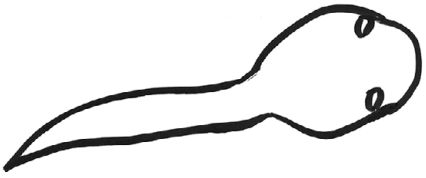
WHAT I USED TO BE



WHAT I AM NOW

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5 Our Bodies, Our Genes and Our Wellbeing

I have chosen to be happy because it is good for my health.

Voltaire

Our thoughts, feelings and behaviour do not occur in disembodied space – they occur in our bodies. This chapter investigates four big questions about the relationship between mind and body:

- Can we locate our feelings in the brain?
- How does our mental wellbeing affect the rest of our body?
- How in turn does our body affect our mental wellbeing?
- How do our genes affect our mental wellbeing?

Feelings and the Brain

Neuroscience is still in its infancy. But we can already locate **areas in the brain** where people experience their mental wellbeing and their distress. The method is to ask many people how they feel and then to correlate their answers with the electrical activity in different parts of their brains. The best method of measuring electrical activity in different parts of the brain is by functional Magnetic Resonance Imaging (fMRI).[1] Using fMRI, significant correlations have been found between measures of wellbeing and electrical activity in a number of different brain areas. For example, Richard Davidson and his colleagues at the University of Wisconsin have found a strong relationship with activity in the ventral striatum (and within it the nucleus accumbens).[2] The relationship holds both over time for the same person (as when a mother is shown a picture of her child) and also, more importantly, across people. Thus people with higher sustained activity in the ventral striatum report higher psychological wellbeing (see Figure 5.1). Their adrenal glands also produce less cortisol – another positive sign of wellbeing (see below).

[1] This measures the rate of glucose metabolism (which is related to the firing of the neurons).
[2] Davidson and Schuyler (2015).

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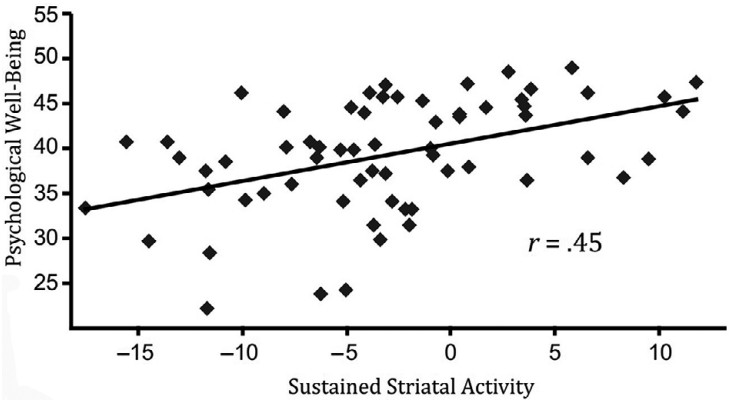

Figure 5.1 Sustained activation of the ventral striatum predicts psychological wellbeing
Source: Heller et al. (2013)

The ventral striatum is a subcortical area that humans share with other mammals. But neuroscientists have also found correlation between wellbeing and activity in different parts of the pre-frontal cortex (including the ventro-medial and the dorso-lateral).[3] Researchers have also identified a Default Mode Network in the brain that takes over when nothing much else is going on. It is focused on the self, and people where the network is more active report themselves to be less happy.[4]

So there are objective measurements that correlate with reports of subjective experience, and this confirms that there is some objective information content in the reports of subjective wellbeing. But our understanding of the neural correlates of wellbeing is still quite partial.

More advanced is the neuroscience of pain. The main area where people feel distress when they are in pain is the anterior cingulate cortex (ACC), which is another subcortical area. **Physical pain** has two components – the 'sensory' aspect and the 'emotional' aspect. The sensory aspect informs us where the pain comes from (back, leg etc.) and its nature (e.g., pulsating, continuous, hot, cold), and it is recorded in the somatosensory cortex. But the emotional distress felt as part of the pain is located in the ACC.[5]

So too, is the emotional distress resulting from **social pain**.[6] So it is not surprising that the pain-killing drug paracetamol (Tylenol in the United States) has the same

[3] Volkow et al. (2011). [4] Raichle et al. (2001).

