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The primary purpose of this book is to provide a broad snapshot of recent findings showing how the environment and genes influence behaviour. At face value, this should be uncontroversial but unfortunately, the history of genetics includes eugenic movements and Lysenkoism. As a result, discussions of how nature and nurture affect behaviour have been dogged by polemic disputes because ideological views about their contributions have tended to cloud what is really an empirical question. This is in some ways exemplified by the book Not in Our Genes (Lewontin et al. 1984), which begins with a political confession from the authors – we are committed socialists – and starts with a chapter on right-wing politics and determinism. For us, the evidence, and not political or any other beliefs, is what counts and any ‘belief’ approach puts the desire for the world to be a certain way ahead of the evidence that it is not so, ultimately committing a version of the naturalistic fallacy – if something is ‘natural’, it is morally correct, which is clearly rubbish (also see Chapter 10). Infanticide, cannibalism, forced copulation (rape), and killing other members of your species (murder) are rife in nature, but it would be difficult to convince anyone of intelligence that these acts are moral because they are natural. Furthermore, ‘politically’ motivated arguments against ‘reductionism’, reducing complex behaviours to single causes, are frequently concocted to protect against a biological determinism that must be fought at all costs. However, as we hope to explain, acknowledging that there are genes underlying behaviour, even genes of large effect, is imperative if that is what the data tell us. After all, it is no use playing music to cows if milk yield is totally determined by genes and unaffected by the environment, and as we outline below, in a polygenic world that includes inevitable environmental effects and all manner of interactions, prediction is tricky and determinism dubious because of the probabilistic and complex nature of the gene–behaviour link. But again, even if single genes were completely responsible for single behaviours, which they cannot be in the strictest sense (see below), let us not fall into a naturalistic fallacy.

Rather than engage in further fruitless arguments about world-views, this book explores exciting new findings about behaviour and where we go from here. Before moving on to these new advances and the interesting questions that arise from them, we wish to make another – a final? – attempt to kill the nature versus nurture polarity.
that has plagued the study of behaviour. This dichotomy is largely, but not totally, dead in academic circles but still haunts many debates outside academia, from views on teaching and punishment to politics and the media more generally. It potentially has grave consequences and is a serious distraction to the much more fruitful and interesting discussion about the determinants and influences of behaviour.

Most behaviours, like any aspect of the phenotype, are not influenced by either nature or nurture but by both and by the statistical interaction between nature and nurture (see reviews in Boake 1994; Sokolowski 2001; Bucan and Abel 2002; van Oers et al. 2005; Hunt and Hosken 2014; Anholt and Mackay 2015) (see also Chapters 4, 6, and 7). To explain, starting with the genetic effects, behaviours (and other characters, for that matter) are typically polygenic (Anholt and Mackay 2004). That is, they have complicated genetic architecture that involves many segregating genes with pleiotropic effects and are characterized by complicated epistatic interactions (Anholt and Mackay 2004). In other words, there are lots of genes, each can affect many characters, and the effects of any one gene frequently depend on the other genes it is associated with. There are exceptions to some of this (see Chapter 5), with, for example, foraging movement in *Drosophila melanogaster* having two distinct behavioural phenotypes that are largely determined by a single gene (reviewed in Sokolowski 2001), and aggression being altered by transposon upregulation of a cytochrome P450 gene (Rostant et al. 2017). However, even these large single-gene effects can be complicated by epistasis (gene–gene interactions) (e.g. Smith et al. 2011; Rostant et al. 2015).

Nonetheless, most behaviours are influenced by many genes, often of small effect, and because of this, we may never uncover all the precise genes that influence a behavioural phenotype. As a result, a statistical approach is needed to describe the average effects of genes on a behaviour and, importantly, to show how genes affect the variation around the mean. The distinction between an average effect and the variation around it is crucial, because for the most part there is not a single gene for phenotype A or B; rather, there are many genes that alter the probability of expressing phenotype A or B. Thus, many interesting traits do not vary discretely but are continuous (Falconer 1981; Roff 1997; Lynch and Walsh 1998), and genes influence the likelihood that an individual will express more or less of the trait in question.

The simplest statistical approach to understanding these relationships involves partitioning the variation in the behaviour of interest into the sum of the genetic effects and the variance unexplained is then due to the environment (which includes maternal/paternal effects, indirect genetic effects, ecology and abiotic factors like temperature, food, and water), or alternatively, testing a range of genotypes across environments and then partitioning effects into genes, environment, and their interaction (how genes and environment affect each other to determine phenotypic variation) (see Chapter 4). This reveals exactly how genes, the environment, and their interaction can affect phenotypes, including behavioural phenotypes.

