defined by their speed, transdisciplinarity and commercial context (Thacker, 2005; Barnes and Dupré, 2008). This raises questions such as: What is 'the research community'? Who is included in the conversation? In what space might such a conversation take place? What would consensus look like? And, how would this labor be rewarded?

## Conclusion

The purpose of this article has been to describe, for a broad science studies audience, an emerging confluence of research trajectories that may lead to a reinvigoration of genetic claims about differences in intelligence between racial groups, to analyze the central claims of and responses to this research, and to motivate and imagine alternatives to polarizing oppositional critique. A convergence of contextual factors, technologies and research frameworks in the genomics of human brain and cognition has generated a new postgenomic model for study of race and IQ. At the center of this model are three unassuming, widely shared and, for those

who accept that there may be genes that influence variation in normal human intelligence, relatively uncontroversial premises: that brain genes are candidate human intelligence genes; that genes involved in human intelligence may have undergone recent positive selection in contemporary human populations; and that genetic variation among contemporary human populations may provide clues to the evolution of the human brain, cognition and culture.

We cannot tell for sure whether evolutionary cognitive genetics is a passing trend or the groundings of a new invigoration of race and IQ claims, but the toolkit is robust enough, and the context permissive enough, to warrant careful advance consideration of the potential proliferation of claims about genetic differences in cognition between traditionally defined racial and ethnic populations. Indeed, many working in the various research fields discussed herein openly predict that in the area of genetic variation in intelligence 'the genes are coming'.[21] Based on the case study of Lahn's 2005 claims about microcephaly genes, race, brain size and intelligence, I have argued that there is a lack of rich discussion around the implications of new genomic research relevant to race and IQ that could lend itself to transformative conversations and the articulation and refinement of community standards for conducting this research. Mapping the disciplinary, explanatory and discursive terrain of this emerging area of research, this article invites such a conversation.

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21 Nicholas Wade reported in a 2005 article in *The New York Times* that Lahn 'expected more such allele differences between populations would come to light, as have differences in patterns of genetic disease. "I do think this kind of study is a harbinger for what might become a rather controversial issue in human population research," he [Lahn] said' (Wade, 2005).

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