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Genes and Human Psychological Traits

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This chapter discusses the role of quantitative genetic models in modern biology, Such models are impartial with regard to estimating genetic and environmental influences and are flexible thereby allowing for the estimation of multiple influences on a character simultaneously. Application of these models across a very large number of quantitative characteristics of an equally large number of species leads to the conclusion that almost all quantitative characters are heritable (the first law of quantitative genetics). I illustrate this truism for the major domains of normal human individual differences — mental ability, personality, psychological interests, and social attitudes. I show that in comparison with main effects in social psychology, ecology and evolution, as well as psychological assessment and treatment, known quantitative genetic influence on human psychological trait qualifies as a large effect size. I refute the argument that "there are no genes for behavior" using "clockwork" genes as an example. I also illustrate the fact that finding genes for a quantitative character can be very difficult using the example of corn oil. I conclude by pointing out that molecular genetics will not replace quantitative genetics, but rather the two levels of analysis will fit together seamlessly.

1. The role of contemporary quantitative genetic models in modern biology

Contemporary quantitative behavior genetic models are the same ones that provided the basis for the agricultural revolution that has taken place over the last hundred years, recognizing, of course, that the models have been elaborated as new knowledge developed. Some have argued that models that work for experimental agricultural genetics are simply inapplicable to natural populations and to behavior. The evidence based on studies of the evolution of animal behavior simply does not support the argument (Boake et al., 2002). Indeed, the findings of moderate heritabilities reported from recent quantitative genetic studies of "avian personalities", are eerily similar to those found for "human personalities" (Drent, van Oers, & Noordwijk, 2003; van Oers, 2003; van Oers, Drent, de Goede, & van Noordwijk, 2004).

In addition, evolutionary quantitative genetics is a thriving field (Roff, 1997). If one is interested in empirically driven theoretical complexity I recommend Lynch and Walsh's (1998) nearly thousand page tome entitled "*Genetics and analysis of quantitative traits*". This volume is only an introduction to the theory. Modern application of this theoretical frame of reference to artificial and natural selection will be treated in a volume in progress entitled "*Evolution and selection of quantitative traits*".

1.1 Some advantages of quantitative genetic models

1.1.1 Quantitative genetic models are impartial

Quantitative genetic models are impartial and have no predilection to favor either an environmental or genetic explanation. This evenhandedness is demonstrated nicely by the near zero heritability of the specific religion to which one belongs as opposed to the very

significant heritability of the trait Religiousness (Bouchard et al., 2004) and by the low heritability of political party affiliation as opposed to one's level of Conservatism (Alford, Funk, & Hibbing, 2005). Specific kinds of behavior genetic designs face practical problems that necessitate the violation of assumptions which may introduce biases. For example, adoption designs under-sample poor quality environments and thus may under estimate particular environmental effects (Stoolmiller, 1998, 1999). But, this difficulty can be and has been dealt with (Johnson, McGue, & Iacono, submitted). The magnitude of such biases can also be evaluated by using other designs that do not require the same underlying assumptions (Loehlin & Horn, 2000).

Quantitative genetic models force the investigator to state the hypotheses being tested in an explicit fashion making them refutable. The assumptions are also explicit and different models allow for different tests of the assumptions. Explicitness in research designs is a strong plus in a field characterized by what has been called "incurable vagueness" (Feigel, 1962) and a practice my colleague Marvin Dunnette has argued is designed to insure a "higher likelihood of a pet theory's long life" (Dunnette, 1966).

1.1.2 Quantitative genetic models using many different kinships allow for the examination of multiple genetic and environmental influences simultaneously.

Twin studies are the primary tool of human behavior geneticists. They are a powerful tool (Boomsma, Busjahn, & Peltonen, 2002; Bouchard & Propping, 1993; Martin, Boomsma, & Machin, 1997) and certainly more informative than simple family and epidemiological studies. They are not, however, sufficient to answer many questions and should be supplemented by additional kinships whenever possible (Coventry & Keller, 2005; Keller & Coventry, 2005). Surprisingly in some domains there are numerous twin

studies and not enough family studies. Avenevoli and Merikangas (2003) provide a lucid discussion of this issue with regard to smoking and the evidence in favor of environmental risk factors for smoking. I provide an example of the estimation of multiple genetic and environmental influences simultaneously using a model that includes eighty kinships in the section on social attitudes.

1.1..3 Quantitative genetic models are flexible

Virtually any hypothesis that can be stated clearly can be formulated in the language of quantitative genetics. Many criticisms of quantitative genetic models are really directed at quantitative models of all sorts, more specifically Analysis of Variance (ANOVA) models (Sesardic, 2005). If these criticisms were taken seriously they would prelude most quantitative research in the social sciences. It is difficult to think of an environmental or genetic effect that a quantitative genetic model could not describe (Crusio, 1990; Posthuma et al., 2003). Consider the common criticism that twin studies overestimate genetic influence on traits because MZ twins include both dichorionic and monochorionic twins. Monochorionic twins are allegedly more similar because of environmental biological factors at work during the prenatal period. This is a criticism of the experimental design, not of quantitative genetic methods. The answer to the criticism is a better study, not abandonment of the method. The design to test for this effect has been implemented for IQ, with the finding that, on IQ, chorion effects are trivial and may even be nonexistent (Jacobs et al., 2001).

2. The "First Law of Quantitative Genetics"

Turkheimer (2004) has argued that "Variation in all behavior, including everything from schizophrenia to marital status has a genetic component..... These findings are no

longer in dispute" (p. 161), and enshrined this fact as the "First law of behavior genetics" (see also, Turkheimer & Gottesman, 1992). Many knowledgeable researchers agree with him. Rutter (2002), for example, has argued that "any dispassionate reading of the evidence leads to the inescapable conclusion that genetic factors play a substantial role in the origins of individual differences with respect to all psychological traits, both normal and abnormal."(p. 2). These claims may appear excessive, but they are not. The findings in behavior genetics are simply a special case of a much broader finding that should be called the "First law of quantitative genetics". As Lynch and Walsh (1998) have put it, "If one's sole interest in performing a quantitative-genetic analysis is to demonstrate that the character of interest is heritable, there is probably little point in expending the effort. The outcome is virtually certain. Almost every character in almost every species that has been studied intensively exhibits nonzero heritability" (p. 174).

