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Understanding the Genetics of Intelligence: Can Height Help? Can Corn Oil?

Wendy Johnson

Department of Psychology and Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, and Department of Psychology, University of Minnesota–Twin Cities

Abstract

Although the subject is controversial, identifying the specific genes that contribute to general cognitive ability (GCA) has seemed to have good prospects, at least among psychological traits. GCA is reliably and validly measured and strongly heritable, and it shows genetically mediated physiological associations and developmental stability. To date, however, results have been disappointing. Human height shows these measurement characteristics even more strongly than GCA, yet data have indicated that no individual gene has more than trivial effects and this is also true for corn oil. The potential for environmental trigger of genetic expression, long recognized in evolutionary and developmental genetics, as applied to these seemingly disparate traits, can help us to understand the apparent contradiction between the heritability of intelligence and other psychological traits and the difficulty of identifying specific genetic effects.

Keywords

heritability, general cognitive ability, height, genetic variation

Although the subject is controversial, identifying the genes associated with general cognitive ability (GCA) has had relatively good prospects compared to identifying genes for other psychological traits. GCA is measured reliably and shows good population variance. Its accumulated validity data across different specific measures and a broad range of life outcomes are voluminous, and a host of studies demonstrate strong genetic influences accounting for as much as 80% of population variance (i.e., heritability) in adulthood (Deary, Johnson, & Houlihan, 2009). Moreover, correlations among diverse specific cognitive abilities are generally genetically mediated, as are developmental stability and associations between proposed intermediary physiological markers of GCA such as brain size, white matter integrity and volume, gray matter volume, and cortical thickness (Segal & Johnson, 2009). The conventional wisdom has been that, together, these characteristics should make the search for the specific genetic variants involved rather straightforward. That is not how it has worked out so far though; despite many concerted and well-designed efforts, at this writing we know of no genetic variants reliably associated with variation in normal range GCA (Deary et al., 2009). Arguably, it is possible, even likely, that the difficulties pervading the search for the specific genes that contribute to GCA are the same difficulties plaguing the searches for the specific genes that contribute to other psychological traits and disorders. Thus, thinking more deeply about reasons for those difficulties may be of benefit in the search for the specific genes involved in psychological traits more generally.

Parallels Between GCA and Human Height

Height is not a psychological trait, but it shares many similarities with GCA that may be relevant to the apparent paradox that measurement properties are so good and heritability is so high but that there do not seem to be any genes with any substantive effects. Height can be measured even more reliably, is about 80% heritable (Visscher, 2008), and shows good population variance. As with GCA, developmental patterns are genetically mediated (Silventoinen et. al, 2008) as are associations between height and other anthropomorphic measures such as limb length, weight, length of spinal column, and shoulder breadth (e.g., Malkin, Ermakov, Kobyliansky, & Livshits, 2006). That is, to an even greater degree than intelligence, height shows the properties that conventional wisdom has

Corresponding Author:
Wendy Johnson, Department of Psychology and Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, 7 George Square, Edinburgh, UK EH8 9JZ
E-mail: wendy.johnson@ed.ac.uk

suggested should make even traits involving many genes yield their secrets to our molecular genetic tools. Three recent genome-wide association studies of height, including some 63,000 people, however, reported a total of 54 specific genetic variants involved (Visscher, 2008). Together, these variants might have accounted for about 5% of the variance in height. Two of these variants were reported in all three studies, and eight were reported in two. A subsequent study showed replication accounting for 1% of the variance (Sammalisto et al., 2008). Do we need more and larger studies to uncover all the genes involved, or have we learned enough to conclude that there are few, if any, specific genes with any consistently meaningful effects on height? Might GCA be similar?

