

A Role for the *X* Chromosome?

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A Role for the *X* Chromosome in Sex Differences in Variability in General Intelligence?

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ABSTRACT—There is substantial evidence that males are more variable than females in general intelligence. In recent years, researchers have presented this as a reason that, although there is little, if any, mean sex difference in general intelligence, males tend to be overrepresented at both ends of its overall distribution. Part of the explanation could be the presence of genes on the *X* chromosome related both to syndromal disorders involving mental retardation and to population variation in general intelligence occurring normally. Genes on the *X* chromosome appear overrepresented among genes with known involvement in mental retardation, which is consistent with a model we developed of the population distribution of general intelligence as a mixture of two normal distributions. Using this model, we explored the expected ratios of males to females at various points in the distribution and estimated the proportion of variance in general intelligence potentially due to genes on the *X* chromosome. These estimates provide clues to the extent to which biologically based sex differences could be manifested in the environment as sex differences in displayed intellectual abilities. We discuss these observations in the context of sex differences in specific cognitive abilities and evolutionary theories of sexual selection.

The possibility that greater male variability in general intelligence could be at least partly explained by genes on the *X* chromosome is a frequent topic of speculation among geneticists. Of course, the hypothesis that there is greater male variability in general intelligence is itself controversial, particularly at the high end of the distribution. The evidence for it is substantial, however (Johnson, Carothers, & Deary, 2008), and certainly strong enough to generate speculation about causal genetic and/or environmental explanations. Moreover, the reasons for considering the possibility of a role for the *X* chromosome reach deep into human evolutionary history and involve gene-environment transactions that have fundamental implications both for how human psychology has been shaped in the past and how it continues to develop today. The purposes of this article are to review the evidence for a role for the *X* chromosome in greater male variability in intelligence, present a model of gene action that uses data about sex differences in variability from two population-representative samples to estimate the proportion of genes for general intelligence likely to be present on the *X* chromosome, and describe how individuals with *X* chromosome anomalies may be able to help us test speculative ideas about the development and manifestation of the specific cognitive abilities involved in general intelligence in the population at large.

The subject of sex differences in any aspect of general intelligence is emotionally and politically charged. Thus, before we begin, it is important that we lay out our definitions of the terms we use. Our usages are not the only ways in which these terms can be and often are used. We intend them only to be brief and clear and to avoid struggles over “correct” terminology. We use the term *general intelligence* to mean the ability to use combinations of preexisting knowledge and abstract reasoning to solve any

of a variety of problems designed to assess the extent to which individuals can benefit from instruction or the amount of instruction that will be necessary to attain a given level of competence. These problems can be posed either verbally or figurally, and we assume that tests comprised of such problems are to varying degrees valid measures of the construct to which we refer, particularly within cultural groups sharing a common place and time. We use the term *sex* to refer to males and females generally, regardless of the sources of any differences between them. Finally, we emphasize that manifestation of genetically influenced traits is contingent on the existence of the environmental circumstances that make trait development possible. In fact, reviewing the ways in which the *X* chromosome could possibly be involved in the manifestation of general intelligence requires explaining many environmental processes that intervene between the genotype and the phenotype more generally. Understanding these processes is thus important in understanding how genes can be involved in psychological traits more generally.

This is not an article about values. Values create the emotionally charged climates pervading discussions of sex differences, creating suspicion of motives for politically incorrect speculations such as those in this article and making it difficult to evaluate their scientific basis objectively. Values are extremely important and appropriately form the basis of many actions and social contracts, but the laws of nature may not be consistent with our values. We can only develop coherent and realistic actions and social policies that will actualize our values if we understand the laws of nature as they exist. With this goal of understanding in mind, we believe that it is valuable to review the speculative basis for the idea that the *X* chromosome is involved in general intelligence. A primary reason for this is that, through testing these ideas, whatever the outcome, we can learn

much about the genetic and environmental processes involved in the development of the important (Gottfredson, 1997) human trait of general intelligence in both sexes.

WHY THE X CHROMOSOME?

Before focusing on general intelligence in a speculative way, it makes sense to review briefly the reasons that genes on the *X* chromosome can be expected to create greater variability in males in any trait they influence. This material is not speculative. The reasons have nothing to do with general intelligence per se. Males carry one *X* chromosome and one *Y* chromosome, whereas females carry two *X* chromosomes. The *X* chromosome is of medium size and contains 3.4% of the total amount of genetic material in the haploid human genome (McKusick, 1966; Skuse, 2005). In contrast, the *Y* chromosome is very small and carries little beyond the genetic instructions for maleness. This alone will tend to produce greater variability in males than in females. This is because males carrying alleles that are expressed in proportion to their presence in the genome will either present the trait or they will not. Female heterozygotes who carry one copy of each allele, however, will present some intermediate level of the trait, reducing their population variability. Males more often express traits from recessive alleles that are expressed only if unopposed. This is because males will express the trait at the level of the allele frequency in the population, whereas females will express it at the square of this frequency. For example, if the population gene frequency is 10%, the trait incidence in males will be 10%, but the incidence in females will be 1% (0.1×0.1). This also means less variability in females than in males. For dominant alleles that are expressed regardless of the other allele, males will express the trait at the level of the allele frequency in the population, but females will express it at a higher level. The maximum

variance for both sexes will occur when the allele frequency is .5, and even then the male variance of .25, $.5 \times (1.0 - .5)$, will be greater than the female variance of .188, $.75 \times (1.0 - .75)$.

The actual situation involving gene frequency for multivariate traits like general intelligence may be complicated by factors about which we currently have little clear understanding, such as epistasis, or interactive effects among genes, and gene imprinting, or selective transmission according to parent of origin (Wilkinson, Davies, & Isles, 2007). But we know for certain that it is made more complicated by the fact that most of the genetic material in females on one of the *X* chromosomes in each cell is inactivated to compensate for the greater dosage of *X*-linked gene products that females would otherwise receive. The inactivation takes place early in gestation when the embryo consists of about 100 cells, so that most of the genes on the inactivated chromosome are not expressed. The inactivated chromosome is randomly selected, so far as we know. This results, on average, in the expression of one of the *X* chromosomes in half the cells in the body and the expression of the other *X* chromosome in the rest of the cells in the body, with the patterns of expression following the developmental lines of the cells of origin.

