

Myth 1: We come into the world as a blank slate: how we think and feel is determined only by experience

Myth 1 is the 'standard social science' model of thinking and emotion.¹ It is just as wrong as saying that everything we think, do and feel is determined by our genes. The mind is the last battle ground for these two polarised positions. Although general medicine ditched this false dichotomy years ago – realising that human anatomy and physiology, health and disease, were all a product of an interaction between both genes *and* environment, psychology has been struggling to find this middle ground when it comes to defining both mental health and mental illness. We seem reluctant to examine what parts of our minds are determined by evolution and what parts seem to be unique to the individual or his experiences. You could assert that this is because there are no fossils of human behaviour. However, for centuries now we have been documenting human behaviour in all contexts around the globe. Furthermore, there are traditional hunter-gatherer communities to examine in the present day, whose cultures might, to a certain extent, represent 'living fossils' of our ancestors, despite the influence of observers and the insidious seep of modern urban life. We can look for certain 'universals' of thinking and emotion which are common to all cultures. We can also examine the behaviour of other mammals from which we presume to have evolved.

So, this type of study, what we might call evolutionary psychology (EP), provides the middle ground between nature and nurture that a proper understanding of psychiatry and psychology requires. We have the capacity to learn and improve on what we started out with, but what we started out with was shaped by evolution and genetic variation. EP goes beyond examining the individual and starts to explore what it means to be human. In the language of the evolutionary psychologist, this equates to the difference between the 'proximate' and 'ultimate' causes of human conditions. A young woman might have a phobia of snakes that is so severe that even seeing pictures of the creatures in a book makes her heart pound and her throat turn dry with fear. The proximate cause might have been coming across a snake while hiking as a young girl. The ultimate cause, however, would be to do with her ancestors on the plains of Africa, and the impact that being bitten by a poisonous snake would have had on their survival (and

hence the propagation of their genes). The eminent psychologist Martin Seligman demonstrated decades ago that we have an increased tendency to develop phobias of snakes (and spiders) over much more modern dangers, like guns. He called this phenomenon 'preparedness'.² Emotional 'programs' like these evolved over millennia and are not going to be modified quickly. Psychologically, we are still living in the caves.

Another influential psychologist called Garcia demonstrated how our ability to learn to avoid foods that make us ill several hours later, and after just one trial, does not conform to the idea that all human behaviour starts from a blank slate.³ Before Garcia's experiments most psychologists were wedded to 'behaviourism', the scientific movement which seemed to be able to prove that all human learning conformed to certain rules – the rules of 'classical' or 'operant' conditioning. In summary, in order to learn, behaviour had to be closely paired in time with a consequence. Also, a negative consequence would need to occur several times before it extinguished a formally learned behaviour. Eating is normally rewarded with feeling satiated, and if a food tastes good we will not hesitate to eat it. However, if that food contains something noxious, and we later vomit as a result, we will immediately acquire a distaste for that particular food that lasts pretty much indefinitely. The evolutionary implications of this are clear – if you don't learn to associate the taste with illness after one trial, and some time after tasting the food, you will not live very long. Garcia proved this 'preparedness' for a unique type of learning in a laboratory experiment. He showed that he could make rats learn a distaste for sugary water by making them ill with radiation several hours later.

To give an example closer to depression, the proximate cause of grief is the death of a loved one, but the ultimate cause is to do with the importance that emotional bonds have played in the history of social mammals, and how grief might be an important way of processing and accepting a loss. It makes perfect sense, from an EP point of view, that the loss of an intimate partner should cause more distress than the loss of a remote relative, because the effect on reproductive success is more direct in the former case. Negative feelings initially help us to strive for the lost person, but if the loss is due to death the pining is futile, and we will need to go through the process of accepting the loss. The closeness that we had prior to the loss must be unlearned; otherwise we can not seek out new bonds that are important for propagating our genes.