To use a simple morphological example to make this point very clearly while noting the principles are exactly the same for behaviour: if we could take three plant-clones (three distinct plant genotypes (Figure 1.1) and grow each of them in two highly controlled environments that only differed from each other by how much water was available and all else was exactly the same, then the differences in plant heights within each environment would be due to just the genes, and the average difference in heights between environments would be due to environmental differences alone.
Figure 1.1 A pictorial explanation of genotype-by-environment interactions (GxE). In (a) we show a plant GxE – for simplicity’s sake (see explanation below) – and in (b) cricket calling behaviour as a hypothetical behavioural example. (a) Three plant genotypes (clones) grown in two environments that only differ in how much water each plant receives, but everything else about the environments is identical. This means that each plant experiences exactly the same conditions within each environment and differences in water between environments. Therefore, plant size differences within each environment are due to just the genetic differences between plants. However, because each plant genotype is found in each environment, any difference in the average plant phenotype across environments is due to the environmental (water) differences alone. The changes in relative size across environments (i.e. Clone 1 is biggest in Environment 1, but smallest in Environment 2) represents a genotype-by-environment interaction. So plant size variation is due to genetic differences, environmental differences and an interaction between the genetic and environmental differences. The same principles apply to any phenotype, including behaviour. (b) This figure shows the same interaction-type across cricket calls where the sonograms above and below the cricket images show the hypothetical songs females of each hypothetical genotype are most attracted to across two imaginary environments. In Environment 1, call rates are slower than in Environment 2 (there is an environmental effect on preferred calls), and each genotype prefers different calls (a genetic effect), but the type of call preferred depends on the environment sampled (gene-by-environment effect).

And if the effects of the genes on the phenotype varied across the two environments (i.e. the biggest genotype in environment 1 is the smallest in environment 2), then we have a genotype-by-environment interaction (we additionally include a hypothetical behavioural example as well; see Figure 1.1). To put that into the simplest terms:

\[ P = G + E + GxE \]

(1.1)

where \( P \) = the phenotype, \( G \) = the genotype, \( E \) = the environment, and \( GxE \) = the interaction between genotype and environment, and this is as true of behaviour as it is of morphology. And if we are talking about variation around average behaviours, then we have:

\[ V_P = V_G + V_E + V_{GxE} \]

(1.2)
where \( V_p = \) phenotypic variation, \( V_G = \) genetic variation (averaged over environments), \( V_e = \) environmental variation (averaged over genotypes), and \( V_{GxE} \) = the variation due to the interaction between G and E. From this, we can estimate the proportion of variation in the phenotype (deviation from the mean) that is due to variation in the genes as the ratio of the genetic variation divided by the phenotypic variation as a whole (\( V_G/V_p \)), which is known as the broad-sense heritability (\( H^2 \)) – a measure of the heredity of a phenotype (Falconer 1981).

Thus, by this simple variance partitioning exercise, we can attribute phenotypic variation into a genetic and an environmental component, and if \( H^2 = 100\% \) then (ignoring maternal effects, for example) the variation between individuals within a population is all due to variation in genes (i.e. the phenotype equals the genotype) and if it is 0\%, then all variation is due to the environment variation (the phenotype does not accurately describe the genotype). Note that these are local estimates – they are population, environment(s), and time specific because they depend on the genotype and environment distributions of the population sampled at that point in time. It is equally important to remember that just because a trait has zero heritability this does not mean it has no genetic component. Remember that heritability describes variation determinants and, for example, finger number has zero heritability despite being clearly determined by genes because there is (effectively) no variation in finger number due to genes – everyone (to a first approximation) has five fingers per hand at birth.