Inactivation makes no difference to the presence or absence of expression for loci where the two alleles are the same (though it limits dosage effects to 1 in both sexes). It only affects heterozygotes, or those with an allele of one kind and another allele of another kind at a particular locus. In heterozygous females, the effects of any activated deleterious allele from one *X* chromosome in some cells of the body are likely to be offset toward the population mean by the effects of the corresponding allele on the other *X* in

other cells of the body. Males experience no such buffering effects: any deleterious alleles on their single *X* chromosome are expressed in all cells throughout the body. Thus females express the full range of genetically influenced trait variability only when they carry the most deleterious alleles on both their *X* chromosomes. Males express this range as long as their single *X* chromosome includes these deleterious alleles, and this occurs more frequently. Across a population, this combination of different levels of trait frequency and *X* inactivation tends to produce overall greater variability in males than in females at both ends of the trait distribution.

Red–green color blindness can serve as a simple example. If the recessive deviant allele that produces it has a frequency in the population of about 5%, then 5% of males will show the trait, but only 5% of 5% or 0.25% of females (those who carry the deviant allele on both their *X* chromosomes) will show the trait. For a binomially distributed trait like this, variance among males will be $.05 \times .95 = .0475$, whereas variance among females will be $.0025 \times (.9975) = .00249$, which is much smaller than male variance. At a more complex level, something analogous will happen with a polygenic continuous trait such as general intelligence that is probably influenced by many genes located on the autosomes and the *X* chromosome in addition to environmental factors. There can be exceptions to these effects of *X* chromosome alleles, as when the presence of one deviant and one nondeviant allele in females has a greater effect than either two deviant alleles or two nondeviant alleles (overdominance).

We now turn our discussion to the more speculative applications of this material to general intelligence, beginning with better established applications related to mental retardation (MR) and working from there to more speculative applications.

HOW MIGHT THE X CHROMOSOME BE INVOLVED IN GENERAL INTELLIGENCE?

The general theory that major genes for intellectual function might be located on the *X* chromosome was first proposed by Lehrke (1972), though it was mentioned as early as 1945 by Roberts. Lehrke's proposal was actually quite specific, and it is worthwhile to review it in greater detail. It consisted of four hypotheses: (a) that there are major gene loci related to human intellectual function on the *X* chromosome; (b) that mutations in the alleles at these loci can lead to poor intellectual functioning, including MR, that is transmitted within families in an *X*-linked manner; (c) that at least one of these gene loci relates particularly to verbal functioning; and (d) that the genetic deficits involve primarily the central nervous system itself rather than some metabolic error resulting in damage to brain cells. As evidence for these hypotheses, Lehrke pointed to an excess of males diagnosed as mentally retarded, a greater variability of general intelligence in males than in females, and a study he conducted of 10 families in which nonspecific MR was segregating in an *X*-linked manner. The general theory was regarded with such skepticism that publication of Lehrke's paper was accompanied by two invited commentaries (Anastasi, 1972; Nance & Engel, 1972). Both were highly critical, but neither provided any real data that could be used to refute Lehrke's propositions. Since then, though the involvement of *X*-linked genes in increased general intelligence has remained controversial (Morton, 1992; Turner, 1996; Turner & Partington, 1991), it has become well established that *X*-linked genes disproportionately affect males and that there many kinds of *X*-linked MR and impaired cognitive functions are transmitted in the

standard *X*-linked manner (Gecz & Mulley, 2000). Thus, Lehrke's first (at least with respect to decreased general intelligence) and second hypotheses have been confirmed.

MR is defined as an IQ less than 70 accompanied by limitations in adaptive functioning (Raymond, 2006). As most IQ tests are structured, the IQ criterion translates to 2 *SDs* below the mean of 100. Prevalence rates for MR are commonly given as 2%–3% of the population, which is consistent with the normal distribution of IQ presumed in structuring the tests. Observations of actual prevalence rates for mild retardation, however, vary widely from 0.5% to 8.0% (Roeleveld, Zeilheis, & Gabreels, 1997; Simonoff et al., 2006), and those for more severe retardation vary even more. The reasons for this variation are not clear, but rates tend to be lower in groups of higher socioeconomic status and higher in population-based samples than in samples collected from educational programs or for test-norming purposes.

MR has many known causes. It is often a marker of a more complex syndrome (e.g., Down's syndrome, fragile *X* syndrome), metabolic disorder (e.g., phenylketonuria), neuromuscular disorder (e.g., Duchenne muscular dystrophy), or even psychopathology (e.g., autism). Not all who suffer from these disorders meet the IQ criterion for MR, however, even when intellectual function is impaired relative to other family members. These syndromal forms of MR are usually distinguished from what is termed *nonspecific MR* (NMR) or cases in which limited postnatal development of intellectual function is the only condition of note. The lines of distinction between these two categories of MR are blurring, however, as we learn more about the etiologies of NMR (Chelly, Khelifaoui, Francis, Cherif, & Bienvenu, 2006). Cases of syndromal MR are generally omitted from norming samples for IQ tests, so some of the large variation in reported rates of MR may

reflect differences in the extents to which syndromal cases are included in samples. In general, syndromal causes account for about 50% of cases of moderate to severe MR and a much lower proportion of cases of mild MR (Chelly et al., 2006).

MR (especially NMR) arises due to both genetic and environmental influences, and many of the environmental causes, such as hypoxia and exposures to toxins including drugs of abuse, have been identified. That said, to date, roughly 300 genes associated with MR have been identified (see Inlow & Restifo, 2004, for a recent review). This is generally considered to represent an underestimate of the actual number involved (Chelly et al., 2006). Perhaps 75% of these involve syndromal MR (Gecz & Mulley, 2000). More than 60 of the 300 genes identified, or some 20%, are located on the *X* chromosome (Ropers & Hamel, 2005). About 3.4% of all genes are located on the *X* chromosome (Skuse, 2005). If the 20% figure fairly represents genes involved in general intelligence, the human *X* chromosome contains a vastly disproportionately large (6:1?) concentration of them. It is, however, relatively easy to identify genes on the *X* chromosome because males carry only one copy of the *X* chromosome, whereas females carry two, and the overrepresentation we see today probably reflects this to some unknown degree. On the other hand, though some of the identified *X*-chromosome genes clearly involve syndromal MR (fragile *X* syndrome is one well-known example), the majority of them apparently do not. We are just beginning to discover autosomal sources of NMR (arising from genes located on chromosomes other than the *X*; Gecz & Mulley, 2000; Laumonnier, Cuthbert, & Grant, 2007). Still, the combination of observations to date suggests that the genetic influences on NMR could be heavily *X*-linked.