Hence, to what degree evolution plays a role in our functioning today

is the real question for EP, and the real question underlying this book. Different functions of the brain – like recognising a smile, fearing a snake, getting sexually aroused by the particular shape of a man or woman’s body, manipulating a tool, solving a problem, and learning a language – have all been shaped by different evolutionary pressures, with some overlaps. These human capacities, coded for by a few or by many genes, some constant in shape and effect, some varying, will interact with each other and the environment, and will vary across the general population. On average they will have persisted because they gave the ancestors carrying those genes a competitive advantage over others – leading to a better share of resources and sexual partners, and hence giving the vessel of those genes the edge in terms of survival and reproductive success. Some complex but apparently inherent skills, like musical ability, seem at first glance to be difficult to explain in terms of evolution. Why should a concert pianist pass on his ability to his child? Environment is not enough to explain this alone – some people will be pushed into music classes from an early age but will never achieve the level of concert pianist. In the case of rare musical ability the gifted musician will have co-inherited a number of characteristics like good hand–eye coordination, optimal hearing, emotional sensitivity and good communication skills. In other words, musical ability is a pleasant by-product of more important skills that would have increased the reproductive success of our ancestors in the close-knit hunter–gatherer environments of their time.

A human quality that directly influenced differential reproductive success in a given environment is what Darwin coined an ‘adaptation’. Qualities that are inherited alongside adaptations are by-products, or genetic ‘fluff’.⁴ The evolutionary psychologist’s role is to identify which characteristics of the human mind are adaptations and which are fluff. So, when considering the ultimate cause of the common condition of depression we might conclude that it is just fluff, a consequence of the development of emotional sensitivity, empathy, language and other social aspects of intelligence. It is possible that you can not have these other human qualities without a tendency to experience depression.

On the other hand, depression may have directly influenced reproductive success because it led to some competitive advantage.

Myth 2: Evolutionary psychology implies that behaviour is under genetic control

EP is accused of genetic determinism – the proposition that genes and only genes determine and limit our successes and failures.⁵ Although sociobiologists of the past might have taken up such an extreme position, EP at its best is sociobiology ‘grown up’ – it has absorbed all the evidence, which has emerged more recently, which confirms that psychological functioning is affected by both environment *and* genetics. Thus, EP has room to acknowledge that depression may have an ancient biological blueprint while also acknowledging that it may be on the increase due to modern cultural pressures. Both can be true at the same time. One aspect does not invalidate the other. In fact, modern theories of evolution recognise the phenomenon of genetic and cultural ‘co-evolution’, whereby changes in culture, like changes in the notion of beauty, lead to the selection of different genes for that culture, or when a new genetic mutation that allows for, say, the development of language, lead to cultural changes, which then make genes coding for social awareness more important, and so on.

Also, EP is the preferred model for exploring the idea that human conditions like depression may have been modified over time: depression may have become more malignant and more chronic in the last few centuries due to many major cultural shifts, although the evidence for this is lacking at present. An evolutionary perspective helps us to take in to account the following facts when thinking about prevention of depression:

- the human genome was selected in ancestral environments which were very different from the modern environment, at least in the developed world
- cultural development now occurs too quickly for genes to adapt, resulting in a split between our genes and our lifestyles
- this mismatch between biology and lifestyle can bring about illness, make illness worse, make an illness last longer, or change the nature of an illness.
- adaptations will always represent a balance between benefits and costs because they are never perfect. The erect human spine causes a lot of problems but it is interesting to speculate on the benefits that must have outweighed these costs. There is no reason why we

cannot speculate in the same way about psychology and behaviour. The brain is a physical structure, after all, and one that has been under the control of gene selection.

Myth 3: Evolutionary explanations for human psychology have no basis and can not be proved

EP is often attacked for coming up with fanciful, un-testable biological explanations for complex human behaviour. It is accused of making up 'just so stories', that can neither be proved nor refuted.⁶ This is to belittle the honest process of hypothesis formation – the creation of a consistent, coherent theory, based on what we now know, that we may test in the future.⁷ Darwin's original ideas were mere hypotheses, but they were consistent with the evidence at the time and they were held together by rational argument.⁸ He was struck, like his predecessors, by the observation that animals, like the giraffe or woodpecker, seemed to be so well adapted to their environments, but he also noted the similarities between the structures of the hand, the flipper and the wing. Subsequently, more and more evidence emerged to support his view, so his ideas developed beyond a theory towards being accepted as a 'best fit' model in the world of science. Darwin is not criticised for developing a theory of how things might have been hundreds of thousands of years ago, based on what he recorded in the nineteenth century. Similarly, Noam Chomsky's conclusions on the universal rules of language, which led to his proposition that we had evolved a 'language acquisition device' (in other words we have certain rules of language structure *imprinted* in our brains which mean that we learn language much more quickly than would be possible just by trial and error), were based on a reasonable hypothesis and careful observation. He did not need to travel back in time.