Height shares some other characteristics with GCA that make this a reasonable conjecture. Like GCA but unlike some other continuous traits such as skin color, height is a developmental trait, normally monotonically increasing throughout childhood and then stabilizing in adulthood until old age. This means that there are opportunities throughout development for environmental perturbations that could either interrupt or accentuate expression of their contributing genes. Height and GCA are also correlated, about .2, and the correlation is primarily genetically mediated (Silventoinen, Posthuma, van Beisterveldt, Bartels, & Boomsma, 2006), so some of the same genes might even be involved. Both height and GCA are affected by known mutations considered abnormal, but these are rare and do not account for much ''normal'' population variance. Both show considerable national mean differences, and the national means vary with national income and national income disparity (Lynn, 2008; Steckel, 2008), suggesting either the presence of national differences in relevant gene frequency or important environmental variables, or both. At the same time, both height and GCA also show well-documented increasing secular trends (the so-called Flynn Effect for GCA; Flynn, 1994; see, e.g., Steckel, 2008, for height), reinforcing the notion that environmental variables may contribute directly to changes in means without contributing to variances—that is, they may have the same effects on everyone.

There are some other puzzles involving height that are potentially relevant to GCA. Though its secular trend has generally been increasing, there have been many exceptions. For example, northern Europeans in the 11th century averaged 3 inches taller than their descendents in the 18th, which is generally attributed to increased urbanization and industrialization during this period, which initially resulted in poorer nutrition and living conditions due to crowding, absence of light, and so on. Over the last 140 years, due to stagnation of increases in U.S. height but continuing increase in the Netherlands, average U.S. height has gone from being 3 inches taller than the Dutch to 3 inches shorter (Komlos & Lauderdale, 2007). Average Canadian height, however, continues to increase, although not as fast as Dutch (Cranfield & Inwood, 2007). South Koreans average about 6 inches taller than North Koreans (e.g., Schwekendiek, 2009), a difference almost impossible to attribute to genetics. Taken together, all of these puzzles suggest large environmental effects on this 80% heritable trait. That is, depending on environmental circumstances, the height of any particular individual could range considerably, but within population groups, most observed differences are strongly genetically influenced because relevant environmental circumstances are similar. Could this also be true of GCA? How might this situation arise?

Considerations Involving Heritability of Developmental Traits

It has been known for quite some time that the heritability of GCA increases with age (Deary et al., 2009). Many studies have shown that shared environmental influences account for perhaps 35% of its variance in early childhood, and genetic influences account for about 30%. As children move toward adolescence and then adulthood, shared environmental influences gradually fall to about 0% and genetic influences gradually increase to as much as 80%. Figure 1A shows this general pattern. Heritability of height with age has not been as extensively examined, but one study (Silventoinen et. al, 2008) has shown something similar, though the changes were much more concentrated in early childhood. Figure 1B shows this general pattern.

Why does this matter? All estimates of heritability rely on the assumption that genetic and environmental influences are independent, yet the more we learn about genetics, the clearer it is that this assumption does not hold. In particular, it does not hold for developmental traits like GCA and height, precisely because of the continual possibilities of environmental perturbations that could either interrupt or accentuate expression of their contributing genes. Moreover, the environment is never constant, and individual humans always exert at least some control over the environments they experience. This means that genetic and environmental influences tend to be correlated: Genes influence the kinds of environmental exposures people receive. It also means that genetic and environmental influences tend to interact: Genes will influence individual differences in the effects environments have on people. Because the control individuals have over the environments they experience increases with age, these two forms of gene–environment interplay tend to coexist (Johnson, 2007), and they do so to increasing degrees as people grow older. Where environmental effects are harmful, all those who can move to escape them, and the most sensitive, move first. Where environmental effects are beneficial, all those who can move toward them, and again the most sensitive, move first. These movements create correlations between whatever genetic influences constrain or facilitate movement and exposure to the environmental effects, and they create interactions between the environmental effects and genetic sensitivity to them. That is, both those most genetically sensitive and those most genetically able move first.