[5] Strictly, it is in the dorsal ACC (the top part, the dACC). If the dACC is somehow severed from the rest of the brain, the distress from physical pain disappears but the sensation of disturbance continues. Equally, if the somatosensory cortex is disconnected, the nature of the pain becomes unclear but the distress is still experienced.

[6] Eisenberger et al. (2003); and Lieberman (2013). With social pain, if this is induced (e.g., by a rejection in an online game) the dACC is stimulated. If Tylenol is taken, this dACC response does not occur, nor do people report as much distress. For qualifications to this analysis, see Ferris et al. (2019).

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dulling effect on the experience of both physical pain and social pain. In each case, the drug is moderating the electrical activity in the same part of the ACC.

So we already know something about where our conscious feelings of wellbeing and pain are experienced. But how does our mental life affect the rest of our body?

How the Mind Affects the Body

The clearest effect of the mind on the body is its effect on **longevity**. In September 1932, the mother superior of the American School Sisters of Notre Dame decided that all new nuns should be asked to write an autobiographical sketch. These sketches were kept, and much later psychologists independently rated them by the amount of positive feeling they revealed. These ratings were then compared with how long each nun lived. Remarkably, the amount of positive feeling that a nun revealed in her twenties was an excellent predictor of how long she would live. Of the nuns who were still alive in 1991, only 21% of the most cheerful quarter died in the following nine years, compared with 55% of the least cheerful quarter of the nuns.[7] This shows how happiness can increase a person's length of life.

More recently, a random sample of English adults over 50 were asked questions about their happiness, and they were also asked whether they had been diagnosed with any long-term physical illness, such as heart disease, lung disease, cancer, diabetes or stroke.[8] They were followed for nine years to see if they died. The crude results are shown in Figure 5.1. The least happy third of them were three times more likely to die than the happiest third. And, even when controlling for all their initial physical illnesses, the least happy third were still some 50% more likely to die. Another study traced everybody in a Norwegian county over a six-year period. At the beginning, they were all diagnosed for their mental state and also asked other questions, such as whether they smoked cigarettes. Over the next six years, it turned out that diagnosed depression was as powerful a predictor of mortality as smoking was (Figure 5.2).[9]

What explains this effect of mood upon physical health? The clearest channel is through the effects of **stress**. The body has a mechanism that responds to stress in a similar way whether the stress is physical or mental. This is sometimes called the **'fight or flight'** response: our heart rate, blood pressure and breathing rate increase; we sweat more and our mouths go dry.

This response begins in the brain, which is linked to the rest of the body by two main sets of nerves. One set includes the sensory nerves and the motor nerves, which give conscious instructions to our limbs about what to do. But the other set is the 'autonomic nervous system', which is largely outside our conscious control and regulates the workings of all our internal organs.

The autonomic system has two main branches: the sympathetic and the para-sympathetic. It is the **sympathetic nervous system** that initiates the fight or flight

[7] Danner et al. (2001), Table 3, rows 5 and 8.
[8] Steptoe and Wardle (2012).
[9] Mykletun et al. (2009) Tables 1 and 2.

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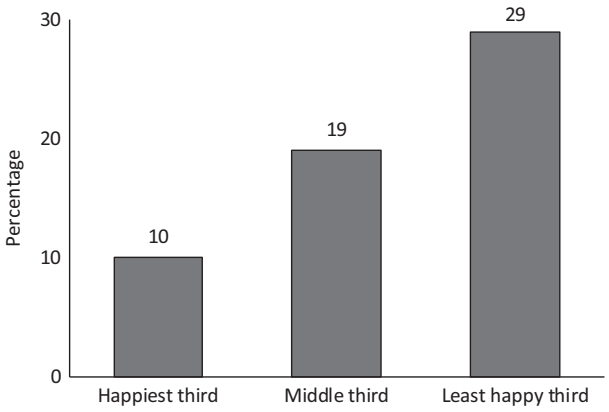

Figure 5.2 Percentage dying over the next nine years (People originally aged 50 and over)
Source: Andrew Steptoe. See also Steptoe and Wardle (2012)

response. It immediately instructs the adrenal gland to produce what Britons call **adrenaline** and Americans call **epinephrine**. This is a hormone (Greek for messenger) that enters the bloodstream and galvanises the whole body for action. It also mobilises the immune system to produce pro-inflammatory cytokines in case they are needed to handle possible infections. The **parasympathetic nervous system**, in contrast, calms the body. When it is active, the body's organs become less active. For example, in meditation or breathing exercises the vagus nerve is active in reducing the heart rate. But, so long as stress is maintained, it is the sympathetic system that is most active.

