Genetics of Social Behavior

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…I think the usual discussion [of] "vitalism-mechanism" puts the question upside down by asking "if we start with physics and chemistry can we explain the whole of biology?" Whereas the real question should be "if we analyse biological systems shall we come across anything which physics and chemistry cannot eventually accommodate?" This second form of question itself implies of course that physics and chemistry are themselves growing and developing subjects.

…There is a lot of biology which is [sic] at present is as far from basic physics as the gas laws are from the dynamics of the individual gas molecules. …The field of natural selection and evolution is one example and… the morphogenesis of large scale structures such as bones will quite likely turn out in the same category. …New bodies of theory will have to be developed to deal with such phenomena, but this does not imply… that the new theories cannot be fully incorporated into an expanded body of physics. …Looking at a few pieces of wire and plastic from the point of view of ordinary physics, I would not easily come to the conclusion that they could beat one at chess. Suitably assembled and programmed they could do so, and their behavior then is not "non-physical" but is I should say "super (conventional) physics."
--- Conrad Hal Waddington, letter to Francis Crick, 27 December, 1967

Substitute biology and genetics for physics and chemistry and psychology of social behavior for biology in the above. You have to change the examples too, of course. Maybe Mendel's laws of inheritance for the gas laws, and gene expression for the dynamics of individual gas molecules? Maybe the Big Five model of personality for natural selection and evolution, and self-esteem for bones? We'll leave the chess-playing computer example at the end alone. It's perfect as it is. Whether those particular substitutions for Waddington's examples from physics, chemistry, and biology float your boat or not, together the substitutions make Waddington's passage strikingly relevant to the role of genetics in social behavior. That is, because human beings are biological organisms and all biological processes involve gene expression of some kind, there is a level at which nothing about social behavior makes sense except in light of biology, or more specifically, genetics. At present, however, we frankly don't know much about that level.

The problem is that many people, even many working in the life sciences, assume we do know a lot about it. They believe that outcomes in which genes are involved emerge through fixed sets of genetic processes beyond control of the individual and basically independent of the environment. They consider these genetic influences to be the fundamental cause of the outcome, so that presence of the relevant genetic variants automatically assures presence of the outcome, and absence of the relevant genetic variants precludes it. They assume that each gene is linked one-to-one to some trait(s) and that everyone who shares a genetically influenced trait to the same degree carries the same combination of involved genetic variants, expressed in the same ways (so-called isomorphism). All this tends to make the outcome seem natural, inevitable, and pre-determined, so that moral and ethical considerations must accommodate its presence (which, even if genetic determinism were true, is Moore's (1903) natural fallacy; 'ought' does not follow from 'is'). And it tends to make many consider all those who share a particular genetically

influenced characteristic to be generally homogeneous in other ways as well. None of this, however, is the case.

If none of this is the case, why is this interpretation of genetic influences so pervasive? Some, such as Dar-Nimrod & Heine (2011) have argued that this interpretation is a form of psychological essentialism, or the human tendency to group the world into categories defining the causes, immutable features, and innate potential of objects and organisms (Gelman, 2003). This kind of thinking emerges in early childhood, and is thought to be highly useful to young children in learning language and the fundamental concepts of their cultures. Over-used, however, essentialism is thought to be one of the primary sources of bias and stereotype (e.g., Bastian & Haslam, 2006). It is of course possible that essentialism contributes to the deterministic interpretation of genetic influences, but it is not necessary that it do so (and this proposal, ironically, could in itself be an example of over-use of essentialist thinking). An alternative explanation for the pervasiveness of the deterministic interpretation of genetic influences is that many people, again even those working in the life sciences, believe this interpretation to be scientifically accurate.

After all, this interpretation has been standard teaching in biology classes and science textbooks and even journal articles for many years. And it is not completely wrong even today, though its limitations have become glaringly clear. For example, humans carrying an extra chromosome 23, or even just particular parts of an extra chromosome 23, do express a series of quite specific facial features, health and growth problems, and intellectual disabilities known as Down's syndrome, and we do not know of any way to alter this, though we have learned much about how to help these people achieve greater intellectual skills. Those who carry at least some minimum number of DNA base sequence repeats in a particular position on chromosome 4 will,

at least at this stage of our medical knowledge, inevitably develop the serious condition Huntington's disease in middle adulthood. Even timing of the onset of the disease is quite reliably associated with the number of repeating DNA sequences in excess of that minimum. But the vast majority of traits and conditions that show genetic influences, which includes virtually every behavioural tendency that can be reliably measured (Turkheimer, 2000), do not do so in this kind of one-to-one completely penetrant and deterministic fashion. To be more specific, we know of no social behavior that follows this pattern. Social behaviors are genetically influenced, but not 'hard-wired', genetically determined, or innate. One way to reduce the pervasiveness of the assumption that the empirical presence of genetic influences on social behaviors means that their manifestation is determined may simply be to update educational programs to reflect current understanding more accurately. This takes time to work into the social fabric, but progress in doing so could be accelerated through greater care than is often taken in discussing new findings about genetic influences in the media, and even in presentation of such findings by scientists authoring journal articles.

How to Think About Genetic Influences Instead?

