

How Jews Became Smart: Anti-“Natural History of Ashkenazi Intelligence”¹

R. Brian Ferguson

Department of Sociology and Anthropology, Rutgers-Newark
bfergusn@rutgers.edu

Abstract

Natural History of Ashkenazi Intelligence (NHAI) provides a novel answer to a long-standing question: why do Jews of Ashkenazi ancestry carry so many recessive genes for harmful conditions? It argues that in heterozygotes, these alleles substantially increase intelligence. For 800 years, Ashkenazi were confined to professions demanding high cognitive skills. Those with the alleles prospered, and had more surviving children, thus selecting for the alleles in the population. This thesis has received widespread media and web attention, and represents a growing tendency to explain psychological differences between populations as due to different genes.

This article challenges NHAI, showing so many points of improbability, that the entire hypothesis is highly unlikely. The main criticisms are: (a) Contrary to NHAI’s argument that the inherited conditions are due to selection, population bottlenecks and drift remain strong explanations of their frequency, and consistent with historical information. (b) In NHAI, less than half of all inherited conditions have even a suggested pathway to higher intelligence. (c) The inference that genes which stimulate aspects of neural growth are linked to higher intelligence is pure speculation predicated on a simplistic view of neurological development. (d) The claimed connection between three specific conditions and higher IQ has virtually no empirical support whatever. (e) The demonstrated IQ advantage of Ashkenazi Jews as a whole is less than asserted. (f) The multi-point IQ boosts proposed for specific genes are very inconsistent with current research on the genetics of IQ. (g) Even within the mainstream of IQ research, which emphasizes genetic/biological bases, the extent of Ashkenazi IQ advantage is easily accommodated as due to environment. (h) The “Talmudic Tradition” of emphasizing learning and abstract reasoning provides a clear cultural explanation for higher IQ among Ashkenazi. In Ashkenazi history, NHAI’s assumption that higher intelligence led to greater income is contradicted by (I) a rigid system of social stratification, (j) the critical importance for amassing wealth of capital, social connections, and political patrons, and (k) the absence of any evidence that success in business required anything more than average intelligence.

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Introduction to the Online Posting

This paper was researched and written from March 2006 through January 2007. In February 2007 it was submitted to an anthropology journal, and in December it was rejected. It became clear that this paper was simply unpublishable in anything like its present form. It is well over twice the length of a standard journal article, yet nowhere near a book. It is long because NHAI involves numerous claims about population genetics, neurobiology, psychology, and history, and an adequate critique must adequately cover all those areas. Another problem is the multidisciplinary character of this essay, and the single disciplinary character of reviewers. A population geneticist (apparently) said there should be more about population genetics, while curtailing the rest. An anthropologist (apparently) said it should focus on the anthropological literature criticizing genetic explanations (even though this article is an example of just that). Also, positions are very polarized. One reviewer said I did not recognize the strengths of NHAI, while another said that population geneticists regard it as obviously false, so that it may not merit such a published response.

To my knowledge, no challenge to the thesis of NHAI has appeared anywhere. It lives, on the web, and in people's minds. A few months ago I fell into a conversation with a young anthropologist, who was arguing for the need to bring genetics into the study of culture. I asked why. He replied by citing NHAI as the perfect example of the power of that approach. As for myself, I am deeply involved in other research, and not about to go back to rework this topic. Rather than letting NHAI go unanswered, I took this course, of posting the paper on my departmental web page. I have incorporated numerous clarifications suggested by the journal's anonymous reviewers, and to them I am grateful. Footnotes have been added for this posting, where I refer or respond to substantive points made by reviewers. Footnotes also note relevant research that appeared in the last year and a half, and somehow came to my attention, but no new literature search was conducted. In section 9, I have made some major changes to correct an error pointed out by one reviewer. Otherwise and in substance, the paper stands as completed in February 2007.

One critic's response does need to be addressed here at the start. He identifies himself as one of the authors of NHAI. Naturally, he argues with critics of NHAI's selection theory, and dismisses researchers who support environmental components of IQ. But more significantly, he offers a very different version of the NHAI argument than appeared in print. He says it is not important if most of the Ashkenazi conditions are associated with higher IQ, although that is postulated in the article; and de-emphasizes NHAI's proposition that some conditions give boosts on the order of 5 IQ points. He says the inherited conditions discussed in NHAI are just the "tip of the iceberg" of Ashkenazi intelligence genes, and that there are probably many more besides those that are currently invisible to us. He adds that they did not make this point explicit in NHAI, and should have done so. He also says they do not believe the alleles for known conditions were necessarily selected for intelligence--though that is claimed in NHAI--only that

they were selected for *something*, possibly for something we have not yet guessed.

All are reasonable points, but together they make for a very different presentation than that of NHAI. In this reviewer's version, only some of the inherited conditions might confer small increases in IQ, but no greater than many more alleles not associated with known conditions, while a good number of inherited conditions may have nothing to do with intelligence at all. If that was the message of the published NHAI, I would not have argued with it. Then, I might not have heard about it, because it would not have gotten all that publicity. The paper posted here takes issue with NHAI, as it appeared in print.

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“Natural History of Ashkenazi Intelligence” hypothesizes that Ashkenazi Jews evolved a genetic adaptation to the financial/managerial niche to which medieval gentiles confined them, and that is why their descendants suffer from harmful inherited conditions, and have relatively high IQ. Both come from the same genes. The NHAI hypothesis is built upon assertions about evolutionary population genetics, the neurobiology of the brain, the psychology of IQ, and medieval history of Jews. To evaluate how well this chain of assertions holds together, each of those areas must be considered. I am not a population geneticist, neurobiologist, psychologist, or medieval historian. Having no expertise myself, I read the what experts have written. This paper reports what I found. My conclusion is that the deductive chain of NHAI is made up of nothing but weak or broken links. I wrote this not because investigators in those fields have taken NHAI seriously—from what I can see, few do—but because NHAI became a major story in published media and on the web, it has reached a huge public, and the message that has been communicated is that different human populations vary in their social characteristics because their genes give them different human natures. This is a message that needs careful scrutiny.

1. The Public Story

The most recent example of a society's possible genetic response to its circumstances is one advanced by Dr. Cochran and Henry Harpending, an anthropologist at the University of Utah. In an article last year they argued that the unusual pattern of genetic diseases found among Ashkenazi Jews (those of Central and Eastern Europe) was a response to the demands for increased intelligence imposed when Jews were largely confined to the intellectually demanding professions of money lending and tax farming. Though this period lasted only from 900 A.D. to about 1700, it was long enough, the two scientists argue, for natural selection to favor any variant gene that enhanced cognitive ability (Wade 2006a)

So wrote Nicholas Wade, The New York Times' expert on genetics, in the Sunday Week in Review, March 12, 2006. He outlines a bold new—or very old—view on how to explain cultural

and historical differences. “The Twists and Turns of History, and DNA” claims that “scientists and historians” are coming to a surprising conclusion: that the reason why the people of one culture or time period think and act differently from those of another could be because they are *born* different. Past circumstances led to the evolution of genetic differences, which manifest themselves in patterns that formerly were attributed to culture itself. Different peoples have different human natures.

“Since it looks like there has been significant evolutionary change over historical time, we’re going to have to rewrite every history book every written,” said Gregory Cochran, a population geneticist at the University of Utah. “The distribution of genes influencing relevant psychological traits must have been different in Rome than it is today,” he added. “The past is not just another country but an entirely different kind of people” (Wade 2006a).

The lynchpin of Wade’s story, and the focus of my article, is the essay by Cochran, Hardy, and Harpending (2006), “Natural History of Ashkenazi Intelligence” (NHAI). NHAI offers an entirely untested hypothesis to explain a long-standing puzzle: why Jews of middle and eastern European origins, the Ashkenazi, are afflicted with numerous inherited diseases. It argues that these deleterious alleles—though some are lethal when inherited from both parents—in heterozygotes act as IQ boosters. Because the gentiles who surrounded them for seven or eight centuries channeled Jews into financial and managerial professions which required higher IQs, the smarter heterozygotes were more successful within the Jewish community, and had more offspring than less intelligent, non-carrier Jews. Their reproductive success was so pronounced that it offset the obvious disadvantage of sometimes having children who die young or are disabled. Thus, Jews of Ashkenazi origin have evolved greater verbal and mathematical ability, higher IQ. That is why they do better in school, win so many Noble Prizes, etc.

One might ask why an untested hypothesis was considered science news at all. Yet this was *big* science news. The article quoted above was the second time NHAI was reported in The New York Times, but earlier coverage (Wade 2005) appeared in the Science Times section. The Week in Review represents a much higher profile—that is where important news for all readers appears. The New York Times was not alone in giving serious space to NHAI. Stories also appeared in The Economist (2005), New York magazine (Senior 2005), several other newspapers, and National Geographic News (Owens 2005). Wade takes it up again in his book, Before the Dawn (2006b; and see Weiss and Buchanan 2006). More recently, after noting another article about recent evolution of lactose tolerance in East Africa, the Times editorial column itself weighed in to support the idea that broad cultural differences may be due to genetic adaptation to past situations: “The dynamism of human culture has always seemed to move faster than evolution itself, but this discovery suggests otherwise” (New York Times 2007).²

² See Wade (2007), “Humans Have Spread Globally, and Evolved Locally,” for more about the “new” perspective on lactose intolerance. The frequent commentary on the discovery of a genetic basis to lactose intolerance is curious. Anthropologists and biologists working together had established that adult lactose tolerance was a genetic adaptation to a “milking” subsistence orientation, decades before the genomic era (Johnson 1981; Simoons 1981). I learned about that in my first semester of graduate school. What is being filled in now is the

Prominent commentators lined up behind NHAI. Evolutionary psychologist Steven Pinker has done much to legitimize and publicize NHAI, while remaining carefully agnostic on its truth value. In the Times: “It would be hard to overstate how politically incorrect this paper is,” said Steven Pinker, a cognitive scientist at Harvard... Still, he said, “it’s certainly a thorough and well argued-paper, not one that can easily be dismissed outright” (Wade 2005). His article about NHAI in The New Republic concluded that it “meets the standards of a good scientific theory, though it is tentative and could turn out to be mistaken” (Pinker 2006:27). A blogger (Your Lying Eyes 2006) reported a public talk by Pinker on NHAI (“Jews, Genes, and Intelligence”). “Overall Pinker emphasized the reasonableness of the author’s hypotheses, the generally better quality of the genetic evidence over the environmental, the non-rational basis of much of the opposition, and the paper’s strong foundation in the current state of knowledge.” Pinker is credited with formulating Edge’s Annual Question for 2006: “What Is Your Dangerous Idea... dangerous not because it is assumed to be false, but because it might be true?” His own answer is “Groups of people may differ genetically in their average talents and temperaments.” Of his four illustrations, one is Cochran and Harpending’s argument on Ashkenazi intelligence.

J. Phillippe Rushton--President of the Pioneer Fund and advocate of the idea that black people are genetically inclined to lack of parental caring, criminal tendencies, promiscuity, etc. (Rushton 2000; cf. Tucker 2002)--also likes NHAI. In an article about how Rushton is becoming more mainstream (Duffy 2005), he is said to believe that

work on the Ashkenazim, has lent respectability to his own work. “Here is another ethnic group (the Ashkenazim) that has been identified, genetically, as possessing a higher IQ,” he says. “So if nature has not made every population group in the world exactly equal in mean IQ, if there is one somewhat above, then it’s quite possible to find one or two somewhat below” (pg. 3).

“Evolutionary conservative” Steve Sailer (2005), on his blog calls NHAI “potentially epochal,” and its coverage by the Times and Economist a “startlingly courageous” step toward liberation from “the deathgrip of political correctness.” Even H. Allen Orr (2006:22), who severely criticizes Wade’s (2006b) adaptationist story telling in The New York Review of Books, says of NHAI: “Such a hypothesis is certainly possible; the critical issue is the strength of the empirical evidence.” The article gets hundreds of links through Google. Working on this article, I was a shocked by how many people have heard of this idea. It is a big story.

This paper will show that there is no good reason to believe that the argument of NHAI is likely, or even reasonably possible. The proper response to the thesis of NHAI is neither to ignore it, or attack it on political grounds, but to evaluate it on the merits of its theory and evidence. That is the purpose of this article. My conclusion is that in the step by step construction of NHAI’s argument, flaws at each step are sufficient to bring the thesis down. NHAI continues the tradition of creating adaptive parables on very thin evidence, then relying on fit with popular preconceptions to carry the idea to a broad public. For all its elaboration, NHAI is just another “just so story.”

We begin with an outline of the issues as they existed before the NHAI hypothesis, what Cochran et al. picked up, and what they added. This is necessary because much of NHAI’s

actual biology of it.

persuasive power comes from emphasizing long-standing puzzles. The existence of puzzles is not in question. NHAI's solutions are. After this overview, each element will be critically scrutinized.