So, returning to depression, the results of large-population follow-up surveys of children and young adults are being published in this millennium, which give new and exciting opportunities to test the possible utility of depression, because they can examine functioning in many areas of life – work, recreation, relationships – before, during and after a major depression, like the NEMESIS study carried out in Holland.⁹ They will challenge the idea that evolutionary theories are not testable.

Myth 4: Depression must be a disease because it is unpleasant and undesirable

At first it seems absurd to consider that depression could be considered anything other than unhelpful, but this is due to social conditioning – what you could call the ‘assumption of disease’. Major depression is considered a disease because it is socially undesirable, interferes with work efficiency, makes the sufferer feel ill, is associated with more physical illness, causes distress and is linked to suicide. Let me take each point in turn:

1. Although depression is often incompatible with functioning well at work, there is a difference between modern and ancestral environments that may make depression seem unhelpful in the urbanised world. Depression could still be helpful in other, more important ways following recovery, or it may have been adaptive in the past, but not now. Modern urban life might cause people to become ‘trapped’ in stressful situations, so that depression is perpetuated and becomes more severe. In the latter case, depression is not being allowed to function as perhaps it should (as a period of ‘time out’) and urbanisation is at fault, not the depression. Depression may have been shaped by urbanisation into something more severe and less helpful.
2. Depression is associated with subjective distress, but this does not imply disorder. The feeling of disgust is subjectively unpleasant, as is the process of vomiting, but no-one would doubt the survival value of these universal human capacities. In other words, unpleasant but instinctive mental reactions, just like the physical examples given above, could be defences rather than disorders. A fear of heights is unpleasant but it is understood to be protective. Similarly, the absence of fear in the face of possible injury or death is considered abnormal. It is equally possible to regard the absence of depression in reaction to certain life events as abnormal.
3. Depression is regarded as socially undesirable, but this does not imply an unhelpful process. People often withdraw from social contact when they have flu, or if they are grieving over a loss, but this is for a good reason. They want to recuperate before returning to the fold.
4. Feeling ill does not equate with disorder. A short term illness can lead to long term benefits. When we suffer a stomach illness after

food poisoning is this a disorder or a normal reaction? Would we consider a fever to be a normal or an abnormal reaction to infection? Similarly, should we consider depression to be an abnormal reaction to stress or loss?

The existence of severe melancholic depression (see definitions) does not necessarily effect depression's adaptiveness overall. A good analogy is the functioning of the immune system: The immune system works well most of the time but occasionally works against us. Mast cells are part of the immune system that work in the nasal passages and skin to protect us from invading 'foreign bodies', like grass pollen. For the majority, the mast cell reaction to grass pollen is proportionate. However, in a minority of people, there is a propensity for the hypersensitivity reaction that we call hay fever. Hay fever is inconvenient but not life-threatening. Rarer still are those people who have a very severe form of allergy known as anaphylaxis. Such people react to the allergenic substance, say shellfish, with angioedema (swelling of throat and larynx), and an asthma-type reaction in the lungs which can obstruct breathing. Extreme reactions can cause death within minutes. The bottom line is that the immune system is very useful to the large majority of us because otherwise we would be killed by any invading toxin. The fact that some people with extreme allergic reactions may die does not significantly affect the propagation of the genes controlling the immune system for the population of humans as a whole. So, it can be argued that severe clinical depression, like anaphylaxis, is a consequence of genetic variation. Human adaptations need only be helpful 'on average' for their genes to survive and propagate. We must also remember that the process of natural selection does not end with the present day. The most disabling types of depression may be bred out in the future.