If not determinism, what understanding should be conveyed about genetic influences on social behaviors? Among genetically influenced medical conditions, only about 2% can be considered monogenic, or caused by single genetic variants (Jablonka & Lamb, 2006). In the other 98%, many, even perhaps thousands, of genetic variants are involved. Genetic variants are any differences in the DNA of individuals within a species, be they substitutions of base pairs ('letters') of the DNA or small structural differences. The frequencies of the different variants (alleles) in the population can be anywhere from common to extremely rare (even specific to single families or individuals). The same individual allele can be expressed differently in

different environmental circumstances, with the environment of any one allele including the rest of the organism's genetic variants. These conditions emerge through transactions among many genetic variants in some environmental circumstances but not others, and those as well as other genetic variants themselves can influence the kinds of environments individuals seek, end up experiencing, and which subsequently influence later genetic expression. This is the pattern of genetic influence shown by social behaviors. There is not one social behavior that appears to act like the 2% of genetically influenced medical conditions that are considered monogenic. This general situation was long suspected by many developmental geneticists (Johnson, 2012, 2013), but hard evidence began to creep in about 15 years ago as linkage and candidate gene studies failed to replicate, and studies observing gene-environment interactions began to pile up. The advent about 10 years ago of genome-wide association studies that survey quite closely spaced markers located throughout the genome has made the overall situation very clear (Johnson, Penke, & Spinath, 2011): though genetic influences on social behaviors are pervasive in some general sense, it is almost impossible that they determine any social behaviour in any cohesive way.

A dramatic source of evidence for this comes from, of all things, corn oil. One of the longest-running experiments of all time has provided evidence that has upset traditional understanding of genetics (Johnson, 2010; Le Rouzic, Siegel, & Carlborg, 2007). Since 1896, geneticists at the University of Illinois have been studying corn's response to artificial selection for oil contenst (Hill, 2005; Laurie, et al., 2004). Like many social behaviors, corn oil production varies continuously across individual plants, is influenced by many different genetic variants, and individual plants thrive though producing many different levels of oil. In this experiment, geneticists cannot control variation in seasonal temperature and rainfall from year to year, but all

of the corn lines have been planted in the same area, so they have been subject to very similar conditions, particularly in any single generation.

Geneticists had expected that, over time, the genetic variants involved in high oil production would gradually go to fixation in the lines so selected, and the genetic variants involved in low oil production would be eliminated. They expected the opposite for the lines selected for low oil production. This is not, however, what they have observed. After 50 years of breeding, they reversed the selection for some of the lines. That is, they started to select some of the corn plants that had been bred for high oil production instead for low oil production, and vice versa. Figure 1 shows what happened. The orange line of the graph shows the rate at which oil production increased in the the corn selected for high oil production. The line is jagged because of variation from year to year in weather conditions and their consequences, indicating the sensitivity of genetic expression to strictly environmental conditions, but it has a clear overall slope. This slope indicates the heritabity of oil production, or the proportion of total variance in oil production that can be attributed to genetic influences. The green line shows the rate at which oil production decreased after 50 years of selection for high oil production. Its downward slope is about as exactly the same as the upward slope of the orange line as one could ever hope to see. The same is true for the purple and blue lines that show what happened to the corn selected for low oil production, and after 50 years of that, for high oil production.

Selection was again reversed after another 10 years in some of the corn that had been selected for high oil production for 50 years and then low oil production. This produced the red line in the graph. The diversity of genetic variants involved in corn's production of oil has apparently not been diminished by the 50 and 55 years of directional selection. This could only have taken place if some genetic variants contribute to oil production against some genetic

backgrounds and not others, or if there is a steady infusion of new gene mutations that contribute to oil production. But all the evidence to date about genetic mutation rates indicates that they are way too low and unsystematic for this to occur (Le Rouzic, Siegel, & Carlborg, 2007). Moreover, after 100 years of selection, the levels of oil production in the corn lines represented by the green and blue lines are almost identical. Given the very different selection histories in these two lines, it is very likely that few if any of the same genetic variants contributed to these levels at 100 years of selection. Such genetic heterogeneity might be ubiquitous. There is no reason to suspect that the genetics of corn oil are in any way unique, and if anything social behaviors should be more complex traits than corn oil production. This means that is it very likely that two people can display the same levels of genetically influenced social behaviors, but share none of the genetic variants that contribute to individual differences in these variables in humans.

This example of genetic selection in corn for oil production is dramatic for its clarity and consistency, but many recent experiments of much shorter direction point in very similar directions. Their results suggest that the genome is very 'deep' and its expression very flexible in many ways. By 'deep' we mean that the genome contains considerable redundancy: one genetic variant may be actively involved in some trait when it is present, but if it is not, some other genetic variant may serve the same role, perhaps even just as well. This other genetic variant could be 'silent' or inactive if the other variant is there, or its product could simply be 'surplus' to this trait and used in some other trait. By 'flexible' we mean that environmental conditions independent of the genome do matter too: the jags in the lines can be substantial. For example, Figure 1 suggests that swings in environmental conditions within periods of 1-2 years appear capable of creating as much difference in oil production as about 30 years of selection (the sharp

downward then upward spike in the orange line corresponds at its lowest to about overall level at generation 30, and at its highest to about overall level at generation 60)..

