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The secular rise in IQ: Giving heterosis a closer look

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Abstract

Although most discussions today start from the assumption that the secular rise in IQ must be environmental in origin, three reasons warrant giving the genetic phenomenon heterosis a closer look as a potential cause. First, it easily accounts for both the high heritability and low shared environmental effects seen in IQ, findings that are difficult to reconcile with environmental hypotheses. Second, numerous other highly heritable traits, both physical as well as psychological, have also undergone large secular changes in parallel with IQ, which is consistent with the occurrence of broad-based genetic change like heterosis. And third, a heterosis hypothesis for the trend can be tested in several straightforward ways. The paper also provides a hypothetical example, based on data from a real population, of how heterosis can result from demographic changes like those that have taken place throughout the developed world in recent history and shows that under certain conditions, even a small demographic change could cause large genetically based phenotypic changes.

Keywords: Heterosis; IQ; Intelligence

1. Introduction

Identifying the cause of the secular rise in IQ test scores represents one of the most puzzling questions facing the field of intelligence research today. This paper explores the potential role of one possible cause, the genetic phenomenon heterosis, often referred to as hybrid vigor. Although heterosis has occasionally come up in discussions of the IQ trend, it has never been examined in any detail. For example, both Jensen (1998, p. 327) and Kane and Oakland (2000) list heterosis as one of several possible causes of the trend; Jensen also provides a brief description of the phenomenon. Flynn (1998) and Dickens and Flynn (2001) state that heterosis may have been a partial cause of pre-1950 IQ gains,

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although they provide no detailed explanation as to why the effects of heterosis must have stopped in 1950. However, the most common treatment of heterosis in current discussions of the IQ trend is to simply ignore it. Such discussions usually start from the assumption that IQ trend must be entirely environmental in origin thus dismissing genetic factors like heterosis without even explicitly stating that they have been ruled out (Fernandez-Ballesteros, Juan-Espinosa, & Abad, 2001; Neisser, 1998; Wahlsten, 1997).

Three reasons justify giving heterosis a much closer look than has been the case up to now. These reasons, which together form the outline of the present paper, are as follows:

1. Because it is a genetic mechanism, heterosis easily accounts for the high heritability, the lack of any secular change in estimates of the heritability, and the low shared environmental effects seen in IQ, findings that are difficult to reconcile with environmental hypotheses.
2. Because it alters a very fundamental aspect of a population's genetic structure, heterosis has the potential to account for a number of other unexplained secular trends that have largely paralleled the IQ trend. Like IQ, several of these other traits have also displayed consistently high heritability and low shared environmental effects.
3. Because it posits a very specific causal factor, a heterosis hypothesis for the IQ trend generates several straightforward predictions, which if confirmed would be difficult to explain except as the result of heterosis. That is, a heterosis hypothesis for the trend is testable.

2. The "IQ paradox" and the need for a "factor X"

In a recent article, Dickens and Flynn (2001) use the term "IQ paradox" to refer to the fact that estimates of the heritability of IQ have remained consistently high over extended periods of steadily rising IQ in many countries. This finding would seem to suggest that any presumed environmental factor(s) responsible for the IQ trend could not have been a measurable source of environmental variance within any given generation. There are two different ways to look at the situation, depending on the specific type of environmental causal factor proposed. Some environmental hypotheses view IQ trend as resulting from the gradual alleviation of some environmental deprivation, such as poor nutrition. If, however, this potent deprivation had varied at all in earlier generations, then alleviating it should have also removed a significant source of environmental variance, thereby raising heritability estimates over time. Other environmental hypotheses, by contrast, view the trend as an artificial effect resulting from something like more test-taking practice. However, unless this artificial IQ-boosting factor is today affecting all individuals equally, then its gradual introduction into the IQ environment should have also represented the introduction of a new source of environmental IQ variance, thereby lowering heritability estimates over time.

In addition, the finding of a limited role for shared family environment in IQ, especially adult IQ, is also paradoxical for environmental hypotheses. Studies of adopted children suggest that sharing the same household has a significant effect on IQ in childhood but that the effect diminishes with age to essentially zero by adulthood (Loehlin, Horn, & Willerman, 1989; Scarr & Weinberg, 1978). What makes this finding so puzzling is that these adoption studies were conducted in countries that were in the midst of ongoing secular increases in adult IQ at the time, such as the United States (Kane & Oakland, 2000). Therefore, whatever aspect of the environment was presumably changing over time across the

entire population could not have varied significantly among families because any such variance should have shown up as a shared family environmental effect on adult IQ.

In addition, any between-family variance in the environmental factors responsible for the IQ trend over time would cause MZA twins to be less alike when reunited. It would also cause siblings to be more alike. However, there is no evidence of any secular trends in either MZA twin (Bouchard, 1998) or sibling correlations (Jensen, 1998, pp. 322–323). As with the results of adoption studies, these findings suggest that any environmental factor(s) responsible for the IQ trend over time never varied among families at any single point in time.