Myth 5: Depression can not be regarded as helpful because it causes suicide

It is commonly said that depression lies behind most suicide, and therefore depression is a bad thing. National and international mental health organisations talk about the need to treat depression in order to get suicide figures down.¹⁰ This perspective is fundamentally misguided because it fails to grasp the real relationship between society, depression and suicide.

Passive suicidal ideas – thoughts that life is not worth living, for example – are a reasonably common symptom of depression, but forming the genuine intention to die is an uncommon feature, and taking real steps to carry out suicide is rarer still.

It is commonly believed that anyone who takes his or her own life must be depressed. This is a fallacy and an oversimplification of reality. In fact only about 30% of suicides are committed during a depressive illness, and there are usually co-existing problems and social causes. Suicide is also importantly linked to schizophrenia, drug misuse, alcoholism, an impulsive or psychopathic personality, or a combination of all these problems.¹¹ Also, not all those who commit suicide have a mental disorder or illness. Surprisingly, only a quarter of people who commit suicide are under psychiatric care in the year before their death. Suicide is often regarded as an understandable reaction to having a chronic, painful and incurable illness. Suicide is also associated with being a member of a cult or holding extreme religious or political views (consider the Palestinian suicide bombers, or partners in the mass suicide pact in Waco, Texas). Some people who kill themselves for a political or religious cause, like the suicide bombers of Hamas, believe that their actions will be rewarded in the afterlife. From their perspective suicide is a rational choice, born out of strong religious beliefs and cultural allegiances. In a public debate at the Maudsley Hospital in London in 2001 it was proposed that the reaction of most Americans to the terrorist atrocity of 11 September was ‘madder’ than the acts of the Al Qaeda terrorists themselves. Following a far-reaching debate, those in favour of the motion won the final vote. It is a mistake to assign the label of madness to a group if their actions are understandable within a cultural context, even if that culture seems twisted and abhorrent. A person who dies in such an action firmly believes that he is a martyr, who will be rewarded in the afterlife by Allah. So, we can not imply that all suicides are committed by people of unbalanced mind. Very often the cause is cultural, or has a sociological explanation.

At a national level depression rates and suicide statistics often fail to match up. For example, a country like Iran has more depression than was previously thought to be the case – but relatively low rates of suicide.¹² This may be largely due to religious and cultural taboos, which not only prevent suicidal behaviour but distort official statistics. For example, in unclear cases coroners may choose, either deliberately or unconsciously, to record likely suicides as death by ‘undetermined’ cause in order to spare the added burden of shame on the bereaved

This was historically common practice in Ireland, and might still be a common practice in Portugal.¹³ However, variations in suicide rates may reflect variations in levels of cumulative stress and differences in community values. The common denominator in most cases of suicide, including those associated with depression, is isolation or social adversity.

The absence of constraining social contacts is important, as reflected in indicators like divorce, the number of close friends, and loss of crucial significant others through bereavement. As important as stress and negative life events is the feeling that no-one cares if you live or die.¹⁴ Hence, societies that have more cohesive communities may have lower rates of suicide than societies with a lot of segregation and fostering of independent aspirations to succeed. Durkheim, the eminent sociologist, explained suicide in terms of this kind of societal pathology, (which he called the 'anomic society'), rather than the pathology of the individual.¹⁵ His focus was not on depression, but on the social conditions which breed isolation and despair. Such conditions would not have been common in the close knit groups of our ancestors, where there was a very strong sense of interdependence. These were the conditions in which depression evolved.

The modern phenomenon of 'entrapment' may combine with increasing isolation to cause an abnormally persistent or severe form of depression and other mental health problems, which then become associated with suicide. Entrapment is a term introduced by social scientists to describe the phenomenon of being trapped in a stressful situation. On an individual level, modern examples include the single mother with four young children and no support, or someone financially dependent on an exploitative employer. On a society level they include War and societies in transition. So, while the website of the Suicide Prevention department of World Health Organisation states that mental disorder is present in 90% of suicide cases (however they define 'disorder'), it then goes on to say that suicide results from 'many complex sociocultural factors and is more likely to occur particularly during periods of socioeconomic, family and individual crisis situations'. Hence social circumstances and cultural phenomena are the common mediating factors that drive both mental illness and suicide. Figure 1 shows this idea in diagrammatic form.