At the same time, a second hormone is produced in another part of the adrenal gland: **cortisol**. A message goes from the brain's hypothalamus to the pituitary gland to the adrenal gland, which releases cortisol into the bloodstream, and this then stimulates the muscles by releasing their store of glucose.

The stress response is totally functional when the stress is brief. But when the stress is persistent, it can lead to **over-activity of the immune system** (especially of C-reactive protein and IL6) and to persistent **inflammation** around the body, which eventually reduces life expectancy.[10] It also leads to more fibrinogen in the blood – intended to cause blood clots in a wound but unwanted in the longer term. In Western countries the most common sources of prolonged stress are psychological, and increased inflammation has been observed as a result of marital conflict, caring for demented relatives, social isolation, social disadvantage and depression.[11]

Another cause of stress with long-lasting effects is **child abuse**. One study followed children born in Dunedin, New Zealand. Those who had been abused in the first ten years of life had increased markers of inflammation twenty years later.[12]

[10] Murabito et al. (2018). [11] Wilson et al. (2018).
[12] Danese et al. (2007). The inflammation index is based on an aggregate of high-sensitivity C-reactive protein, fibrinogen and white blood-cell count. See also Steptoe et al. (2007).

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These findings on child abuse are confirmed in a meta-analysis of many comparable studies.[13] By contrast, optimism and purpose in life protect against coronary heart disease and stroke.[14]

Perhaps the simplest evidence of the effect of mind on body comes from a simple experiment. In it people were given a small **experimental wound**. Those who were depressed or anxious took the longest to recover.[15] In another experiment, people were given injections, and people in the greatest psychological distress developed the fewest antibodies.[16] Since we can affect the mind by psychological intervention, we can also affect the body that way. For example, mindfulness meditation reduces the level of pro-inflammatory cytokines.[17] It also increases the production of telomerase, which increases life-expectancy.[18]

Perhaps the most striking effect of the mind on the body is the **placebo effect**. For many diseases, up to 30% of people given a placebo pill (with no active ingredient) will recover.[19] People recover because they believe that they will.

So the mind has profound effects on the body. These are a major part of how our wellbeing is generated, and the different chemicals involved provide useful **biomarkers** of how our wellbeing is developing.

How the Body Can Affect the Mind

But there is also a stream of causation going in the opposite direction – from our body to our wellbeing. Bodily events can alter our mental state. Healthy living is vital for our mental wellbeing and this means plenty of exercise, enough sleep and good sense in drinking and eating.[20] Equally, physical illness and dementia reduce our wellbeing.

But one of the clearest examples of the effect of the body on the mind is the power of **drugs**, be they recreational or psychiatric. These work by affecting the operation of chemical **'neurotransmitters'**, which are crucial to the working of the brain. The brain consists of about 100 billion brain cells or neurons, and each neuron is connected to thousands of other neurons. Messages travel round the brain one neuron at a time. When that neuron 'fires', an electrochemical impulse travels from one end of the neuron to the other. But then it reaches a gap between that neuron and the next neuron. This gap is called a 'synapse' and the message is carried across the gap from the sending neuron to the receiving neuron by a chemical neurotransmitter.[21]

[13] Baumeister et al. (2016).
[14] Kubzansky et al. (2018) and references therein. See also Steptoe, Wardle and Marmot (2005) on the correlation of positive affect with reduced levels of neuroendocrine, inflammatory and cardiovascular activity.
[15] Kiecolt-Glaser et al. (1995). See also Cole-King and Harding (2001). [16] Cohen et al. (2001).
[17] Cresswell et al. (2016). [18] Schutte and Malouff (2014). [19] Evans (2003).
[20] See Chapter 14. There is also growing evidence that the microbiome in our gut affects our mood. See Michels et al. (2019).
[21] The receiving neuron has 'receptors' designed to receive the neurotransmitter.