When genes and shared environments interact, the variance associated with the interaction gets thrown into the heritability estimate, thereby exaggerating it. When genetic and shared environmental influences are correlated, the variance tied up in the correlation gets thrown into the estimate of shared environmental influences. When genetic and nonshared

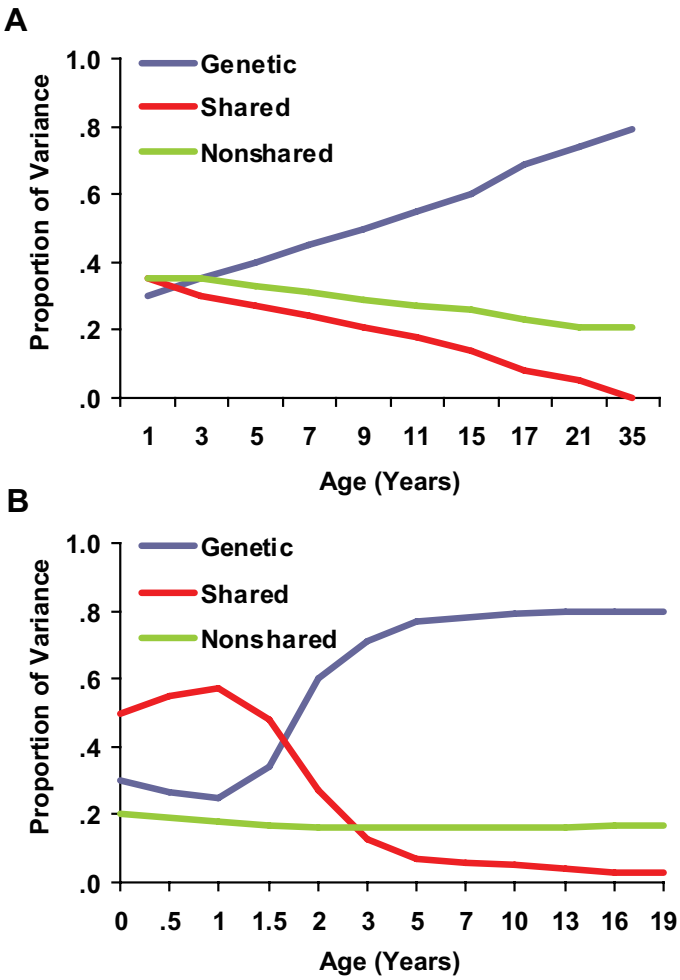

Fig. 1. Proportions of variance attributable to genetic and shared and nonshared environmental influences with age in (a) general cognitive ability (GCA) and (b) height. Data for GCA are summarized across many studies. Data for height are from Silventoinen et al. (2008), smoothed and adjusted to a more commonly observed 80% heritability for height in adulthood. Note the more detailed X-axis at the youngest ages for height than for GCA. Such violations of the assumption that genetic and environmental influences are independent have systematic effects on estimates of genetic and environmental influences (Purcell, 2002).

environmental influences are correlated, the variance tied up in the correlation gets thrown into the heritability estimate. Although we know almost as little about what specific environmental influences matter to GCA and height as we do about the specific genes involved, we know that genetic and environmental influences on GCA are correlated: Those who are brighter tend to use larger vocabularies in talking to their children, read more books to them, and are more likely to expose them to intellectual experiences of all kinds. To the extent that height is a marker of health (Tanner, 1990), similar gene–environment correlations may be involved. One way to interpret the developmental patterns in both GCA and height is that some major proportion of their population variance (35%?) is tied up in gene–environment correlations and their associated interactions, with shared environmental influences in early childhood that gradually become nonshared environmental influences in adulthood. The techniques we use to identify specific genes involved in polygenic traits rely heavily on the existence of direct main effects between genes and traits; the presence of such environmental conditioning makes detecting them much more difficult. It also makes the specific genes involved less relevant: If sorting on environmental experiences is accentuating the manifestation of genetic differences in traits like GCA that matter to life outcomes, it may be more important to understand the environmental sorting processes than to understand the genetic differences.