2. Existing Issues, and NHAI's Contributions

The Ashkenazi are Jews of central and eastern Europe. In this paper I use the term to apply both to those populations, and their descendants today. Ashkenazi origins are unclear, despite extensive historical and genetic investigation. It is believed that sometime very widely around 600 A.D., Jews moved, probably from the Middle East and/or Italy, into the Rhineland and northern France. Their skills and extensive social ties suited them for long distance trade with Islamic lands and elsewhere. For that reason they were actively recruited by local potentates, who brought them in to promote local commerce. In time, they became more involved in money lending, partly because of Catholic restrictions against lending money at interest. "[T]he story of Jewish usury is a continuous alternation of invitation, protection, protestation, and condemnation" (Parkes 1976:360). In the twelfth and thirteenth centuries, Jews were massacred and/or expelled from lands of England, France, and Germany. Some—how many is an issue—moved east to join existing Jewish communities in Poland and Lithuania, which became the Ashkenazi heartland for 500 years. In the east, many began as money lenders, but over time branched into a variety of financial and managerial professions (Ben-Sasson 1976a; Della Pergola 2001; Encyclopaedia Judaica 1977:VIII 875-880; Jagur-Gradzinski 1997; Ostrer 2001, 892; Parkes 1976; Weinryb 1972).

90% of American Jews are descendants of Ashkenazi (Ostrer 2001, 891). In this paper, American Jews are roughly equated with Ashkenazi, because data on American Jews usually do not discriminate origins. (Globally, about 80% of all Jews are of Ashkenazi ancestry--Motulsky 1995:99). Ashkenazim are burdened with over 40 inherited conditions with Mendelian patterns of transmission—that is, two alleles with simple dominance/recessive expression. A number of these are shared by other populations. Ostrer tallies 18 distinctively Ashkenazi diseases. These conditions fall into five clusters, along with some independent conditions. Besides lysosomal storage diseases—the main focus of NHAI--there are clusters involving DNA repair, glycogen storage disease, clotting factor disorders, and disorders of adrenal steroid biosynthesis. Several of these conditions have more than one mutation with the same effect, making their genetic history even more curious (Ostrer 2001, 893-894).

For over three decades, there has been a very active debate as to why the Ashkenazi have so many inherited diseases (Goodman and Motulsky 1979; Risch et al. 2003, 812; Spyropoulos 1981). Could it be that those mutations, though often lethal in homozygotes (two copies of the recessive allele), conferred a positive selection advantage in heterozygotes (one copy of the recessive allele)? When the fitness of heterozygotes exceeds that of either homozygotic state, a balanced polymorphism may result. In this perspective, Ashkenazi conditions may follow the model of the sickle cell trait—as NHAI claims (Cochran et al. 2006, 666)--where positive selection increased the recessive's frequency because heterozygote carriers enjoy resistance to malaria.

The alternative to positive selection is that these alleles spread among the Ashkenazi through chance, a combination of founder or bottleneck effects, and drift. The concept here is that

the mutations were present in members of a small founding community, or among those who passed through a major constriction or bottleneck in a population's history. Since both founder and bottleneck effects are possible in Ashkenazi history (see section 3), the terms are used interchangeably. In a small founder or bottleneck population, the frequency of alleles may increase by genetic drift. No selection benefit is involved. (Both selection and founder effect arguments share the assumption that gene frequencies are shaped by a history of within-group marriage among the Ashkenazi).

Most of the focus has been on four lysosomal storage diseases (LSDs)—Tay-Sachs, Gaucher, Niemann-Pick type A, and mucopolysaccharidosis type IV (see Goodman and Motulsky 1979; Walkley 2001; Zlotogora 2006). Lysosomes are sack-like structures inside cells, within which enzymes break down old macromolecules into simpler elements for re-use. In LSDs, a mutation leads to an absent or poorly functional enzyme, so the substance it would target accumulates—stores—in cells. The Ashkenazi conditions involve a class of LSDs where sphingolipids accumulate, so NHAJ refers to them as sphingolipid disorders. Accumulation of these compounds can damage a variety of tissues in a variety of ways (Ginns 1985; Walkley 2003).

Tay-Sachs involves degeneration of brain neurons, usually leading death before age three. Estimates of Ashkenazi heterozygote (carrier) frequency are 1:25 to 1:30. However, locations vary, suggesting the importance of founder populations. Heterozygote frequency for Toronto is 1:14, but Boston is 1:37 (Spyrolopoulos et al. 1981:366). Niemann-Pick type A also leads to brain damage, and homozygotes rarely survive 18 months. Heterozygote frequency is about 1:90. The Ashkenazi form of Gaucher (Type I) has no neurological impact. Its expression varies greatly in individuals, and can appear at any age, involving the spleen, liver, bone marrow or other tissues. “In other cases, patients with Gaucher disease may live until old age totally unaware of the disorder until the diagnosis is made incidentally in the course of the investigation of some other health problem” (Beutler 1979:159). It is the most common of the Ashkenazi LSDs, with heterozygote frequency estimates from 1:7 to 1:18. Mucopolysaccharidosis type IV (MLIV) is a less-understood condition, leading to psychomotor retardation and ophthalmological abnormalities developing in the first years of life. Heterozygote frequency is 1:100 to 1:112 (Ostrer 2001:893-894; Zlotogora 2006a;b).³

These four different conditions involve mutations of different genes, but all lead to the accumulation of glycolipids. This is a very curious--some would say impossible-- coincidence, and it has long led to arguments in favor of positive selection. A 1988 article titled “Selection in Favor of Lysosomal Storage Disorders?” concluded, probably yes, without hazarding a guess at the nature of selection (Zlotogora et al. 1988). Jared Diamond (1992, 291), then a commentator for *Science*, considered three of these diseases, adding in the several different mutations known to lead to Gaucher disease, and concluded “lightening has struck Jewish lysosomes not once, not three times, but at least eight times.” A laboratory offering genetic tests Ashkenazi mutations lists 16 different mutations leading to the four conditions, though many are rare (Mayo 2006). It

³ One journal reviewer whose field is lysosomal diseases added information about MLIV. Although it may involve lysosomal disorder, it is not a sphingolipidosis. Its main effects on the brain are reduced axon extension in the corpus callosum and degenerative process in the cerebellum.

is a genuine puzzle. But if the prevalence of these diseases represent the tracks of selection, then selection for what?

Previously it was suggested that these LSDs might somehow increase resistance to tuberculosis, based on apparent differences in Jewish/non-Jewish susceptibility (Myriantopoulos and Aronon 1967; Myriantopoulos and Melnick 1977). A follow up study, however, indicated that if Ashkenazi Jews did have better resistance to TB, it was not associated with carrying the Tay-Sachs allele (Spyropoulos 1981; and Motulsky 1979, 305-307). In the 1990s, the interpretation of genetic researchers was swinging against selection explanations and toward founders, bottlenecks, and drift as the cause of lysosomal and other disorders. NHAI takes direct aim at founder/drift arguments. It has to. If bottlenecks can explain these conditions, then there is no mystery to solve, and there is no need to invoke positive selection.

Another building block of NHAI is the long-noted fact that American Jews do relatively well on intelligence tests compared to non-Jewish white Americans, and do so with an unusual cognitive profile: they score highly on verbal subtests, relatively poorly on performance subtests. Scholars have debated whether, or how much, this relative advantage was due to culture, or to genes. On the cultural side, there was a more than two-thousand year cultural history valorizing scholarship (Patai 1975, 149-156). On the genetic side, several possible selective forces were considered that could have led to more “high-IQ genes:” marriage preferences that conferred greater reproductive success on accomplished students of sacred texts (the “marry a rabbi” hypothesis), the need to cope with a long history of sometimes lethal persecutions, general circumstances of long-term concentration in urban environments, and/or selective survival and migration out of Eastern Europe to America during the holocaust (Motulsky 1979, 307-309). NHAI is not new in proposing that Jews evolved higher intelligence.

NHAI takes a hard line on Ashkenazi intelligence. It opts for high-end estimates of American Jewish cognitive advantage. It comes down strongly on “nature” rather “nurture” as a general explanation of individual and group IQ differences. But what is really novel in NHAI is the substance of its argument about the selection process that led to elevated IQ. NHAI hypothesizes that from about 800 to 1650 A.D., Ashkenazim were forced, by the larger non-Jewish populations around them, into restricted fields of commerce, management, tax farming, and (especially) money-lending; that success in these areas was due to higher intelligence; that the smarter did better and so had more surviving children than others; and that the genes for financial and reproductive success were the same genes that burden Jews with inherited conditions today.

Cochran (et al. 2006, 660) first date this time from around 800 AD, when the Ashkenazi begin to emerge in historical records, up to “1600, after which we think many of the unique selective pressures were relaxed.” Later they put it as “roughly AD 800 to AD 1650 or 1700, [when] the great majority of the Ashkenazi Jews had managerial and financial jobs, jobs of high complexity, and were neither farmers nor craftsmen.” There remain serious questions about how exclusive was this occupational restriction at any time—somebody had to be the butcher—which I will not address. Leaving that aside, Hundert’s (1992, 46-68) discussion of the Jews of Opatow Poland in the 18th century makes clear that the restriction of Jews to cognitively demanding financial and managerial positions was a thing of the past well before 1700. By then Jews made up a very large proportion of the urban population, and large numbers of Jews were engaged as craftsmen or peddlers. Expansion in those “ordinary-intelligence” jobs continued markedly

through the 18th century, and the class of poor Jews expanded. For this paper, the time period when, following NHAI, there was strong selection for intelligence will be 800-1650 AD.

Back to the inherited diseases: A few of those genes, LSD or not, are known or suggested to be involved in neuronal growth. A few of those genes, LSD or not, are claimed to be associated with unusually high IQs. NHAI proposes that *most* of these 18 or so inherited conditions may be associated with elevated neuronal development, and consequently, heightened IQ for heterozygotes. Between 800 and 1650 AD, it claims, higher IQ led to financial success, and financial success led to reproductive success. In the currency of genes, that success outweighed the genetic losses of those homozygotes actually afflicted by the diseases. That is the thesis of NHAI. That is how the high IQ genes became associated with the population. That is how Ashkenazi Jews became smart. Now to examine the different components of this hypothesis.

3. Selection or Drift in Inherited Diseases?

Three recent studies used different methods to consider selection regarding lysosomal storage disorders are considered in NHAI. Risch (et al. 2003, 819; and see Wade 2003) took the position that if LSDs were under positive selection, then they should exhibit different patterns of number of mutations, allele frequencies, and geographic distribution than non-LSDs, i.e. the statistical profile of these measures should be recognizably different from recessive Ashkenazi conditions not under positive selection. They found no major difference between the LSDs and non-LSDs, but they did find different localizations of alleles, suggesting multiple founder events (Risch et al. 2003:815).

Frisch (et al. 2004) evaluate the most common mutation for Tay-Sachs disease in 55 unrelated Ashkenazi individuals. Conserved haplotypes (the specific DNA sequences around the gene) indicate a common ancestor in the 8th-9th centuries. The frequency of this allele shows “the absence of a determinant positive selection (heterozygote advantage)”, and the authors conclude genetic drift is “a robust and parsimonious hypothesis” (pg. 366). Slatkin (2004) statistically tests the founder effect hypothesis, and concludes it is sufficient to account for the frequencies of LSDs.

NHAI takes issue with these studies and conclusions (Cochran et al. 2006, 671-674, 682-684). Comparing 652 neutral genetic markers for Ashkenazi and other Europeans who were not from small founder populations, they found very little difference between the two. From this they infer there is “*no suggestion of any bottleneck at all*” (pg. 672, emphasis added; and pg. 660). “The genetic distance between Ashkenazim and other Europeans computed from IQ is roughly one hundred times greater than the distance from polymorphic markers” (Cochran et al. 2006:673). Ashkenazim are not characterized by a broad genetic difference, as would be expected from a population whose genes came from a small founding group. The difference is in IQ.

NHAI authors dispute Slatkin’s statistical assumptions and analysis (pg. 683). They offer their own statistical test of the likelihood of the functional clustering of Ashkenazi disorders arising by chance, and conclude it is highly unlikely (pg. 685). From this too, they rule out founder effects and drift. They accept Risch et al.’s basic finding of no difference between LSDs and non-LSD patterns, but with an entirely different interpretation of the similarity. “Our hypothesis in this paper is precisely that most of these are the result of the same selective force, on

IQ” (pg. 684). (A few possible exceptions are noted, such as the mutation for cystic fibrosis which might confer disease immunity--Cochran, Hardy and Harpending 2006, 684; Ostrer 2001, 895; cf. Risch et al. 1995, 15). The idea that most of these inherited conditions are IQ boosters is necessary for NHAI to counter Risch et al.’s argument against selection (unless most have some other, unsuspected benefit). But more importantly, only by bringing in non-LSD’s can the frequency of inheritance possibly explain a generally elevated Ashkenazi IQ. The four LSDs would effect approximately 15% of Ashkenazi Jews. But totaling estimated frequencies for 13 conditions, they calculate “the probability of having at least one allele from these disorders is 59%” (Cochran et al. 2006, 675).⁴

The idea that clustering of similar mutations is evidence against drift was actually raised against Risch et al., before NHAI, although on a more limited basis. Zlotogora and Bach (2003), unlike Cochran et al., accept that most Ashkenazi conditions are indeed due to founder effects and drift, but argue the LSDs are different. The common biochemical pathways of the four, combined with the number and frequency of multiple mutations for the same conditions, they argue, indicates the existence of some unknown selective pressure—in other words, the selection argument as it stood pre-NHAI. Risch and Tang (2003) reply that such frequencies and clustering of similar mutations also occur in other populations known for founders. So they note that Romani, roughly contemporaries of Ashkenazi, have a comparable but non-overlapping spread of inherited conditions, including three different sensory neuropathy syndromes. Different Arab populations have a great spread of highly localized founder conditions, and several relatively common diseases with multiple mutations. So the clustering of similar mutations among Ashkenazi may be puzzling, but it is not unusual in founder populations.