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Table 5.1 Some recreational drugs and their effects

Effect	Drug	Effect on main neurotransmitters
Stimulant	Ecstasy (MDMA)	Increases serotonin
	Cocaine, amphetamines	Increases dopamine
	Nicotine	Mimics acetylcholine
Sedative/relaxant	Alcohol, barbiturates	Increases GABA
	Cannabis	Increases endocannabinoids*
Pain relief	Opiates (heroin, morphine)	Mimics endorphins**

* Can also increase dopamine, acting as a stimulant. **Endorphins are endogenous morphines, hence their name.

Table 5.2 Some psychiatric drugs and their effects

Problem	Drug	Neurotransmitter action
Depression	Prozac	Increases serotonin
Schizophrenia	Chlorpromazine	Reduces dopamine
ADHD	Ritalin	Increases dopamine
Anxiety	Diazepam	Increases GABA

So the different circuits in the brain are operated by different neurotransmitters:

- Serotonin produces a good mood.
- Dopamine and acetylcholine are neurotransmitters that stimulate (and can heighten) mental activity. (A 'high' feeling is generally associated with a rush of dopamine).
- GABA (gamma aminobutyric acid) and endocannabinoids reduce mental activity.
- Endorphins reduce pain.

Drugs affect the operation of a neurotransmitter. Some stimulate production of a neurotransmitter, others reduce its production, while others bind to the same receptors as some neurotransmitters and thus have a similar effect as the neurotransmitter that they mimic. Table 5.1 shows how the main **recreational drugs** alter our mental state, through the way in which they alter the operation of the neurotransmitters. Unfortunately, all these drugs can be addictive.[22]

There are, however, other drugs that can make people feel better: **psychiatric drugs**. These are generally less addictive. Table 5.2 shows some of the psychiatric drugs recommended for different psychiatric conditions. Take one example, Prozac. This is a psychiatric drug that increases the flow of serotonin and thus activity in the circuits that it serves. It does this by inhibiting the reuptake of serotonin, thus increasing the supply of serotonin and improving mood. In other words, it is a selective serotonin-reuptake inhibitor (SSRI). In many depressed patients, Prozac improves mood. Clearly dopamine is a tricky neurotransmitter: increasing it can be

[22] Whether they should therefore be banned is a separate issue. For one view on this, see Layard and Ward (2020) pp. 156–58.

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stimulating, but an excess of dopamine leads to schizophrenia (and a deficiency of dopamine to Parkinson's disease). Like psychological therapy, psychiatric drugs do not always work. They are, however, strongly recommended for severe depression, where they achieve 50% recovery rates. But they have no effects on relapse unless they continue to be taken.

A key fact about the brain is the ease with which it can be altered by experience[23] – in other words, there is a high level of '**neuroplasticity**'. Blind people use part of their visual cortex to hear. And London taxi drivers have to remember so many streets and routes that they develop abnormally large hippocampal areas in their brains.[24] There are many places in this book where we shall report the effect of wellbeing interventions on brain activity.

How Our Genes Affect Our Wellbeing

The one part of our body that never changes is our **genes**. They were determined at the moment of our conception and, apart from mutations, we keep the same genes throughout our life. The same genes are present in the nucleus of nearly every cell in our body. It is the continuity in our genes that explains much of the continuity in our personality, our appearance and our behaviour over our life.

But how do we know this, and how far do differences in our genes explain the differences in wellbeing in the population? To explore these questions, we shall proceed this way.

(1) We shall examine twins and show that identical twins (with the same genes) are much more similar to each other in wellbeing than non-identical twins are (with many different genes).
(2) We shall look at adopted children and show that they still resemble their biological parents in many ways.
(3) We shall show that the genes and the environment do not have independent effects on our wellbeing – they interact. Therefore, it is not possible to say in any meaningful way how much of the spread of wellbeing is due to differences in genes and how much to differences in environment.
(4) We shall describe the pioneering search for the specific genes that affect wellbeing.
(5) Finally, we shall examine the inter-relation between genes, personality and wellbeing.