2.1 An aside on heritability?

Many, but certainly not all behavior genetic studies compute a heritability statistic to describe a trait or the relationship between traits. Heritability (h^2) is a relatively straightforward descriptive statistic that indexes the degree of population variation in a trait that is inferred from the particular research design (i.e., a study of twins, adoptees, a breeding design) to be due to genetic differences. The concept of heritability was largely developed by agricultural geneticists concerned with the response to selection, or realized heritability (Bell, 1977; Lush, 1945). In modern quantitative genetics heritability is defined theoretically and derived from genetic theory (Lynch & Walsh, 1998). The complement of heritability ($1-h^2$) indexes variation contributed by the environment (plus error of measurement) to population variation in the trait. A common criticism of work in

human behavior genetics is that estimation of heritability is uninformative. A widely cited reason that it is considered uninformative is that it is a population statistic and it supposedly could vary from one population or environment to another. Somehow on the basis of this claim we are left to infer that heritability is not worth calculating. Of course we won't know how variable this statistic is if we don't calculate it. More importantly, this same argument applies to any descriptive statistic, the mean, standard deviation, etc., yet one virtually never hears the argument that these statistics are uninformative. The fact that a descriptive statistic varies as a function of some other variable may well be an important empirical finding. For example, the mean raw scores on mental ability tests used to assess IQ have risen dramatically in modern industrialized populations, a phenomenon called the "Flynn effect". This phenomenon has generated a plethora of research and the cause or causes of the "Flynn effect" remain to be explicated (Dickens & Flynn, 2002; Loehlin, 2002; Neisser, 1998; Rowe & Rodgers, 2002). I have already referred to another example involving heritability. The heritability of IQ increases with age and this phenomenon is clearly related to the interesting developmental neuroscience finding that the brain continues to undergo change well into early adulthood (Giedd et al., 1999; Kuhn, 2006; Shaw et al., 2006). This phenomenon needs a name to help make it as well known as the Flynn Effect. I suggest the “Wilson effect” in honor of Ronald Wilson who first described it in detail (R. S. Wilson, 1978, 1983a).

Once a trait has been shown to be heritable a cascade of epidemiological questions follow, What kind of gene actions is involved?, How many loci?, Is there sex linkage or sex limitation?, Are there joint genetic and environmental influences (interactions, correlations)?, Do different genes come into play during the development of the trait?,

etc. (c.f., Bouchard & McGue, 2003, Table 1). Given the plethora of evidence in support of the first law of quantitative genetics these questions should encompass a large part of the research program of students of individual differences.

In my view criticisms of the heritability statistic are largely rhetorical, and more often than not, simply reflect a hostility to the underlying idea that genetic factors influence human behavior. Philosophers of science have been particularly hard on the concept of heritability. According to Downes in the Stanford Encyclopedia of Philosophy (Downes, 2004), "The consensus among philosophers of biology is that broad heritability measures are uninformative but there are a few dissenting voices (Sesardic, 1993)". Sesardic has expanded his challenge to the philosophical consensus into book form (2005) and I highly recommend it.

3. Human intelligence

3.1 The g factor

The idea of a general intellectual factor or more appropriately, the *g* factor (Jensen, 1998) continues to be controversial among many intellectuals. Among specialists, however, there is much less controversy. It has often been argued that different instruments (IQ tests) give dramatically different results and often measure "different things". Put in theoretical terms, the argument asserts that different tests measure different *g*'s. This assertion is false. A reasonable sample of diverse measure of mental ability provides a good measure of *g* (Johnson, Bouchard, Krueger, McGue, & Gottesman, 2004). It is also very likely that the genes the influence *g* are the same genes that influence the various learning disabilities (Plomin & Kovas, 2005).

Until quite recently evolutionary psychologists were quite hostile to the idea of g, arguing that all mental modules were highly specialized to solve specific problems (Bock & Cardew, 1997). This view is no longer so widely shared (Miller, 2000; Nesse, 2000) but see (Kanazawa, 2004). Similarly, in the animal literature until recently it was assumed that there was no *g*. This view is changing rapidly (Anderson, 2000; Matzel et al., 2003).

The practical validity of *g* has also been well documented. Hunter and Schmidt (2004) have summarized the findings regarding *g* as they relate to perforamce in the world of work. Gottfredson (2004) and Deary and his colleagues (2004) have demonstrated the importance of *g* in the domain of health epidemiology. Kuncel, Hezlett and Ones (2004) have demonstrate the importance of g for academic performance and creativity.

3.2 Genetic and environmental influence on g

It has been traditional to attempt to generate an overall estimate of heritability for intelligence (c.f., Devlin, Daniels, & Roeder, 1997). This practice has begun to change as it has become clear that heritability changes with age. As noted earlier Ronald Wilson, using data from the Louisville Twin Study was the first to unequivocally demonstrate that the heritability of mental development increased with age (R. S. Wilson, 1983b). Latter McGue, et al (1993) carried out a meta-analysis of the world's twin data and confirmed Wilson's findings. They also summarized the world literature on unrelated individual reared together (URT's) and demonstrated a striking age effect. URT's assessed early in life show a correlation of .26, suggesting that 26% of the IQ variance is due to shared environmental factors. URT's assessed latter in life, however, show a correlation of only

.04. It is worth noting here that critics often assert that twin studies overestimate the heritability of IQ and underestimate shared environmental influence. The available URT data (there are only 14 such studies, ten of children and four in adulthood) suggest that may not be the case. Teasdale and Owen (1984) report the highest heritability for IQ in the published literature (.96, see Bouchard, 1998, Table 3) using five kin-ships not including twins. Plomin and his colleagues (1997) have also confirmed the effect of age on the heritability of IQ with parent-offspring data for children 1 to 16 years of age. Finally, Boomsma and her colleagues (2002) have replicated the McGue, et al (1993) findings with a series of twin studies in the Netherlands. It appears that the heritability of IQ asymptotes somewhat after 20 years of age (See figures in, Bouchard & McGue, 2003). These findings are entirely consistent with the recent Magnetic Resonance Imaging work on brain size and IQ and genetic influence on brain size. A recent meta-analysis of the brain size x IQ correlation (McDaniel, 2005) shows that the correlation is about .33, but it is higher for adults than for children.