Many geneticists are currently interested in understanding genetic involvement in physical and mental illness as well as behavioural traits. In recent years they have conducted technically powerful genome-wide association studies (GWAS) of as many as 1 million common genetic markers with a wide variety of traits and conditions of interest. Though these studies have turned up a few important 'hits', they have not generated anywhere near the expected harvest of understanding about the particular genetic variants involved in anything. Every study has generated a pile of possibilities, but the pile has generally differed quite dramatically from study to study of the same condition. Moreover, even within a single study, the pile of possibilities cannot account for anything close to the existing heritability estimates. This has led to frequent discussion of reasons for the 'missing heritability' (Maher, 2008). Many suggestions have been offered, but consensus remains distant. Our own take is that, if the situation in corn is at all common, which we believe to be likely, this kind of genomic flexibility and density, along with some inflation of heritability estimates due to the presence of gene-environment interaction and correlation as discussed below, could easily account for a substantial amount of the missing heritability. The rare variants GWAS studies do not examine no doubt also contribute.

What Does this Imply for Genetic Influences on Social Behaviors?

Of course we do not practice genetic selection on humans as has been done in this experiment in corn, so our evidence of genetic influences on human social behavior is less direct. We can derive similar evidence from species considerably closer than corn, however. For example, most of the major dog breeds have been established and maintained based at least as much on breeding for behavior as for appearance. This was described specifically in books

dating back to 1576 and 1656 (Plomin, DeFries, McClearn, & McGuffin, 2008), and such breeding for behavior continues today. For example, in England, most dogs have been bred for hunting, and there are 26 breeds of hunting dogs, each specialized to a particular kind of hunting. And it took only about 40 generations of breeding to create a new breed of fox that is tame enough to have become a popular house pet in Russia (Trut, 1999). Many laboratory experiments with rodents have also demonstrated genetic influences on behavior. Most of these experiments involve selection for high and low levels of the behavior of interest, as well as unselected control lines. For example, there are large individual differences among mice when they are placed in a large, brightly lit box that has come to be known as the 'open field'. Some freeze, defecate, and urinate, while others run around actively exploring it. Breeding across 30 generations for high and low levels of activity in the open field generated graphs strikingly similar to Figure 1 (DeFried, Gervais, & Thomas, 1978).

Most of the evidence for the presence of genetic influence on social behavior in humans comes from twin studies. These studies rely on the observation of greater behavioral similarity in mono- than in dizygotic twins. Monozygotic (MZ) twins are effectively genetically identical, as they develop when a single fertilized egg divides early in gestation to go on to produce two individuals rather than the usual one. Dizygotic (DZ) twins are as genetically similar as full singleton siblings who share on average half the genetic variants (i.e., the genetic variance among humans), as they develop when a woman emits more than one egg during a single ovulatory cycle and two of them are independently fertilized. Much has been written about the limitations of twin studies in establishing genetic influences on behaviors, but these limitations, though real, are much more relevant to the magnitudes of estimates of genetic influences than they are to the question of presence versus absence of genetic influences (Johnson, Penke, &

Spinath, 2011; Johnson, Turkheimer, Gottesman, & Bouchard, 2009). The implication of this is that, surprising as it may seem, the most relevant difference between corn and humans with respect to understanding and measuring genetic influences is *not* that we routinely breed corn however we please and develop heebie-jeebies at even the *prospect* of doing *any* breeding at all in humans. Rather, the most important difference is that humans move through and actively and passively, consciously and unconsciously, select among various environmental opportunities throughout their lives (Johnson, 2007; 2012, 2013; Penke, 2010), while corn is stuck with whatever environmental conditions into which its seed happens to fall.

To understand the implications of this, it is helpful to run through the assumptions on which twin studies rely, and thus the limitations on interpretations of their results. This is especially the case because these assumptions and their attendant limitations are often glossed over in twin study reports. The first is that twins are representative of the more general, mostly singleton, population. There are questions about this for some traits, especially in early childhood, as twins are often born a little early and at lower birth weights than singletons, and are more likely to encounter birthing complications (Crosignani & Rubin, 2000). Most effects these leave, however, are generally outgrown by age 6 or so (Christensen, et al., 2006). There has been evidence that twins may average slightly lower IQs than singletons (Voracek & Haubner, 2008), but the most recent and careful studies contradict this (Webbink, Posthuma, Boomsma, de Geus, & Visscher, 2008). There are at least two reasons for this. With improvements in medical care, birth complications that used to leave lasting effects may no longer do so, and the more recent studies have had the advantage of comparing twin and singleton IQs within families, making for more accurate assessment (Webbink, et al., 2008). Past early childhood, there is little evidence for any differences between twins and singletons in personality, which is probably the

area closest to social behavior in which the subject has been addressed. One of the largest studies (Johnson, Krueger, Bouchard, & McGue, 2002) found no differences except for the trait of social closeness, for which mean levels were slightly higher in twins. There is evidence that the twin relationship may be particularly close (for perhaps obvious reasons; Segal, 2000), but little or no evidence that twins respond differently than singletons to social stimuli when they are on their own. This is what would be most relevant to the question of the representativeness of twins in studying social behavior in the larger general population.