Evidence from twins

To see that genes matter we have only to look at the following data on Norwegian middle-aged, same-sex twins (see Table 5.3). Some of the twins are **identical**: both

[23] Dahl et al. (2020). [24] Maguire et al. (2000).

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Table 5.3 Correlation in life satisfaction between each twin and his/her co-twin (Norwegian adults in mid-life)

Identical twins	0.31
Non-identical twins	0.15
Difference	0.16

Source: Roysamb et al. (2018)

come from the same egg. They therefore have the same genes and look more-or-less identical. The other sets of twins are **non-identical**: each twin comes from a separate egg. So half her genes are the same as her co-twin's but half are different (just as is the case with any other pair of siblings).[25] And what a difference that makes! As Table 5.3 shows, if the twins are identical, their life satisfaction is fairly similar (with a correlation across the 2 twins of 0.31). But if the twins are non-identical, their enjoyment of life is much less similar (with an across-twin correlation of only 0.15).

What could account for this difference? Clearly it must be because the identical twins have genes that are more similar. Even though both sets of twins were raised together, their final wellbeing was very different. This suggests that the family environment has a smaller influence than many people suppose.

Countless twin studies testify to the effect of our genes not only on our wellbeing but also on our mental health. For example, if you are bipolar and you are an identical twin, then the chance that your co-twin is also bipolar is 55%. (That is the degree of 'concordance'.) But, if your co-twin is not identical, the chance is only 7%.[26] Thus if someone has bipolar disorder, the risk to the co-twin is a huge 48 points higher (55%–7%) if the co-twin is identical rather than non-identical. Figure 5.3 gives comparable numbers for many other types of mental illness. In every case, the difference is substantial. That is an astonishing testimony to the power of genes.

Evidence from adopted children

A different approach to the same issue is to look at **adopted children** and ask, Are they more similar to their adoptive parents or to their biological parents? Until the 1960s it was assumed (largely due to Freud) that mental illness was chiefly caused by how our parents behaved. But in 1961, Leonard Heston of Oregon University

[25] At each gene locus, I have 2 representations of the gene (2 'alleles'). One came from my father and 1 from my mother. For the same reason my father also had 2 alleles at that locus. But I only got 1 of my father's 2 alleles. Which of the 2 I got was randomly determined. The same is true of my sibling. Thus if I got any particular allele, the chance that my sibling also got it was ½. So we have half our father's alleles in common. The same is true of the alleles we got from our mother. Thus ordinary siblings 'have roughly half their genes in common'. But identical twins come from the same egg and have all their genes in common. (This analysis applies to those genes that differ between people, the so-called polymorphous genes. These genes comprise about a quarter of all our genes; the other three-quarters are the same for all humans.)

[26] Plomin et al. (2013) p. 246.

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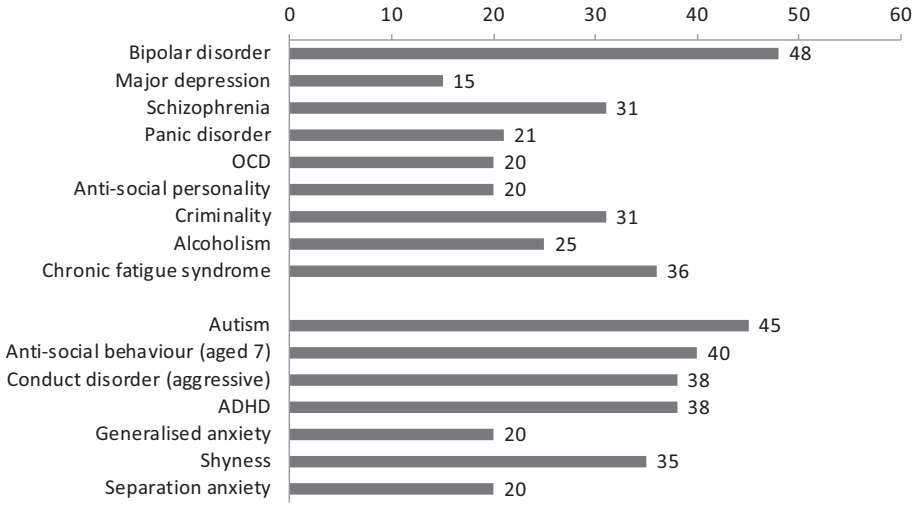