So, saying that a suicide is caused by depression is no more enlightening than saying that a death by heart attack was due to a blocked coronary artery. The heart attack was ultimately due to a

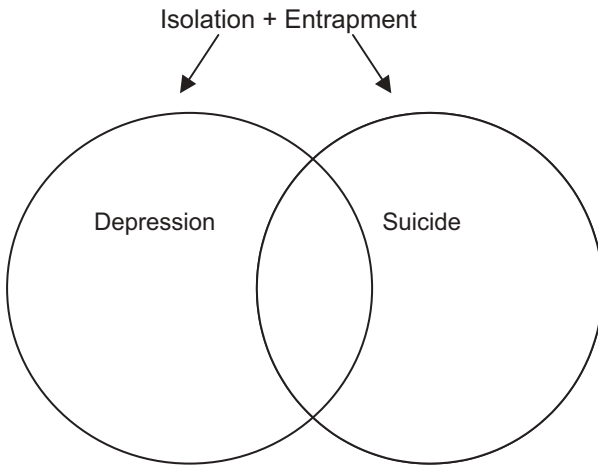


Figure 1 Suicide and depression

combination of an ancient instinct to eat calorific foods and a modern western lifestyle. Additional proximate causes would have been to do with chosen lifestyle, work demands and culturally sanctioned behaviours resulting in stress, lack of exercise, drinking alcohol to excess, or smoking. Similarly, an act of suicide might be tapping in to certain ultimate causes of depression, but in addition there will be the overlay of the social conditions of entrapment and isolation, leading to something different to just misery – a strong sense of hopelessness.

During a radio debate on the evolutionary theories of depression an argument was put forward for a 'fitness advantage' for human suicide.¹⁶ It has been argued that if you are not productive yourself due to any kind of disability or illness, if you have reached a certain age, and procreated, and if being alive puts a strain on the resources of those around you, it would be useful if you were to kill yourself and let your offspring get on with the job of propagating your genes. This is not such a strange theory if you consider how the life of a honey bee comes to an end. However, it does come from the extreme end of adaptationist theory. In my view, it is dangerous to try to posit a fitness advantage for a rare event. It is not justified. Otherwise you might suggest an evolutionary explanation for multiple sclerosis, or diabetes. In the UK in 2002, your risk of suicide was 0.015% if you were female, 0.043% if you were male.¹⁷ So, it is a rare event in anyone's book. And while the rate of suicide is going down in the UK, according to the official statistics, the

rate of depression is going up, suggesting a less than perfect relationship.

Recent research suggests that the neurobiology of depression and suicide are distinct and separate. Gambling tests (the IOWA gambling task is often used) suggest that people with a history of depression but no suicide attempts perform better, i.e. win more money, than those with a history of suicide attempts.¹⁸ This is thought to be due to a greater tendency in the suicidal group to act impulsively. These tests are being combined with neuro-imaging to see if differences can be found in blood flow to different parts of the brain.

Depression and suicide seem to have different patterns of inheritance within a family tree. The Amish communities of the *United States* and *Ontario, Canada*, who are known for their plain dress and limited use of modern devices such as *automobiles* and *electricity*, separate themselves from mainstream society for religious reasons. The result of this isolation is a well defined gene pool and as a result the genetic determinants of many human characteristics have been examined using the Amish. Studies on suicidal behaviour reveal that the family histories of suicide separate from the family histories of depression. In other words, there might be a genetic predisposition to suicide which is separate from the genes coding for depression. Depressed people commonly have thoughts of suicide but mostly stop short of committing the act. Self-preservation instincts usually take over, even in the depths of despair. Suicide 'completers' seem to have an impulsive aspect to their personalities, which propel them towards the final act. A greater propensity to express anger and emotional instability is also thought to be linked to suicide. It should also be noted, when thinking about how suicide relates to depression, that although women experience more depression than men, suicide is overwhelmingly more common in men than in women. This can be partly, but not entirely, explained by the fact that men tend to use more violent and lethal methods than women.

To sum up, depression is unfairly 'blamed' for suicide, which deserves a more sophisticated sociological explanation. Those severe forms of depression that may have a more direct association with suicide are rare, and are outside the scope of this book.