The correlations between IQ and size of various regions of the brain tend to be similar to those obtained with total brain volume (Colom, Jung, & Haier, Available online 2 March 2006; Haier, Jung, Yeo, Head, & Alkire, 2004; MacLullich et al., 2002; Thompson et al., 2001) and the correlation is genetic in origin (Posthuma et al., 2002; Toga & Thompson, 2005).

On the basis of all the literature (Bouchard, 1998; Bouchard & McGue, 2003; Devlin et al., 1997; Johnson et al., under review) I estimate that the heritability of IQ in normal adults is to be in the range .65 to .80.

3.3 Genetic influence on IQ may be moderated by environmental circumstances

A few recent studies suggest that the heritability of IQ may vary with environmental circumstances (Guo & Stearns, 2002; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Both studies used young twins and unfortunately confound race and environmental circumstances. The Turkheimer et al. study used 7-year old twins from the National Collaborative Perinatal Project who had completed the Wechsler Intelligence Scale for Children. The authors used rather sophisticated analytic methods, but for illustrative purpose they divided their sample (a mixed sample of both Black twins and white twins) into high and low socioeconomic status (SES) groups and used the standard twin method to compute the heritability for each group. In the low group (most likely heavily weighted with Black twins) the heritability was .10. In the high SES group the heritability was .72. They did not report heritability by race. Guo and Stearns (2002) made use of a sample of participants in the National Longitudinal Study of Adolescent Health who were in grades 7 to 12 and had completed the Peabody Picture Vocabulary Test. They did not provide an overall analysis of the influence of SES but rather report the influence of various components. In the model that includes three factors simultaneously and therefore allows a direct comparison of the influence of each component (Model 4, Table 8) we find the following results.

1. For three levels of family income (<16k, >16k but <100k. >100k) the heritabilities were .526, 572 and 586.

2. For three levels of Mother's education (less than high school, high school graduate, greater than high school) the heritabilities were .557, .527 and .480. These results are NOT in the direction predicted by the authors.

3. For biological father absent vs. biological father present the heritabilities were .509 vs. .565.

It seems likely a standard composite measure of SES (typically father occupation, fathers education and family income) would yield a very modest moderation of heritability. These findings do not replicate the much stronger effects reported by Turkheimer et al. (2003). It is not well known but in large scale studies (samples of thousands) father's education is correlated as well (sometimes higher) with child's IQ as the mother's education (Table 6. Bouchard & Segal, 1985). This is of course due, in part, to the genetic correlation between level of parental education and IQ. Consequently information on the influence of father's education and/or a composite of father and mother education would have been of interest. Guo and Stearns report a heritability of .576 for Blacks and .72 for whites. It should be noted that the sample sizes for some of the comparisons in this study are quite modest.

There are additional mental abilities over and above the general factor (Carroll, 2003; Johnson & Bouchard, 2005) and we provide an illustrative study demonstrating genetic influence on them latter in this chapter.

4. Personality

4.1 Theories of personality

Unlike the domain of mental abilities there is no general factor of personality, rather there are a number of competing models, in particular three factor vs. five factor models (Markon, Krueger, & Watson, 2004). Bouchard and Loehlin (2001) provide a brief overview of the major theories and a summary of the genetic findings. We prefer the

Tellegen three factor model of personality and provide a large scale illustrative study of genetic influence on Tellegen's Multidimensional Personality Questionnaire (MPQ) and will use it to illustrate the evidence for genetic influence on personality. As with the domain of intelligence recent animal work has demonstrated the pervasiveness of personality traits across the animal kingdom. Work has been done with birds (Sol, Lefebvre, & Rodriguez-Teijeiro, 2005; van Oers, de Jong, Drent, & van Noordwijk, 2004), squid (Sinn, 2005), fish (Azuma et al., 2005), macaques (Maestripieri, 2003), chimpanzees (J. E. King & Landau, 2003), dogs (Gosling, Kwan, & John, 2003; Svartberg & Forkman, 2002) and horses (Morris, Gale, & Duffy, 2002) Gosling (2001) provides a comprehensive review of what we can learn about personality from animal studies and Gosling (1999; Gosling & Vazire, 2002) provides additional references to phenotypic studies of the Gorilla, Hyena, Cat, Donkey, Pig, Rat, Guppy and Octopus. Some of this work involves genetics and the work with birds has demonstrated the Darwinian adaptiveness of such traits (Dingemanse, 2003; van Oers, Drent, de Goede et al., 2004; van Oers, Drent, de Jong, & Noordwijk, 2004). It is worth noting that one reason the dog genome has recently been sequenced (Ostrander, Giger, & Linblad, 2006) is because of the investigators interest in behavior (Wayne & Ostrander, 2004).

4.2 Genetic and environmental influence on personality

A powerful quantitative analysis of genetic influence on personality traits is provided by Finkel and McGue (1997). Their study utilizes the MPQ and makes use of twelve kinships (N=4,298 pairs). This study also illustrates how nicely the heritability estimated from multiple kinships converges with data from monozygotic twins reared together and monozygotic twins reared apart. The data set comes from the Minnesota Twin Family

Registry and the participants range in age from 27 to 64 (mean=37.8). The findings are shown in Table 1.