Figure 5.3 Difference between identical and non-identical twins in the concordance between twin and co-twin
Source: Plomin et al. (2013) pp. 245, 249, 251, 252, 259, 265, 290
Note: For each condition, we calculate the concordance for identical same-sex twins and for non-identical same-sex twins and report the difference. For OCD, alcoholism and all childhood conditions except autism, we give the difference in co-twins' correlation (on a continuous measure). For rare binary conditions, the concordance and the correlation are very similar

published his classic paper on schizophrenia. Heston studied adopted children and what he found was remarkable. One per cent of these adopted children became schizophrenic (the same as other children) – unless their biological mother was schizophrenic. But in that case 10% became schizophrenic. So it was the mother's genes that made the difference rather than the behaviour of their live-in adoptive parents. And in fact, if you had a mother with schizophrenia, you were no more likely to become schizophrenic if you lived with her than if you did not.[27] Not all adoptive studies are as striking as Heston's. For depression and anxiety disorder, they yield less striking results. But they are not inconsistent with the evidence from the twin studies that we have just looked at.[28]

When studying wellbeing, a key source of evidence has been the Minnesota Twin Registry, which includes twins who were raised together and twins who were raised apart (as adoptees). The study showed that identical twins raised apart were much more similar in wellbeing ($r = 0.48$) than non-identical twins brought up by their biological parents ($r = 0.23$).[29] This again underscores the importance of genetic factors.

[27] Layard and Clark (2014). [28] Plomin et al. (2013) chapter 6.
[29] Tellegen et al. (1988). Wellbeing was measured by indicators of positive affect. For identical twins raised together the correlation was 0.58 – not much higher than for identical twins raised apart.

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Heritability

A natural question now is to ask What fraction of the variance of wellbeing across individuals is due to the genes?[30] In other words, what is the **heritability** of wellbeing? Questions on heritability are usually answered using some strong assumptions, which we will question later.

We assume an additive model with (in its simplest form) two components – G for the genetic component and E for the environmental component. Thus wellbeing (W) is determined by $W = G + E$. It follows that the variance of wellbeing equals the variance of the genetic component plus the variance of the environmental component plus twice the covariance of the genetic component and the environmental component:

$$\text{Var}\,(W) = \text{Var}\,(G) + \text{Var}\,(E) + 2\,\text{Cov}\,(G, E)$$

Crucially, in behavioural genetics this **covariance is considered to be due to the genes**. But in truth, it can be as much due to the environment as to the genes. For while it is certainly true that people with good genes are skilful at finding good environments, it is equally true that in most societies good environments are more receptive to people with good genes.[31] So the first arbitrary assumption in behavioural genetics is that individual outcomes related to genes are caused by genes. The second arbitrary assumption (to which we shall return) is that there are **no gene-environment interactions**.[32]

But if we wish away these two problems, we can show that the heritability of a trait (like wellbeing) equals twice the difference between the correlation of the trait across identical twins and its correlation across non-identical twins.[33] So if we use the numbers in Table 5.1, the heritability of life satisfaction is $2\ (0.31-0.15) = 32\%$. This is a typical estimate of the heritability of life satisfaction obtained from twin studies worldwide.[34]

Genes and environment

So the genes really matter. But so does the environment that we experience. In fact, the environment is generally more important. Even with the most heritable mental trait that we know of (bipolar disorder) only just over a half of the co-twins of bipolar people also have the condition. And for most conditions it is much less.

Moreover, genes do not operate on their own, with the environment just adding further effects. Rather, the genes and the environment interact, with the genes

[30] This clearly depends on the structure of the environment. For example, when educational opportunity improved in Britain after WWII, the heritability of school performance increased dramatically (Haworth et al. 2016).

[31] Wootton et al. (2017).