There is another reason that a disproportionately large number of genes involved in general intelligence may be located on the *X* chromosome. The recent publication of the genomic sequence of the human *X* chromosome has provided new information about the genes it contains and their organization (Ross et al., 2005). It contains about 1,100 annotated genes, of which at least 800 code for proteins. In comparison with other chromosomes that have been similarly sequenced, the *X* chromosome contains relatively few and relatively short genes. They tend to be expressed in testis, brain, skeletal muscle, ovary, and placenta. Laumonier et al. (2007) report that over 500 of the 800 genes on the *X* coding for proteins are expressed in the brain. Though more than 900 potentially functionally variant single-nucleotide polymorphisms have been identified on the *X* (Ross et al., 2005), this is a much smaller proportion of all genetic loci than on the autosomes. It suggests that an unusually high proportion of the genetic loci on the *X* chromosome make no contribution to individual differences of any kind. If these nonvariant loci are involved in fundamental brain organization, this may help to explain how the functional variants that have occurred could be involved in NMR, as alterations of fundamental processes may be particularly likely to have important disruptive effects.

Since the variability hypothesis was first proposed, one source of controversy surrounding it has been the appropriateness of any inference that whatever genes may be involved in MR are also involved in higher level general intelligence. It is clear that the heritability of general intelligence in the general population results from many genes of small effect (Bouchard & McGue, 1981, Butcher, Davis, Craig, & Plomin, 2008; Deary, Spinath, & Bates, 2006), yet we also know that rare mutations (alleles?) in certain genes can have large effects in disrupting the general intelligence of individuals. Of course,

whether these mutations result in diagnosable cases of MR may depend on the genetic background in which they present. For example, if the presence of a particular mutation (or allele) were to result in a 40-point reduction in IQ, an individual with genetic and environmental background otherwise suggesting an IQ of 160 might retain very high cognitive function, even in the presence of the deleterious mutation. This extends well beyond our current understanding of the effects of any of the alleles known to be involved in MR, but it serves to illustrate how these genes could also be associated with normal-range general intelligence. The fact that to date we only have well-established evidence for alleles that disrupt general intelligence may be at least partly because of the social burden created by MR, as there is no a priori reason that there could not be alleles of comparable effect in increasing general intelligence.

The established alleles involved in NMR that are located on the *X* chromosome tend to show greater expression in males because they have only the single *X* chromosome. The *X*-linked alleles that are involved in NMR could be sources of greater male variability in general intelligence in the lower ranges of the distribution but presumably not in the higher ranges, because they act to disrupt general intelligence. Any *X*-linked alleles that act to increase general intelligence would likely be of smaller effect, if only because we have not yet uncovered them. Zechner et al. (2001) have made the case for the existence of such alleles, but to date none have been identified.

There is another, more fundamental reason that individual genes involved in increasing general intelligence may be of much smaller effect than those involved in disrupting it. We have gained some understanding not only of the genes but also of some of the neuronal endophenotypes (Gottesman & Gould, 2003) common in families with

concentrated numbers of members with NMR. One example of this is the postsynaptic proteome (PSP), or the collection of proteins in the postsynaptic terminal that, broadly speaking, are involved in receiving the neural transmissions that produce cognitive ability (Migaud et al., 1998). The PSP contains some 1,180 proteins in several distinct structural and functional complexes (Laumonnier et al., 2007). The largest of these is the postsynaptic density (PSD), which is located directly below the postsynaptic membrane of a neuron and is comprised of more than 1,100 proteins involved in a broad range of cell biological functions such as signaling, adhesion, reception, and transport, as well as RNA metabolism, transcription, and translation. Another is the collection of glutamate receptor complexes, known alternately as NRC or MASC, which are comprised of about 180 proteins, some of which overlap with those in the PSD. The NRC/MASC is the best studied of these large postsynaptic complexes and, with the PSD, is considered a model for understanding the overall PSP. It appears to be a signaling complex involved with the basic process of neural plasticity.

Though the genes involved in these complexes do not appear to be located on the *X* chromosome disproportionately, high proportions of the genes on the *X* chromosome that code for PSD and NRC/MASC proteins (49% and 85%, respectively) have been found to be mutated in psychiatric disorders (Laumonnier et al., 2007). These mutations involve a broad range of the functional groups of proteins, but particularly signaling proteins. The fact that NMR in general, and *X*-linked MR in particular, is associated with many of these same proteins suggests that the overall function of the PSD and NRC/MASC (and other) complexes might be seriously impaired in phenotypically similar ways by mutations causing disruptions in very different proteins within the

complex. An analogy might be that a car's ability to run can be blocked by failure of any of a number of parts, including transmission, engine, electrical system, or simply lack of gasoline. Laumonnier et al. (2007) present evidence that this analogy is apt. It is much more difficult to imagine how one particular protein could similarly enhance the overall function of these complexes.

In summary, despite the skepticism that greeted Lehrke's (1972) proposition that there were major genes on the *X* chromosome associated directly with central nervous system function that in turn affect intellectual function, a substantial body of evidence now supports it. This evidence, however, is limited to the involvement of the *X* chromosome in disrupting general intelligence. This has two implications. The first is that the overall population distribution of general intelligence should not be normal. Instead, it should be negatively skewed, with greater frequency of low levels of general intelligence. The second is that this evidence does not address either the question of the existence of greater male variability of general intelligence at the high end of the distribution or the question of *X* chromosome involvement in it if it does exist. Data from the Scottish Mental Surveys of 1932 (SMS32; Scottish Council for Research in Education, 1933) and 1947 (SMS47; Scottish Council for Research in Education, 1949) are helpful here. They provide evidence that the distribution of general intelligence is in fact not normal and that there is greater variability in general intelligence in males at the high end of the distribution as well (Johnson et al., 2008). As these data are probably the most population representative that have ever been gathered, the evidence they provide is important. We used these data to estimate the proportion of genes influencing general intelligence that might be located on the *X* chromosome.