Height could have something else to teach us about the genetics of GCA. At any given height, the ranges of human weights and sizes of bone structure are large. Some appear relatively "large" for their height, as if height is stunted relative to overall body size. Others appear to be stretched—they are surprisingly tall for their overall bone structure. Perhaps optimal matching of overall body size and height is the genetically relevant trait rather than height itself. This would suggest the presence of some genes controlling variance in overall bone structure and others controlling variance in height given the overall bone structure. Throw in some differences among these genes in sensitivity to environmental conditions, and height itself could be almost irrelevant to the genetic and environmental processes that contribute to it. Figure 2 illustrates the idea for height. What might be analogous to optimal body size in GCA? Perhaps a balance between some kind of information processing capacity and reactivity to stimuli?

Parallels Between GCA and Corn Oil?

Corn oil? Yes, corn oil. Heritability estimation was originally developed to measure the response to selection that could be expected from agricultural breeding experiments. If we select for breeding only those plants or livestock (or whatever) that are at least, say, 1 standard deviation above the mean on some trait, the standarized difference between the mean in the original generation and the mean in the offspring generation is heritability, or the proportion of variance in the original generation that can be attributed to genetic influences. Of course we do not selectively breed humans, but psychologists interested in the presence of genetic influences on behavior realized that they could make use of the underlying quantitative genetic concepts. In selective breeding, the offspring generation presumably is more genetically homogeneous than the original, and thus shows less variance in the trait. Geneticists had thought that, if they kept on doing this, over time all the genetic variance would be eliminated as genes associated with lower levels of the trait were selected out, leaving only fixed genes for higher levels of the trait.

As in all areas of science, however, it makes sense to test even the ideas that seem most obvious. Thus, in one of the longest-running experiments ever, since 1896 geneticists at the University of Illinois have been looking at corn's response to