Table 1 about here

The results are very simple. All 11 personality traits are significantly heritable, with a mean of .44 (range = .26 to .61). These results are entirely consistent with those reported for a wide variety of ordinary twin studies over many years (Bouchard & Loehlin, 2001). The three higher order factors in this study (not shown) yield a heritability of .49 a value also consistent with previous work. The difference between the scale heritabilities and the factor heritabilities is due to the fact that the factors are more reliable. There are very few sex differences in heritability. The authors also report no significant shared environmental influence. This finding is nicely replicated when their MZT twin correlations are compared with the MZA twin correlations. The MZA data were not included in the study. The MZA correlations directly estimate h^2 (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990), whereas the MZT correlations estimate (h^2 + shared environmental influence) as they are reared in the same family. Since the two correlations hardly differ shared environmental influence must be quite small or nonexistent. Note that under these circumstances the MZT correlations alone provide a reasonable estimate of h^2. These results are also consistent with the larger literature. Not shown here is the fact that almost all scales showed significant non-additive genetic variance.

5. Psychological interests

5.1 The structure of psychological interests

It is not widely known, even among psychologists, that psychological interests are quite distinct from personality traits and constitute a domain of their own (c.f, Waller,

Lykken, & Tellegen, 1995). The most widely used theoretical scheme in this domain is the Holland Hexagon (Holland, 1997) which characterizes six types of people (Conventional, Enterprising, Social, Artistic, Investigative, Realistic) in a two dimensional array (Things vs. People; Data vs. Ideas). I prefer to present the behavior genetic findings at the scale level; conceptually just below these "higher order" factors, as was done in the personality domain.

5.2 Genetic and environmental influence on psychological interests

The illustrative data shown in Table 2 is from Betsworth (1994) the only study in this domain that has made use of multiple kinships (monozygotic and dizygotic twins reared together and apart, Adoptive parents x offspring, Adoptive siblings, Biological parent x offspring, Biological siblings). The instrument used was the Strong Vocational Interest Blank, but because the instrument has been revised a number of times and the samples included in the study were gathered at different times, only a small number of items were common to every sample. Consequently the scales used in this study were short and somewhat less reliable than those used in most studies. Thus the heritabilities reported are some lower than they would be otherwise (c.f., Lykken, Bouchard, McGue, & Tellegen, 1993).

Table 2 about here

The mean heritability based on model fitting all the kinships (N=4,002 pairings) is 0.35. The mean MZA correlation is 0.31 (N=59 pairs). Obviously 59 pairs is a small fraction of 4,002 pairs. Nevertheless, as with the personality data the small sample of MZA twins approximate the heritability of the interest measures rather closely. Unlike the personality data this data suggests a modest amount of shared environmental variance

— about 10%. The difference between the MZA and MZT twins, which estimates the same value is 13%. Again the two estimates are quite close. It is worth noting that the intraclass correlation for the 17 full length, and therefore more reliable scales, for the full sample of 78 MZA twins is.41 (unpublished data from the Minnesota Study of Twins Reared Apart).

This data suggests that the heritability of psychological interests when measured with ordinary instruments is around .40 and shared environmental influence is quite modest. This conclusion is consistent with conclusions derived from other data sets (Lykken et al., 1993; Nichols, 1978; Roberts & Johansson, 1974; Waller et al., 1995).

6. Social attitudes

6.1 The Structure of Social Attitudes

The structure of social attitudes is somewhat ill defined in comparison to that of abilities, personality and interests. Three linked traits, Authoritarianism, Conservatism and Religiousness, however, dominate the research domain and appear to be the most important from a psychometric point of view as well (Saucier, 2000). I call this group of traits“ The Traditional Moral Triad” (TMVT) (Koenig & Bouchard, in press). The construct of Authoritarianism was first systematically presented in the famous book, *The Authoritarian Personality*. (Adorno, Frenkel-Brunswick, Levinson, & Sanford, 1950). The most recent body of work on the construct and its relationship to Conservatism and Religiousness has been carried out by Altemeyer (1981; 1988; 1996) whose Right Wing Authoritarianism scale (RWA) currently defines the trait. The psychological meaning of

this cluster has been, and continues to be controversial (Greenberg & Jonas, 2003; Jost, Glaser, Kruglanski, & Sulloway, 2003) as one might expect.

6.2 Genetic and environmental influence on social attitudes.

6.3 Authoritarianism

The very first behavior genetic study of social attitudes was reported by Eaves and Eysenck (1974). They reported heritabilities based on a large twin study (451 pairs of MZ and 257 pairs of DZ twins) for measures "Radicalism" (R) (Radicalism vs. Conservatism) and "Tough-mindedness" (T). (Tough-mindedness vs. tender-mindedness) as measured by Eysenck's Public Opinion Questionnaire (see Eaves, Eysenck, & Martin, 1989, Appendix). The heritability of R was .65 and the heritability of T was .54. Findings in this range for these measures, as well as related measures. have been replicated a number of times (Eaves et al., 1989; Martin et al., 1986). The first behavior genetic study of Authoritarianism was an adoption study by Scarr and Weinberg (1981) who used the original F-Scale (F for fascism) from *The Authoritarian Personality*. They found very significant genetic transmission, but it was largely mediated by the correlation between the F-Scale and IQ. Once IQ was partialled out only a small residual of genetic influence remained. The authors of this study had been unaware of this particular psychometric weakness of the F-Scale (Christie, 1991; Christie & Jahoda, 1954). Altemeyer claims that his RWA scale has overcome this obstacle. We do not agree, as we and others have found a sizeable correlation between RWA and IQ (Bouchard et al., 2004). Using a four group behavior genetic design (MZ and DZ twins reared together and apart) we (McCourt, Bouchard, Lykken, Tellegen, & Keyes, 1999) have found a significant heritability for RWA — between .50 and .64 depending on assumptions. Shared environmental influence

was between .00 and .16 also depending on assumptions. Unlike Scarr and Weinberg when IQ was controlled in the MZA sample the correlation (an estimate of h^2) only dropped from .69 to .59 suggesting that RWA is distinct from IQ and heritable in its own right. We did not have IQ data on the reared together sample. The MPQ Traditionalism scale correlates .76 with RWA. This is the same as the four-year re-test reliability of the RWA scale (Altemeyer, 1988, p. 94) and suggests that the two scales are measuring much the same trait. As Table 1. indicates the h^2 of Traditionalism is about .53. These results based on alternate measures and different samples provide an excellent constructive replication.