**USING SEX DIFFERENCES IN VARIABILITY IN GENERAL INTELLIGENCE
TO ESTIMATE THE PROPORTION OF GENES INFLUENCING GENERAL
INTELLIGENCE THAT MIGHT BE LOCATED ON THE X CHROMOSOME**

As with any model, the model we used to make this estimate required that we make assumptions that may or may not be met. First, we had to assume that the multiple alleles involved in general intelligence act independently and of each other and thus additively (Hill, Goddard, & Visscher, 2008). The most common sources of violation of this assumption involve alleles that are expressed only in the presence of certain other alleles and alleles that are located so close to each other along a chromosome that they are generally carried either together or not at all (linkage disequilibrium) during meiosis and recombination. Second, we assumed that the effects on variance from all other sources besides alleles on the *X* chromosome are the same in both sexes. That is, we assumed that autosomal alleles involved in general intelligence are expressed to the same degree in males and females and that environmental influences on general intelligence are the same in males and females. There is evidence for sexually dimorphic gene expression in tissue throughout the body and brain in mice (Yang et al., 2006), suggesting that this assumption may not hold. And the evidence that socialization processes involving general intelligence may not be the same in males and females is monumental.

Third, we assumed that the gene loci involved in general intelligence each have two alleles, with frequencies p and $q = 1 - p$, respectively. This simplifies the mathematics of the model considerably, but many gene loci have more than two alleles. The presence of additional alleles beyond two does not bias the results from the simplified model. Rather, information about the extent to which alleles involved might

have more than two alleles would refine the precision of our estimates. Fourth, we assumed that all of the pairs of alleles involved are in Hardy–Weinberg equilibrium. That is, we assumed that there is no population stratification or assortative mating involving these alleles. This is a big assumption, as assortative mating for general intelligence is usually observed to be substantial (Vandenberg, 1972). Its violation acts to increase variance in both males and females, but the relative extent to which that is true in the sexes depends on the extent to which any of the alleles involved are dominant over the other alleles at their loci. When one allele is completely dominant over another, its effect is expressed even if only a single copy is present, and the effect of the other allele is expressed only if two copies are present. Dominance does not have to be complete. The model contains a parameter for degree of dominance so the effects can be estimated. When there is no dominance, assortative mating increases variance to the same degree in males and females.

Fifth, we assumed that *X* chromosomes are randomly inactivated. This is the usual general pattern in placental mammals, and it means that two copies of either allele in females have the same effects as single copies of these alleles in males. There is evidence that some of the genes on the *X* chromosome may not be completely inactivated (Pennisi, 2005), but there is no good way to incorporate this into our model, as failure of inactivation appears to apply only to about 25% of the genes on the *X* chromosome, is incomplete, and varies from woman to woman. Finally, we assumed that there were no exceptions to the general pattern of no heterozygotic advantage in females. It is not necessary to follow all of the algebra we present in order to grasp its point.

Given these assumptions and, without loss of generality, effects a and $-a$ of the two alleles and ad of the combination of the two alleles in females (where d is the degree of dominance), we can estimate the variances in males and females. The variance will be $4pqa^2$ for males, and $2pq[1 - (1 - 2q)d + (1 - 2pq)d^2]a^2$ for females (Lyon, 1962). When d is 0, the male–female variance ratio is 2. This applies to multiple loci as well, as long as they are independent, as the variances are simply summed. It also applies to loci that have more than two alleles, as the allele effects remain additive. When d is not 0, then the variances for males and females are not simply related, and they depend on q (the frequency of the more recessive allele) as well. For single loci, male variance will be greater than female variance as long as q is in the range .29 to .71. When q falls outside this range, then d must be restricted to the range $-.62$ to $.62$ if we are to be sure that male variance remains greater than female variance. As long as d is in the range 0 to 1 and $q < .5$ or d is in the range -1 to 0 and $q > .5$, the ratio of male to female variance falls in the range 1.33 to 2.67. Of course the actual situation for gene loci on the X chromosome is likely to be some mixture of loci involving alleles with varying degrees of dominance and differing frequencies—some offsetting and others accentuating each others' effects. The overall variance ratio range of 1.33 to 2.67 for gene loci actually on the X chromosome, however, seems reasonable for estimating the proportion of gene loci on the X chromosome involved in general intelligence given the overall variance ratio.

We define the overall male and female variances to be V_M and V_F respectively, and the components of variance attributable to gene loci on the X chromosome to be X_M and X_F . In keeping with the assumptions itemized above, we define the variance attributable to all other sources to be V , the same in both sexes. Then $V_M = V + X_M$ and V_F

$= V + X_F$. We define the X chromosome variance ratio X_M/X_F to be R and the overall variance ratio V_M/V_F to be r . Some simple algebra yields the proportions of total variance attributable to gene loci on the X chromosome. For females, the proportion is

$$\frac{r-1}{R-1}.$$

For males, the proportion is

$$\frac{R}{R-1} \times \frac{r-1}{r}.$$

With some assumptions about R , the X chromosome variance, based on the range of 1.33 to 2.67 we estimated above, we can use these formulas to estimate the proportion of the full variance in general intelligence that can be attributed to gene loci on the X chromosome as long as we have an estimate of the r , the overall variance ratio. Pooling the variances from SMS32 and SMS47, this variance ratio is 1.155. Table 1 shows the results for three levels of R . With no dominance, $R = 2$. As the X chromosome includes only 3.4% of the genetic material in the haploid human genome (Skuse, 2005), these estimates seem very high. Recall, however, that over 20% of the genes for MR identified to date are located on the X chromosome (Ropers & Hamel, 2005). This does not mean that these genes would be involved in variability at both ends of the distribution of general intelligence, but it does suggest that the assumptions underlying our model may not be distorting our estimates as much as it might appear at first glance.

We can address this with our conclusion that the overall distribution of general intelligence is a mixture of two approximately normal distributions (Johnson et al., 2008). The distributions of general intelligence representing those without conditions that disrupt general intelligence show a pooled variance ratio for the two Scottish Medical Surveys samples of 1.0595. Table 1 also shows the proportions of this variance in general

intelligence that can be attributed to gene loci on the *X* chromosome—again for three levels of *R*. Though still high, these seem much more reasonable. Clearly, however, the most tenuous assumption in our analysis is that the sources of variance in general intelligence from all genetic and environmental sources besides the *X* chromosome are the same in males and females. Violations of this assumption could produce effects of almost any size. Still, our results are not so extreme that gene loci on the *X* chromosome could not explain the greater male variance in general intelligence. Sex differences in variability in specific cognitive abilities, however, could also explain at least some of the overall sex difference in variability. This is the second, even more speculative, explanation of the sex difference in variability in general intelligence that we wish to discuss, and once again we will show that the *X* chromosome may be involved.