Other recent genetic studies not considered in NHAI also run counter to the selection hypothesis. Behar (et al 2004a, 363-364) studying Y chromosomes with more fine-grained data than previously available, find evidence consistent with a bottleneck, but its timing and magnitude cannot be estimated. Behar (et al. 2004b) studying mitochondrial DNA find evidence for an early bottleneck about 100 generations ago, although noting there could be a later bottleneck as well. Behar (et al. 2006, 493) go further regarding mitochondrial DNA, reporting the detection of a small set of only four individual female ancestors, likely from a Hebrew/Levantine mtDNA pool, whose descendants lived in Europe and carried forward

⁴ This paragraph receives special attention in the comments of the NHAI reviewer. Two of his points were noted in the online Introduction: that it is not necessary that most of the Ashkenazi conditions were selected for IQ enhancement, only that they were selected for something; and that it is not necessary for the inherited conditions to adequately account for the posited Jewish IQ advantage, because they are probably only a fraction of all IQ boosting alleles, most of which are entirely unknown. I repeat, this is not the message conveyed in the published article, and it is that article being considered here. His third point is that I do not deal adequately with their theoretical debate with Slatkin. The reviewer writes that NHAI’s statistical analysis demonstrates conclusively that the neutral theory which underlies bottleneck models is wrong. Elsewhere he identifies this as the single most important theoretical claim in NHAI. I am not competent to evaluate this argument, and I hope that a population geneticist will step up to consider it.

their particular mtDNA variants to 3,500,000 individuals in a time frame of <2 millennia. This founding event(s), established here as a dominant mechanism in the genetic maternal history of the Ashkenazi Jews, is a vivid example of the founder effect originally described by Mayr 4 decades ago.

Usher syndrome is a non-LSD condition which leads to hearing and visual loss. Although Ashkenazi do not have a higher frequency of the syndrome than the general European population, about 40% of the Ashkenazi instances are due to a specific allele which is very rare--except among Finns, another population known for founder effects (Ben-Yosef et al. 2003). Alpha-thalassemia is a globally widespread condition, most prevalent in malaria areas, for which it is believed to confer some resistance. But its frequency in a study of Ashkenazi reached the very high level of 1:12, despite the absence of malaria in northern Europe. This is consistent with a founder effect (Rund et al. 2004).

Finally, Service (et al. 2006), although not concerned specifically with inherited conditions, come to conclusions that go against the NHAI claim that, comparing neutral genetic markers for the Ashkenazi and other Europeans, Ashkenazim show no sign of founder effects. This new study compares linkage disequilibrium—an indicator of relatively recent genetic change—and the absence thereof, in the Ashkenazi and an outbred European population (among others). For 2,486 genetic loci along the full length of chromosome 22, the results for the two populations were radically different. The Ashkenazi have much more recent genetic change. They are indeed markedly different from mixed Europeans. Further, the Ashkenazi values fall squarely within the range of other populations known to have small founding populations.⁵ All together, there is a very substantial body of genetic research that goes against NHAI's foundational claim, that there is no evidence of a bottleneck capable of accounting for Ashkenazi inherited conditions.

I should clarify again that, not being trained in population genetics, I am not qualified to evaluate all of these different claims. I am in no position to opine that genetic analysis does, or does not, support the existence of founder/bottleneck effects. My point here is that nine studies by teams of geneticists within the last five years all go against NHAI's foundational position.

Cochran and colleagues consider only genetic data in their assertion of no bottleneck at all. Their assertion is hard to reconcile with the historical record, fraught though it is with unknowns and uncertainties (Della Pergola 2001; Encyclopedia 1971, 875-880; Jagur-Grodzinski 1997; Weinryb 1971). The record, such as it is, seems to indicate at least three major founder or bottleneck periods.

The Ashkenazi originated somewhere between the 4th and 10th centuries around the Rhine Valley, but nothing is known about their numbers. This utterly unknown period is probably the most important for population history. Given the social separation and occupational specialization observed in later history, it is difficult to imagine whole groups of Jews moving into entirely foreign lands. It seems more likely that individuals with capital or commercial assets would be invited or allowed by local potentates. If this was anything like other diasporas, those first established would have brought along family, and over time communities developed. True

⁵ The NHAI-author reviewer attributes the linkage disequilibrium finding by Service et al. to the fact that the Ashkenazi are a population of mixed European and Middle Eastern ancestry.

this is just speculation, but it highlights the real possibility that the Ashkenazi could trace back to a very few people.

After some centuries of growth around the Rhine valley, Jews from this core group gradually moved into what would become England, France, Germany, and elsewhere, appearing in many communities by the late 12th century. From then to the late 14th century, massacres and expulsions greatly reduced those Jewish populations. Large numbers were killed directly, or thrust into existential peril.

The Eastern European Jewish population appears in the late 12th century, from Bohemia to Russia, but concentrated in the future Poland and Lithuania, where later Ashkenazi growth would occur. When they arrive, how, and how many is unknown. The first tombstone date is 1203 A.D. Whatever happened, this must represent only a fraction of the prior Western European Ashkenazi gene pool. Also unknown is how much this Eastern population grew over the next century and a half, and how much it was subsequently reduced by the famines and plagues of 1315-49. (Slatkin [2004, 290] models the population low point as 1348). Weinryb (1972, 9)—the main source of NHAI’s information for differential reproductive success—puts their number in Poland/Lithuania at that point as “a few thousand persons or less” in “a few isolated islands” of settlement. The Encyclopedia Judaica pegs it at 5,000 (1971, 877-88). Jagur-Grodzinski (1997, 80) opts for the low end, concluding the number of extended Ashkenazi-Jewish families which resided at that time in Eastern Europe may have been less than a hundred (assuming 5 person on the average per basic family and 5 basic families on the average per one extended family).” Of course, those families would themselves have their own history of intermarried development.

From 1350, the Polish/Lithuania Jewish population grew. Weinryb’s (1972, 311) very careful assessment of sources, unknowns, and estimates led him to conclude that 150 years later, at the end of the fifteenth century, adding all of the Ashkenazi heartland together, “might bring the total number up to 10,000 or very slightly more... [who] were destined to increase into a community of about three-quarters of a million in less than three centuries.”

Weinryb (1971,10) believes the founding core of East European Ashkenazi was supplemented by substantial immigration from Western lands, and some immigration is clearly indicated in recorded grants. But DellaPergola and Jagur-Grodzinski both find little evidence of large scale immigration, which in the latter’s words would involve historically “trackless blending” (pg. 81-82), unlikely given the fractious nature of known Ashkenazi society. Instead, they see later increases as largely due to growth of the small founding population, which is well-within known growth rates of later Ashkenazi. Factors contributing to locally disproportionate Ashkenazi growth could include better sanitation in food preparation, injunctions for washing and bathing, better care for sick and weak, very young age of girls at marriage, and an absence of celibate religious orders.

No bottleneck at all? From an unknown size founder population (400-800 A.D.), a small stream flowed into East Europe around 1200 A.D. After settling and growing, they were again reduced by famine and plague, to a low around 1350 A.D. This historical evidence fits well with published estimates of the coalescence of eleven Ashkenazi mutation: six from 575-850 A.D. (at 25 years per generation), and two from 1225-1275 A.D. (Risch et al. 2003, 815.) Risch et al. also emphasize the evidence of several localized founders and growth within the Eastern European population, most notably the Lithuanian population, corresponding to two more coalescence dates

around 1700 A.D. (see the discussion of ITD, below). (The remaining published coalescent date is around 1,000 B.C.).⁶

Since all this might seem rather narrow and technical, it is important to recall its significance. The idea that positive selection accounts for Ashkenazi inherited conditions is the key case for the notion of rapid behavioral evolution in human populations. If founder and bottleneck effects can account for the statistical profile of the Ashkenazi conditions, selection need not be invoked. Take out positive selection, and the NHAI thesis is stopped at the start. Several recent genetic research projects have endorsed the existence of founder effects among the Ashkenazi. NHAI says they are wrong. Resolution of this issue will be done by population geneticists. But as we will see, this is only the first of many severe problems for NHAI.

4. Neural Promoters?

NHAI argues that the Ashkenazi genes boost intelligence by stimulating the growth of nerves in the brain. Key for this conjecture are two studies demonstrating that *en vitro*, higher levels of sphingolipids associated with LSDs lead to extension and branching of neurons (Schwartz et al. 1995; Walkley 2003). Those studies themselves do not suggest any heterozygote advantage in intelligence, however. It is NHAI that does that, in a theoretically critical passage that deserves careful evaluation.

Dendritogenesis appears to be a necessary step in learning. Associative learning in mice significantly increases hippocampal dendritic spine density (Leuner et al., 2003), while enriched environments are known to increase dendrite density (Holloway, 1966). It is likely that a tendency to increased dendritogenesis (in Tay-Sachs and Niemann-Pick heterozygotes) or to increased axonal growth and branching (in Gaucher heterozygotes) facilitates learning.

Heterozygotes have half the normal amount of the lysosomal hydrolases and should show modest elevations of the sphingolipid storage compounds. A prediction is that Gaucher, Tay-Sachs and Niemann-Pick heterozygotes will have higher tested IQ than control groups, probably in the order of 5 points (Cochran et al. 2006, 677).

This is the explanatory core of the NHAI thesis, and several points need be made. First, the two studies it cites show that *environmental* stimuli result in dendrite growth, in striking contrast to the later dismissal of environmental impacts on IQ (below). The findings of those two studies are then flipped—from demand to supply side as it were—without any research documentation that chemically stimulated growth augments learning. Second, even if hydrolases

⁶ The NHAI-author reviewer argues these population restrictions are not small enough to fulfill the mathematical requirements of a bottleneck. I would reply that we do not know just how small they were. Weinryb calculates a 75 fold increase from ca. 1500 to 1800. If that rate of growth is projected backward from 10,000 in 1500, to the low point of 1350, it suggests a very tiny starting population indeed. Of course this was added to by later migrants, maybe even some mixing with local gentiles, but we do not know how much or when. Plus, those survivors of 1350 could themselves have had unusual genetics, being descendants of two previous events, the migration eastward, and the original European founding.

are lower, it is not self-evident that this would have any effect on nerve growth. In LSDs, “[i]t is very difficult to quantify residual enzyme function, as very low enzyme activity is sufficient to ensure a normal phenotype” (Maire 2001:59). Third, it should be emphasized for future discussions that NHAI posits about *5 points* elevation for each of these three genes. (No point estimate is offered for other inherited conditions)

Let us accept for argument that heterozygotes do indeed have greater than normal neuronal growth and branching. NHAI’s speculation that this leads to higher IQ might seem plausible—more neural connections, more intelligence. But this plausibility rests upon a simplistic view of neural development. Construction of the human brain involves many distinct processes, including neural proliferation, differentiation, migration, and the selective *elimination* of large numbers of dendritic connections. Axons and dendrites grow, but that growth is guided to proper destinations by other factors, and the establishment of dendritic connections between neurons depends on variables in the interaction between cells. All these processes in the construction of the brain are affected by experience, what the brain does and gets done to it in life (see Dowling 2004, 14-38; Matthews 2001, 430-457; Purves 6, 44-68).

Walkley, who demonstrated the sphingolipid growth effect, discusses the complexity of interactions at the cellular level.

An overall view that emerges from these studies is that the initiation of primary dendrites on neurons is likely controlled by multiple layers of interrelated regulatory mechanisms... Understanding how such mechanisms initially craft and then maintain the unique dendritic arbors displayed by individual types of neurons likely will require attention not only to growth factors and their receptors, related membrane proteins and secondary messenger systems, but also to the specific glycolipid microenvironment of the membranes in which these molecules reside (Walkley et al. 2000, 1035)

Walkley (2003, 94) discusses the problem of “meganeurites” and “ectopic dendrites,” stimulated to sprout at the wrong place or developmental time: “the resulting altered synaptic connectivity was a basis for brain dysfunction, most notably mental retardation” (Walkley 2003, 894).

Cochran et al. might respond that they are talking about more modest elevations of sphingolipids. Yet NHAI does not posit that this elevation-effect would be confined to the early development of the cortex, when elevated sphingolipid levels are normal (Walkley 2003:898). The deficiency of hexosaminidase A in Tay-Sachs heterozygotes is in adults. If that or similar enzyme elevations had the effect NHAI posits—of raising sphingolipid levels and thereby promoting neural growth—it is difficult to understand why life-long stimulation of meganeurites and ectopic dendrites should lead to greater intelligence, rather than pathology. Put in the context of real brain development, NHAI’s equation of more neuron growth with more intelligence seems much less than “likely.”