6.4 Conservatism

One of the most widely used measures of Conservatism is the Wilson-Patterson Conservatism Scale (W-P) (G. D. Wilson & Patterson, 1968). It is worth noting that the item content of the W-P has changed over the years and studies reporting results with this instrument have not always used the same items (Bouchard et al., 2003). The most comprehensive study of this measure made use of 80 distinct kinships drawn from the Virginia 30,000 . Table 3 presents the results.

Table 3 about here

This table illustrates a number of important points. First, as more kinships are added to a study more model parameters can be estimated (nine in this instance, 18 if sex differences are counted). Second, assortative mating is an important issue in the social attitude domain and should be taken into account in genetic modeling. The social attitude domain has the highest level of positive assortative mating of any domain of individual difference and all the studies cited above report relatively high spousal assortment.

Variance due to assortative mating is a form additive variance. Third, additive variance swamps the non-additive variance. Fourth, there is a significant sex difference in heritability. Fifth, heritability is high in both sexes. Sixth, specific forms of familial environmental influence, including an effect due to being twins, are minor and generally near zero. Using twins reared apart and the same instrument with the same items we have found the same overall heritability (Bouchard et al., 2003).

Truet (1993) has shown using MZ and DZ twins that the heritability of Conservatism is age related and that samples younger than age 20 show near zero heritability. On the other hand an adoption study of 12 to 15 year-olds (Abrahamson, Baker, & Caspi, 2002) detected genetic influence on Conservatism as early as age 12.

6.5 Religiousness

A number of different measures of religiousness have been used in behavior genetic and other studies and there is no consensus regarding which measure is the best. We prefer the modified Intrinsic Religiousness scale (Bouchard, McGue, Lykken, & Tellegen, 1999; Donahue, 1985) originally devised by Allport and Ross (1967). The Minnesota Multiphasic Personality Inventory (MMPI) also contains a scale called the Religious Fundamentalism scale (Wiggins, 1966) which we have found to be quite valid (Bouchard et al., 2004; Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990).

Table 4. presents the findings from a range of studies of religiousness using a number of different research designs. Most of the studies converge on a heritability in the .41 to .50 range with little if any shared environmental influence. The Beer, Arnold and Loehlin (1998) study yields quite different results depending on whether or not twins are or are not included in the model and it is clear that the twin data contributes heavily to estimates

of shared environmental influence. We suspect that some of this is an age effect as the twin sample is extremely heterogeneous in age. As note above Abrahamson (2002) detected genetic influence on Conservatism as early as 12. The did not, however, detect any influence on religiousness in their adolescent sample. The other study that is somewhat inconsistent with all others is that of Kendler, Gardner and Prescott (1997). An examination of the items on the two factor scales suggests something peculiar about the factor analysis. Bouchard et al. (2004) discuss the problem of age effects on social attitudes and measurement problems in more detail as do (Koenig & Bouchard, in press; Koenig, McGue, Krueger, & Bouchard, 2005).

Table 4 about here

7. Gene x environment interactions and other complex processes

Current research places a great deal of emphasis on genotype x environment interactions, particularly in the domain of childhood psychopathology (Caspi et al., 2002; Caspi et al., 2003; Lyman et al., 2000; Moffitt, Caspi, & Rutter, 2005, 2006; Rutter & Silberg, 2002). Some of these interactions are quite fascinating and I strongly encourage investigators to continue in this vein. I also suggest, however, that such interactions be put in context. Most are rather small in terms of variance accounted for, a result consistent with experimental work in lower organisms. Unfortunately, they are not always reported in such a way that one can calculate their magnitude. Some, perhaps many, are in the "trivial" range. Others may never replicate. Eaves (2006) on the basis of simulation studies suggests that this domain is plagued with measurement problems that lead to replicable artifactual interactions and that enthusiasm for this new paradigm

should perhaps be tempered. It is also the case that a good number of interactions show up during childhood. This is of course because most of the research is focused on children. It may well be, however, that the importance of some of these childhood effects will fade with time (Werner, 1989, 1997). That is, they are what one might call "developmental disturbances" with little or no importance with regard to "ultimate trajectories".

The idea that development is "complex and contingent" is a seductive one (Lickliter & Honeycutt, 2003), but it must be kept in mind that "individually unique and unpredictable factors in the web of developmental interactions are a disordering threat to normal development. Selection built anti-entropic mechanisms into organisms to orchestrate *transactions* (emphasis added) with environments so that they have some chance of being organization building and reproduction-enhancing rather than disordering" (Tooby, Cosmides, & Barrett, 2003, p. 858). Many geneticist hold a similar view. "Since armchair examples of significant interactions in the absence of additive effects are pathological and have never been demonstrated in real population, we need not be unduly concerned about interaction effect. The investigator with a different view should publish any worthwhile results he may obtain" (Rao, Morton, & Yee, 1974). Psychologists and others have a propensity to talk about interactions with the environment when they mean little more than "transactions" or "co-action". The taking in of ordinary food is not an interaction with the environment. It is a transaction and obviously necessary for growth, development and the maintenance of life. The use of the term "interaction" maintains an unnecessary facade of complexity. I have treated this issue in detail elsewhere (Bouchard, 1993). The obfuscating use of the rhetoric of

complexity in psychology and biology (c.f., Charlesworth, 1992; Orr, 1999) deserves more attention by someone. Critics often argue that "there may be unknown complex interactions" consequently the main effect of genes cannot be estimated. Of course, "everything in the world can be explained by factors about which we know nothing." (Urbach, 1974. p253). Non-specific complexity arguments are less than helpful. They are seldom more than an assertion of ignorance — "we do not know the mechanisms underlying this outcome" — why not just say so. Arguing complexity is simply another form of the "incurable vagueness" cited by Feigel (1962) and as pointed out earlier designed more to ensure a long life to pet theories (Dunnette, 1966)[1] than to advance knowledge.