THE IMPACT OF SEX DIFFERENCES IN THE VARIABILITY OF SPECIFIC COGNITIVE ABILITIES ON THE SEX DIFFERENCE IN VARIABILITY OF GENERAL INTELLIGENCE

As noted above, many studies have observed greater male variability in two of the three fundamental areas of specific cognitive abilities: quantitative and visuospatial abilities. Sex differences in variability in the third area, verbal abilities, have been much less clear. Sample characteristics can have subtle effects on the relations between sex differences in mean levels and sex differences in variability, and because sex differences in variability may be different at the low and high ends of the distributions of ability, it is important to discuss findings in this area using samples that are as clearly population representative as possible. We focus this discussion on the results reported by Strand, Deary, and Smith (2006) because their results were both generally consistent with those

that have been reported in other studies and more detailed. Even more important, however, their results were obtained in a sample of 320,000 that was nationally representative of United Kingdom school pupils aged 11–12 years. Moreover, their results have been substantively replicated in a large sample in the United States (Lohman & Lakin, 2008). In addition to being large and nationally representative, the Strand et al. (2006) sample was also reasonably consistent with the Scottish Medical Surveys in age of assessment and geographic origin. It clearly represented a different cohort, however, as the assessments were made between September 2001 and August 2003. To the extent that results indicate a similar overall distribution of ability, we feel confident in the general relevance of our observations from the Scottish Medical Surveys data.

The Strand et al. (2006) sample was assessed using the Cognitive Abilities Test (CAT; Lohman et al., 2001). The test measures verbal, quantitative, and nonverbal reasoning domains, thus roughly paralleling the areas of specific cognitive abilities that have generally been considered most fundamental (e.g., Johnson & Bouchard, 2005; Snow, Corno, & Jackson, 1996; Snow & Lohman, 1989; Vernon, 1964; but the nonverbal tests specifically avoid spatial mental rotation problems). Each domain is assessed by three different tests. Mean scores are calculated for each domain and overall. As in the Scottish Medical Surveys, overall CAT variability was greater in males than in females; the male–female variance ratio was 1.13 and was identical to that found in SMS32. The variance ratio in SMS47 was slightly larger: 1.19. The male–female tail proportion ratios in the top and bottom 5% and 10% of the distributions were similar to those in the Scottish Medical Surveys as well. Table 2 shows this comparison. In both the CAT and SMS47 data, the tail proportion ratios at the low end of the general intelligence

distribution were greater than those found at the high end. In contrast, the tail proportion ratios in SMS32 were actually greater at the high ends of the general intelligence distributions than at the low ends. The data were thus not completely consistent and could of course indicate demographic shifts in the relative male and female distributions over time. There was, however, some basic consistency in the overall pattern of greater male variability at both ends of the distributions. This basic consistency makes relevant the following discussion of sex differences in variability in verbal, quantitative, and nonverbal abilities from the CAT.

In addition to the variance and tail proportion ratios of the overall CAT scores, Table 2 shows the variability and tail proportion ratios for CAT verbal, quantitative, and nonverbal scores. Though the Scottish Medical Surveys contained items involving these three domains as well, the item-level scores are not available, so we could not compute the analogous ratios for them. In all cases, CAT variability was greater in males than in females, but the variance ratio was smallest for verbal ability. In addition, there was a mean difference in this domain with an effect size of .15 favoring females, and the tail proportion ratios indicated stably greater proportions of females at the high end of the distribution. These were more than offset, however, by greater proportions of males at the low end of the distribution.

In contrast, the quantitative and nonverbal abilities showed effectively no sex differences in means, but there were greater proportions of males at both the low and high ends of the distributions. For nonverbal ability, the ratios were greater at the low end than at the high end, but the reverse was true for quantitative ability. At a minimum, this indicates that the pattern of greater male representation in the highest levels of

achievement in math, science, and technology-related professions is present in the contributing underlying abilities by age 11. There is no question that there have been and continue to be social and cultural influences on sex differences in intellectual performance in all areas (Halpern et al., 2007), including the three major ones assessed by the CAT. It is easy to see how these influences would have produced mean differences in ability, but this is not what the data from the Scottish Mental Surveys suggest for those not affected by conditions that disrupt general intelligence (Johnson et al., 2008). It is also not what the Strand et al. (2006) data suggest for the three more specific cognitive abilities. Instead, the data suggest sex differences in variability. It is more difficult to see how social and cultural influences on sex differences would have produced greater variability in males without producing mean differences favoring males.

HOW THE X CHROMOSOME MIGHT BE INVOLVED IN SEX DIFFERENCES IN SPECIFIC COGNITIVE ABILITIES THAT CONTRIBUTE TO GREATER MALE VARIABILITY IN GENERAL INTELLIGENCE

In his third hypothesis, described earlier in this article, Lehrke (1972) proposed that at least one of the gene loci involved in general intelligence on the *X* chromosome relates particularly to verbal functioning. The different patterns of sex differences in means and variability among the three ability areas of the CAT suggest that there may be something to this hypothesis as well. Moreover, in addition to MR, other conditions, including dyslexia and other reading disabilities (Rutter et al., 2004), autism and Asperger's autism (Baron-Cohen, 2002), and attention-deficit hyperactivity (Hermans et al., 2004), involve both cognitive abilities and disabilities and show much higher incidence rates in males. The existence of genetic and epigenetic mechanisms involving

the *X* chromosome has been suggested as a possible explanation for these conditions (Skuse, 2005) as well as for sex differences in cognitive abilities more generally (Zechner et al., 2001). Much of the evidence for these mechanisms comes from individuals with anomalous *X* chromosomal patterns: females with *X*-chromosome monosomy (due either to loss of one parental *X* chromosome or to the *Y* chromosome, known as Turner's syndrome, with an incidence of about 6 in 10,000 live births) and males with an additional *X* chromosome (presenting as *XXY*, known as Klinefelter's syndrome, with an incidence of about 1 in 500 live births).

These conditions are clear abnormalities generally involving the whole *X* chromosome, so many individual genes are involved. Individuals with these conditions present particular patterns of cognitive and physical malformation and disability, and we must use these patterns to infer the existence of genes for more typical development. In the past, these conditions were associated with MR, but this is much less true today as social acceptance of unusual developmental patterns has improved.

Females with Turner's syndrome display physiological and behavioral anomalies for two related reasons. First, not all of the genes on the *X* chromosome are subject to inactivation in the normal *XX* female. There are two regions on the *X* chromosome that have analogous regions on the *Y* chromosome, and these so-called pseudoautosomal regions are inactivated. They recombine with the *Y* chromosome in the same way that autosomes in normal *XX* females do. Females with Turner's syndrome do not experience this and therefore receive only half the usual dosage of the products from the genes in these regions. Second, some of the *X* chromosome genes that normally remain active contribute to the maintenance of ovarian tissue that supports the production of estrogen.