Another assumption is that environmental influences act to make MZ and DZ twins similar to the same degree. This assumption has generally been considered valid (e.g., Plomin, De Fries, McClearn, & McGuffin, 2008), but it also has primarily been tested by measuring whether MZ and DZ twins are treated alike to the same degree by parents and others around them, and whether these treatments impact on relevant behavior patterns. The assumption is actually quite a bit broader than this, and the way in which this is true makes it more likely violated. That is, the assumption includes the implication that whatever is 'done' to MZ and DZ twins by the environment will affect them to the same degree. As genetic influences are expressed through environmental contexts (including other genetic background) and DZ twins are less genetically alike than MZ twins, this likely is often not true.

Twin studies also generally rely on the assumption that people do not mate assortatively; that is, they do not end up with mating partners that are systematically more similar or dissimilar to them than chance on the trait in question. This allows the consequent assumption that DZ twins on average share 50% of their genetic variants. This latter assumption appears overall to be very accurate (Visscher, et al., 2006). The accuracy of the assumption of absence of assortative mating for social behaviors, however, is a wide-open question. Most studies have shown little of

it for personality (Plomin, De Fries, McClearn, & McGuffin, 2008), but there is generally considerable assortative mating for intelligence (spousal correlations on the order of .3 to .4; (Plomin, De Fries, McClearn, & McGuffin, 2008), and one of the primary reasons it takes place has implications for the potential for assortative mating for social behaviors. Assortative mating for intelligence takes place at least in part because most societies are rather stratified by educational level and people have tended to meet their mating partners while engaged in their day-to-day activities, and what makes up those day-to-day activities is always genetically as well as environmentally influenced. Educational programs such as universities tend to accept students on the basis of measured intelligence, either directly, through test scores, or indirectly, through performance in lower-level educational programs. Many of these programs also demand enough investment of time and energy that they inevitably extend to include social activities, so that they tend to become quite central to participants' lives and thus primary sources of mating partners. The net result is that there end up being genetic influences on presence in particular kinds of situations, such, for example, as attendance at a university campus party or presence in a bar frequented by auto production line workers. This kind of sorting process likely applies to presence in all situations, though we may not have identified or developed measures for the traits that influence people's participation in them. For example, social attitudes also show substantial assortative mating (spousal correlations on the order of .5-.6; Coventry & Keller, 2005). The sorting processes are less formal and do not generally involve qualification processes or explicit enrolment in any kind of program, as is the case with intelligence, so they are more difficult to track. Some studies have begun to address them, however. It is likely that many other social behaviors show similar though perhaps weaker patterns, but this remains speculation at present.

This relates directly to the final and most important assumption: that genetic and environmental influences act independently of each other. At this point, we know that this assumption is often violated. It has two major components, one of which is much more commonly investigated than the other. The more commonly investigated component is gene-environment interaction (GxE). It is worth taking a moment to consider the definition of gene-environment interaction in some depth. This definition can be considered from two contrasting perspectives: genetically controlled differential sensitivity to environmental circumstances, and environmental control of genetic response (Purcell, 2002). The point using either perspective is that, when GxE takes place, people who differ genetically from one another in some relevant way experience some environmental circumstance differently. But which perspective is more relevant in some particular situation matters greatly. When the first perspective of genetic control is more relevant, it is likely that individuals have some (conscious or unconscious) hand in whether or not they experience the kind of environmental circumstance to which they would be genetically sensitive. If they would be positively genetically sensitive, they are more likely to experience it; if they would be negatively genetically sensitive, they are less likely to experience it. A key here is that the reasons for this difference in probability of experiencing the relevant environmental circumstance are at least partially genetic: the relevant genetic background leads an individual to seek or avoid, as appropriate and to the degree possible, the relevant environmental circumstance. In contrast, when the second perspective of environmental control is more relevant, individuals likely have little control over whether or not they experience the relevant environmental circumstance. This means that two people with the same genetic variants involved in a trait may wind up expressing very different levels of the trait if their relevant environmental experiences have been different.

Most studies of GxE have considered it in isolation from gene-environment correlation (r_{GE}), which can also be defined from two perspectives: genetic control of exposure to the environment, or environmental control of genetic expression (Purcell, 2002). The reason for the detailed focus on the two perspectives encompassed by the definition of GxE should now be clear: it is *through* r_{GE} that GxE will be manifest *whenever* individuals have *some* (conscious or unconscious) control over whether or not they experience the relevant environmental circumstance (Johnson, 2007). Because individuals almost always do have *some* control, conscious or not, over the environments they experience, GxE and r_{GE} will generally be linked to at least some degree. This means that, as we come increasingly to recognize that GxE is common, we must also begin to acknowledge that r_{GE} is too.