The point of the above is merely to illustrate the relative ease of hypothesically dissecting behavioral variation into genetic, environmental, and interactive effects using standard analysis of variance (ANOVA) (Anholt and Mackay 2004; and see Zar 1999; Sokal and Rohlf 1981), despite the problems ANOVA has with correctly assigning variation for some gene–environment (G/E) relationships (e.g. with some reaction norms ANOVA can fail to detect G or E effects, instead falsely assigning all variation to one effect or another: Lewontin 1974; Figure 1.2). While this is old and obvious for many, the same is

**Figure 1.2** An example of how ANOVA can in principle fail to correctly assign phenotypic variation to causal factors. Here, the phenotypic reaction norms for two genotypes (1 and 2) are shown across an environmental gradient. There are clear environmental effects since both reaction norms increase across the gradient, and there are clear genotype effects on the phenotype as the reaction norms of the genotypes differ. However, if all environments were considered equally, there would be no overall effect of genotype because the two genotypes would have the same phenotypic mean across the gradient, while if environments towards the origin were sampled more heavily, an effect of genotype would be detected. Source: Redrawn from Lewontin (1974).
Figure 1.3 The complex interplay between nature and nurture that affects a phenotype like behaviour. Here we can see, for example, that the genes expressed in a focal animal (genotype) contribute to the social environment and the social environments (and the genes of other individuals expressed in it) can influence gene expression in the focal individual (genotype), while both also directly act on the (focal) phenotype, which itself can also affect the social environment and genotype. The latter effect is indicated by the smaller dashed arrow, which indicates epigenetic pathways whereby parental phenotypes can alter offspring gene expression, for example. These same pathways exist between the abiotic environment and genotype/phenotype and there is a link between abiotic and biotic environments – social environments can be affected by abiotic factors, for example (e.g. Simmons and Bailey 1990). Note that the social environment is effectively all non-self genes that are part of the broader environment, and this can include endosymbionts. And for simplicity’s sake, we have not included gene–gene interactions, which even with small numbers of genes can be huge (e.g. with 10 genes influencing a trait, there are 180 two-way interactions, 1920 three-way interactions and 3360 four-way interactions. And, for example, the number of four-way interactions with 100 genes increases to $6.3 \times 10^7$). Source: Wade (2000).

not true for everyone. For a more thorough explanation of statistical genetics, consult a dedicated text (e.g. Falconer 1981; Roff 1997; Lynch and Walsh 1998; and see Chapter 4), but the take-home message here is that complex traits will be influenced by genes, environment, and their interaction (e.g. Boake 1994; Anholt and Mackay 2004; Hunt and Hosken 2014). This picture is further complicated by gene–gene interactions (Anholt and Mackay 2004, 2015), which rapidly increase as gene number (loci and alleles/locus) increases (Wade 2000), as well as epigenetic feedbacks and a social environment which influences, and is influenced by, genes in the focal organism (Figure 1.3). All of this makes predicting behavioural phenotypes highly probabilistic because the phenotype of each individual will be determined by the unique biochemistry of their genotype and how this interacts with the unique set of environmental and developmental conditions they experience.

So what evidence is there that behaviours are mostly polygenic and that the variation in behaviours can be partitioned as described above in Eq. 1.2? The fact that many behaviours are not discrete but tend to be continuous is indirect evidence (e.g. Sokolowski 2001), and with the advent of cheap sequencing we can now count the numbers of genes that correlate with behavioural variation – for example, more than 250 genes showed twofold expression differences in divergent Drosophila lines selected for behavioural differences in geotaxis (Toma et al. 2002). Statistical estimates of the relative contribution of G, E, and GxE to behavioural variation have also been undertaken in a wide range of taxa (Table 1.1), with behaviours tending to have heritabilities that are smaller than for general morphology and on a par with life history traits (reviewed in Mousseau and Roff 1987; Roff 1997). Behaviours studied include mate preference, aggression, dominance, and even personality, and clearly show G and E effects, as well
Table 1.1  A small sample of behaviours that have been explored using statistical genetics approaches to determine the heritability (here narrow-sense heritability: the proportion of variation in a behavioural phenotype explained by the additive action of genes) of behavioural phenotypes. Estimates listed here range from 15% to 71% of the variance explained by genetic effects, with the remaining 85–29% due to environmental and interactive effects.