The presence of only one set of these genes in Turner's females leads to insufficient production of estrogen and early degeneration of the ovaries. The result is that those with Turner's syndrome manifest several physical anomalies, including infertility, short stature, high arched palate, neck webbing, broad chest, and cardiac and renal anomalies.

Females with Turner's syndrome also manifest cognitive and emotional deficiencies. They tend to display normal verbal ability, in particular reading ability, but their numerical and visuospatial abilities are generally impaired (Skuse, 2005). They also display subtle executive functional difficulties, such as poor oral fluency due to slower than normal verbal production (Temple, 2002). They usually manifest impairments in social skills and affective discrimination and tend to be socially isolated and to have poor self-concept. Some show difficulties with face and emotion recognition and with interpretation of another's eye gaze and line of sight. Risk of autism is increased at least 200 times (Brown & Greally, 2004). This pattern of social and emotional impairment resembles that seen in cases of bilateral amygdectomy (Adolphs, 2003). The amygdala tends to be enlarged, and many Turner's cases show increases in gray matter volume in the orbitofrontal cortex bilaterally, close to a region thought to be involved in emotional learning (Good et al., 2001). As with many conditions involving chromosomal anomalies, Turner's syndrome shows heterogeneity. About 50% of affected individuals show evidence of mosaicism: some of their somatic cells contain normal *XX* karyotypes, structurally anomalous additional *X* chromosomes, or partial *Y* chromosomes (but not enough of the *Y* chromosome to spur development of male sex characteristics). In these cases, there is generally some proportional amelioration of Turner's syndrome features.

Males with Klinefelter's syndrome also show some physical anomalies such as unusually long limbs, tall stature, infertility, underdeveloped testes, and enlarged breasts. They do so because of additional expression of active genes on the additional *X* chromosome, including genes involved in production of female sex hormones. They tend to display low-normal to normal general intelligence, but this masks a general pattern of preserved nonverbal abilities with impaired verbal abilities and executive functioning (DeLisi et al., 2005), particularly reading and language comprehension and ability to monitor interference. In tests of verbal fluency, they normally produce unusually high rates of overt errors or perseverative responses, suggestive of reduced inhibition of inappropriate responses (Temple, 2002). Many cases show overall reductions of brain volume and reductions of gray matter in the frontal and temporal lobes with accompanying white matter tract abnormalities (DeLisi et al., 2005). Rates of attention-deficit hyperactivity, dyslexia, and schizophrenia tend to be elevated (Vawter, Harvey, & DeLisi, 2007). Klinefelter's cases also display chromosomal heterogeneity, with some cases showing some somatic cells with normal *XY* karyotype and others showing some somatic cells with other anomalous karyotypes such as *XXXY*, *XX*, or *XYY*, but less is known about the impact of this on the manifestation of Klinefelter's syndrome features.

In combination, the features of Turner's and Klinefelter's syndromes suggest the presence of dosage-sensitive (not inactivated) genes in the pseudoautosomal regions of the *X* chromosome involved in enhancing quantitative and visuospatial abilities relative to verbal abilities (Skuse, 2005) and reducing response inhibition and gray matter volume in frontal and temporal brain regions. The idea that there is dissociation between verbal and visuospatial abilities has been documented in normal populations (Johnson &

Bouchard, 2007a, 2007b). A region on the proximal p arm of the *X* chromosome that escapes inactivation has been suggested as one possible location for these genes (Brown & Greally, 2004). It would seem, however, that these genes should have counterparts on the *Y* chromosome. Some counterparts do exist, though they probably represent only about 5% of the genes on the *X* chromosome (Ross, 2005). If the *X*-chromosome genes do not have *Y*-chromosome counterparts, normal *XY* males should display cognitive characteristics similar to those in Turner's syndrome (relatively high verbal ability, deficient quantitative and visuospatial abilities). This is exactly the opposite of the pattern that has been documented in the sex differences literature.

There is evidence for the involvement of another genetic mechanism in Turner's syndrome that may have relevance for the expression of genes for specific cognitive abilities on the *X* chromosome. Some genes are genomically or gametically imprinted (Skuse, 2000). That is, they are differentially marked as being of paternal or maternal origin during spermatogenesis or ovulation, and after fertilization these markings lead to differential gene expression according to the parent of origin. The imprinted allele from one parent is silenced, and development is dependent completely on the function of the allele from the other parent. The imprint is generally erased at some point before the production of gametes, so that it is not transmitted from one generation to the next. Only about 1% of all genes are imprinted (Tycko & Morison, 2002), but they are disproportionately involved in growth, especially placental and brain development, and they have highly pleiotropic effects (over many traits). They are also more vulnerable to dysregulation than are nonimprinted genes. Their expression can be affected by both genetic mechanisms, such as alterations in nucleotide sequence, methylation, and histone

modification (Wilkins, 2005), and environmental circumstances (Dolinoy, Weidman, & Jirtle, 2006). Though maternally and paternally imprinted genes may be involved in sex differences in behaviors, there is no known correspondence between the paternal or maternal source of imprinting and behaviors that are more common in males or females.

Because males invariably inherit their single *X* chromosome only from their mothers, imprinted genes on the *X* chromosome could be involved in sex differences. This could happen in two ways. If only paternally imprinted *X*-chromosome genes are expressed, then expression will normally take place only in *XX* females. If only maternally imprinted *X*-chromosome genes are expressed, then sex differences will occur if the genes are subject to *X* inactivation. In Turner's syndrome, of course, the single *X* chromosome could be either maternally or paternally derived. Skuse et al. (1997) reported that Turner's syndrome cases with paternally derived *X* chromosomes showed significantly less social and emotional impairment than did cases with maternally derived *X* chromosomes. Data from 8 females with partial deletions of the p arm of the *X* chromosome suggest that the imprinted gene(s) involved may escape *X* inactivation and lie on the q arm of the *X* chromosome or on the p arm near the centromere. Skuse et al. (1997) suggested that such imprinted genes could help to explain the greater vulnerability of males to language and social disabilities. Clearly, they could conceivably explain normal-range sex differences in cognitive abilities as well.