The above findings do not necessarily render environmental hypotheses logically impossible. As has often been pointed out in the context of ethnic differences in IQ, a source of variance between populations (in this case, between generations) need not also be a source of within-population variance (Lewontin, 1970). Although no one disputes that an environmental factor with such properties is possible, it has, however, been argued that such a factor is not very plausible, and the term "factor X" has been used somewhat derisively to refer to a hypothetical environmental factor that somehow varies significantly between different groups but never within any group (Flynn, 1980, pp. 54–63; Jensen, 1973, pp. 136–138). Dickens and Flynn (2001) have likewise applied the term "factor X" to potential environmental causes of the secular IQ trend, which, given the consistently high heritability of IQ, would appear to have varied a great deal between generations without varying at any single point in time, either among individuals or among households.

Although for convenience I will follow Dickens and Flynn (2001) in applying the term "factor X" to potential causes of the IQ trend, I would note that ethnic IQ differences are not entirely analogous to IQ differences over time in one important respect. Because full biological siblings inevitably belong to the same ethnic group (however defined), an ethnic factor X cannot be partitioned into shared and nonshared family components; such a factor is, by definition, common to all members of the group. However, because siblings can and usually do belong to different cohorts, it is possible to determine whether a secular trend is occurring only between families or both between and within families. I only mention this point here but will discuss it in greater detail in a later section on testing the heterosis hypothesis.

The most direct way to resolve the IQ paradox without positing an implausible environmental factor X is to posit genetic change. If the IQ trend has been genetic in origin, then one would not necessarily expect any consistent directional change in heritability estimates. Genetic factors could have always been the primary source of IQ variance, with environmental factors playing a minor role, throughout the entire period of rising scores. Also, genetic changes would not be expected to induce any shared environmental effect. One might even say that the so-called "IQ paradox" is only created when one insists that the IQ trend be environmental in origin.

Based on the above arguments, there would seem to be no plausible way to explain the IQ trend without positing large genetic changes. Today, however, advocates of the various environmental hypotheses that have been advanced generally fail to confront or explain these paradoxical empirical findings. One noteworthy exception is the recent article by Dickens and Flynn (2001) in which the authors frankly admit that all of the most frequently suggested causal factors, such as nutrition and better schooling, are implausible because at any given point in time, these factors must have varied across the affected populations. Interestingly, however, Dickens and Flynn do not draw the conclusion (expressed in this paper) that the IQ paradox therefore requires positing genetic change. Instead, the authors have developed a complex analytical model that they claim resolves the IQ paradox without positing either a factor X or genetic change. Briefly, they employ several analytical techniques, such as positing a large

gene–environment correlation and a social multiplier effect whereby an individual's IQ is affected by the mean IQ of the larger population, with each technique being used to account for part of the paradox. Two very good reviews of the Dickens/Flynn model have already been published that raise serious questions about the model's plausibility as well as its testability (Loehlin, 2002; Rowe & Rodgers, 2002), and so a lengthy discussion of the model is not necessary here. Based on these first critiques, however, it is clear that the Dickens/Flynn model currently falls short of a resolution to the paradox; many open questions remain.

One additional point that has yet to be raised in the debate over the Dickens/Flynn model is that the so-called "IQ paradox" is not at all unique to IQ. On the contrary, it is actually difficult to find a heritable human trait that has not undergone large secular change in recent history; several of the most highly heritable of these traits are discussed immediately below. For this reason, even if the Dickens/Flynn model was to eventually evolve into a more detailed, compelling environmental explanation for the IQ trend, we would still need at least a half dozen additional Dickens/Flynn-type models, each tailored to a different secular trend in a different highly heritable trait in order to resolve all the paradoxical trends that have occurred. I would add that some of the techniques used in the Dickens/Flynn model, which are already questionable in the context of IQ, would likely be even less applicable to other traits like those mentioned below.

3. Secular trends in other traits

The genetic phenomenon heterosis increases the ratio of heterozygous to homozygous genotypes throughout the entire genome of a population (Dahlberg, 1942, pp. 180–192; Jensen, 1998, p. 327; see also the discussion in the next section). Because recessive alleles affect the phenotype primarily in the homozygous state but have little effect in the heterozygous state, heterosis tends to decrease the phenotypic expression of all recessive alleles and increase the expression of all dominant alleles in the population and affects all traits that display what is known as directional dominance (Falconer, 1981, p. 227). This is the situation in which more of the alleles influencing a trait in one direction are dominant while more of those influencing the trait in the opposite direction are recessive, with the effect of heterosis being to cause secular trends toward all the generally more dominant phenotypes. Because heterosis represents such broad-based genetic change, a heterosis hypothesis for the IQ trend predicts that other traits would likely undergo parallel secular trends along with IQ, which has in fact occurred.

The following is a partial list of genetically influenced traits that have undergone secular changes that have largely paralleled the IQ trend in their timing, location, and relative magnitudes. Many other traits could have been added to this list. In fact, because heterosis represents such broad-based change, almost any unexplained secular trend in any genetically influenced trait could conceivably represent the effects of heterosis. I have restricted the current list to traits with heritability in excess of 0.6; Dickens and Flynn (2001), after Herrnstein and Murray (1994, pp. 298–299), point out that heritability this low still poses a paradox for environmental explanations for an observed secular trend.