[32] See Plomin et al. (2013) p. 401 for an explanation of how difficult the concept of heritability then becomes.

[33] For derivation, see Annex 5.1 (from Layard and Clark 2014).

[34] Bartels (2015); Roysamb et al. (2018); Van der Weijer et al. (2020). By contrast, the heritability of height is some 90% and of Body Mass Index 70% (Plomin et al. [2013]).

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Table 5.4 Chances of onset of major depression within the month following a severely stressful life event

Condition of co-twin	Chances of depression within the month(%)
Co-twin depressed and identical	14
Co-twin depressed and non-identical	12
Co-twin non-depressed and non-identical	8
Co-twin non-depressed and identical	6

influencing the effect that the environment has on us and vice versa. This we can see clearly in the following study of the impact of negative life events on a sample of twins in Virginia.[35] Negative life events included the death of a loved one, divorce/separation and assault. And the issue was how frequently did those people who had a negative event experience a major depression in the subsequent month?

Therefore, for each individual the study measured

(i) what negative events they experienced,
(ii) whether major depression followed within a month and
(iii) the mental health and relatedness of the individual's co-twin.

As Table 5.4 shows, a person was **more** likely to experience a major depression if their co-twin was depressed (especially an identical twin who was). And they were **less** likely to be depressed if their co-twin was non-depressed (especially if it was an identical twin). This is a clear case where people have bad experiences but the effect depends also on how far their genes predispose them to depression.

Evidence of such interaction is ubiquitous. For example, in one study of adopted children anti-social behaviour was more common in adolescents if their adoptive parents were anti-social. But the effect was greater still when the biological parents were also anti-social.[36]

There is however some encouraging news. As we have seen, people with unfavourable genetic predispositions respond worse to bad events than other people do, but they also **respond better** to good events.[37] For example, children who carry the unfavourable variant of the gene most closely related to depression can respond better to CBT than other children do.[38]

The interaction between genes and environment in determining wellbeing should not come as a surprise. For such interactions are also common in physical health. A classic case is the disease known as phenylketonuria (which produces mental retardation). To get the disease requires two things:

- First, you need the unfavourable gene.
- Second, you have to eat phenylalanine, which is present in many foods.

[35] Kendler et al. (1995). [36] Cadoret et al. (1995).
[37] On the 'differential sensitivity' hypothesis, see Belsky (2016); and Pluess (2015).
[38] The serotonin gene. See Eley et al. (2012).

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If you avoid the foods, you don't get the disease.[39] So, even for people with unfavourable genes, we can greatly improve their lot by improving the environment.

Evidence from DNA

The previous discussion does not rely on any actual data on genes – you just compare the wellbeing of different pairs of twins or adoptees. But today we can sequence the actual **DNA** that each individual carries. There are millions of positions on the string of genetic material, and at each position, one of three variations is present. These variations are known as **Single Nucleotide Polymorphisms** (or SNPs, pronounced Snips). With the aid of this information we are able to get more direct evidence on the roles of genes.

For wellbeing, a comprehensive study of 11,500 unrelated people and over half a million SNPs assessed the genetic similarity of every possible pair of people in the sample. By relating this to the difference in their wellbeing, it showed that people's genes (treated as additive) explained 5–10% of the variance in their wellbeing. This is a minimum estimate since it omits the effect of any interaction between different genes.[40]

A different and potentially important endeavour is to discover which specific genes make the most difference, through **genome-wide-association studies** (GWAS), which look for the effects of each gene upon the trait in question. The first pioneering study was able to find three SNPs that passed the test of significant effects on wellbeing.[41] Each SNP explained 0.01% of the variance of wellbeing. A more recent study has been able to identify 148 significant SNPs, which together explain 0.9% of the variance. This is partly due to larger sample size and precise measurement of the outcome, and further work will then further increase our understanding.[42]

The conclusion is that there is no single gene for happiness, or even a small number of genes. Instead, thousands or more genes are involved, interacting in complicated ways with each other and with the environment. Taken together, these genes predispose people to more or less happy lives.

Personality and Wellbeing

As we have seen, an important way in which genes affect our wellbeing is through our mental health. But a more general way is through all aspects of our personality.