In summary then, there are data suggestive of genes enhancing quantitative and visuospatial abilities common to the *X* and *Y* chromosomes and for imprinted genes on the *X* chromosome that are activated only when they are paternally derived. Though chromosomal regions in which these genes may be located have been identified, the

specific loci or alleles involved have not. The data do not indicate clearly whether the alleles involved are polymorphic (whether they differ normally from individual to individual) nor whether they would be considered to disrupt general intelligence or to contribute to variations in ability that would not be considered disruptive. Moreover, their putative observed effects would appear to be more clearly associated with sex differences in means than with sex differences in variability, and thus these genes would presumably not be included among those we estimated effects for in Table 2. The very specific nature of the apparent effects of these genes and the relatively strong overall robustness of general intelligence to their effects in Turner's and Klinefelter's syndromes, however, indicate that there must be many other genes involved in general intelligence.

EVOLUTION AND THE X CHROMOSOME

We have described how genes on the *X* chromosome tend to create greater trait variability in males than in females, but why might the *X* chromosome be a location for such genes, for general intelligence, or for any other trait? At some level, it might seem obvious that the *X* chromosome would be involved in sex differences simply because it is one of the two chromosomes (*X* and *Y*) that determine sex. But for sex differences, it would be much more straightforward to have the genes involved on the *Y* chromosome so that they would then be expressed only in males. When such genes are on the autosomes or the *X* chromosome, something must act to turn them on only in males to result in genetically influenced sex differences. Despite this, the human *Y* chromosome is small and extremely gene-poor, whereas the *X* chromosome is much larger. This alone suggests that sex differences may not have been of primary importance in human evolution. Still, a gene locus on the *X* chromosome is at least 3 times more likely to be involved in sexual

development than a gene locus on an autosome (Hurst, 2001), which suggests that sex differences have played some role. The same conclusion is indicated by the large literature on sexual dimorphism for size in humans (e.g., Lindenfors & Tullberg, 1998).

Natural selection is conservative—it acts upon existing biological mechanisms rather than creating new mechanisms designed for specific purposes (Kirschner & Gerhart, 2005). This means that the resulting biological mechanisms are often extremely clumsy and inefficient, but they work—they confer a reproductive advantage. At the same time, they confer this advantage primarily through regulatory innovation that conserves core physiological organization and metabolic processes while maximizing the potential for genetically influenced variation in observed traits and behaviors (Gerhart & Kirschner, 2007). This suggests that there should be some reproductive advantage if the *X* chromosome is rich in genes for sexual development. What might that advantage be?

As with all evolutionary explanations, we can only speculate, but the advantage may lie with sexually antagonistic alleles or variations of the same gene locus that differ in the extent to which they are advantageous with sex. One classic example of the kind of trait involved is the lion's mane, particularly its color (West & Packer, 2002). The dark, large and glorious mane is advantageous in the male because females find it attractive and the male who displays it gets more mating opportunities that extend longer throughout life. But it would be disadvantageous in the female, who would have to support it metabolically, endure the higher body surface temperatures it creates, and carry it around without gaining any mating advantage. Even if a recessive allele involved in a sexually antagonistic trait is initially rare and its cost to females is greater than its benefit to males, it will tend to increase in frequency if it is on the *X* chromosome (Hurst, 2001).

This is because the benefit to *XY* males is straightforward, whereas the cost to females is masked by the recessive character of the allele. In contrast, a similar allele on an autosome will only tend to increase in frequency if its benefits to males are greater than its costs to females. As the *X* chromosome allele increases in frequency, there will be more females who carry two copies of it and thus incur its costs. Natural selection will then tend to favor any mechanism, such as imprinting, that silences expression in females but not in males. In other words, unlike genes on the *Y* chromosome, genes on the *X* chromosome can be carried by females as well as by males, minimizing the sexual dimorphism they create. This would especially be true if the costs of carrying the genes are not too extreme or if the mating advantage does not always lie only with males. At the same time, in comparison to genes on the autosomes, genes on the *X* chromosome can respond more rapidly to selection pressures.

Comparison of gene expression between humans and chimpanzees, who share about 99% of their DNA with humans, is used to estimate the rates of evolutionary change over time (and therefore divergence) in the two species. This can be tied to specific chromosomes and particular regions of gene expression such as the brain. Khaitovich et al. (2005) found substantial differences in *X*-linked gene expression in testes between humans and chimpanzees but low diversity in expression within species. Moreover, the relative magnitude of change in gene expression is greater in humans than in chimpanzees and greater in primates than in rodents (Dorus et al., 2004), particularly in the brain. This suggests that genes on the *X* chromosome can evolve rapidly and affect sexual selection if there is an evolutionary advantage to males. We have noted above that a large proportion of the genes encoding for proteins on the *X* chromosome are expressed

in the brain. In addition, among genes expressed in the brain, a large proportion are *X*-linked and show very sex-biased expression patterns (Skuse, 2006). Together, the rapid evolution and concentration of *X*-linked genes in the brain suggest that specialization of these genes could be responsible for much of human cortical complexity and brain size.

But why would these genes have conferred a reproductive advantage to males but not females—the very definition of sexual antagonism? Baumeister (2007) has suggested one explanation. Noting that, among our ancestors, only about 40% of males reproduced but 80% of females reproduced, he suggested that females have been selected for ability to be social in intimate relationships primarily with close kin and for ability to maintain stable environments for their offspring, whereas males have been selected for the ability to stand out from the crowd in some dramatic way that will make them desirable mates for women (Geary, 1998; Miller, 2000). Probably the best way to stand out from the crowd is to be willing to take risks, and the best way to succeed when taking risks is to do it intelligently, particularly using certain kinds of general intelligence. The relatively few males who succeeded in taking risks had many sex partners and many offspring, and the relatively many who failed had few, if any. At the same time, the best way to succeed in maintaining a stable social and physical environment for offspring is also to do it intelligently, though most females did reproduce. Hence enrichment of the *X* chromosome rather than the *Y* for genes involved in general intelligence makes sense. It also makes sense that some of these genes, in particular the ones that conferred some reproductive advantage to females, would escape *X* inactivation, making it possible for females to receive twice as much of their gene products as males.