3.1. Height

Like IQ, average height has risen steadily throughout the developed world (Eveleth & Tanner, 1990, pp. 205–206; Schmidt, Jorgensen, & Michaelsen, 1995; Shay, 1994; Tanner, Hayashi, Preece, &

Cameron, 1982; Van Wieringen, 1986). A good example is The Netherlands, where average male height has increased by 16 cm or just over 6 in. since the mid 1800s representing an overall gain in excess of two SD (Van Wieringen, 1986). Although it has been suggested that the trends in height and IQ have not always precisely overlapped, the discrepancies are relatively minor and others actually cite the parallel nature of the two trends to argue for a common cause (see the exchange between Martorell, 1998, and Lynn, 1998, where Lynn argues for nutrition as the common cause).

Estimates of the heritability of height generally exceed 0.8 (Carter & Marshall, 1978) and appear to have been just as high in even the earliest studies (Pearson & Lee, 1903); nor is there any evidence that shared environmental factors have ever been a significant source of within-generation variance in height (Farber, 1981, pp. 65–66). Even those who argue that the height trend is environmental in origin admit that its cause must be acting like a factor X, varying greatly over time but very little within any single generation (Tanner, 1994) thus creating a "height paradox" identical to that seen in IQ. In fact, it was precisely this height paradox that originally led the Swedish geneticist Gunnar Dahlberg (1942, pp. 180–192) to first suggest that heterosis might be occurring as a result of the ongoing breakdown of small isolated villages. Interestingly, Dahlberg also raised the possibility that heterosis might have a beneficial effect on intelligence thus anticipating the secular rise in IQ some 4 decades before it was fully documented by the scientific community.

3.2. Growth rate

The rate at which children grow and mature sexually has increased dramatically throughout the developed world. Two of the clearest indicators of this process are the age at which girls experience their first menstruation, or menarche, and the age at which the adolescent growth spurt of both boys and girls reaches its peak height velocity (PHV). In many countries, both these milestones of maturation occur today about 2 years (or 2 SD) earlier than they did at the start of the 20th century (Eveleth & Tanner, 1990, p. 207; Tanner, 1990, pp. 156–162). The heritability of age at menarche is approximately 0.8 with no clear evidence of consistent secular change (Bailey & Garn, 1986). In girls, age at menarche and age at PHV are highly correlated (Hoshi & Kouchi, 1981), and evidence suggests that age at PHV is also highly heritable (Fischbein, 1977).

3.3. Myopia

A number of countries around the world have reported large increases in the incidence of myopia (nearsightedness). The following two examples are drawn from a much longer list compiled by Storfer (1999, pp. 159–162). The rate of myopia in Singapore military conscripts tested between 1974 and 1984 was 26.3%, as compared to 43.3% in those tested between 1987 and 1991. In urban areas of Finland, the incidence of myopia in 14-year-olds born in the late 1960s was nearly double (24%) the incidence of those born 50 years earlier. The heritability of myopia is estimated at 0.8 (Hammond, Snieder, Gilbert, & Spector, 2001) with no evidence that these estimates have changed throughout the 20th century (Karlsson, 1974). As with height, advocates of environmental hypotheses for the myopia trend admit that its cause is acting like a factor X (Wallman in Chew et al., 1994).

In addition to documenting the myopia trend, Storfer (1999) also offers a possible explanation for it. His hypothesis is especially interesting because he not only tries to explain more than one secular trend, but also tries to account for the high heritability of the traits in question. Storfer cites the

parallel nature of trends in three traits—myopia, IQ, and brain size—and argues that their common cause is a process known as genomic imprinting, which acts as an essentially Lamarckian mechanism for intergenerational change. Specifically, he proposes that the amount of visual stimulation experienced by one generation affects the phenotype of their children and even grandchildren (p. 205). Although interesting, Storfer's hypothesis currently falls short of what might be considered a complete, convincing explanation for the IQ rise. For one thing, epigenetic processes like genomic imprinting are not yet very well understood (Jablonka & Lamb, 1995). Also, although the hypothesis attempts to explain three separate trends, it is still not clear how it might be applied to trends in other heritable traits. In particular, Storfer specifically does not invoke imprinting as the cause of the height trend (pp. 195–196).

3.4. Asthma

There is a large and growing body of evidence that the incidence of asthma has been increasing in recent decades (Hansen, Rappeport, Vestbo, & Lange, 2000; Magnus & Jaakkola, 1997; Upton et al., 2000). To take one example, in a region of Western Scotland, the incidence of atopic asthma rose from approximately 3.0% in 1974 to 8.2% in 1996 (Upton et al., 2000). Estimates of the heritability of asthma converge on a figure of approximately 0.65 (Sandford, Weir, & Paré, 1996), and the role of shared family environmental factors in asthma appears minimal (Harris, Magnus, Samuelsen, & Tambs, 1997). Although it has been suggested that heritability estimates have risen along with rising incidence (Sibbald, 1997), this apparent increase in heritability may be due to different ascertainment methods used in earlier studies (Duffy, 1992; Sibbald, 1997).

3.5. Autism

There is a growing body of evidence that the incidence of autism has been on the rise in recent decades in a number of countries (California Department of Developmental Services, 1999; Gillberg, Steffenburg, & Schaumann, 1991; Gillberg & Wing, 1999; Powell et al., 2000; Taylor et al., 1999; Webb, Lobo, Hervas, Scourfield, & Fraser, 1997; but see also Fombonne, du Mazaubrun, Cans, & Grandjean, 1997). For example, a series of epidemiological studies in the Goteborg region of Sweden indicates that the incidence of autism nearly tripled, from approximately 4/10,000 in 1980 to approximately 11.5/10,000 in 1988 (Gillberg et al., 1991). The heritability of autism is estimated at 0.9, making it one of the most highly heritable of all psychological disorders (Bailey et al., 1995; Szatmari, Jones, Zwaigenbaum, & MacLean, 1998). It should be noted that despite the large number of studies reporting rising incidence, many autism researchers are still skeptical that a real change in incidence is occurring. Interestingly, it has been argued that the high heritability of autism makes rapid secular change unlikely (e.g., Fombonne, 1996). However, as this list demonstrates, the occurrence of large secular trends in even the most highly heritable traits has been a common occurrence throughout the developed world during the 20th century.