<table>
<thead>
<tr>
<th>Behaviour</th>
<th>Species</th>
<th>Estimation method</th>
<th>Heritability estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Mate preference</td>
<td><em>Drosophila simulans</em></td>
<td>AS</td>
<td>0.26 (± 0.11)</td>
</tr>
<tr>
<td>(2) Mate preference</td>
<td><em>Achroia grisella</em></td>
<td>P-O</td>
<td>0.21 (± 0.13)</td>
</tr>
<tr>
<td>(3) Attractiveness*a)</td>
<td><em>Drosophila simulans</em></td>
<td>P-O</td>
<td>0.29 (± 0.15)</td>
</tr>
<tr>
<td>(4) Foraging ability</td>
<td><em>Panorpa vulgaris</em></td>
<td>P-O</td>
<td>0.15 (± 0.05)</td>
</tr>
<tr>
<td>(5) Post-mating sociality</td>
<td><em>Nauphoeta cineria</em></td>
<td>P-O</td>
<td>0.33 (± 0.28)</td>
</tr>
<tr>
<td>(6) Exploration</td>
<td><em>Parus major</em></td>
<td>P-O/F-S/AS</td>
<td>0.22/0.37/0.54</td>
</tr>
<tr>
<td>(7) Risk taking</td>
<td><em>Parus major</em></td>
<td>AS</td>
<td>0.19 (± 0.3)</td>
</tr>
<tr>
<td>(8) Dominance</td>
<td><em>Pan troglodytes</em></td>
<td>AM</td>
<td>0.71 (± 0.01)</td>
</tr>
<tr>
<td>(9) Cognitive ability</td>
<td><em>Homo sapiens</em></td>
<td>TS</td>
<td>0.41–0.66</td>
</tr>
<tr>
<td>(10) Boldness</td>
<td><em>Ovis canadensis</em></td>
<td>P-O*b)</td>
<td>0.21 (± 0.23)</td>
</tr>
</tbody>
</table>

AM, animal model (pedigree); AS, artificial selection; F-S, full-sib; P-O, parent-offspring regression; TS, twin studies.

a) Includes the sum of all male courtship behaviours.
b) Mother-offspring regression which includes maternal effects.

Source: (1) Sharma et al. (2010); (2) Jang and Greenfield (2000); (3) Taylor et al. (2007); (4) Missoweit et al. (2007); (5) Moore (1990); (6) Dingemanse et al. (2002) and van Oers et al. (2004); (7) van Oers et al. (2004); (8) Weiss et al. (2000); (9) Haworth et al. (2010); (10) Reale et al. (2000).

as GxE interactions when these have been tested (e.g. Jia et al. 2000; Miller and Brooks 2005; Narraway et al. 2010; Ingleby et al. 2013) (see also reviews in Ingleby et al. 2010; Hunt and Hosken 2014).

Human twin studies report generally similar results – there are clear genetic and environmental effects on behaviour (e.g. Bouchard 2004; Haworth et al. 2010). These studies often follow identical twins that were separated from birth that can then be compared with twins reared in the same home, thus providing essentially the experimental design discussed above (see Figure 1.1). This is because identical twins are genetic clones, and hence similarities across environments (different homes) largely represent the effects of shared genes, for example. These and related pedigree studies find that, as expected, G, E, and GxE all influence a range of human behaviours. And even in cases where genes of large effect, or gene regions that affect behaviour, have been identified, this does not imply strict determinism, for the reasons outlined above.

So for the vast number of studies that have undertaken measurements of behaviour within an appropriate genetic design, it is abundantly clear that many behaviours are determined by genes and environment (and their interaction), and this is true of human behaviour too. We hope (but with some trepidation) that is the end of the false dichotomy of genes versus environment and an end to denials of exclusive genetic underpinnings for behaviour. Does, as appears to have been the fear, the fact that genetic variation underlies behaviour mean that apparently complicated behaviours are in fact
deterministic, thereby destroying the notion of human free will and responsibility (‘my genes made me do it’)? We hope that the above discussion puts this anxiety to rest too. The fact that there are GxEs in one very real sense means that the effects of genes on behaviour are unpredictable and that the smallest variation in environment can fundamentally alter the effect of genes on behaviours (even ignoring gene-by-gene effects). Understanding that the social environment provided by other members of a society is also continually changing shows that the GxE interaction is also always on the move even if G remains constant (which it will not). Furthermore, development itself, where local developmental-environment and gene feedback occurs to lead from zygote to fully differentiated multicellular organisms, ensures that relationships between genes and outcomes are inherently probabilistic. And as Figure 1.3 shows, the multiple pathways that link genes and phenotype coupled with the environmental effects and all the feedbacks, including inherited epigenetic links (which are just another maternal/paternal effect), really do mean we are dealing with probabilistic rather than deterministic outcomes (for an example of complicated maternal/social-biotic-environment interactions, see Tregenza et al. 2003). Finally, it is hoped that we all now broadly accept that to be human ultimately means rising above the imperative of the genes (Dawkins 1976). So with that out of the way, we can now move on to consider some of the (more interesting) topics discussed here more fully in subsequent chapters.

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References