That is the case for neural stimulation within the LSD cluster. What about the other 14 inherited conditions? Some could seemingly fit the pattern. Canavan disease is an Ashkenazi disease not in any of Ostrer’s (2001,893-4) five clusters. It affects the myelin sheath that covers nerve fibers, leading to early death in homozygotes. Heterozygote growth stimulation is at least conceivable. But in torsion dystonia—another condition especially significant because of claims of heightened IQ (below)—the allele involved actually *inhibits* the extension of neurons (Hewett et al. 2006). And what about Factor XI deficiency, a blood clotting disorder (Goldstein et al. 1999)?

This is very common. The two different types of this condition (II and III) are together carried by about 1 in 10 Ashkenazi, although expression is highly variable. How is a tendency to bleed supposed to boost IQ?

In NHAI, the entire case for other mutations being neural promoters rests on one gene in the DNA repair cluster, BRCA1. It's frequency among Ashkenazi is about 1:100. No case of BRCA1 homozygosity has ever been found, presumably because that leads to early fetal death (Denic and Al-Gazali 2002). Female heterozygotes have an increased risk of breast and ovarian cancer. Risk estimates vary by study, and apparently by time. King (et al. 2003) found among a large Ashkenazi sample that the risk of developing breast cancer before age 50 was 24% for those born before 1940, but 67% for those born after 1940. For the entire sample, by age 50, there was a 39% chance of breast cancer and 21% chance of ovarian cancer. A mutation of the distinct BRCA2 gene has comparable frequency and effects. Thus heterozygosity for BRCA mutations significantly impacts life expectancy during female reproductive years.

For NHAI, the important fact is that the BRCA1 mutation has also been associated with neuronal growth.

What could the selective advantage be? It seems unlikely that any degree of disruption of a basic housekeeping process such as DNA repair could ever be favored by selection. In fact BRCA1 and BRCA2 heterozygotes face cancer risks. Still, those risks come fairly late in the reproductive schedule and have only a slight effect on fertility, so heterozygote advantage is not impossible. Our original speculative notion of how these DNA repair mutations might in some cases give heterozygote advantage was inspired by the fact that BRCA1 is expressed in embryonic and adult neural stem cells and is involved in cell proliferation... It seemed possible that a defective BRCA1 gene might, in heterozygotes, slightly unleash neural growth in a way that might favor cognition. Assuming that there is anything to this notion, the other Ashkenazi HRR-path DNA repair mutations may have similar effects (pg. 681).

To support this idea, Cochran et al. (2006, 681) turn to work by Bruce Lahn and colleagues that indicates BRCA1 is structurally and functionally connected to the gene known as microcephalin. That study (Evans et al. 2004:1140) found:

that BRCA1, like microcephalin, has a critical function in the proliferation and differentiation of neural progenitor cells, raising the possibility that positive selection on BRCA1 was actually directed toward its activity in brain development rather than its function in tumor suppression (Evans et al. 2004:1144).

It is only because of the microcephalin connection that BRCA1 can be suggested as promoting smarter brains in heterozygotes. "Ashkenazi DNA repair mutations in genes such as BRCA1 may be the most recent manifestation of an evolutionary trend that goes back many millions of years, from lemurs to human subpopulations" (Cochran et al. 2006, 681). But the lesson of microcephalin now appears to be heading in a very different direction, one that shows the hazards of simple genetic explanations of cognitive functions.

Microcephalin, and also the gene ASPM, have recessive alleles which in homozygotes lead to microcephaly: that is, abnormally small brains with some retardation but otherwise normal functioning. Both genes appear to have been under strong positive selection in our past. Patching those findings together has led to speculation that the genes are involved in the evolutionary

growth of human cognitive capacity, and that is why they have been selected (Evans et al. 2004, 1114; 2005, 1717; Mekel-Brobov et al. 2005; Wang and Su 2004; Zhang 2003, 2063). Beyond that, more recent selection in different geographic areas has been suggested as linked to major steps in societal evolution (“the explosion of symbolic behaviors” and the rise of cities), and to cognitive differences among living populations (Balter 2005).

Wade emphasizes that possibility in his book (2006b, 98), and told a National Public Radio audience what it signified:

[W]e’ve already found several brain genes that have been under evolution in the last 50,000 years. And this surely matches the growing complexity of our societies. I mean it’s much more complex to operate in one of our societies, to perform the difficult jobs that we all do than to be a hunter and gatherer; and even though hunters and gatherers know an enormous amount about their environment and the plants and the animals, they’re very skilled resourceful people, but nonetheless, there are many concepts that they never had that you get with settling down. And there is surely greater need for intellectual capacity and our brains surely has [sic] been evolving fast over the last 15,000 years, certainly since settlement, and will continue to do so (NPR 2006, 6).

Its like the Ashkenazi, but on a global scale—with the “less civilized” populations lagging behind.

Contrary to this build up, however, other studies indicate that these genes perform other functions, and could have been selected for those, rather than any effect on cognition (Kouprina et al. 2004, 657; Kouprina 2005; Trimborn et al. 2004). More: Positive selection on *ASPM* began 7-8 million years ago, and so “Our finding that selection on *ASPM* begins well before brain expansion suggests that the molecular evolution of *ASPM* in hominoids may indeed be an example of molecular ‘exaptation’ (Gould and Vrba 1982), in that the originally selected function of *ASPM* was for something other than large brain size.” More: fMRI scanning of subjects identified for presence or absence of both alleles of microcephalin and *ASPM* found no association between them and brain volume (Woods et al. 2006, 10).

While the role of recessive mutations of both of these genes in producing microcephaly is undisputed, our findings suggest that it is potentially misleading to refer to either of these genes as controlling, regulating, or determining human brain size outside the context of the microcephalic state. The titles of several recent papers notwithstanding, evidence of positive selective pressure acting on genes known to be associated with human microcephaly and mental retardation does not necessarily imply that such genes account for the variations in brain size or intelligence that are encountered in normal human populations.

This deflation of great initial claims is typical for behavior genetics. Individual gene-based differences, are routinely either not replicated, or their effect found to be quite small, on the order of 2% or lower of some measure of individual behavior (Hamer 2002; Plomin et al. 2003). In a meta-analysis of studies of eight non-Mendelian genetic diseases, Ioannidis (et al. 2001, 306) conclude: “The typical situation is that a very strong association is proposed by the first study, which becomes gradually less prominent or even disappears as more data accumulate.”

These cautions refer to associations with individuals *within* one population. Taking the idea of genetically based differences in cognitive orientations or behaviors, that may exist *between* different populations, immeasurably complicates these problems. Even those who advocate the

biological reality of race, such as Neil Risch, are clear about this, and the need for great caution in positing between-group differences.

[T]he skepticism of some scientists of the early 1970s regarding our ability to find genes (if such genes exist) underlying group differences in behavioral traits such as intelligence seems warranted. We are far from characterizing the contribution of genes to between-group variations of any complex trait and are likely to continue struggling in the future... [Consequently] there is a need for stringent criteria for drawing conclusions regarding the contribution of genes to between-group difference. Generalizations and assumption are unwarranted and may exacerbate group disparities. We therefore advocate standards for statements regarding genetic contributions to between-group differences (Mountain and Risch 2004, 552).

We really do not understand what microcephalin and ASPM do, why they were selected (if they were), or what variations between different populations imply. But take away the microcephalin-intelligence connection for BRCA1, and there is nothing to suggest this gene somehow increases intelligence. With that gone, there is nothing to suggest that any non-LSD is connected with neural growth. Given the lack of any evidence that heterozygosity for LSDs actually stimulates axonal growth or dendritic branching; and the implausibility that, if it did, the result would be beneficial rather than pathological, the neural promotion component of the NHA1 hypothesis appears to have even less foundation than the argument against drift.⁷

5. IQ Boosters?

Moving from neuronal growth to the main issue, IQ itself, just three inherited conditions are claimed in NHA1 to be associated with greater measured intelligence: Gaucher disease, idiopathic torsion dystonia, and congenital adrenal hyperplasia. Under scrutiny, none provide substantial backing for the claims.

Gaucher is an LSD which, as we saw, NHA1 claims to be associated with increased axonal growth and branching. The additional claim to higher intelligence is based solely on data provided by a Gaucher disease clinic at Shaare Zedek Medical Center in Jerusalem. Occupations of 302 patients show them heavily over-represented in brainier fields compared to the general Israeli population. 15%, for example, are engineers or scientists (Cochran et al. 2006, 677).

Alternative explanations for the clinic's experience must be considered. Could relative endogamy within certain professions (e.g. academics) concentrate the allele? Or, given the fact that Gaucher can have few overt symptoms, could more intelligent individuals be aware of the

⁷In the face of mounting criticisms, Lahn's team set out to directly test their hypothesis that ASPM and microcephalin are associated with IQ. The hypothesis was not confirmed: "intelligence, as measured by these IQ tests, was not detectably associated with the D-allele of either" (Mekel-Bobrov 2007,601). Independently, Rushton and colleagues made a similar test, of those alleles with brain size and mental ability: "no relationship was found between the genes and any of the criteria" (Rushton et al. 2007,157). Thus the posited connection of ASPM and microcephalin to IQ—the bridge that possibly connected Ashkenazi BRCA1 and BRCA to mental development—now appears to be dead.

condition, and knowing of family instances, be more likely to seek out counseling? Or, since many diagnoses of Gaucher are incidental to some other medical treatment, could those who can afford to pay for better doctors and second opinions be more likely to be diagnosed?

NHAI expects that Gaucher heterozygotes are more intelligent than non-carrier siblings. In fact, the Shaare Zedeck clinic has already investigated that possibility, with IQ and genetic testing.

In unpublished studies by our group that asked the question whether carriers of Gaucher disease enjoy a selective advantage of increased intelligence, we administered the Wechsler Intelligence Scale and Raven Matrices to appropriately age- and sex-matched carrier and normal siblings of adults and children with Gaucher disease. Both groups had high intelligence scores (Elstein et al. 2005; reference to unpublished study omitted).

No scores or other details are included, and I was unable to obtain them from the clinic. But the published result is very significant for evaluating the hypothesis NHAI: contrary to its expectations, non-carrier siblings also have high IQs. Clearly, there is something other than the Gaucher allele involved in higher intelligence of the clinic patients.

Idiopathic torsion dystonia (ITD) is a non-LSD condition, which results in involuntary postures or movements in various parts of the body. Cochran et al. (2006, 679) claim that “the many observations of increased intelligence among people with ITD strongly suggest that increased fitness resulted from increased intelligence.” They rely on a cluster of publications in the 1970s by Roswell Eldridge and colleagues, which repeatedly made the association of ITD and higher intelligence. This then would seem to be a very supportive case for their hypothesis. On closer inspection, however, the evidence for IQ elevation associated with ITD among Ashkenazim is weak to the point of non-existence.

The genetics and expression of ITD are complex, variable by population, and not well understood. Different muscle groups can be affected, to different degrees, and at different ages of onset. As reports of individual cases of ITD accumulated over the last century, scientists and physicians inferred that it came in two varieties. Some instances appeared with no known prior family history of the condition (although information was usually restricted to just one or two generations). These were believed to occur from a recessive gene. In others, the symptoms were known in parents or grand-parents. These were believed to be from a dominant gene.

This was the interpretive framework used by Eldridge (1970) and colleagues in discussing IQ. Subsequent research concluded that around a dozen different alleles, most dominant but a few recessive, lead to ITD in different populations. Among Ashkenazim, however, there is only one, or very largely one, allele for ITD. This is an autosomal dominant, with limited penetrance. About 30% of the carriers show symptoms, about 10% to a crippling degree (DBGET:OMIM; Muller et al. 1990). What this means is that Ashkenazi cases of ITD cannot validly be divided into two different genetic conditions, dominant and recessive. It is one disease. That fact makes a big difference for evaluating published claims of higher IQ.

What is the evidence for elevated IQ? Eldridge’s (1970, 56; 1976, 108) thorough literature review found that 10 different publications on ITD, including one of the first in 1911, commented on the maturity and/or intelligence of the patient. But this means very little. It is from a survey of over 200 publications, most of them individual case reports, of all populations and varieties of the illness. This same survey also found 22 instances of ITD being associated

with below-average intelligence or retardation. Because of that, *retardation* was thought to be “an integral part of the autosomal dominant disease.” Since we now know that the Ashkenazi carry just one version of the autosomal dominant condition, the sum total of these earlier reports is just a big mess.

Seemingly stronger evidence comes from a study that compiled data on 14 ITD patients of Jewish ancestry, their siblings, and Jewish controls. The patients have an average IQ of 121, compared to 111 for their controls. This is a statistically significant correlation at the $p < 0.03$ level, and it is that fact that has been reported. 10 siblings of patients have an average IQ of 119, compared to 112 for controls. Yet as close as those two sets of numbers are, the sibling’s advantage over controls is not statistically significant, highlighting the problem of very small sample size. Sample size causes other problems, as well.