3.6. Attention deficit hyperactivity disorder (ADHD)

ADHD has gone from being virtually unknown 30 years ago to now being diagnosed in approximately 5% of school-aged children in the United States. As with autism, there is currently

considerable disagreement among experts in the field as to the meaning of this apparent increase, with some even questioning whether ADHD is a legitimate diagnosis (see the exchange between DeGrandpre & Hinshaw, 2000). However, I have chosen to include it in this list primarily because of the disorder's high heritability, which is estimated at 0.8 (Tannock, 1998, pp. 85–89).

3.7. Head circumference

A review of five studies conducted in different parts of the UK throughout the 20th century by Ounsted, Moar, and Scott (1985) revealed generally increasing head circumference over time. For example, in the only two studies conducted in the same locality, the head circumference of male 7-year-olds increased approximately 1 cm in the 27 years between the two studies for an average rate of gain of approximately 0.25 S.D. per decade, which comports well with the rates of gain generally seen in both height and IQ. Estimates of the heritability of head circumference include values of 0.74 (Clark, 1956) and 0.5 (Weaver & Christian, 1980).

In connection with the evidence of increasing head circumference, it is worth noting that there is also evidence of a secular increase in brain size. A number of cross-sectional studies have reported negative associations between brain size and age, which could represent an age-related decline in brain size, but which Storfer (1999, pp. 187–191) has argued for several reasons to be likely due to a secular increase in brain size (see also Miller & Corsellis, 1977). There is a real need for a more definitive answer to this important question of whether brain dimensions are actually changing, especially in light of the fact that IQ is rising and the incidence of at least two cognitive disorders, ADHD and autism, appear to be rising at alarming rates. I would like to join Storfer (1999, p. 274) in suggesting that individuals who were given MRIs many years ago be retested to provide data on the extent to which brain size declines with age, and which could then be compared to cross-sectional studies to quantify the magnitude of any secular trend.

3.8. Head breadth

The situation with head breadth represents one of the few real inconsistencies for the heterosis hypothesis. Specifically, head breadth appears to have trended in different directions in different parts of the world. In Japan, Korea, and the southern regions of Italy, head breadth has increased (Formenti, Cavagna, & Basso, 1991; Kouchi, 2000). However, from central Italy north through France and Germany, head breadth has actually declined (Billy, 1980; Giot, 1949; Vercauteren, 1990; Zellner, Jaeger, & Kromeyer-Hauschild, 1998). A full discussion of the possible explanations for this finding is beyond the scope of this paper. I would only note here that, although puzzling from the perspective of the heterosis hypothesis, the disparate changes in head breadth are also not easily explained by any simple environmental hypothesis (Kouchi, 2000).

At present, no compelling environmental explanation exists for any one of the trends listed above. Furthermore, considering that several of the trends were first documented many decades ago, the likelihood that convincing environmental explanations for all of them will be found any time soon seems extremely remote. This is not to suggest that the search for possible environmental explanations should be abandoned. However, because heterosis represents such broad-based genetic change, it has the potential to explain all of the trends listed above (as well as numerous others) and therefore deserves a closer look.

4. Conditions necessary for heterosis

This section provides an example of how the process of heterosis can cause secular trends in traits like IQ. The example starts with genetic data from a real population. It then posits demographic changes similar to those that have occurred in recent history throughout the developed world and estimates the likely genetic effects of such changes. It is intended primarily to identify the basic conditions that must be in place for heterosis to occur; I will then argue that these conditions have very likely been in place in those countries, and at those times, that IQ gains have been recorded. The example is also intended to clear up some common misconceptions about heterosis and to detail an important theoretical point regarding the association between the genetic changes associated with heterosis and their potential effect on the phenotype of polygenic traits like IQ.

In the late 1960s and early 1970s, Neel (1994) and his colleagues, working among the Yanomama Indians of Northwestern Brazil, collected some of the most detailed data now available on the genetic structure of a population. Fig. 1 depicts the frequencies of 16 alleles (occurring at nine different loci) in 48 villages of the Yanomama (Neel, 1994, p. 193). Each dot in the figure represents the frequency of a