Why Does r_{GE} Matter in Studying Social Behavior?

r_{GE} is the elephant in the room, to use the idiomatic expression for a problem or risk or ugly possibility no one wants to bring up, let alone discuss. There may be a too-apt analogy between social scientists' behavior and that of the detectives in Mark Twain's *The Stolen White Elephant* (Twain, 1882), which is likely one major source of the expression: in that story, a white elephant in transit from India to Britain as a gift to the Queen is lost in New Jersey. The local police force springs into action, with detectives ineptly searching far and wide in ridiculous places when in fact the elephant had never left the spot at all. Given Twain's usual snide use of humor, there is little question that the fact that the elephant in the story was white was no accident: 'white elephant' is an idiomatic expression for a possession that is too valuable simply to junk but which entails a burden of upkeep way beyond its worth so its owner cannot get someone else to take it on for a price that would recoup its value. This only makes the analogy with social scientists' behavior that much more apt: if we were to find that r_{GE} is behind any of

the social ills we experience, it would pose a huge social burden we cannot easily unload, one that is considered politically poisonous. But why would r_{GE} be so bad?

This can be made clear through further definitions associated with r_{GE}. These gene-environment correlations are often classified as passive, evocative, or active (Scarr & McCartney, 1983). They are passive when individuals both inherit genetic variants influencing behavior and tend to find themselves, through *no* action of their own (the second perspective in the definition above, environmental control of genetic response), in environmental circumstances that reinforce that behavior. Examples used to illustrate this form of r_{GE} usually focus on childhood, when most live with the biological families into which they are born, and parenting activities: e.g., children of antisocial parents are likely to inherit genetic variants associated with antisocial behavior from those parents and are also more likely to experience at their hands maltreatment that reinforces the children's own propensities toward antisocial behavior. In contrast, r_{GE} is active when people actively (though not necessarily consciously) seek out and experience environments that reinforce genetic inclinations (the first perspective in the definition above, genetically controlled differential sensitivity to the environment); common examples of this are tendencies for bright people of any age to engage in intellectually stimulating activities that build and reinforce already-genetically-influenced intellectual skills (and the less felicitous opposite tendency for less bright people to do things like watching TV instead). Evocative r_{GE} falls in between the two definitional perspectives: something about the genetically-influenced ways an individual behaves tends to evoke certain kinds of responses from the environment that reinforce the genetic influences on that behavior. So, for example, people genetically inclined to be friendly make more friendly overtures to others, which in turn elicits friendly responses from others, so that these people end up with more friends than those who do not make so many

overtures. Similarly, children genetically inclined to act out anger in disruptive ways get punished for this by parents and teachers and other caretakers, often making them yet angrier. Or people expressing genetic variants involved in higher physical attractiveness might be treated better by others, possibly resulting in the development of a greater sense of entitlement and tendency to be demanding in social interactions (Sell et al., 2009).

As the examples highlight, all these forms of r_{GE} involve reinforcement by the environment of expression of pre-existing genetic differences. This means that, over time in the presence of r_{GE}, influences of genetic variation that originally make only very small differences will tend to make increasingly large differences through development and, yes, learning and behavior reinforcement schedules. With time, these differences may become quite large and every bit as firmly stable and hard to change as if they were genetically 'hard-wired'. To the extent this is the case, it will tend to mean that societies become stratified for genetic variants influencing the traits the societies reward and punish.

For most industrialized societies, this means positive stratification for genetic variants influencing traits like intelligence, physical attractiveness, and ability to market one's skills and abilities to others; and negative stratification for traits like antisocial behavior, vulnerability to mental illness, and difficulty controlling impulses. Such a situation would undermine all our aspirations to an open society that makes equal opportunity available to all, so even acknowledging the possibility is about as politically incorrect as one can get. Yet nature does not 'care' about our ideals of equality or standards of political correctness. So if the elephant is in the room, it will 'poo' on us (exert its effects) whether we are willing to acknowledge its presence or not. And it will be a *white* elephant – valuable but 'high maintenance' - in that the effects of r_{GE} will potentially be very powerful but difficult to control.

Complicating the situation, it would currently be almost impossible to refute the all-too-likely possibility of r_{GE} and its attendant population genetic stratification. Doing so would involve demonstrating that there are no individual genetic markers associated reliably with socially positively and negatively valued behavioral traits that differ in frequency in various segments of the population to the appropriate degrees, and this is well beyond current technology. Any prospect of doing this is only made more difficult given our present understanding that behavioral traits are influenced by a mixture of many common genetic variants of individually small effects that accumulate to rather substantial effects and genetic variants with potentially substantial effects that are extremely rare in the population (Gibson, 2012). In addition, any of these genetic variants may be expressed in some circumstances and not in others, to the point where two individuals could have the same level of some trait, say intelligence, but share *none* of the genetic variants that contribute to that trait.

Moreover, the statistical techniques and twin studies that have demonstrated the general presence of genetic influences on behavior cannot refute r_{GE} anywhere near as clearly. One of the best of these is the discordant twin study (also called twin difference design) that explores whether twins, especially MZ twins, discordant for some environmental condition are also discordant for some outcome that is associated with that condition in the more general population. This provides much stronger evidence than possible in any general population sample that the environmental condition is actually causal for the outcome. The problems are that most twin samples are quite short on MZ twin pairs discordant for environmental conditions, so power is generally low (McGue, Osler, & Christensen, 2010), and most such studies to date have found that the association between the environmental condition and the outcome is much smaller in MZ twins than in the general population, providing evidence *for* the presence of $r_{GE.}$ It

is almost as impossibly difficult at present to find evidence clearly supporting the presence of r_{GE} as well. This is because we do not know of any individual genetic variants reliably associated with behavioral traits, and it would take even more statistical power to demonstrate that any such genetic variants differ in frequency across social strata than it would to identify them in the first place. All this only hinders us from acknowledging the elephant that is all too likely in the room.