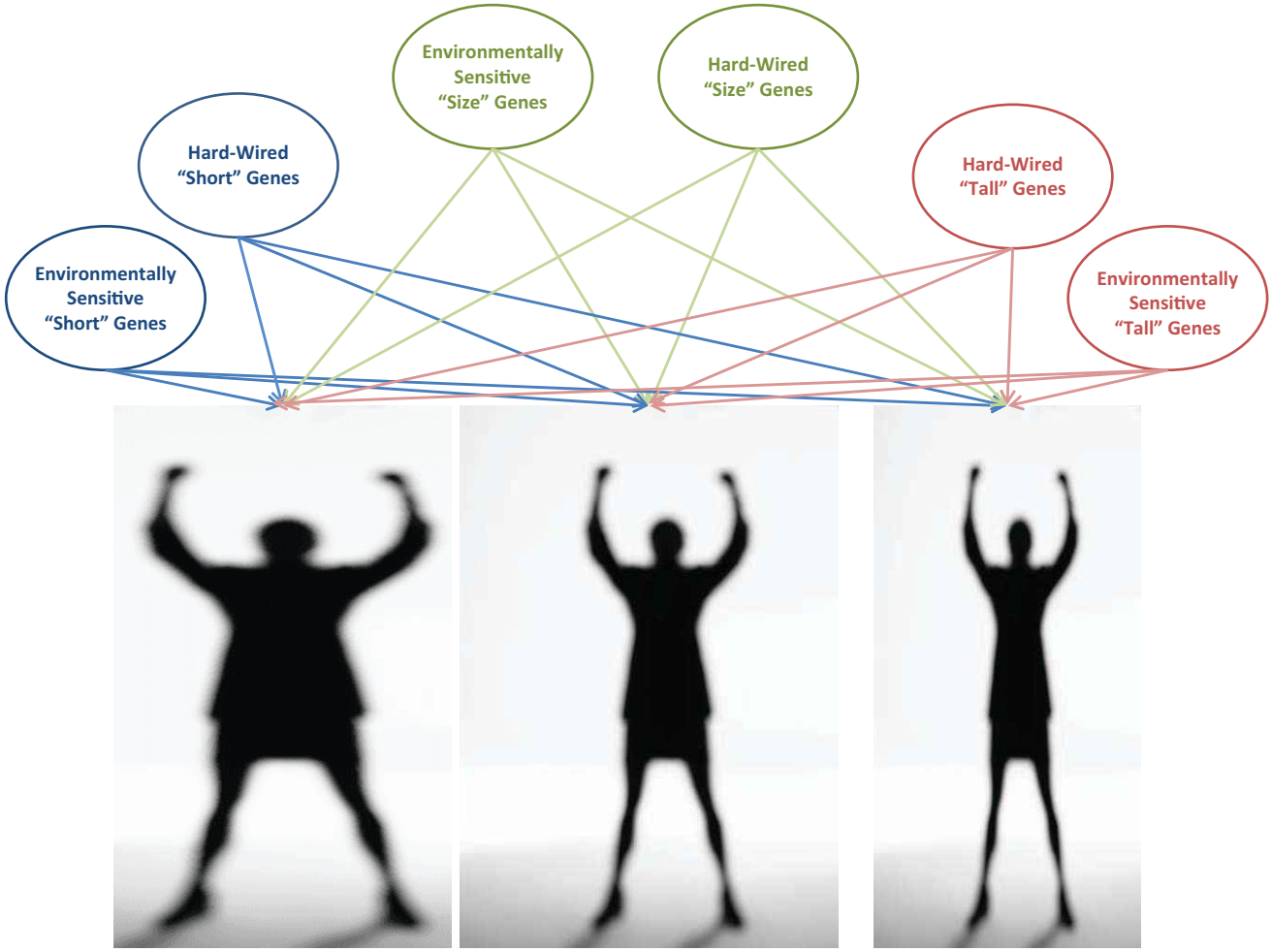

Fig. 2. Hypothetical contributions of genes to height and overall body size.

selection for oil content (Hill, 2005). Like GCA and height in humans, corn oil production is a continuous, polygenic trait, and corn survives well with a broad range of oil production levels. Unlike GCA and height in humans, researchers in this experiment have gotten to exert a lot of control over the corn's environment. They cannot control seasonal variation, but all the corn lines have been planted in the same area, so the rest of their environments have been quite uniform over time and especially within each generation of plantings.

The results have been a surprise to geneticists. Figure 3 (Hill, 2005, supplementary material) shows the history of corn's response to selection for oil content. The orange line indicates oil content over time when the plants producing the highest 20% oil content were selected. Oil content initially averaged about 5% and, with selection, has increased to more than 20% on average. The increase has been generally steady, with sporadic variations probably mostly reflecting seasonal variations. The slope of the line indicates the heritability of oil content, which was estimated as 96% after appropriate scale transformations (Laurie et al., 2004). The purple line indicates oil content over time when the lowest-producing 20% plants were selected. Beginning at the same average 5%, oil production decreased steadily and reached effectively 0 at about the 84th generation, at which point these plants were no longer viable. After 50 years, some of the corn that had been selected for high oil content was selected instead for low oil content, and vice versa. This produced the green and blue lines in Figure 3, respectively. After another 5 years, selection was reversed again for some of the corn on which selection had been reversed at 50 years. This produced the red line in Figure 3.

Geneticists had expected that, as each selected line became more genetically homogeneous from generation to generation, its ability to respond to selection in the opposite direction would decrease because the genes involved in opposite levels of corn oil would no longer be present in that selected line. That was not, however, what happened. In fact, the responses to selection after 50 and 55 generations were as great as the original responses (the slopes of the lines beginning at 50 generations in Fig. 3). The only ways for this to occur are for genes to contribute to oil production against some genetic backgrounds but not others, or for newly arising genes (in the form of mutations) to become involved. As we currently understand them, mutation rates are too low and unsystematic to account