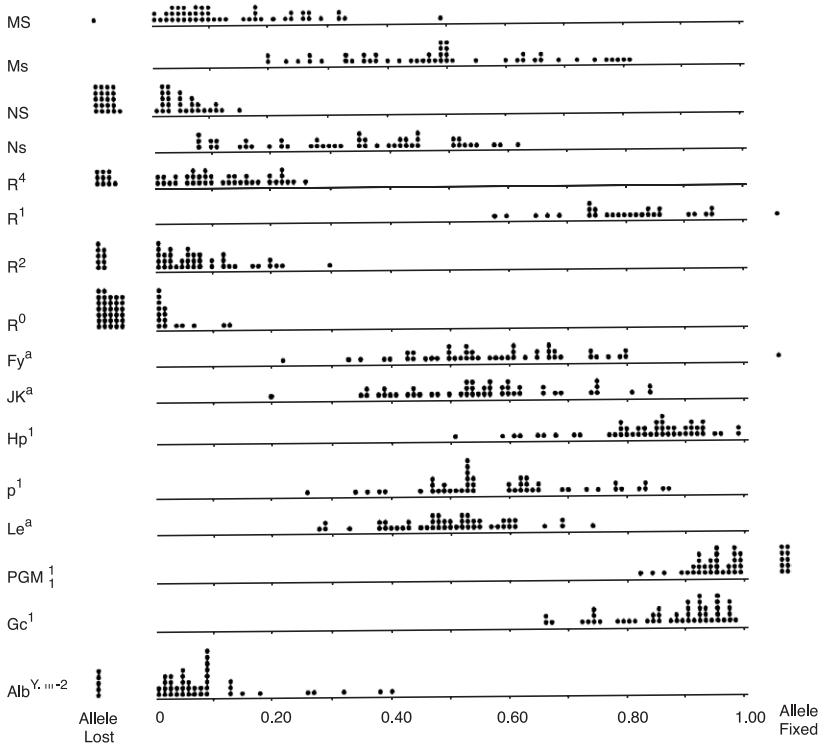

Fig. 1. Allele frequencies in villages of the Yanomama. This material is used by permission of John Wiley & Sons, Inc. and author J.V. Neel; © 1994 by John Wiley & Sons, Inc.

particular allele in a single village. There is a common misconception that if a group of people shares the same language, culture, and ethnic identity, they must also be homogenous genetically. However, Fig. 1 demonstrates that such superficial indicators of homogeneity can mask considerable genetic heterogeneity. Clearly, each Yanomama village is not genetically identical; instead, there is a strong tendency for allele frequencies to fluctuate from village to village. This type of small-scale genetic heterogeneity that occurs between towns or villages is referred to as microdifferentiation (Neel, 1994, pp. 191–209), and it represents the fuel for future genetic change through heterosis.

Now, let us imagine that demographic changes take place among the Yanomama that break down the barriers to communication and mating between villages. Such changes might include urbanization or improved transportation between villages. If such a trend were to continue long enough, the Yanomama would eventually approach panmixia, whereby mating takes place at random across the whole population rather than mostly within the 48 separate and genetically distinct villages depicted in Fig. 1. According to the principle from population genetics known as Wahlund's principle, this kind of demographic change would be accompanied by genetic changes. Specifically, Wahlund's principle states that when two or more genetically distinct subpopulations combine to form a new amalgamated population, there will be an overall decline in homozygotes and a corresponding increase in heterozygotes (Hartl, 1981, pp. 97–101).

In the case of the Yanomama, it is a simple matter to estimate the decline in homozygotes that would occur if the population demography were to shift from the microdifferentiation depicted in Fig. 1 to complete panmixia. For any randomly mating breeding population, the likely percentage of the population that will be homozygous for a given allele is simply the square of the allele frequency. Under microdifferentiation (i.e., 48 separate breeding populations), the overall percentage of homozygotes can be estimated by first squaring the allele frequency of each village separately then averaging the resulting squared values. By contrast, under an assumption of panmixia (i.e., a single breeding population), the percentage of homozygotes is estimated by first averaging the allele frequencies of all the villages then squaring the resulting average value (the above procedure assumes that each village is approximately equal in size).

Table 1 lists the percentage of the entire Yanomama population that would likely be homozygous for each of the 16 alleles studied, both under microdifferentiation (column 5) as well as under complete panmixia (column 6). As expected from Wahlund's principle, the overall expected percentage of homozygotes in the population is lower under panmixia for every allele at every locus. Although not calculated here, the decline in homozygotes shown in Table 1 would of course be accompanied by a corresponding increase in heterozygotes at each locus.

Table 1 serves to demonstrate an important point about the relationship between a given demographic change and its likely effect on the phenotype. First, it turns out that in this type of simplified model of isolate breaking, the decline in homozygotes is equal to the variance of allele frequencies among the villages (i.e., column 5 − column 6 = column 4; see Hartl & Clark, 1997, pp. 122–124 for a detailed discussion). For the 16 alleles studied, this absolute decline in homozygotes tends to fall within a relatively narrow range from 0% to 3% of the total population, meaning that for every 1000 individuals in the population, a shift to completely random mating would mean the loss of 0–30 homozygotes for each allele. However, although this absolute decline in homozygotes is roughly similar for all the alleles, the relative decline that this represents naturally tends to be much larger for rare alleles than for more common alleles. This can be seen quite clearly by examining the last two alleles (Gc^{1} and Alb^{Ya}) in Table 1. Although the absolute decline in homozygotes is the same for both alleles (about 0.8% of the