Considering only those cases where patients also have IQ-tested siblings (i.e. taking out the patients without siblings), the average scores of patients (117.6) is almost identical to siblings (116.9). This means that patients who have the gene are nearly identical to siblings with only a 50% chance of having the gene. If *either* individual patients #1 or #2 (with IQs of 151 and 150) were removed from the sample, the IQ advantage of patients over controls would *not* be significant at the $p < 0.03$ level, and siblings (with half the chance of carrying the allele) would actually beat patients by about 2 points (Eldridge et al. 1970; Eldridge et al. 1971). If even one of two individuals had said no to this study, there would have been no report of higher IQ associated with ITD.

A second study was a continuation of the previous work including one of Eldridge’s co-investigators (Cooper). This tried establish the intellectual advantage of the ITD gene among Jews, specifically (Riklan et al. 1976). Its support for heightened IQ is even more qualified. Working with neurological institutions around the country, researchers garnered impressions of about 222 patients, 95 Jewish, 127 non. 46% of Jewish patients were informally estimated as being above average in intelligence, but only 24% of the non-Jews. However, when 68 patients were followed up with actual IQ tests, the results are very different. The Jewish average was 104.9, and non-Jews 104.6—indistinguishable (pg. 190-191).

This study divides Jewish and non-Jewish groups into cases with negative or positive family histories of ITD, conforming to the dominant/recessive distinction then assumed to exist. Among Jews, 14 negative-histories had an average IQ of 110.7, vs. 99.4 for the 9 positive-histories. There was no difference by this factor among non-Jews, which both average 104. The inference was that among Jews, the “dominant” gene lowered IQ, while the “recessive” gene raised it. Analyzed a different way, among Jews, 10 children with onset between 0 and 8 years averaged 95.2, while 11 with onset between 9 and 13 years averaged 113.6. Again, no similar pattern was found for non-Jews.

The authors conclude regarding IQ:

No significant differences were found between Jewish and non-Jewish groups nor between patients with positive or negative family histories. Only when age of onset was assessed as an independent variable, or in relationship to Jewish negative family history, did a statistically significant difference occur. This finding of higher IQ scores in this subgroup tends to corroborate the reports of Eldridge et al. and Cooper with respect to a differential intellectual function in the Jewish group with an apparent recessively inherited form of

dystonia (pg. 195).

Now we know that there is only one disease among the Ashkenazi, of dominant inheritance. That means that any IQ advantage of Jews with ITD can only be associated with time of onset. Given the uniformity of the allele among, time of onset must be credited to something other than the allele itself.

Thus the evidence for higher IQ being associated with ITD among Ashkenazi comes down to two studies, one where the relationship barely reaches statistical significance, and another where it does so only using distinctions which are not valid for the Ashkenazi condition. (Two more recent studies of cognitive function and ITD found no significant difference between patients and controls, but these were not focused on Ashkenazi, and did not compare IQ itself—Jahanshahi et al. 2003; Taylor et al. 1991).

Another problem is that the ITD allele is too recent for the NHAI hypothesis. Its coalescence is estimated around 1650 AD, with a range of 1500-1750 (Motulsky 1995, 99; Ostrer 2001, 895). That puts it near or after the end of the period of selection for higher intelligence posited in NHAI. This late date greatly undercuts the position that these mutations had sufficient generations to rise in frequency because of the selective advantage of higher intelligence in money lending and similar occupations. On the other hand, Risch (et al. 1995) argue the spread of ITD can be explained by drift in recently expanded sub-populations. In sum, the total support ITD offers for NHAI boils down to a single individual in one study—without patient 1 or 2, there would be nothing.

The final claimed evidence for IQ-boosters is another non-LSD condition, non-classic congenital adrenal hyperplasia (CAH). NHAI's (2006, 679) claim is based on a review (Nass and Baker 1991) which describes 7 investigations showing CAH patients and family members had an average IQ of 107 to 113. Non-classic CAH is the most common of all Ashkenazi inherited conditions (Zlotogora 2006; Ostrer 2001, 894). In NHAI, CAH is crucial for increasing the number of Ashkenazi mutations. By their count (Cochran et al. 2006, 675), all the LSDs and DNA repair cluster mutations count for only 15% of Ashkenazi, other mutations bring that up to 32%, but with CAH added, it reaches 59%. (Other heterozygote frequency estimates for CAH are considerably lower, e.g. 17% of the Ashkenazi—Zlotogora 2006b, 6). At first glance, one wonders why the whole NHAI thesis was not built around CAH rather than the less common LSDs. Further examination reveals why.

A central point of the Nass and Baker review is that all 7 IQ studies have methodological biases, such as sampling from higher socioeconomic levels, which cloud any association of genes vs. other factors. Other investigators have been skeptical of an IQ advantage with any form of CAH, reporting significantly lower IQs in patients than in controls (Helleday et al. 1994; Johannsen et al. 2006). "Although there have been occasional reports of elevated IQ among CAH patients, this has not generally been observed" (White and Speiser 2000, 253).

But more important, the Nass and Baker review (1991, 189) is about *classic* CAH, a more severe condition which leads to genital masculinization among females and/or chronic salt-wasting. They specifically note (pg. 192) that one of their 7 studies may include non-classic cases. Classic CAH is *not* found among the Ashkenazi, but among Moroccan Jews. The Ashkenazi condition is *non-classic* CAH, which often has such mild expression (e.g. delay of menarche or hirsutism in females, reduced fertility or stature in both sexes) that it is never

diagnosed (New 1998, 316-320; Ostrer 2001, 894; Zlotogora 2006). (Non-classic CAH may be the most common inherited condition in the world [New 1998, 317; White 2000, 255]). If IQ scores reviewed by Nass and Baker were taken at face value, the implication would be that Moroccan Jews, not Ashkenzi Jews, should have higher intelligence. NHAI has got the wrong condition. Yet without it, the population carrying one of the IQ booster genes, by their count, shrinks to around a third of Ashkenazi Jews.

One other IQ connection is not mentioned in NHAI. Bloom's Syndrome, in the DNA repair cluster—short stature, sun sensitivity, predispositions to infections and cancer—has been associated with *lower* intelligence. “Several children with BS have been slow learners and required special schools. Three are frankly retarded in mild degree. It is my impression that, in general, intelligence is toward the low side of the normal range” (German 1979, 127). True, this is just one report, and presumably about homozygotes. But it is in the opposite direction than expected by NHAI, and it is the only intelligence estimate of any sort related to any of the DNA repair cluster.

Finally, there is the dog that did not bark, as Holmes might put it. NHAI offers no report, nor could I find any, of Tay-Sachs carriers having elevated intelligence. That is remarkable. Tay-Sachs has been recognized and studied since 1881 (Volk 1964, 1). It was identified with Eastern European Jews in 1905 (pg. 9), found to be based on glycolipid retention in the 1940s (pg. 5), understood as an autosomal recessive condition by 1955 (pg. 5). The method for identifying carriers was developed in 1969 (Yoo 1993), and has been used for countless thousands of Ashkenazim since. The question of some possible selective advantage was raised in the 1960s, by which time there was also a theory (“marry a rabbi”) that Ashkenazim had evolved higher IQ. The connection of ITD to higher IQ was publicized in the early 1970s. Yet despite all these studies, tests, and suggestions, no one—to my knowledge—has reported any IQ advantage to Tay-Sachs carriers, much less something like 5 points. How could that go unnoticed?⁸

This dog's non-bark is particularly loud. Tay-Sachs provides a perfect case for an initial test of the NHAI thesis. Do heterozygote carriers have significantly higher IQ's than non-heterozygote siblings? A positive finding would not confirm the NHAI thesis, which is made up of many other claims besides this one. But the claim of a Tay-Sachs boost is central and unambiguous. Failure to find a relationship would be a compelling disconfirmation of the NHAI thesis.

On the association of inherited Ashkenazi conditions with higher IQs, NHAI's argument once again is remarkably weak, coming down to no more than one individual establishing statistical significance for ITD, and the curious job distribution found in a Gaucher clinic, but where non-carriers also have high IQs. But then, how much of an Ashkenazi IQ advantage is there to explain, really?

6. Ashkenazi IQ

⁸ The reviewer who works in field of lysosomal storage diseases, comments that there is no evidence that glycolipids enhance IQ, while there are many signs that IQ may be reduced by their accumulation.

NHAI is quite firm on the Ashkenazi IQ advantage:

Ashkenazi Jews have the highest average IQ of any ethnic group for which there are reliable data. They score 0.75 to 1.0 standard deviations above the general European average, corresponding to an IQ of 112-115... although a recent review concludes that the advantage is slightly less—only half a standard deviation (Cochran et al. 2006, 3)

The “recent review” they mention conveys a very different impression from that forceful assertion (Lynn 2004).

It has often been asserted that Jews have a higher average level of intelligence than non-Jewish whites of European origin... Despite these assertions, the purported high IQ of the Jews has never been systematically reviewed... Despite the widespread consensus on the high Jewish verbal ability, not all studies have shown that Jews have a higher verbal IQ than gentiles. Furthermore, virtually all the existing studies are unsatisfactory because the samples have been unrepresentative, very small or for other reasons (pgs. 201-202)... There is only one study of the intelligence of American Jews in the last century which appears to be representative and had a reasonable sample size. This is Backman’s (1972) analysis... The mean IQs of the Jew in relation to gentile white means of 100 and standard deviations of 15 were as follows: verbal knowledge (described as “a general factor, but primarily a measure of general information”...)--107.8, English language 99.5; mathematics—109.7; visual reasoning (“a measure of reasoning with visual forms”)—91.3; perceptual speed and accuracy—102.2; memory (short term recall of verbal symbols)—95.1 (pg. 203).

Lynn compares this result with an analysis of vocabulary scores using national survey data broken down by categories including Jewish and non-Jewish white. This he translates into a verbal ability score (not full-scale IQ) of 107.5 for American Jews, compared to 100.0 for gentiles (pg. 204).

Other estimates come in higher or lower. MacDonald (1994, 190)--whose three volume series is dedicated to demonstrating a Jewish evolutionary strategy of promoting their own reproductive success at the expense of gentiles—comes in at the top, with a general IQ around 117 and a verbal IQ around 125. Hughes (1928, 90), cited in NHAI, made a large study of London school children under the supervision of Cyril Burt. He found Jews scoring about 10 points higher than gentiles, regardless of economic well-being. But Hughes also refers to previous studies of Jewish immigrants to the U.S.: “four of them find that Jews are not more intelligent than the non-Jewish American population, and four of them find that they are.” Patai and Wing (1975, 146-149) review studies, most of which give Jews higher IQ, but a few of which do not. That same year, Dershowitz and Frankel (1975, 127) compiled other findings. The average verbal IQ (WISC) from two studies of American Jews was 8.4 point higher than Italian-Americans, 7.5 points higher than Irish-Americans, and 3.1 points *lower* than WASP-Americans. For performance IQ, Jews came out lower all around, 2.7, 2.6, and 9.2 points below, respectively. For full scale IQ, Jews beat Italians and Irish both by 3.2 points, and lost to WASPs by 6.8 points. Another study by the authors of 185 Israeli Jewish children found a much lower score than for the Americans, with an average IQ of 96.13. Taking all the information together, it is fair to say that most, though not all, studies give Ashkenazi descendants a higher IQ than non-Jewish whites. How much? Take your pick.

NHAI also focuses on the high end of the curve, how many very, very gifted there are among Ashkenazim (Cochran et al. 2006, 661). In this they follow Patai (1977, 321-342) and others who tallied the disproportionate number of Jews winning Nobel Prizes. Cochran et al. add world chess champions, and winners of ACM Turing awards. This clear over-representation is cited frequently in print and internet discussions of NHAI, as clear demonstration of the power of Jewish genes. But these impressive totals provide no evidence in favor of the NHAI hypothesis.

They would *only* support that conjecture if it were shown that a disproportionate number of those winners were heterozygote carriers of one of the supposed IQ-booster genes, as compared to the normal distribution of these alleles among the Ashkenazi. If 1/3 of the Ashkenazi have one of these alleles, then most of the winners should be from that 1/3. That would suggest the exceptionally gifted among Ashkenazim are so smart because they have the alleles. But if the winners did not have such an over-representation, if they were pretty much like the Ashkenazi in general, then the fact of so many excellent minds would turn into decisive falsification of the NHAI thesis. The alleles would not be related to these indicators of especially high intelligence. No one can say which way the evidence would turn, but the hypothesis itself certainly does not constitute evidence.⁹

Commentators on Jewish IQ regularly note the unusual pattern of their measured abilities (Backman 1972; Dershowitz and Frankel 1975; Levinson 1977; Patai 1977, 292-294). “They have high verbal and mathematical scores, while their visuo-spatial abilities are somewhat lower, by about one half a standard deviation, than the European average” (Cochran et al. 2006, 661). NHAI emphasizes this, quoting Hans Eysenck (1995), longtime champion of inherited class and racial differences in intelligence. Eysenck makes clear that this large a discrepancy on different scores is *unique*: “there is no other group that shows anything like this size difference.” NHAI folds this into its theory: “Verbal and mathematical talent helped medieval businessmen succeed, while spatio-visual abilities were irrelevant” (Cochran et al. 2006, 671).