Another Reason r_{GE} Matters in Studying Social Behavior

But there is another important reason that r_{GE} matters in studying social behavior. It involves the kind of lab experiments dear to the hearts of social psychologists and generally considered the gold standard for causal inference. Such experiments typically assign participants to artificially constructed laboratory situations. This is generally done purportedly randomly, under the assumptions that it does not matter who is placed where because on average everyone would respond the same to any given situation. It is considered ecologically valid under the implicit assumption that situations develop out of aggregated actions of many circumstances over which no one individual has any control. Increasingly, these experiments are being designed with recognition that all do not respond the same, but then a single measured difference in response tends to be the focus of study, examined as a statistical interaction.

If GxE is common, however, the assumption that everyone will respond the same is often violated, and specific interaction effects incorporated in the study design will likely not capture all of it. Moreover, the links between GxE and r_{GE} and the common occurrence of r_{GE} in its own right mean that situations do *not* just develop out of aggregated actions over which no one individual has any control. Instead individuals exert considerable control over whether and how a situation develops at all as well as how it turns out. After all, we can think of life as no more or less than an ongoing series of situations. The control may not be conscious and may be the

outgrowth of long-standing patterns of behavior that have little overt association with entry into any particular situation. An example may help to clarify this.

Wendy and Lars, the two authors of this chapter, were walking back towards downtown Edinburgh and their respective homes one Saturday night around 3 am from a friend's party. The walk took about an hour, which they spent engaged in animated conversation about, what else, some aspect of psychology. Perhaps about halfway there, someone clearly at least slightly drunk approached and tried to pick a confrontation with Lars, striking him on the arm in the process. Lars responded to put him off, ignored the fact that he had been struck, the potential confrontation fizzled, and the man, and Lars and Wendy, continued on their respective ways. It might be easy to see how Lars exerted control over the outcome of this situation, but how did he exert control over finding himself in it?

Well, Lars is a rather large man, both tall in height and large in build. He may not have had any kind of direct control over this development, but size shows large genetically influenced individual differences, and individuals learn to accommodate their behavior to their sizes and the responses in others their sizes create (Sell et al., 2009). In Lars' case, we can speculate that some of his other characteristics have been at least partially accommodations to his size. Whether or not this is true, other, also genetically influenced, characteristics of Lars relevant to his entering this situation include enjoyment of parties with friends, willingness and even eagerness to stay at them 'til late at night and undertake long walks home through all parts of town, never-ending interest in animated discussion, a distinctive style of dress (always all in black with his long hair tied back in a ponytail), self-confidence and assertiveness that shows in how he carries his body, a habit of talking with assurance in quite a loud voice, and, though very fluent, an obvious German accent in his English speech. He would definitely never have encountered this situation

that night if he had gone home much earlier with his wife when she took their son home in their car, and he very well might not have encountered it if he did not have as many habits of speech, dress, and carriage that call the attention of others in particular ways. There is little question as well that, once entered, this situation could have had much different outcomes if Lars was not also willing to let small affronts just pass. Lars did not think consciously about entering this situation in any way, and would not even say that he consciously contemplated response alternatives once in the situation, but both his presence in the situation and his response to it reflected long-term behavior patterns that show large genetically influenced individual differences.

Wendy's presence (or rather really basically absence) in this situation was similarly characteristic of her. She was happy to have Lars' company for the long walk back to town, but would have done it on her own if he had not been there and did proceed alone to her own flat when their routes parted downtown. She was completely ignored by the man during his encounter with Lars, and the fact that this is typical is no doubt part of what has led her to feel, over many years, free to walk around alone at night as a woman: she is small but not short, far from glamorous in appearance or dress but also far from decrepit, carries herself as if she is just on her own business, seems not to attract much attention at all as she goes along, and almost reflexively deflects what little attention she does attract. (Of course no one controls the situations they enter or do not so completely that she should truly feel safe, but that is also where the genetically influenced characteristic of being willing to accept some risk comes in.) Everyone has analogous patterns of behavior and preferences that contribute directly but often not consciously to the situations they enter and do not enter.

Control over entry into situations is never complete, and people have many different, often contradictory goals and motivations. This means that they commonly find themselves, consciously or unconsciously, having entered situations they do not prefer. Some examples of this, such as being rear-ended at a stop light while a passenger in the car, are clear, but presence in many situations involves more complex combinations of choice and lack of choice. An extreme but not uncommon example might be a not-particularly-naturally-nurturant husband who becomes caretaker when his wife develops Alzheimer's disease. As has been well documented, this is a very stressful role for most. Few would choose it, but many accept it at least for some period of time out of love, loyalty, and sense of duty. Entering any non-preferred situation means tolerating the stress generated by the need to behave in ways appropriate to it, which always has individually characteristic repercussions on genetic expression that may influence other aspects of a person's life and even physical and mental health as well (Schmalhausen, 1949). Waddington (1953), quoted in opening this chapter, provided one of the first experimental demonstrations of this, in fruit flies, and we can see its traces in humans (e.g., Hicks, South, DiRago, Iacono, & McGue, 2009; Johnson & Krueger, 2005, a,b; Johnson, Kyvik, Mortensen, Skytthe, Batty, & Deary, 2010). We have little understanding of any specific mechanisms at present, however, and associations involving individual specific genetic variants are likely to measure out as at best wobbly (e.g. Duncan & Keller, 2011; McGuffin, Alsabban, & Uher, 2011; Risch, et al., 2009; Uher & McGuffin, 2007).