Table 1
Decline in homozygosity under panmixia

Allele	Villages (n)	Mean frequency	Variance (absolute decline in homozygotes)	Frequency of homozygotes		Relative decline in homozygotes (%)
				With microdifferentiation[a]	Under panmixia[b]	
MS	48	0.139	0.012	0.031	0.019	39
Ms	48	0.494	0.029	0.273	0.244	11
NS	48	0.033	0.0015	0.0026	0.0011	58
Ns	48	0.336	0.024	0.137	0.113	18
R^z	48	0.096	0.007	0.016	0.009	44
R1	28	0.803	0.011	0.655	0.644	2
R2	48	0.070	0.005	0.010	0.005	50
R^o	48	0.011	0.0008	0.0009	0.0001	89
Fy^9	47	0.577	0.021	0.354	0.333	6
JK^a	48	0.553	0.018	0.324	0.306	6
Hp^1	48	0.826	0.012	0.694	0.682	2
P^1	48	0.590	0.020	0.368	0.348	5
Le^1	48	0.499	0.010	0.259	0.249	4
PGM^1	43	0.953	0.002	0.911	0.909	0.2
Gc^1	48	0.866	0.008*	0.758	0.750	1*
Alb^{Ya}	47	0.097	0.008*	0.016	0.008	50*

p_i is the allele's frequency in each of the 48 Yanomama villages depicted in Fig. 1.

[a] $\left(\sum_{i=1}^{n} p_i^2\right)/n$.

[b] $\left(\left(\sum_{i=1}^{n} p_i\right)/n\right)^2$.

*see discussion in text.

population), this represents only a 1% reduction in the number of homozygotes in the case of the more common allele Gc^1 but a 50% reduction in the case of the rarer allele Alb^{Ya}. This disproportionate effect on rare alleles is important because it suggests that even a small demographic change could have a very large effect on the phenotype under certain conditions. Specifically, if there is a large number of relatively rare recessive alleles that influence the trait, then even a minor demographic shift toward more random mating could, in theory, cause a large change in the phenotype by eliminating a large fraction of the double recessives influencing the trait.

A common misconception about heterosis is that it must involve either an increase in mating between individuals of different ethnic groups or a decrease in mating between partners who are of some very close known relation, like cousins. For example, Flynn (1998) suggests that heterosis could not have been a very large source of post-1950 IQ gains presumably because the frequency of cousin marriages became quite rare in the industrialized world after that time, nor were there many interethnic marriages. However, the above example shows how this misconception can be wrong. First, all of the heterosis effect that occurred in the example took place among members of a single ethnic group, the Yanomama; no interethnic mating was posited. One can also imagine how heterosis effects could continue even after the frequency of cousin marriages becomes rare. For example, if, instead of 48 villages, our example involved the breakdown of several hundred or several thousand European villages, the frequency of cousin marriages could drop to almost zero well before complete panmixia was achieved across the entire population. In fact, in theory at least, heterosis could continue until there is complete panmixia across the whole species, a situation that is still a long way off.

The above example also serves to demonstrate the three basic conditions that must be present in order for heterosis to occur. First, the population must not be genetically homogeneous in its initial state but must instead be genetically heterogeneous to some extent. Second, demographic changes that move the population away from the genetically differentiated state toward a closer approximation to panmixia must occur. Importantly, this process need not occur instantly but can take place over many generations, with each incremental increase in heterozygosity causing commensurate changes in the affected traits. Finally, the affected traits must display directional dominance. Note that in the above example, the demographic changes do not alter the overall frequency of any of the alleles in the total Yanomama gene pool. The demographic changes merely reassort existing alleles into a new configuration. Therefore, if the alleles that influence a trait were to always combine in a strictly additive manner, then merely reassorting them in a different way will not change their mean effect on the phenotype. There would also be no mean effect if the number of recessives that influence the trait in one direction equals the number of recessives that have the opposite effect.

In order to rule out heterosis as a potential cause of some observed secular trend, one would have to show that at least one of the three necessary preconditions listed above was not present in the population in question during the period of secular change. However, although current investigations often start from the assumption that the secular rise in IQ must be environmental in origin, there is actually good reason to believe that all the conditions necessary for heterosis have been in place in those countries (and at those times) that IQ gains have been recorded.

First, one might argue that the kind of village microdifferentiation seen among the Yanomama, a Stone Age culture until recent times, has little relevance to the modern developed countries that have experienced IQ gains during the 20th century. In fact, however, the classic study of village microdifferentiation was conducted by Cavalli-Sforza (1966) and his colleagues in the Parma Valley area of Italy in the late 1950s. There, the researchers examined three blood groups (ABO, MN, and Rh) and found that the frequencies of the various alleles studied often fluctuated markedly from village to village. Interestingly, they found that the most pronounced microdifferentiation, as measured by the deviation of the village frequencies from the mean regional frequencies, occurred in the sparsely populated mountain regions where the kind of semi-isolated village lifestyle that was still common a few generations ago was still largely intact. The fact that genetic microdifferentiation could still be detected within a single region of a European country well into the 20th century should dispel the misconception that groups that appear homogeneous from the perspective of language, culture, or ethnic identity cannot differ at the level of the genes. At the very least, such a finding demands that one actually look at the genetic makeup of the population before assuming genetic homogeneity.