Additive genetic variance is that part of the variance in a trait that can be transmitted reliably from parent to off-spring. It is an important source of variance for evolutionary change. Interactions of various sorts cannot be transmitted in this manner although they may also be important from the point of view of evolution (Grigorenko, 2003; Wolf, Brodie, & Wade, 2000). I have no "in principle" objection to the idea of nonadditive

[1] The problem is not unique to psychology. The following quote from a distinguished cell biologist at the 1958 Conference on Biophysics, at Boulder Colorado certainly sounds like some of the current arguments against behavior geneticists. "No two cells give the same properties. Biology is the science of heterogeneous systems…You know there are scientists; and there are people in science who are just working with these oversimplified model systems — DNA chains and in vitro systems — who are not doing science at all. We need their auxiliary work: they build apparatus, they make minor studies, but they are not scientists" (Platt, 1964, p. 348). DNA chains and in vitro systems seem to have served us well in the last 44 years.

variance. My colleagues and I have developed the idea of "emergenesis" (Lykken, McGue, Tellegen, & Bouchard, 1992), a form of interaction at the trait level. It is an idea that has been incorporated by Simonton (1999; 2001) into his theory of the development of talent. The important fact is that genetic variance is ubiquitous and the amount of additive variance is seldom trivial.

8. What is the magnitude of genetic effects relative to other known effects in biology and psychology?

This is an easy question to answer. All we need to do is compare quantitative behavior genetic findings to typical findings in the social sciences.

8.1 Meta-Analyses of Meta-Analyses

8.1.1 Social Psychology

Richard, Bond and Stokes-Zoota (2003) recently published and article entitled "One hundred years of social psychology quantitatively described". It is a meta-analysis of meta-analyses. They report an effect size (Pearson correlation) for "social psychological effects" of .21 (S.D.=.15). Following Hunter and Schmidt (2004), they show that much of the variance in effect size is simply sampling variance. There was much less variation in effect size from one literature to another than one might expect. The smallest effect sizes came from "Social Influence" studies (.13), the largest came from "Group Process" studies (.32). These correlations can be interpreted directly or squared to indicate amount of variance accounted for (coefficients of determination).

8.1.2 Ecology and evolution

Møller and Jennions (2002) carried out a meta-analysis of meta-analyses in ecology and evolution. *Studies of heritability were specifically excluded* (emphasis added). They point out that, "In our analyses, the weighted mean Pearson |r| across all estimates at the meta-analysis level was 0.19, equaling a mean coefficient of determination of 2.5%. There was very little variation from field to field. "Looking at all the different possible analyses, the 95% confidence intervals for mean |r| always fell between 0.14 and 0.22 across a range of fields in biology." (p. 497). They interpreted these effect sizes as small.

8.2 Meta-Analyses

8.2.1 Psychological assessment and treatment

The results of two large meta-analyses of the psychological literature (Psychological Assessment and Treatment) have been reported by Hemphill (2003). He used correlations for his scale of effect sizes. Similar effects were found for assessment and treatment so they were combined. The lower third of the distribution of correlations ranged from -.08 to .17. The middle third of the distribution ranged from .18 to .29 and the upper third of the distribution ranged from .29 to .78. These three ranges might well be called Small (<.20), Medium (.20 to .30) and Large effects (>.30).

Quite independently Lubinski and Humphreys (1997) pointed out that Cohen's (1988) effect sizes (Standard Deviations differences or *ds*) of .20 — small, .50 — medium and .80 — large, correspond to correlations of .10, .24 and .37 respectively. Hemphil's empirical results and Cohen's largely intuitive criteria converge nicely.

It is obvious that genetic influences on behavior fall mostly in the large category and are larger than typical effects reported in a wide variety of domains of psychology and

biology. Note that heritability estimates are estimates of proportions of variance and not correlations; consequently they are not to be squared (Bouchard et al., 1990).

9. Can we find genes for behavior? The solvable and the difficult.

Critics of quantitative behavior genetics often argue that "there are no genes for behavior", "we don't know how to get from genes to behavior", "statistical studies and studies of the influence of specific genetic mechanisms have nothing to do with each other". I call this the "incompleteness argument". The implication of the argument is that unless you know everything you know nothing. It is a bogus argument. Let me give two examples to illustrate why the argument is too facile: one suggests the problem is solvable and the other indicates how difficult the problem may be even though there is not question regarding the importance of genetic influence.

9.1 The problem is solvable: Clockwork genes

The first example involves what must be one of the earliest behavioral traits to have evolved: circadian behavior under the influence of "clockwork" genes. As Hastings and Mywood (2000) put it, "When seeking to explain the evolutions of clocks, it was suggested that their original adaptive role was to ensure that phases of DNA replication sensitive to damage by the high UV levels of the primitive solar day were protected by being synchronized to night " (p. 29-30). An enormous amount is now known about clock genes (Stelling, Gilles, & Doyle, 2004). The literature is huge and growing. I put genes AND clock into the Web of Knowledge and got 1,777 hits. Psychologists have been studying circadian behavior under the term "*entrainment*" for a very long time. The classic work on the genetic system of the circadian clock in mammals and its molecular basis was done by Joe Takahashi (D. P. King & Takahashi, 2000; Lowrey et al., 2000;

Prolo, Takahashi, & Herzog, 2005), who suggested that it was a polygenic trait in mammals although the core mechanism was the same in all organisms.