Lab experiments make a mess of these 'normal' conditions of social situations. They commonly make use of university students, a segment of the population selected for genetically influenced traits of intelligence, conscientiousness, absence of antisocial behavior, and other traits (Heinrich & Heine, 2010; Sears, 1986). These samples are at best not representative of the

general population for these observed traits, but they are also likely not representative of the general population for whatever *genetic variants* are involved in these traits. Moreover, they are assigned during the experiment to artificial situations they might never experience or choose, consciously or unconsciously, to enter on their own. In the lab they know the circumstances are artificial and have received the blessing of some ethical review panel as being unlikely to confer any lasting real discomfort on them. In addition, they often involve academic credit that may prime them to be motivated to please the experimenters by behaving in whatever way they think the experimenter expects (Klein, et al., 2012). This means that the behaviors these individuals display may be different in ways all too relevant to whatever is under study from any behaviors they would usually display, while at the same time it means that whatever behaviors they are displaying may not be representative samples of those the full population would display. And the lab experiment does not follow them to gather any information about how they deal with whatever stress this odd combination of choice and lack of choice of situation has generated. Your guess of what this means for interpreting results is as good as ours, but the likelihood that it often has relevance should not be ignored.

Implications for Theory and Explanation in Social Psychology

All this has two kinds of implications for theory and explanation in social psychology. The first concerns how to understand and interpret presented evidence that the heritability of some social behavior is some amount like 35% (which is very typical of the levels of heritability that can be expected for all social behaviors; Johnson, Penke, & Spinath, 2011). Such estimates confirm the presence of genetic influences and by now there is no doubt that such influences are ubiquitous (Turkheimer, 2000). They directly imply that explanations of differences between individuals in social behavior cannot ultimately be completely environmental. But they say

nothing about how exactly genetic differences are involved. They refer only to the variance in the trait across the population, not to anything about trait levels in any individual or even within the sample used to generate them. The specific magnitude of any estimate is very likely wrong because it was calculated based on assumptions that probably did not hold. It is not possible to say whether an estimate is high or low, however, without knowing which assumptions were violated and how. Even simple psychometric aspects, like how reliably the behavior was measured, influence the sizes of heritability coefficients. This really does not matter though, because the particular magnitude of any estimate does not tell us much of anything about the trait. This is because heritabilities are ratios of genetically influenced variance to total variance, with environmentally influenced variance and measurement error making up the rest of the total. Heritability can be high simply because relevant environmental variance happens to be low temporarily or in the sample in question, or because violation of the assumptions about the independence of genetic and environmental influences happens to be great, rather than because the trait is inherently genetically determined (Johnson, Penke, & Spinath, 2011). Thus, the primary value of heritability estimates is the evidence they provide that genetic influences transmissible from one generation to the next are important and substantial, but not much more.

Epigenetic phenomena are currently commonly offered as possible ways in which it could appear that genetic influences are transmitted from one generation to the next when in fact it is an environmental influence that is transmitted. Epigenetic phenomena involve differences in gene activation and expression not tied to genotypic variation. For example, mice (*mus muscalis*) commonly show coats of either yellow or agouti (ticked, with different colors on single hairs) fur. Yellow color is not due to variation in the gene that controls coat color, but due to suppression of expression of that gene. The suppression of expression is controlled in turn by a

genetic variant lying upstream along the DNA from the color gene. But it can also be induced by feeding the mice a particular kind of otherwise-nutritious food. Female mice that have yellow coats due to consumption of this food are more likely to have offspring with yellow coats through at least the next two generations (Blewitt, Vickaryous, Paldi, Koseki, & Whitelaw, 2006).

Such epigenetic phenomena are likely involved somehow in GxE and r_{GE}, but at present we know very little about how or to what degree. Epigenetics is sometimes discussed as if it were an alternative to genetic influences, or as a mechanism by which environmental influences could override genetic differences and make environmental influences on behavior transmissible across generations. These claims are grossly overstated. We do have evidence for environmental influences that affect gene regulation and translate to behavioral differences, mostly from model organism studies (Hoffmann & Spengler, 2012), but little is known reliably about similar influences in humans, though it is highly unlikely such influences are absent. What we do know points towards interactions between genetic differences and epigenetic influences, which are often genetically influenced themselves, not to epigenetic influences as alternatives to genetic influences (Murgatroyd & Spengler, 2012). Furthermore, while inheritance of epigenetic differences is possible, it is likely not very stable across generations and not a good explanation for the observed genetic influences on psychological traits and behaviors (Slatkin, 2009).