With respect to the second precondition for heterosis, few would dispute that dramatic demographic changes have occurred in the populations that have experienced IQ gains. As recently as a century ago ($\sim$ 4 generations), it was still common for individuals throughout the now developed world to live their whole lives in the same small village in which their ancestors had been residing for many previous generations. With increased urbanization and greater population mobility, the rural village lifestyle that used to be so common has virtually disappeared in many countries. For example, one of the largest and best-documented IQ gains occurred among Dutch military recruits on a version of the Raven matrices; the gain was approximately 21 points between those tested in 1952 and 1982 (Flynn, 1987). However, because they were tested at age 18, the recruits actually represented the offspring of marriages that took place decades earlier, circa 1930 and 1960, respectively. This 30-year period encompassed the Great Depression, World War II, and a postwar period of rapid industrialization in The Netherlands (Bax,

1990). It is difficult to imagine how these events could not have had the effect of reducing the number of marriages in which the partners shared significant common village ancestry. Although the specific historical events have varied from country to country, the general trend in all the populations that have experienced IQ gains has been toward a breakdown of small rural communities throughout the 20th century.

Finally, although far from definitive, there is considerable evidence from inbreeding studies supporting a role for positive directional dominance in the alleles that influence IQ (see Bouchard, 1993; Jensen, 1983 for reviews; but see also Kamin, 1980). What is typically done in such studies is to first divide the population into discrete groups that are likely to differ in average heterozygosity, with the division being based on some attribute of the subjects' parents. Ideally, some effort is made to control for possibly confounding environmental factors. Lower average IQ in the inbred group is then taken as evidence of directional dominance for the trait in question (some authors might refer to such studies as outbreeding or hybridization studies, depending on how the population is divided, but the basic methodology is still the same).

Interestingly, the magnitudes of the effects on IQ inbreeding studies have tended to be relatively small. In Japan, for example, Schull and Neel (1965) compared the offspring of cousin marriages (some as distant as second cousins) to the offspring of couples who were of no known relation and found the more inbred group to be 3.7 IQ points lower on the Wechsler Full Scale IQ after controlling a number of environmental factors. Agrawal, Sinha, and Jensen (1984) obtained a similar result (seven points on the Raven matrices) in a study of the male offspring of first cousin marriages in an Indian–Muslim community. Nagoshi and Johnson (1986) compared the offspring of interethnic marriages (Americans of European and Japanese ancestry) to those of intraethnic marriages in the Hawaii Family Study of Cognition. Although there were no significant socioeconomic differences between the groups, offspring of the interethnic marriages scored higher on 13 out of the 15 tests administered in the cognitive battery (e.g., 0.30 S.D. on a version of the Raven and 0.22 S.D. on the PMA Vocabulary Test). Also, although most would not call it an inbreeding study, Schreider (1969) found a negative association between the frequency of consanguineous marriages and the IQ of military conscripts from different regions (départments) of France. This result is consistent with positive directional dominance if one assumes that the frequency of consanguineous marriages is a reasonable indicator of the extent to which the regions' mating patterns deviated from panmixia.

Results of inbreeding studies also support a role for directional dominance in height (Damon, 1965; Hulse, 1964; Mange, 1964; Schreider, 1969; Schull & Neel, 1965; Shapiro, 1936, pp. 222–223), growth rate (Schreider, 1967; Wolanski, Jarosz, & Pyzuk, 1970), and infant mortality (Bittles & Neel, 1994), which has also declined in many countries in parallel with increasing height (Schmidt et al., 1995). Thus, four human traits for which there is reasonably good evidence of directional dominance have all undergone large parallel secular trends in the direction consistent with increased heterozygosity. Furthermore, I am not aware of any strong evidence that any of the other traits that have undergone secular change do not display directional dominance or display it in the wrong direction. That is, the results of inbreeding studies conducted to date provide no obvious discrepancies with the expectations of the heterosis hypothesis.

Therefore, there is at least preliminary evidence that all three of the conditions necessary for heterosis have been in place in the populations that have experienced rising IQ. Although merely stating that conditions have been ripe for heterosis does not prove that it is the cause of the trend, it does make heterosis a viable candidate. It also requires those who would dismiss heterosis to clearly state which of

the three conditions they believe have not been present. Most importantly, the fact that conditions conducive to heterosis have been in place means that it deserves closer scrutiny as a potential cause of the secular trends.

5. Testing the heterosis hypothesis

Because heterosis is a very specific causal factor, a heterosis hypothesis makes several clear predictions that can be tested in straightforward ways. This section discusses three approaches to testing the heterosis hypothesis for the IQ trend, as well as the trends in other traits. Although there are other possible tests of the heterosis hypothesis, the three approaches described below are likely to yield the most definitive results. That is, positive results on these tests would be difficult to explain without positing a role for heterosis in the IQ trend.

5.1. Approach 1—More and better inbreeding studies

As mentioned above, the results of existing inbreeding studies provide no obvious discrepancies for the heterosis hypothesis. Clearly, though, more studies involving more traits would provide a much stronger test of the hypothesis. If every trait and disorder that have undergone change also show evidence of positive directional dominance in inbreeding studies, it would require positing a very great coincidence without accepting a significant role for heterosis in the trends.

I would add that inbreeding studies of the type that have been carried out to date could be greatly improved by the use of direct genetic testing of the subjects. Thus far, these studies have tended to rely on proxies of likely heterozygosity such as parental relatedness, birthplace, or ethnicity. Because these proxies can only be a crude measure of the true genetic distance between parents, their use inevitably introduces some error into the studies, which, although unlikely to alter the direction of the result, could very well alter its magnitude. We are fortunate to live in an age in which we have excellent genetic tests that can determine the zygosity of a large number of loci in any individual or the number of alleles that any couple shares in common, so there is really no need to rely on genetic proxies.