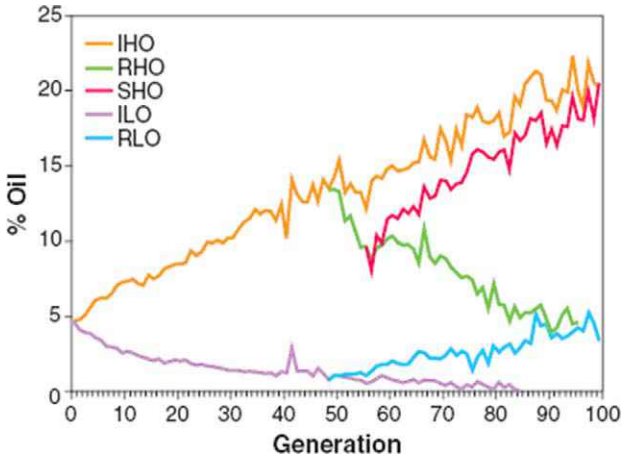

Fig. 3. Corn oil content as a response to selection over 100 generations. Line IHO was selected each generation for high oil content, and line ILO was selected (to generation 87) for low oil content. The direction of selection was reversed in lines RHO (started from IHO) and RLO (started from ILO) at generation 48. In line SHO (derived from RHO), selection was switched back to high oil content at generation 55. Reprinted from "A Century of Corn Selection," by W.G. Hill, 2005, *Science, 30*, Supplementary Material. Copyright 2005, American Association for the Advancement of Science. Reprinted with permission.

for the depth and consistency of the observed response to selection (W.G. Hill, personal communication; Le Rouzic, Siegel, & Carlborg, 2007). This implies that we have to consider seriously the possibility that gene expression patterns are heavily dependent on both genetic background and environmental circumstances. Moreover, consider the approximately 5% oil content level indicated by both the blue and green lines at the 95th generation. Given their very different selection histories, it is quite likely that this same level of oil content is produced by very *different* combinations of genes in the two lines. About 50 identified genetic loci appear to account for about 50% of the genetic variation in corn oil content (Laurie et al., 2004), but the others remain unknown. Despite the much greater level of control possible in evaluating the genetics of corn oil and the huge commercial payoff that could be realized by identifying the genes involved, we have done only marginally better in understanding the genetics of corn oil content than we have in understanding the genetics of human height. There is no reason to think that what goes on with the genetics of corn oil is *necessarily* relevant to human height, let alone GCA or any other psychological trait, but it is very reasonable to think that it might be. With the inevitable extensions involving greater environmental variation in humans, we have to consider the possibility that attempting to identify genes for traits like human height and GCA, not to mention other less stable, less clearly measurable, and apparently less heritable psychological traits, may simply not be the best way to understand them.

Conclusions and Food for Further Thought

The fact that genetic background and environmental circumstances can release previously unexpressed genetic variation has been known for well over 50 years, and many examples are documented in model organisms such as *Drosophila*. In recent years, however, the extent to which this fact is relevant to understanding both evolutionary processes and genetic influences on currently manifested traits has transformed the field of genetics. At the same time, much of the new technology that has been developed to explore the genome, sophisticated though it is, is still rooted in the idea—rapidly becoming outdated—of one-to-one associations between genetic loci and proteins and therefore traits. It is all too easy to be dazzled by the availability of our current molecular genetic technology. Together, however, the developmental natures of GCA and height, the likely influences of gene–environment correlations and interactions on their developmental processes, and the potential for genetic background and environmental circumstances to release previously unexpressed genetic variation suggest that very different combinations of genes may produce identical IQs or heights or levels of any other psychological trait. And the same genes may produce very different IQs and heights against different genetic backgrounds and in different environmental circumstances. This would be especially the case if height and GCA and other psychological traits are only single facets of multifaceted traits actually under more systematic genetic regulation, such as overall body size and balance between processing capacity and stimulus reactivity. Genetic influences on individual differences in psychological characteristics are real and important but are unlikely to be straightforward and deterministic. We will understand them best through investigation of their manifestation in biological and social developmental processes.

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