It fits, but not without very inconvenient baggage. This lopsided specialization goes against the idea which Cochran et al. (2006, 662) otherwise endorse and build upon: that there exists one, biologically based *g*, or “general intelligence.” “*g* is what diverse cognitive abilities have in common” (Plomin 2003:183). And even if their posited logic of selection sounds plausible, the biology of it all becomes much more complicated. The posited neuronal stimulation would boost only some capabilities, and simultaneously retard others. The complications do not end there. Dershowitz and Frankel (1975, 133), discussing Israelis of Ashkenazi origin, argue that specific cultural values and practices could explain this internal pattern of subtest scores. Burg and Belmont (1990), working with Israeli Jews of four different geographical origins, encountered the Ashkenazi pattern, but also found Jews of the three other

⁹ I note again here the NHAI-author reviewer’s point--that there are probably many other entirely unknown Ashkenazi IQ genes besides those associated with inherited conditions. For that reason, he disagrees that the comparison suggested here would be any kind of a test of the NHAI thesis. He adds that he now sees that this point about other possible IQ genes should have been made explicit in NHAI itself. Again, I am responding to the published article.

nationalities each had their own distinctive pattern of mental abilities.¹⁰

After discussing these and still other difficulties, Lynn (2004, 205) concludes “At present it is doubtful whether any conclusion can be reached about the intelligence of American Jews except that their verbal intelligence... is about 107.5.” Because performance scores regularly are lower, full scale IQ would be well below that.¹¹ To go back to the question that ended the last section, how much IQ elevation is there to explain, the answer is—not as much as NHAI or some others claim. Yet if we take a 5 to 10 point advantage over gentiles as a reasonable estimate, that

¹⁰ David and Lynn (2006) review ten studies from Israel that compare the IQs of Jews of Ashkenazi ancestry with Jews from the Middle or Near East, or North Africa, to test NHAI’s hypothesis that only the Ashkenazi situation led to selection for higher intelligence. The median difference of these studies give the Ashkenazi an advantage of 14.6 points. This is presented as confirmation of NHAI. Eight of those ten studies are unavailable to me, either unpublished reports or published in Hebrew. The two I could obtain have more debatable results.

Lieblich (et al. 1972) administered a Hebrew version of the WISC IQ test to Israeli born children of European or Middle Eastern/North African background. The Europeans had an average IQ of 106.0, and the “Orientals” combined of 92.8, for a 13.2 point difference. Comparing the two after sorting into categories of high or low socioeconomic status reduced that gap to just under 10 points difference. That supports NHAI. But this study also attempted to compare IQs for the second generation of Israeli born children, those whose parents were born in Israel. “It was found that mean I.Q. for Oriental-Israeli children was 101.3, and that of Western-Israeli children was 109.6. Two points should be made: 1) First generation Oriental children had mean I.Q. of about 92, and Western children of about 106. The increase of I.Q. level for the second generation Oriental children is almost 10 I.Q. points, and for the Western children about 4 I.Q. points. This may be interpreted as an indication of environmental impact on intelligence levels” (pg. 167). That goes against NHAI.

Gross (1978) administered the same test to small samples of kindergarten children of Ashkenazi and North African-ancestry Jews in one middle class community. The Ashkenazi score was 121.56, the North African 111.26. As the author notes, this is a major increase compared to Lieblich et al. For the North African sample, it is almost 20 points higher, and 11 points above the high SES subgroup. Still, a 10 point gap between the two ancestries persists. Gross concludes that environment is more than just SES status. Understanding IQ performance will require “looking for different emphases within the two subcultures, divergent motivational systems, aspirations, cognitive styles, or learning commitments” (pg. 810).

What closer examination of the other eight studies would show, I cannot guess. These two studies can be interpreted in different ways. NHAI finds support in the significant and persisting IQ differences between the two groups. Against NHAI, these studies indicate a major role of environmental influences, with no telling how far those could go.

¹¹ In a more recent review covering that same material, Lynn and Longley (2006, 542) present 110.4 as the median IQ reported in four publications on American Jews. Settling on the median discards the reservations previously expressed by Lynn. Surveying several British studies, they calculate 110 as the rounded median IQ British Jews.

is still significant. That shift in the curve can account for the over-representation of Jews at the very high end of intellectual accomplishment. The more important question is, is this difference genetic, due to biology rather than culture?

7. IQ, Biology and Culture

NHAI takes a hard biological line on IQ, against environmental explanations. “In general the search for social and nutritional causes of IQ differences has not led to any convincing results and most workers now regard IQ as a biological rather than a social variable.” But here is how one IQ “worker” characterizes the situation regarding intelligence: “the hotly contested ‘nature versus nurture’ debate can officially be declared a draw with both genetic and environmental factors contributing roughly equally towards observed variation in ability” (Payton 2006, 44).

Cochran et al. (2006, 662) emphasize that IQ is highly heritable, and “genetic manipulation can raise intelligence in mice.” Yes, IQ is highly heritable, compared to other measurable psychological traits (Luciano et al. 2006, 45; cf. Richardson 2000). Thus, many researchers are convinced that there are genes that influence IQ. But finding IQ genes for normal or high intelligence, such as those NHAI posits, is proving to be extremely difficult. From within the IQ research mainstream: “Between 40% and 80% of the variation in human intelligence (IQ) is attributable to genetic factors. Except for many rare mutations resulting in environmental cognitive dysfunction, attempts to identify these factors have not been successful” (Posthuma et al. 2005, 318).

The quest for IQ genes began in 1994, and quickly turned up two candidates (Plomin et al. 1995). In 1998, a breakthrough seemed at hand, with the apparent discovery of the first gene leading to higher IQ (Wade 1998). After that announcement, the original researchers waited four years for replication by some independent lab. Since no one else checked the claim, they set out to replicate it themselves, with the same methods, except adjusted for a newly discovered polymorphism that might have affected their original finding. The result was a failure to replicate—no association of the allele and higher cognitive ability was found (Hill et al. 2002).

Realization set in that there could be hundreds of genes involved in IQ, and their individual contribution to variance on the order of 1 or 2%, or less (Geus et al. 2001, 490; Plomin et al. 2001, 507). Even highly refined search techniques have been frustrated. One massive, careful genome scan found no IQ enhancing genes that survived its rigorous methodology, although it did show how frequently false positives should be expected (Plomin et al. 2001). Plomin (2002, 194-195), a leader of the IQ hunt, explains that as currently conceptualized, IQ genes may be easy to miss: “[i]f the 50% heritability of g is due to 50 QTLs [quantitative trait loci] with the average effect size of 1%...” —then he goes on to worry that the effects of individual genes may be below 1% and so continue to escape detection. Currently, a dozen years after project began, progress consists of developing new search techniques which might be able to pinpoint IQ genes in the future (Craig and Plomin 2006; Luciano et al. 2006; Posthuma et al. 2005; Yu et al. 2005).

Who could doubt that many genes are somehow involved in whatever it is that gets measured as IQ? But NHAI posits that three specific alleles each confer a 5 point advantage in heterozygotes (Cochran et al. 2006, 677). That truly would be a monumental discovery, one

entirely different from the empirical slogging that has bogged down the quest for IQ genes so far. As Wade put it on National Public Radio: "One of the most fascinating things about the Ashkenazi hypothesis that I described in such detail is that this pinpoints for us exactly which of the 20,000 or 10,000 genes active in the brain are the ones you need to increase intelligence; if the hypothesis is correct" (NPR 2006, 7).

Contrary to NHAI, many of those who argue for the genetic determination of the neurobiology of *g*, fully acknowledge the importance of environment in shaping IQ. For instance, Toga and Thompson (2005) are firm that intelligence depends "on structural differences in the brain that are under very strong genetic control" (pg. 17). But without contradiction they also affirm:

The genetic contribution to intelligence therefore differs in different environments—a caveat against general inferences based on heritability data... Positive environmental influences on intelligence are hard to identify, in part, because of the inevitable confounding of variables in large-scale epidemiological studies of cognition... The significant influence of heredity on IQ has been misinterpreted to imply that there is little point trying to educate or be educated, or that IQ is somehow impervious to change. This is a fallacy because many environmental factors, including family rearing environments, socioeconomic status, diet, and schooling, influence IQ (pg. 15).

Cochran et al. (2006, 663) wave off such concerns. They do acknowledge "there are apparently some environmental effects on IQ," but marginalize the idea. They dismiss the home, or "shared" environment, as explaining IQ variance. But in a mainstream view, before age 16 (and not after), shared environment can explain a large part of IQ scores, even 50% at age 5 (Posthuma et al. 2003:143). So in considering IQ scores, it is important to ask: how old when tested? Cochran et al. passingly mention "non-shared environment" as a factor to be considered. Non-shared environment refers to effects experienced outside the home. IQ researchers (Plomin and Asbury 2001, 274), emphasize the potency of those outside influences. "The bottom line is that nonshared environment is where the environmental action is in terms of behavioral outcomes. There is a lot to be done before we understand either what it is or how it operates" (pg. 277) "No matter how difficult it may be to find specific nonshared environmental factors, it should be emphasized that nonshared experience is how the effective environment operates to create individual differences" (pg. 278).

All this refers to individual variation within one group—not touching on inter-group difference, which is of course the issue in NHAI (and in Wade's "Twists and Turns of History, and DNA"). Gray and Thompson, for example, are squarely in the hereditarian corner on many things. Yet they also note that "heritability within a group does not imply that group differences are due to genetic factors. Environmental factors could completely explain group differences, even in a case where genetic factors completely explain within group differences;" and "That the genetic contribution to intelligence differs in different environments is a caveat against general inferences on heritability data" (2004, 477). One striking example of that is a recent twin study that found environment accounted for 60% of the variance in impoverished families, and close to zero in affluent families (Turkheimer et al. 2003).

Then there is the Flynn Effect—named after James Flynn—the discovery that performance on intelligence tests has drifted steadily upward over time by about .3 IQ points per year. Cochran

et al. (2006, 663) mention the Flynn effect. They say some rise in IQ may be due to biological factors, such as diet; more due to increase in school attendance and test familiarity. They note the increase may have stopped. Anyway, they argue, since the Flynn Effect applies across ethnic groups and classes, it cannot explain group difference.

But the evidence of large environmental effects on measured IQ cannot be dismissed. In an article reviewing 20 years of research, Flynn (1999) laid out what has been found, and what it means about group differences.

Massive IQ gains over time revealed that the present generation has a huge IQ advantage over the previous generation. Yet the IQ advantage did not seem to be accompanied by a corresponding achievement advantage... IQ differences between the generations are clearly environmental in origin. Yet heritability of IQ within generations is robust, which suggested that high within-race heritability estimates do not signal a genetic gap between Black and White populations... (pg. 5) Data from 73 studies containing 7,500 participants ages 2 to 48 years showed that between 1932 and 1978, White Americans had gained 14 IQ points. The rate of gain was about 0.30 IQ points per year, roughly uniform over time and similar for all ages... [Contrary to his expectations, test data] showed that from 1930 to the present, the largest IQ gains were on culture-reduced tests like Ravens... (pg. 6). This deals a stunning blow to our confidence in the ability of IQ tests to compare groups for intelligence, at least when those groups are separated by cultural distance. Can anyone take seriously the notion that the generation born in 1937 was that much more intelligent than the generation born in 1907... (pg. 7) Some years ago, Jensen envisioned tests running from the detour problem through an adapted form of Ravens, which would allow us to measure the intelligence of cats and chickens, Kalihari Bushmen and Polar Eskimos, even extraterrestrials. Today we know that Ravens cannot bridge the gap between the Dutch of 1982 and the Dutch of 1967... (pg. 8)

What has caused this upward drift remains a puzzle, though many ideas have been offered (Neisser 1998). It is noteworthy that NHAI invokes better schooling and test familiarity, yet otherwise denies the impact of environmental factors. Diet does indeed appear to be a substantial contributing factor, among others, as rising test scores have correlated with increasing height (Lynn 1998; Sigman and Whaley 1998). Daley (et al. 2003), in rural Kenya, document a Flynn Effect of 26.3 point in 14 years, which they attribute to "parents' literacy, family structure, and children's nutrition and health" (pg. 218). Dietary iodine alone has been linked to major differences in IQ scores in less developed countries: 12.45 points between low and high iodine areas in China (Qian 2005, 35). Even in southern Spain, lower iodine levels were positively correlated with lower IQ scores (Santiago-Fernandez 2004). But in developed nations, IQ gains continued even after height leveled off, so nutrition alone cannot explain the rise (Martorell 1998). In some of those same countries, the Flynn affect appears to have stopped, as some predicted it would (Norway, Sundet et al. 2004) or even reversed (Denmark, Teasdale and Owen 2005). That fact in no sense diminishes the demonstrated significance of non-genetic factors in population IQ scores.

Dickens and Flynn (2001) offer formal models to account for the paradox of high heritability combined with massive changes over time. They discuss gene-environment interactions, masks and multipliers, and the impact of individuals shaping their own cognitive

environment over a lifetime. They show that IQ, while not accurately reflecting real-world intelligence, may be strongly influenced by how much *abstract problem-solving*—of the sort measured in IQ tests— is emphasized within a population.