The use of direct genetic testing might also provide insight into why inbreeding studies conducted thus far have shown relatively small effect sizes, which is something of a problem for the heterosis hypothesis. It may be that when we actually look at the genes, the parents categorized as outbreeding in the studies might not be much more distant genetically than those considered inbreeding. That is, there may be unexpected and counterintuitive phenomena going on at the genetic level as a result of complex demographic and social phenomena.

5.2. Approach 2—Testing for increasing heterozygosity over time

The heterosis hypothesis posits increases in the ratio of heterozygotes to homozygotes in the populations affected by the various secular trends. That is, older individuals should be more homozygous, on average, at virtually every locus as compared to younger individuals. Again, because we have good genetic tests, it should be a relatively straightforward process to measure such genetic changes if they have occurred. In fact, researchers around the world are currently collecting

genetic data from multigenerational samples in the search for the genes that influence various traits and disorders; such data should be suitable for testing the heterosis hypothesis' prediction of increasing heterozygosity over time (see, e.g., Hirschhorn et al., 2001; Plomin et al., 1994; Risch et al., 1999).

Although relatively straightforward, efforts to test for increasing heterozygosity face at least one methodological difficulty serious enough to mention here. The difficulty is that of possible population substructure and/or ethnic stratification. That is, different segments of the population (e.g., local, regional, ethnic, or religious groups) might be generally more or less heterozygous depending on specific events in their demographic history such as founder effects or genetic bottlenecks. One would therefore have to be careful not to inadvertently capture such differences when looking for secular genetic changes.

Probably the best way to avoid the potential problem of population substructure would be to restrict intergenerational comparisons to those within individual families. The ideal test would therefore take a large sample of families from a population that has undergone documented secular changes. According to the hypothesis, the children in the majority of families should be more heterozygous than their parents. By always comparing children to their own parents, the problem of population substructure would be largely avoided. Also, by measuring the IQ (and other traits) of the various family members, such studies could easily be turned into inbreeding studies. The heterosis hypothesis would predict that in those families where the children are more heterozygous than their parents, intergenerational increases in IQ should be observed on average. Conversely, when the children are more homozygous than their parents, a mean intergenerational decline in IQ should be observed.

5.3. Approach 3—Within-family studies

Another approach to testing the heterosis hypothesis is to determine the extent to which the secular trends have occurred within families (i.e., within sibships). If the IQ trend has been the result of changes in the environment common to all children, one would expect successive siblings to display gradually increasing IQ from the earlier-born to the later-born siblings, reflecting the presumably improving conditions over time. By contrast, a genetic hypothesis like heterosis would predict no such within-family trends, even when the siblings are born far apart in a population that has undergone rapid secular changes in the intervening years between their births.

Rodgers (1999) has already suggested this approach as a way to better understand the nature of the IQ rise. He also points out that studies of the effect of birth order on IQ appear to indicate that the IQ trend has occurred primarily, perhaps solely, between rather than within sibships, which is consistent with heterosis. Although cross-sectional studies show an apparent IQ advantage to earlier-born siblings, longitudinal studies, which are the only methodologically sound ones, show no birth order effect (Rodgers, Cleveland, van den Oord, & Rowe, 2000). It is important to emphasize that looking at birth order alone is not the ideal test of the heterosis hypothesis. Instead, within-family studies should also look at the age difference between siblings, as well as the rate of secular change in the intervening years between the siblings' respective cohorts. Environmental hypotheses for the trend would predict that the advantage to later-born siblings should be larger when the age difference between the siblings is greater and/or when the rate of secular IQ gains in the population is rapid.

It is worth noting that, in addition to explaining the lack of any birth order effect on longitudinal studies, a heterosis hypothesis also provides a possible explanation for the apparent birth order effect on cross-sectional studies for the following reason. Consider a group of same-age children. Those in the group who are the firstborn in their family can have only younger siblings and will therefore tend to have generally young parents. By contrast, those in the group who are, say, the fourth born will likely have older parents because they must have three older siblings. In a population experiencing the effects of heterosis, younger couples will have children who are generally more outbred (and therefore higher in IQ) than those of older couples, which would create an apparent IQ advantage for earlier-born children but only on cross-sectional and not longitudinal studies.

6. Conclusion

IQ and a number of other highly heritable traits, from height and growth rate to autism and myopia, have shown large parallel secular trends in populations across the industrialized world. At present, no compelling environmental explanation exists for even one of these trends. In the cases of several trends, decades of research have brought science no closer to an explanation than when the trend was first found to be occurring. Because the populations exhibiting these trends have undergone dramatic demographic changes, it is possible that the trends are not environmental in origin but are instead due to the genetic phenomenon heterosis. A heterosis hypothesis has several distinct advantages, the greatest of which is that it is testable, and this paper specifies three approaches to testing that could be conducted with existing data or data that are currently being collected for other purposes.

In scientific investigation, incorrect assumptions that go unquestioned are often a greater impediment to progress than frankly admitted ignorance. Given our current lack of knowledge as to the potential role of heterosis in the secular rise in IQ, continued perpetuation of the false assumption that the trend must be entirely environmental in origin should cease until the heretofore neglected factor heterosis has been given a much closer look.

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