Surprisingly, there is only one published paper on the heritability of circadian behavior (often called Owl-Lark behavior as measured by a Mornigness-Eveningness questionnaire) in humans and it is from my laboratory (Hur, Bouchard, & Lykken, 1998). We estimated that the heritability was about .54 with no shared environmental influence. These results are in the same range as for most other psychological traits. The MZA correlation alone, which estimates h^2 directly, was .47 (N=55 pairs). We also collected spouse data. The correlation was .25 (N=79 pairs). The degree of similarity between spouses was uncorrelated with years of marriage (r=.01), suggesting that cohabitation does not drive the correlation and that it is probably a function of assortment at time of marriage.

Michael Rosbash (2000, http://www.hhmi.org/lectures/), a key molecular biologist in the domain of circadian genes, argues that he is working in the field of *personality* even though one of his specialties is Drosophila genetics. I agree with him. Our paper on Morningness-Eveningness was published in *Personality and Individual Differences,* which has also published numerous other papers demonstrating the construct and predictive validity of self-reported circadian behavior (c.f., Mecacci & Rocchetti, 1998; Song & Stough, 2000; Tankova, Adan, & Buela-Casal, 1994). Circadian period, a fundamental property of the human clockwork, is correlated with the self-report measure of morningness-eveninglness (Baehr, Revelle, & Eastman, 2000; Duffy, Rimmer, & Czeisler, 2001). Regarding morningness-eveningness which is generally studied as a single dimension in human, Rosbash and his colleagues have recently shown

"…that the timing of morning and evening activity in *Drosophila* derives from two distinct groups of circadian neurons: morning activity from the ventral lateral neurons that express the neuropetide PDF, and evening activity from another group of cells, including the dorsal lateral neurons. Although the two oscillators can function autonomously, cell-specific rescue experiments with circadian clock mutants indicate that they are functionally coupled." (Stoleru, Peng, Agosto, & Rosbash, 2005, p. 682).

Thus, at least in fruit flies, two different mechanisms functionally linked giving us a beautiful reductionist picture of the underlying mechanisms. We also know that a mutation in one of the clockwork genes (Per for Period) in humans causes *"Familial advanced sleep phase syndrome"* (Toh et al., 2001; YingXu et al., 2005).

9.2 The problem may be very difficult: Corn oil

My second example, which demonstrates how difficult it may be to find genes influencing a quantitative trait, does not deal with behavior, but with something that might appear to be simpler — corn oil. Corn is the world's largest crop in metric tons grown (White & Johnson, 2003). It is of enormous economic importance to both developing and highly industrialized countries. An extremely valuable component of corn is its oil. What do we know about corn oil? For one thing we know it is "heritable". The longest running selection experiment in the history of genetics (100 years) involves corn oil. Lines have been selected systematically for high and low oil content.

Hill (2005) has quantified the effect, "The two maize lines differ by about 32 standard deviations. Divergent selection in separate lines for kernel protein concentration gave similar responses, except that the low line reached a plateau at about 5% protein" (p.

683). In their *Genetics* paper (Laurie et al., 2004) the authors of the original work pointed out that their method accounted for ~50% of the genetic variance and that this suggested about 50 QTLs are involved. QTLs are quantitative trait loci. Specifically they state, *"The QTL effect estimates are small and largely additive"* (p. 2141). Hill went on to point out that. "The recent studies of selected maize and broiler lines were extensive, and the QTL effects identified were small. These appear to conform to the infinitesimal model of genes of small effect assumed in much quantitative genetic theory (Lynch & Walsh, 1998) which predicts the observed continuous steady response to artificial selection." (p. 684).

What is the bottom line? According to Hill's perspective article,

> "We have yet to discover how such QTLs work, but several of the SNPs associated with oil concentration were candidate loci...so there are opportunities to find out. It is a challenge for geneticists to identify the genes and the molecular changes in them that cause these many small but important differences in quantitative traits." (p 684).

How interested might these people be in solving this problem? Well the first 7 of the 10 authors of the *Genetics* paper (Laurie et al., 2004) work for the Monsanto Corporation. I would presume that they are intensely interested. The genes for corn oil have not been identified even though powerful breeding experiments have been available for a long time. It appears to us that the argument that no one has discovered genes for behavior as an argument against behavior genetic research is both wrong (such genes are known) and premature (we know it is a difficult problem).

10. Will molecular genetics replace quantitative genetics?

It has repeatedly been argued that quantitative genetics is "old fashioned", "obsolete", "lacks precision", "ignores the real effect of genes", etc.. I hope the previous discussion will dissuade the reader of such thoughts. The methods used in the corn (and poultry studies) cited above are precisely the same ones being used by contemporary quantitative behavior geneticists.

As the study of clockwork genes shows there is no doubt that molecular genetics deals with important issues including the structure, function and expression of genes. Molecular genetics deals with "the parts list". It will help us understand the design or the wiring diagram of cells, organs, the body, and most importantly the brain. Molecular genetics, however, does not deal with a large number of important issues of variation and transmission that are of interest to students of the causes of individual differences. First and foremost students of individual differences are *whole-organism* scientists (along with naturalists and many other biologists) who study the developed *non-Mendelian phenotypes*[2] of individuals in populations and particular ecologies. They are interested in the *transmission* (genetic and non-genetic) of characteristics both vertically (parent to offspring, whether genetically related or not, teacher to child, etc.) and horizontally (sib to sib, peer to peer, etc.). The study of individual differences (Differential Psychology) is a synthesis of quantitative behavior genetics, epidemiology and evolutionary psychology (Bouchard, 2006) not an extension of molecular genetics. In addition,

[2] I prefer the term non-Mendelian phenotypes to complex phenotypes because the latter term carries too much baggage. Whether a trait is complex of not is matter of perspective. Single gene (Mendelian) influences on phenotypes are generally mediated through a long chain of biological processes that are not "simple".

molecular genetics has little to say about an issue of considerable importance to behavior geneticists and differential psychologists, namely our understanding of the multivariate associations among a number of characteristics (phenotypic and latent).