[39] Plomin et al. (2013). One particular form of gene-environment interaction is epigenetics. This occurs when environmental factors determine whether a gene gets 'expressed' or not (e.g., methylation in a gene's promoter region prevents the gene having any effect).

[40] Rietveld et al. (2013). This is equivalent to the share of explained variance in a multiple regression of wellbeing on all the SNPs. After allowing for measurement error the estimate rises to 12–18%.

[41] Okbay et al. (2016). The test of significance is demanding because it has to take into account the problem of multiple testing.

[42] Baselmans et al. (2019). A meta-analysis by Jamshidi et al. (2020) estimates heritability based on GWAS to be between approximately 0.5% and 1.5%.

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Table 5.5 Correlation between twins and their co-twins in various aspects of personality (Norwegian adults in mid-life)

	Identical twins	Non-identical twins
Neuroticism	0.56	0.27
Extraversion	0.46	0.27

Source: Roysamb et al. (2018)

Psychologists find that much of the variation in character that we experience in those we encounter can be described by five dimensions. These are Openness, Conscientiousness, Extroversion, Agreeableness and Neuroticism (which spell **OCEAN**). Some of these dimensions appear to be poorly correlated with our life satisfaction. But two are highly correlated with life satisfaction. These are neuroticism (as you might expect from our earlier analysis) but also extroversion. So, if we revert to the mid-life Norwegian twins we discussed at the beginning of the chapter, personality overall explains about a third of the variance in wellbeing. And personality itself is partly determined by our genes (see Table 5.5). So a significant part of the heritability of wellbeing comes from the heritability of personality.

This said, it is crucially important to recognise that personality (like wellbeing) varies substantially over the life course. Not only do we on average become more conscientious, agreeable and emotionally balanced over time, but we also become less open and less extrovert. And we also change a lot relative to our contemporaries – due to differences in how life treats us.[43] Genes are important, but from now on we concentrate on the effect of what policy-makers **can** affect – namely our experience of the environment in which we live.

Conclusions

(1) Self-reported wellbeing is correlated with activity in a number of brain areas. The sensation of pain is most clearly experienced in the anterior cingulate cortex (ACC), which registers both physical pain and social pain.
(2) The mind affects the body. Wellbeing predicts mortality as well as smoking does. Prolonged psychological stress leads to excessive production of adrenaline/epinephrine and cortisol, over-activity of the immune system and excessive inflammation in the body. Mindfulness meditation reduces these effects and increases life-expectancy.
(3) The body affects the mind. The most obvious effects are those of drugs, recreational and psychiatric.

[43] Specht et al. (2011).

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(4) Genes have important effects on our wellbeing. We know this in two ways.
   - Identical twins (who have identical genes) are much more similar to each other in their wellbeing than are non-identical twins (who share only 50% of their genes).
   - Adopted children are more similar in mental health to their biological parents than to the parents who raised them.

   It is important that parents and professionals realise the importance of these genetic effects and do not automatically blame parents' behaviour for the problems of their children.

(5) It is, however, not possible to neatly separate the effects of the genes and the environment for two reasons:
   - Genes and environment often interact in their effects on wellbeing.
   - Genes and the environment are correlated, and there is no simple way to apportion that part of the variance of wellbeing that comes from the covariance of genes and the environment.

And we should never assume that, because a problem is partly genetic in origin, it cannot be treated as effectively as one that is primarily environmental.[44]

This completes our review of some basic processes common to all humans – our behaviours, thinking styles, physical processes and genes. It is time to turn to the impact on us of specific features of our experience.

Questions for discussion

(1) Does the evidence from the brain make people's self-reports any more credible?
(2) How important is the mind in explaining physical health?
(3) How far can drugs of all kinds improve our wellbeing?
(4) Does it help to know how far a person's wellbeing is affected by their genes? In what ways might it help?
(5) What does it mean to say that for most purposes genes and environment interact to determine character?
(6) Why is it so difficult to say what proportion of the variance of wellbeing is due to the genes?
(7) If something significantly influenced by genes, is it automatically more difficult to change than something mainly due to the environment?

[44] Haworth and Davis (2014).

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Further Reading

On Brain Measurement

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