If this is even partly correct, we suggest that it could also have led to coevolution. This is the name given to the process by which biological and cultural factors transact to transmit culturally influenced behaviors and attitudes from one generation to the next through natural selection pressures (Cavalli-Sforza & Feldman, 1981; Durham, 1979). It is a form of genetic correlation. More than that, however, it includes explicit recognition that individuals seek out and evoke environmental experiences that are compatible with genetically influenced traits. This in turn creates adaptive niches within broader groups of individuals (Johnson, 2007). The existence of coevolution has been extensively documented in both human and nonhuman species (Laland, Kumm, Horn, & Feldman, 1995). A common human example is the greater adult lactose tolerance that exists today in certain cultural groups that have long-standing traditions of domestication of cattle and dairy and cheesemaking activities. There is evidence that the cultural selection processes involved in the development of these activities sufficiently altered the environments of the groups involved to invoke genetic selection processes for greater lactose tolerance (Aoki, 1986). This illustrates the point that culture involves the intergenerational transmission of knowledge in a manner that serves as an extra-genetic inheritance system (Cavalli-Sforza & Feldman, 1981). Such a system can act to accelerate genetic change through the evolutionary processes of natural and sexual selection by unleashing the expression of underlying genetic variation through tenuous and circumstantial linkages between regulatory signals and gene responses (Gerhart & Kirschner, 2007).

Much of the knowledge involved in culture is social (language, standards of conduct, values, technology), and there is evidence that the ability to make use of this cultural knowledge is evolutionarily adaptive (Betzig, Borgerhoff, Mulder, & Turke,

1988; Chagnons & Irons, 1979; Geary, 2005). The involvement of evolutionary adaptation also suggests genetic involvement in the transmission of cultural processes. Of course, the ability to accumulate and make use of cultural knowledge, as with any form of knowledge, is fundamental to the notion of general intelligence, with its well-established genetic influences (Bouchard & McGue, 1981; Deary et al., 2006). Moreover, the evidence that males and females go through different cultural socialization processes and accumulate different forms of cultural knowledge is massive. In conjunction with the potential for environmental stimulation of differences in genetic expression patterns (Kirschner & Gerhart, 2005), this suggests that small sex differences in genetic hardware can underlie large differences in observed behavioral outcomes. It also suggests a large potential for very rapid genetic evolutionary change in this area.

CONCLUSIONS AND FUTURE DIRECTIONS

In this article, we reviewed the reasons that many geneticists speculate that the *X* chromosome may be involved in general intelligence and the often-observed differences in variability between males and females. We presented evidence that many genetic conditions that disrupt general intelligence involve genes on the *X* chromosome, which helps to explain the greater incidence of these conditions among males. We also, however, presented evidence that there are genes on the *X* chromosome involved in special abilities that may affect general intelligence more generally (high and low) as well as sex differences in the specific ways that general intelligence is manifested. We showed that two very comprehensive data sets on sex differences in variability in general intelligence indicate that genes on the *X* chromosome are overrepresented among all the genes likely involved in general intelligence, but we also presented substantial evidence

that such overrepresentation may exist. Finally, we pointed to epigenetic and genetic regulatory mechanisms and genetic and environmental coevolutionary processes that might be involved in the evolution of both sex differences in patterns of abilities and sex differences in variability of general intelligence, and we noted that there is evidence that these kinds of mechanisms and processes are subject to rapid evolutionary change in response to environmental circumstances. This means that these mechanisms and processes are very sensitive to the environment.

Despite the emotional hostility with which Lehrke's (1972) hypotheses about the involvement of the *X* chromosome in general intelligence have been received, there is much still to be learned from their objective exploration. Individuals with *X* chromosome anomalies such as Turner's and Klinefelter's syndromes provide rich sources of information about the ways that genes direct the development of general intelligence in the contexts of environmental experiences and surrounding genetic background, as do individuals with *X*-linked MR, whether syndromal or not. At the same time, understanding the functions of endophenotypes (Gottesman & Gould, 2003), such as the postsynaptic density and the roles of environmental influences on genetic expression patterns and epigenetic processes (of which genetic imprinting is only one example), is likely to prove fruitful.

To date, conventional genetic linkage and association (including genome-wide association) studies have been singularly unsuccessful in identifying the genes associated with general intelligence (e.g., Butcher et al., 2008). Many candidate genes have been identified, but replication across samples has been poor. None of the candidates proposed

to date is on the *X* chromosome. We suggest that the *X* chromosome be targeted for chromosome-wide (as opposed to genome-wide) association studies.

Though many genetic and environmental conditions that can disrupt general intelligence have been identified, it is also plain that general intelligence is very robust to major disruption. Major chromosomal anomalies such as those involved in Turner's, Klinefelter's, fragile *X*, Down's, William's, and other syndromes have clearly remarkable features involving general intelligence, but the ranges of intelligent function among individuals with these conditions, the overlap of features among these disparate kinds of syndromes, and the degree to which individuals with these conditions retain intelligent function is at least as remarkable. Moreover, there is no evidence to date for a sex difference in the overall proportion of variance in general intelligence that can be attributed to genetic influences. This is consistent with the notion that general intelligence is a very general systemic trait and suggests that understanding the transactions between genes and developmental experiences is crucial to understanding its manifestation.

The more we learn about how genes exert their effects, the clearer it becomes that those effects depend on overall genetic background and environmental context (Carlborg & Haley, 2004). In this article, we have focused on the reasons for speculations that the *X* chromosome may be involved in sex differences in variability in general intelligence not to discount the importance of environmental influences, but to illuminate some of the reasons that biological explanations for those differences persist and, in particular, to increase understanding of the complexity of genetic mechanisms. *X* chromosome involvement in sex differences in general intelligence would not minimize the very important roles of motivation, experience with subject matter, and occupational interest

in achievement in science-related fields (Lubinski & Benbow, 1992, 2006, 2007). Nor would it minimize the importance of sex differences in self-confidence (e.g., Deaux, 1976; Heatherington et al., 1993) or the personal and professional trade-offs required to maintain high-level careers (Halpern et al., 2007). And it would not minimize the effects of overt and covert sex discrimination (e.g., Bowen, Swim, & Jacobs, 2000; Davison & Burke, 2000; Swim, Borgida, Muruyama, & Myers, 1989) or the very early establishment of sexual identity and at least rudimentary understanding of its social manifestations in cognitive development (Gottfredson, 1981). Sex is clearly both a biological and social construct. We will only understand sex differences in general intelligence (or the lack thereof) when we understand how all of our genes, including those that distinguish the sexes, are involved in its development. Until then, psychological scientists of all disciplines can best further progress by maintaining open minds to all possibilities in designing studies and interpreting their results, by learning as much as possible about the rapid changes in our understanding of the genetics of developmental biology, and by encouraging genetic as well as environmental explorations of human behavior and abilities.

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