Consider the twin study of the heritability of the Wechsler Adult Intelligence Scale (a widely used measure of intelligence), its subtests, factors and *g* by Dorrette Boomsma's group in Holland (Rijsdijk, Vernon, & Boomsma, 2002) shown in Figure 1.

Figure 1 about here

We can see from this analysis that the majority of the genetic variance in any subtest is due to genetic influence on *g*. Thus the heritability (h^2) of Vocabulary (Voc) is .72, but .52 is due to the general factor (Ag), .17 is due to the broad Verbal Comprehension factor and only .03 is due to the specific test (Asp). This analysis nicely illustrates the fallacy of believing that because a study includes a measure of vocabulary, vocabulary or verbal comprehension is actually the ability under study. The high heritability of verbal ability is due largely to the fact that verbal ability is heavily loaded with *g* and *g* is highly heritable.

Nevertheless, as I have pointed out elsewhere, "At a fundamental level, a scientifically impressive theory must eventually describe the specific molecular mechanisms that explicate how genes transact with the environment to produce behavior" (Bouchard, 2004, p. 151). Such a theory, however, will not be inconsistent with quantitative genetic models. Rather, molecular and quantitative levels of analysis will fit together seamlessly.

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Table 1.
Broad heritablity (h^2) of Multidimensional Personality Questionnaire (MPQ) scale scores and intraclass correlations for monozygotic twins reared apart (MZA) and together (MZT)

MPQ Scale	h^2			MZA	MZT
	Women	Men	Mean	N=74	N=626
Well-Being (Happiness)	0.40	0.40	0.40	0.50	0.40
Social potency (Dominance)	0.54	0.53	0.54	0.54	0.53
Achievement (Hard work)	0.38	0.32	0.35	0.33	0.35
Social closeness (Sociability)	0.47	0.44	0.46	0.44	0.46
Stress reaction (Neuroticism)	0.45	0.43	0.44	0.47	0.44
Alienation (Feels vistimized)	0.39	0.61	0.50	0.35	0.48

Aggression (Reverse Aggreableness)	0.39	0.35	0.37	0.40	0.37
Control (Reverse Impusivity)	0.33	0.47	0.40	0.46	0.40
Harm avoidance (Reverse Sensation-Seeking)	0.45	0.46	0.46	0.45	0.45
Traditonalism (Reverse Autoritarianism)	0.55	0.52	0.54	0.52	0.54
Absoption (Openness to Experience)	0.44	0.26	0.35	0.56	0.33
Mean	0.44	0.44	0.44	0.46	0.46

Note: Heritability data and MZT correlations from Finkel and McGue (1997), MZA correlations from Bouchard and McGue (2003). Common names in parentheses added by the author.

Table 2.
Broad heritablity (h2) and shared environmental variance (e2) of Strong Vocational Interest Blank (SVIB) scale scores and intraclass correlations for monozygotic twins reared apart (MZA) and together (MZT)

SVIB Basic Interest Scale	h^2	e^2	MZA N=59	MZT N=1960
Nature	0.41	0.05	0.28	0.47
Mechanical activities	0.43	0.05	0.33	0.49
Science	0.35	0.12	0.43	0.47
Mathematic	0.50	0.00	0.40	0.51
Medical service	0.39	0.06	0.38	0.45
Music/dramatics	0.37	0.15	0.30	0.51
Art	0.40	0.07	0.17	0.47
Writing	0.38	0.09	0.36	0.47

Teaching	0.31	0.10	0.33	0.41
Social Service	0.34	0.06	0.23	0.41
Athletics	0.39	0.10	0.45	0.49
Public speaking	0.37	0.07	0.36	0.45
Law/politics	0.39	0.12	0.28	0.50
Merchandising	0.29	0.06	0.35	0.34
Sales	0.19	0.11	0.13	0.29
Business management	0.25	0.08	0.35	0.32
Office practices	0.26	0.17	0.12	0.43
Mean	0.35	0.09	0.31	0.44

Note: Heritability data and twin correlations from Betworth, et al, (1994).
N=number of pairings.

Table 3.
Estimates of Sources of Variance (%) for Males and Females for Conservatism Scores based on data from the Virginia 30,000.

Sources of Variance	Males	Females
Genetic		
Additive	35.5	19.8
Assortative Mating	22.2	12.4
Non-additive	6.7	12.5
Total Genetic	64.5	44.7
Environmental		
Maternal	1.5	0.1
Paternal	0	0
Sibling	0	5.2
Twin	0.1	4.2
Residual	40.1	36.6
Total Environmental	41.7	47.2
G by E Covariance	-6.2	8.1

Table 4. Adult Twin Estimates of Genetic and Environmental Influence on Trait Measures of Religiousness.

Scale	Sample	Heritability (genetic influence)	Shared Environmental Influence
Religious Fundamentalism (MMPI)[1]	MZA, DZA, MZT & DZT	.54	.00
Religious Occupational Interests[1]	MZA, DZA, MZT & DZT	.44	.00
Religious Leisure Time Interests[1]	MZA, DZA, MZT & DZT	.57	.00
Religious Activities (SCII)[1]	MZA & DZA	.43	.00
Religious Values[1]	MZA & DZA	.46	.00
Intrinsic Religiousness[2]	MZA & DZA	.43	ne
Extrinsic Religiousness[2]	MZA & DZA	.39	ne
Religious Fundamentalism (MMPI)[3]	Adoption Data	.28	.26
Religious Fundamentalism (MMPI)[3]	Adoption, MZT & DZT	.41	.50
Personal Devotion[4]	Female MZT & DZT	.29	.24

Personal Conservatism[4]	Female MZT & DZT	.00	.45
Religiousness[5]	Male MZT & DZT	.44	.18

1. Bouchard, et al. (2004), 2. Bouchard, et al. (1999), 3. Beer, et al. (1998), 4. Kendler, et al. (1997), 5. Koenig, et al. (2005)