We believe that it is not only people’s phenotypic IQ that influences their environment, but also the IQs of others with whom they come into contact. The latter is influenced by society’s average IQ. Therefore, if some external factor causes the IQs of some individuals to rise, this will improve the environment of others and cause their IQs to rise. We call this the social multiplier, and it can play an important role in determining the impact of society-wide changes (pg. 347).

NHAI’s bulwark against the Flynn Effect is that gains were registered across all groups, so it has no relevance to between-group difference. This is illogical. The issue is whether environmental differences between groups can explain the difference in IQ. Referring to the long debated difference in “white/black” IQ scores, Flynn (1999, 15) concludes: “Therefore, an environmental explanation of the racial IQ gap need only posit this: that the average environment for Blacks in 1995 matches the quality of the average environment for Whites in 1945. I do not find that implausible” (Flynn 1999, 15). Or as Wahlstein (1997, 78; quoted in Marks 2005, 223) puts it: “More recently born children exceed the raw intelligence of their own parents at a comparable age by almost the same average amount as Americans of European ancestry exceed Americans of African ancestry.” From this perspective, to explain—say—a 9 point IQ advantage of Ashkenazim in 1950, one would only need to posit that their average, total environment—from intellectual stimulation to diet—was as favorable for IQ as the average environment of gentiles in 1980.¹²

¹² More evidence of environmental impact on IQ is provided by a massive study of birth order among Norwegian conscripts, involving 63,951 adjacent sibling pairs (Kristensen and Bjerkedal 2007). It has long been observed that birth order is correlated with IQ, i.e. first-borns on average score higher than second, second-borns higher than third. Several theories have been offered to explain this finding, which the new study is able to discount. For instance, it was proposed that the association was false, showing up in cross-family comparisons because less intelligent parents had more children. The new work confirms the birth order affect with within-family comparisons. It has been argued that the birth order affect was biological, a result of increased maternal anti-body attacks on fetal brains in successive pregnancies. This studies demonstrates IQ follows social, not biological order. A second born child whose older sibling died has virtually the same average score as a first born; a third born whose two older siblings died has the score of a first born.

The Norwegian study demonstrates that there is something different about the family environment for successive siblings, that leads to a 3.4 point average difference between first and third born children (Bjerkedal et al. 2007, 512). This analysis also identifies several variables that have measurable effect on the size of the birth order gap. “The difference in mean standardized scores between brothers of adjacent birth orders increased with higher maternal level of education, was highest between brothers of married women, higher with paternal income and decreased with sibship size and longer spacing between births” (pg. 503). How those variables impact IQ is unknown, and some complex intra-family dynamics have been suggested

8. The “Talmudic Tradition”

We know that environment has a major effect on IQ. We have an empirically grounded, if speculative, argument that cultural variation in cognitive styles can lead to major differences in IQ scores between populations. Is there any evidence of a cultural emphasis on abstract reasoning associated with the Ashkenazi? The answer is an emphatic yes—what has been called the Talmudic Tradition. Originating before the time of Christ, brought with Ashkenazim to northern Europe, and lasting up until modern times, this tradition conferred both respect and authority on those who excelled in scholarship.

An emphasis on broader secondary education began to develop in Palestine during the first century BC. This occurred as part of a competition for leadership between the more aristocratic Sadducees and the Pharisees. The former put more emphasis on ritual and sacrifice performed by priests in the Temple, the latter on study of the Torah. The Sadducees, and the Temple, were destroyed around 70 AD for the revolt against Rome. The Pharisees did not participate in the revolt, survived, and were given permission to establish an academy “where they replaced the cult of the Temple with study and prayer” (Botticini and Eckstein 2003, 13). For the next six hundred years, Jewish communities expanded—and taxed for—academies (yeshivot) for study of Torah, Mishna, and Talmud. Primary education for boys became nearly universal, and higher education common.

This early history is important, because without it one could argue that the emphasis on learning was an outgrowth of Jewish higher intelligence. The tradition of education developed while Jews were almost entirely farmers, before there were any income advantages to be had. NHAI itself argues that there was nothing cognitive separating Jews from their farmer-neighbors during this pre-Diaspora period (Cochran et al. 2006, 667). Yet by the second half of the first millennium, most Jews were literate, in striking contrast to other farming populations around them. Consequently, as urbanization developed by the middle of the 8th century, Jews were able to move into rapidly expanding niches including commerce and money lending, and were actually encouraged in that direction by the educational tax. Progressively fewer Jews remained farmers. It was this series of developments that created the social basis for Ashkenazi occupational specialization (Botticini and Eckstein 2003; 2005).

Talmudic academies were open to boys of ability and dedication, and offered a clear avenue of social mobility (Safrai 1971; Urbach 1971). It has long been speculated that this social elevation of intellectuals might have selected for the evolution of higher IQ (Hughes 1928, 94). Patai (1977, 305-306) summarizes what some call the “marry a rabbi” argument:

Until the Enlightenment, the Jews considered Talmudic scholarship the greatest of all

(Sulloway 2007). Like the Flynn Effect, this study provides very strong evidence that environment does indeed affect IQ. Seen in this birth-order perspective, all that would be necessary for the Ashkenazi-gentile IQ difference to be environmentally caused, would be for the average environmental difference between the two populations to be about two and a half times greater than the average environmental difference for first born and third born siblings.

achievements. The appreciation of the scholarship was inculcated into the children to such an extent that, generally speaking, all the boys who had the mental capacity endeavored to achieve—and many actually did achieve—scholarly status. The most distinguished among the many budding scholars obtained coveted positions as rabbis of Jewish communities or as heads of yeshivot (Talmudic academies). Wealthy Jews sought out the promising young rabbinical scholar to be their sons-in-law. A rich man's daughter, on her part, considered it a great distinction to be chosen as the bride of such a young luminary. Thus excellence in Talmudic study (which this argument considers a mark of high intelligence) enabled a young man to obtain a better economic situation, marry earlier, have more children, give them better care, and thereby save more of them from infant and child mortality...

A nice, neat theory. Cochran et al. (2006, 660) briefly note this idea, but conclude that the number of rabbis was just too small to have much impact on population genetics. It certainly was not too small, however, to promote a cultural emphasis on abstract reasoning.

Jews today are not living in the middle ages, but the cultural tradition continues. They live in the larger secular world, where intellectual achievement—not just Talmudic study—is prized and encouraged. Patai (1977, 302-303) is eloquent on “the Jewish Home Environment.” (Although this would be classified as “shared” environment, what happens in the home reflects the value system of the larger community.)

Whatever studies have been made comparing the average Gentile and the average Jewish home with respect to the factors listed have all yielded one typical result: the quality of the home environment maintained by the Jewish family differs from that in the Gentile family even if both belong to the same socio-economic stratum... The same pride which a Jewish mother of the eighteenth or nineteenth century felt when she thought or spoke of her son the great Talmudic luminary filled the heart of her granddaughter in the twentieth when she could refer to “Mein Sohn der Doktor”... In the modern world, the road to intellectual achievement led through the gates of academic professions. This was almost intuitively grasped by the millions of East European Jewish immigrants who arrived in America... [T]he poorest home of the most ruthlessly exploited sweatshop worker was a place permeated by the age-old Jewish emphasis on learning... because both father and mother did everything they could to enable their children to study, to stimulate them to study, and if necessary, to drive them, push them, force them to study.

NHAI asks ‘why are there so many smart Jews?’ Before NHAI, we already had a very good answer—that Jews today partake of a cultural tradition emphasizing scholarship and abstract thought that may be without parallel in the Western world. That is a fact. The heterozygote intelligence boosters are very shaky speculation. Theoretically, this cultural-environmental explanation of existing IQ advantages among Ashkenazim is entirely compatible with mainstream research on IQ that recognizes the significance of the environment. On the other hand, NHAI's claim of specific alleles which give a large jump to IQ—on the order of 5 points—seems far outside of that mainstream.

9. A Medieval Meritocracy?

This section focuses on a different question: in medieval Ashkenazi society, did greater

wealth lead to greater reproductive success, and—leaving aside the rabbinical route—did higher intelligence lead to greater wealth. The answer to the first question is an unsurprising yes. There is good though quite limited evidence that the wealthy had far more surviving children than others. On Jews in eastern Europe, Weinryb (1972, 313) writes:

some fragmentary information seems to indicate that more children survived to adulthood in affluent families than in less affluent ones. A number of genealogies of business leaders, prominent rabbis, community leaders, and the like—generally belonging to the more affluent classes—show that such people often had four, six, sometimes even eight or nine children who reached adulthood, although there were also families with only two children. On the other hand, there are some indications that poorer families tended to be small ones (pg. 313)

Weinryb then notes some figures of children per Jewish family for the 18th century: innkeeper, 2.5; house owners, 1.2; tenants .6. Hundert (1992, 78) refers to data from one exceptionally well-documented 18th century Polish town, indicating that of families with 5 or more children, roughly half had resident servants. This is not a lot of data, and much of it from after the NHAI window of selection, but it is consistent with what one might reasonably expect in olden times.

The elite does seem fruitful, relatively and absolutely. How concentrated was this reproductive advantage? Weinryb (1972, 70) calculates that among Jews of Breslau in the 14th century, the very rich constituted about 7% of the total population, the poor at 10%, and the rest in between. We have no information at all on how far, or how much, the elite reproductive advantage went beyond the top tenth or so. The top tenth is all NHAI needs for its model.

The question is not whether the rich had more surviving children, but whether a higher IQ enabled one to become rich. NHAI claims that is why all those recessive conditions were selected for, despite their evident liabilities. That brings us to the only major substantive change between this posting and the earlier version of this paper. The NHAI-author reviewer pointed out an important error in my presentation of their argument. I had understood NHAI as suggesting a pattern of upward mobility among ancestral Ashkenazi, by which those heterozygotes with higher IQ regularly rose from less to higher wealth, and therefore enjoyed greater reproductive success. The reviewer pointed out this was not true, NHAI did not posit a pattern of upward mobility.

That criticism is correct. NHAI does not discuss upward mobility. Their model (Cochran et al. 2006:664) accounting for long term increase in population intelligence is as follows. Using as assumptions the correlation of IQ and income, and the heritability of IQ, as found in our own society, and assuming the top 10% of Ashkenazi society had twice the average number of surviving children, population IQ would increase by about .8 points per generation. An alternative scenario is that Ashkenazi males with an IQ of 80 or lower did not reproduce (at least not as Jews), and that would produce a similar increase per generation.

Nevertheless, the central point of NHAI is that the increased intelligence conferred by heterozygosity for harmful or lethal conditions is what led to the financial success that brought higher reproduction. “Jews who were particularly good at these jobs enjoyed increased reproductive success” (Cochran et al. 2006, 670). Unlike typical pre-modern societies, achievement among the Ashkenazi was achieved, rather than ascribed. “To the extent that status and wealth were inherited rather than earned, the correlation between cognitive traits and reproductive success in elite groups may have been quite weak” (pg. 671). That, according to NHAI, is what sets the Ashkenazi apart.

Against that proposition, the historical record shows that Ashkenazi society was highly stratified, that status and wealth *were* inherited within a largely closed elite, and that the central achievement in life was being born to the right parents.

During the Middle Ages the leadership of Jewish society was almost always in the hands of clearly defined and cohesive groups... Throughout the Diaspora the leadership circles were known for their aristocratic family consciousness, although the rigidity of the ancient Near East ('Babylonian') Jewish aristocracy had vanished... In all areas there were specific families that led and guided the people for centuries (Ben-Sasson 1976, 511).

Weinryb (1972, 76-77) tells us that stratification appears less in the early years of Jewish settlement in Poland, but increased as the self-enclosed communities grew over time (Weinryb 1972, 76-77). In Poland and Lithuania after 1100, the wealthiest few exercised broad powers.

The taxpayers, that is the large taxpayers, were the important people. The leadership (*parnasim*, elders) were elected or nominated from among the most wealthy. These elected leaders sat in court as judge with or without the rabbis. They heard mainly financial cases, apportioned taxes, represented the community before the authorities, controlled the right of settlement, oversaw the economic as well as some other facets of life, and served as administrators of community property (1972, 74).

Socioeconomic stratification of the Ashkenazi communities thickened into a wide and dense network of kin ties. One example is the Landau family, based in Opatow.

The importance and the influence of the Landaus was not limited to Opatow, where they dominated the *kahal*; their presence was felt as well in the regional (*galil*) institutions of Jewish autonomy... Members of the family occupied important lay and rabbinic offices and formed marriage ties with others in similar positions from Tykocin (*Tiktin*) to Hamburg and Prague, and from Miedzyrzecz in Podlasie to Lwow and Cracow (Hundert 1992, xiv).

Stratification was a steep wall within money lending. Emery's (1959) detailed study of notarial registers from 13th century Perpignan in Aragon identifies 228 Jewish males. 78% are noted as lending money. Besides the 22% who do not show up as lenders, a "large number of Jews... appear as money lenders only once or twice." A total of 31 individuals or families (some with several men, though women loaned too) are noted as involved in 15 or more loans. Fourteen men who each acted as officials of the Jewish community had an average of 32 loans each. This politically connected 6% of the males were involved in 27% of all loans (451 of 1,643) (pg. 26-27). Those who made more loans made larger loans, on average, and those who made fewer, made smaller. Of the 31 cases with 15 loans or more, 10 of these individuals or families loaned a total of 173,819*s*, while the next 21 lent a total of 66,049*s* (or an average of 17,382*s* vs 3,145*s*). These 31 lenders accounted for 1,239 loans, leaving the remaining 404 for everybody else (Emery 1959, 26-27, 30-31). Of course this snapshot does not show process, but it does show how the deck was stacked.