So what *do* we get from estimates of genetic influences? Well, that brings us to the second kind of implications. These concern how the ubiquitous presence of genetic influences, GxE, and r_{GE} should be taken into consideration in developing theories in social psychology. It means recognizing that the ways in which individuals differ genetically will tend to be expressed most freely when their behavior is least constrained by social norms and conscious goals. This

might mean they may tend to be expressed more clearly in priming and in implicit than explicit attitudes. It also means genetic differences likely distort the representativeness of behaviors in lab experiments. It means their influence on traits like intelligence that contribute to what counts as success or failure in any situation must be taken into consideration in theories about the development and maintenance of self-esteem. Similarly, it means that their influence on temperamental traits that contribute to emotional experience and response must be taken into consideration in understanding the development of early attachment and its influences on later social relationships. In short, their traces pervade all areas of social psychology, but we are only beginning to understand this, never mind just how. As Waddington said so well in the introductory quote, this is going to mean developing new theories, as well as new methods to test them. Of course social psychological phenomena are remote from the level of gene action, but social psychological theories still need to be consistent our understanding of genetic variation and the ways in which their actions transact with those of the environment. The end result could well transform understanding in the field as much as appropriate assembly and programming transforms the capabilities of wire and plastic when we build them into a computer that can be programmed to beat us at chess.

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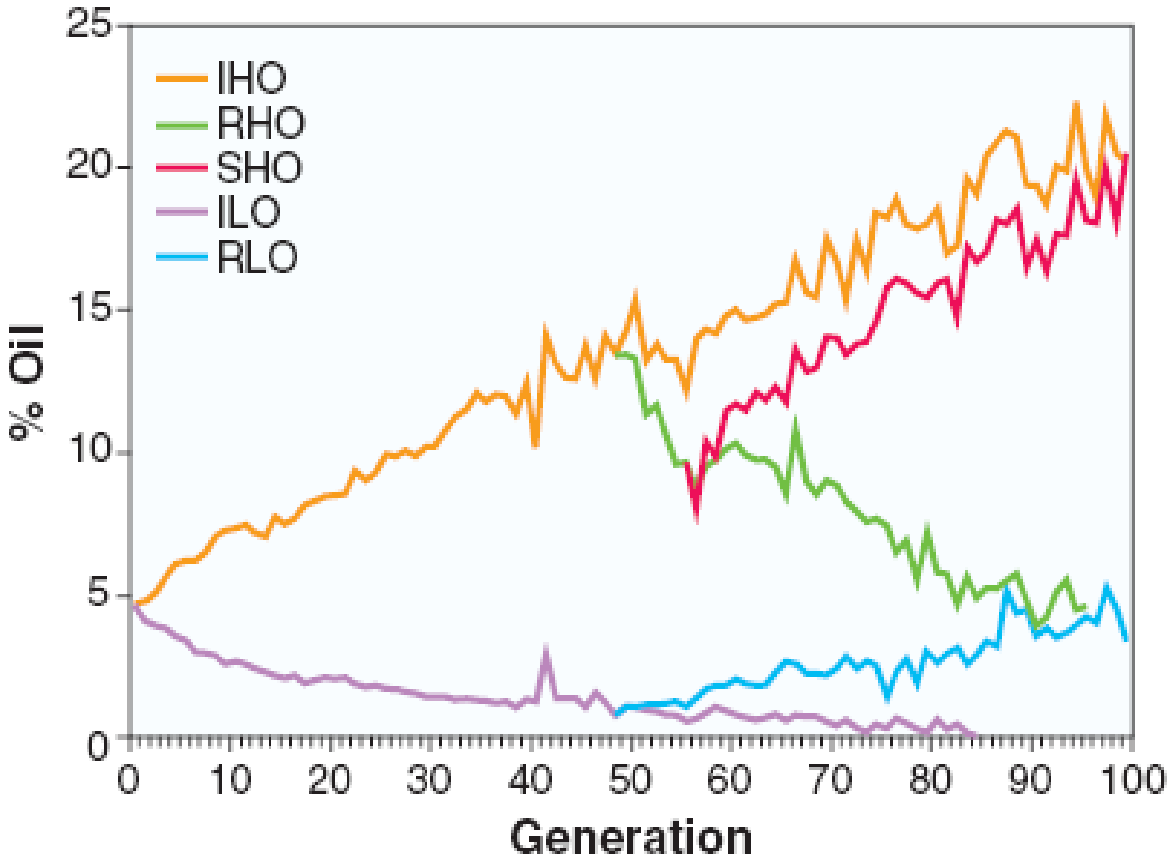

Figure 1, from Hill, W. G. (2005), supplemental material. The graph shows oil content by yearly generation of initially identical seed plantings in a single field in Iowa throughout the 20th century. The top line is oil content by generation in seeds bred consistently for high oil content. The bottom line is analogous, in seeds bred consistently for low oil content. Oil content fell below a level at which the corn could survive around generation 85. The line that breaks off from the top line at generation 50 is oil content in seeds bred consistently for the first 50 years for high oil content. The generation-50 seeds were then bred for low oil content for the next 50 years. The line that breaks off from the bottom line at generation 50 is oil content in seeds that experienced an analogous switch at that time from breeding for low to high oil content. The line that breaks off at generation 60 is oil content in seeds that experienced a second switch in breeding for low to high oil content. The bumps and wiggles in all the lines result from differences primarily in weather conditions from year to year.