In sum, for centuries, a wealthy, self-consciously aristocratic, intermarrying elite conducted the lion's share of business, controlled financial courts, determined tax rates, acted as intermediaries with political authorities, determined who could live where, administered economic matters and community property, and filled secular and rabbinical offices. Of course, even granting this level of stratification, one could still make a plausibility argument that being smarter still led to making more money. The available evidence, however, points to factors other

than intelligence as key to higher incomes.

10. How Much Did IQ Matter?

The Perpignan case suggests that what determined income from money lending was not possession of higher intelligence, but possession of capital. Parkes (1976, 339) makes that point explicitly.

It was no question of aptitude that created moneylenders, but the fact of possessing cash to lend. Four classes lent, regular and secular clergy, tax-collectors, and merchants. Each class exercised the profession as long and as widely as it could. The limited victory of the Church [in prohibiting money lending] reduced the lending powers of the first two classes during the very period in which the fourth came into prominence and acquired immensely enhanced facilities for practicing this profession. It is as members of this fourth class that the Jews came also to practice this activity.

Ben-Sasson (1976, 470), in discussing the shift away from more varied commerce to money-lending after the First Crusade, also emphasizes capital.

Personal connections—the epoxy of social class—were also very important for financial success. Weinryb (pp. 58-65) provides abundant evidence that major Jewish financiers and managers of all sorts prospered due to high-level political connections. They also benefitted from established ties to others of wealth.

Solidarity and contacts played a considerable role in economic activity. The strength and structure of an enterprise, firm, or partnership were conditioned by group solidarity, which also may have helped in terms of development and seizing new opportunities. Jews went into partnership as moneylenders or merchants and toll farmers on a larger scale. (There were also some Jewish-Christian partnerships, but these seem to have been less frequent)... Jews from different parts of the country may have formed partnerships or done business together, thus utilizing resources and opportunities to be found in widely separated places (Weinryb 1972, 97)

There were at times specific niches for entrepreneurs. In Poland in the 14th century, advantages went to some Jews who moved first into southern borderlands. “In fact, the Jewish toll and revenue farmer was sometimes the first Jew to settle in a place.” But should that be credited to a high IQ? A penchant for risk taking, or the plain bad luck of being driven out of someplace else, would seem better candidates. Or, “ore mines, the salt mines, and often the mint were farmed out by princes and kings to entrepreneurs who paid a fixed sum annually and then usually took in a much higher amount” (Weinryb 1972, 63). But that of course required the initial capital, and the squeezing that ensued also would require personality characteristics other than intelligence.

Did success in financial/managerial pursuits require disproportionately high intelligence? Cochran et al. (2006, 670) talk of “cognitively demanding jobs... the Ashkenazi niche was so specifically demanding of accounting and management skills.” Wade emphasized the complexities of money lending on NPR.

If you try and work out—remember, in the Middle Ages we didn’t have the concept of zero. If you try and work out 17 percent of 3,000 without using zero, it’s not a straightforward computation. So for these reasons, it seems very possible that the mutations that were

favored in the Ashkenazi communities were ones that promoted extra intelligence (NPR 2006, 5)

Sounds intimidating. But does it represent reality?

Here is an actual loan contract, from England in 1179. (There are two others with it much the same.)

Know, &c., I Herbert, parson of Wissenden, owe Aaron Jew of Lincoln 120 marks to be returned at the second feast of St. Michael after the death of Richard de Luci in six years, vis: each year 20 marks at two terms of the year, at Rogations 10 marks and at the chain of St. Peter 10 marks, and so on, from year to year, till the whole debt is paid. The first term for receipt is at the second Rogations after the death of Richard de Luci. And if by chance any one of those terms shall pass, I will give him every week twopence interest for every pound, so long as I shall hold the debt by his grace, and I make my affidavit, and have confirmed it with my seal (Parkes 1976, 405).

This isn't string theory.

Emery (1959, 82-83), reading between the notarial register lines, deduces some behind-the-script maneuvering in loan management. One way to increase profit was to let the loan run longer than agreed, though to get around legal limitations on interest, some loans had to be re-contracted. But even from those detailed records, one cannot tell how the real business of money lending worked. In this evidentiary vacuum, it is not inappropriate to consider the operations of contemporary loan sharks. Joe Valachi, Mafia turncoat, describes (Maas 1969, 168-171) the extension of an outstanding debt as the sweetest of deals. Valachi described other ways and means, none of which require a super brain. As Valachi tells it, the key to success was maintaining personal ties, and being a perceptive judge of character to avoid bad borrowers. A shark did well by getting good customers, who kept coming back. You might say his type of money lender prospered by being a "people person."

It will not do to overemphasize money lending in Ashkenazi history. That activity pops up with special frequency in media and blog commentary, much more than the other financial and managerial occupations also discussed in NHAI. As with, but more insidiously than the "smart Jew," the "Jewish moneylender" is a popular stereotype. Shylock. But money lending as a speciality was most prominent only in restricted times and places.

In England [Jews] flourished as moneylenders from the beginning of the twelfth to the middle of the thirteenth century, but they were ruined before their expulsion in 1290. In France they flourished at various periods from the end of the twelfth to the end of the fourteenth century, but frequent expulsion prevented this from being called a period of continuous prosperity. In Germany they do not appear to have become important until the thirteenth century, and they were probably completely ruined by the beginning of the fifteenth (Parkes 1976, 345)

In Poland, Jewish money lending decreased after the 1430s (Weinryb 1972, 60).

At other times and places, Jews were involved in many areas of commerce and management (tolls, mills, mines, etc.). Large scale commerce and management surely can involve complex calculations. Yes, those calculations needed some brains. A wealthy businessman could hire those brains. Speaking of international trade, Weinryb (1972, 67) once again notes the importance of initial capital, but also describes who was doing the calculations.

Only the wealthy had the means to risk such high stakes, which admittedly promised great

profits. But connected with this limited number of entrepreneurs were many other Jews who took wares from them on consignment, bought and sold for them, or served as clerks, bookkeepers, and the like (pg. 67)

More generally,

Wealthy Jews with extended business connections in banking, trade, or toll and tax farming apparently employed a considerable number of clerks and assistants, although the sources yield very scant information on this point... These staff members—secretaries, clerks, and collectors—were undoubtedly Jews, for some of the toll registers and many receipts were written in Yiddish or Hebrew (pg. 68).

Even in money lending, in some places and times, the number of staff members may have outstripped the capitalists.

In general, according to documentary evidence, the number of Jews involved in money lending was small... Only a small part of the Jewish population seems to have been engaged in such activities, although the big bankers and lenders may have employed a number of assistants or agents (pg. 61).

Here we see a clear niche for scholarly accomplishment being hooked up to the wheels of capital. But it would seem very unlikely that such work—“secretaries, clerks, and collectors”—would make a man rich. For the really smart but not well-off, the Talmudic route could seem a more secure avenue for (more limited) success and prosperity.

What about those at the pinnacle, did they need high IQ's? No doubt, it took cunning to see good opportunities. But other personality factors besides intelligence could lead to fortune. One could even keep this with a psychological Darwinian orientation by suggesting that risk taking, or aggressiveness—both traits often claimed to have genetic bases—led to great profit. Yet more than any individual qualities, the most important factors leading to greater financial success were possession of capital, social connections, and political patrons. And let us not forget luck--circumstances that lead to a huge payoff, or sudden ruination.

I do not dispute that unusually low intelligence would be an obstacle in business. My point is that there is no evidence that it took anything more than *average* intelligence to make more money, if those other factors just discussed were working in one's favor. There is nothing in this record to suggest that those fecund few at the top were any smarter than those staff members who labored below them, or that they had acquired their wealth by special perspicacity. NHAI argues that financial success was based on higher intelligence, that those with greater *g* prospered and disproportionately reproduced, overcompensating for the undeniable liability of carrying alleles for harmful conditions, and pulling the entire population's IQ upward. A closer look at Ashkenazi society makes this scenario extremely unlikely.

11. Conclusion

The thesis of NHAI has multiple problems, tottering at one theoretical step after another. Each step must support the theory, or it fails. The main criticisms are: (a) Contrary to NHAI's argument that the inherited conditions are due to selection, population bottlenecks and drift remain strong explanations of their frequency, and consistent with historical information. (b) In NHAI, less than half of all inherited conditions have even a suggested pathway to higher intelligence. (c) The inference that genes which stimulate aspects of neural growth are linked to

higher intelligence is pure speculation predicated on a simplistic view of neurological development. (d) The claimed connection between three specific conditions and higher IQ has virtually no empirical support whatever. (e) The demonstrated IQ advantage of Ashkenazi Jews as a whole is less than asserted. (f) The multi-point IQ boosts proposed for specific genes by NHAI are very inconsistent with current research on the genetics of IQ. (g) Even within the mainstream of IQ research, which emphasizes genetic/biological bases, the extent of Ashkenazi IQ advantage are easily accommodated as due to environment. (h) The “Talmudic Tradition” of emphasizing learning and abstract reasoning provides a clear cultural explanation for higher IQ among Ashkenazi. In Ashkenazi history, NHAI’s assumption that higher intelligence led to greater income is contradicted by (i) a rigid system of social stratification, (j) the critical importance of capital, social connections, and political patrons in amassing wealth, and (k) the absence of any evidence that success in business required anything more than average intelligence.

Beyond these specific problems, NHAI suffers because it represents a fading notion about genetic causality. In the post-genomic world, we know that genes do not simply read out to phenotype as once imagined. Starting with epigenetics (Jaenisch and Bird 2003) and proteomics (Patterson and Aebersold 2003), and going all the way to the developmental construction of the brain (Lickliter and Honeycutt 2003; Westermann et al. 2006), genes play out in a system of systems, all with environmental inputs (Oyama et al. 2001). Leading researchers are now calling for a new “cultural biology” (Quartz and Sejnowski 2002). Even with many developmental disorders, growing appreciation of the role of the environment often precludes any one-to-one mappings of genes and conditions (Karmiloff-Smith et al. 2002). The three conditions NHAI links with higher IQ—Gaucher, ITD, CAH—are each examples of this enormous developmental variance in expression (as are LSDs in general—Maire 2001). “It’s all in the genes” now sounds quaint.

NHAI and its public reaction should be of concern to anthropologists. They challenge more than a century of anthropology premised on psychic unity, the idea that humans are all born with essentially similar mental capabilities. They illustrate just how marginal cultural anthropology has become in wider public discourse. For a long time, it has been common to hear, in everyday conversations, that genes explain behavior—of individuals, of humans in general, of women, of men, or of races, although the last may only be whispered. But we have not commonly heard that same idea applied to ethnic or national differences. We are right on the edge of that now. This is the whole point of Wade’s (2006a) New York Times article. Where The New York Times leads, others follow. We have a very short distance to go before it becomes ‘common knowledge’ that ‘scientists have shown that different peoples are just born different in how they act.’

Consider what that could mean. Throughout this paper, I have avoided raising political issues. That does not mean they do not exist. To start, NHAI says that the mind of Jews evolved to acquire more money. I see a problem with that. Sure, bigots will use or ignore any information as it suits their purpose, but this is just lobbing them a softball. Gilman (1997) has shown that the seemingly flattering image of “smart Jews” goes hand in hand with the imputation of lesser virtue, and the need to set Jews apart.

Beyond the image of Jews, in the current vogue of “cultural explanations” in international relations thinking, this approach suggests that cultural gaps may be bred in the genes. In the ultra-Darwinian social world projected by sociobiology and evolutionary psychology—which Nicholas

Wade has presented regularly in the Times over the years—genetic difference means intractable competition. The “Clash of Civilizations” could come to be seen as an evolutionary struggle for survival. We have heard that before. The psychological variation espoused in NHAI and The New York Times has the potential for pushing the recent scientific controversy on race into more politically loaded form. Will future debates on immigration ask what behavioral tendencies may lurk in Mexican genes? What subterranean proclivities could we imagine going along with the physical features profiled as “a Middle Eastern type”? NHAI itself is quite temperate in restricting the analysis to an very unusual situation of social selection, but consider how author Gregory Cochran explained the implications in the Times: “we’re going to have to rewrite every history book every written” (Wade 2006).

No one can say that it is impossible for population differences in genes to influence aspects of psychology, and through that, culture. It is often said about ideas such as this, that if it is true, then we must accept it. That is disingenuous rhetoric, because there is no possible way to know, for true, that different genes in different populations lead to different predispositions. In the vast theoretical plain that stretches from gene to behavior, multiplied by the complexities of defining and comparing human populations, can we expect to demonstrate convincingly that some posited genetic effect “really is true”? The untested NHAI hypothesis has already spread far and wide-- a “scientists say” idea floating around in the collective consciousness. More ideas like this will come. Anthropology needs to address them.

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