Because the dangers of depression are sometimes very apparent – like suicide, or weight loss, or psychotic symptoms (experiencing hallucinations or delusions) an assumption is often made that *all* depression is unhelpful. 'Mistakes' accompany the average functioning of most human characteristics, due to genetic variation. Depression

is probably no exception. Most depressive illnesses are of mild to moderate severity, and occur as a reaction to stress. Although the risk of suicide in a severely depressed person must be taken seriously, most suicides are not carried out in the midst of a depressive illness. Many suicides are erroneously and retrospectively attributed to depression, while social factors are overlooked.

Myth 6: Depression makes us more prone to other diseases

An association does not prove a cause.

A large body of research has suggested that depression lowers our resistance to infection, by suppressing certain parts of the immune system.¹⁹ Other research suggests that depression increases our risk of suffering from conditions like heart disease, or increases the severity of these diseases following their onset and leads to premature death. These assertions, if true, support the disease model and challenge the 'depression-as-adaptation' idea.

Our natural 'killer' cells help us to kill other body cells that have been infected by viruses. One study showed that natural killer activity was reduced in depressed people, but doubled after two weeks of treatment with an antidepressant, and returned to normal levels upon recovery.²⁰

Other research has suggested that depressed people may be more susceptible to a reactivation of chicken pox, in the form of shingles, due to immune system suppression.²¹ However, evidence to show that the immune system changes observed in the laboratory actually influence the risk of shingles in humans is still lacking.

The relationship between depression, immune system activity and infection is complex. Animals exposed to inescapable stress seem to adopt a behavioural state akin to depression (more of which later). There is compelling evidence that when in this state animals have an increased susceptibility to viral diseases such as herpes simplex and influenza, due to immune system malfunction.²² However, because depression is often a *consequence* of chronic stress, it is hard to separate

the effects of depression *per se* on immunity from the effects of chronic stress and anxiety that pre-date it. If we were to accept that depression is *not* synonymous with chronic stress, it would be hard to make the case that depression has an independent effect on infection risk. It seems that very stressed individuals are at a greater risk of infection, *regardless of whether or not they become depressed*. When a depression resolves, immune system function tends to return to normal, which is beneficial in the longer term. Furthermore, Michael Irwin, an expert in this area, has concluded that 'stress *and* depression can induce *increases as well as decreases* of immune function . . . depending on the immune measures and the chronicity [duration] of the stress' [my italics].¹⁹

Similar defences can be made when depression is accused of increasing the risk of most physical diseases, including heart disease, and of increasing mortality. Evidence has been presented which suggests that depression increases the risk of dying following a heart attack, for example.²³ Depression seems to predict mortality whether it is considered a disorder or an ongoing risk factor. This association with early death is not just limited to specific physical diseases – there is an increased risk in the mixed-disease populations of hospitals or nursing homes.^{24,25} Complex models have been proposed which attempt to explain how depression can lead to disease. For example, the increased risk of heart disease in depressed patients is explained by depression's influence over increased blood pressure, variation in heart rate, increased platelet 'stickability', and increased immune system attack on our own artery walls.

However, all conclusions of the kind 'depression increases one's risk of developing disease', it 'speeds the progression of disease' or it 'increases disease severity' are beyond the data, and may be driven by a desire to medicalise the condition. Not only is there contradictory evidence, none of the research evidence can separate the effects of depression from the independent effects of the stress that pre-dated the depression, and which can impact on both physical and mental disease at the same time.

Furthermore, patients with depression generally have other risk factors for chronic disease. One has only to consider the association between depression and unemployment or poverty. Such social adversity can independently affect disease rates, not only through stress, but also through its association with poor diet, smoking, drinking and inactivity. Increasingly sophisticated studies of premature death have

tried to control for these factors but apply to the elderly only and still have problems with method.²⁶

Let us consider some of the contradictory evidence. Other studies have shown that there is no increased risk of death in depressed people with cancer or end-stage kidney disease.^{27, 28, 29} One study, published in the *American Journal of Geriatric Psychiatry*, concluded that the experience of subclinical depression had no effect on the lifespan of elderly men, and was associated with *reduced* mortality in elderly women.³⁰ Furthermore, a study attempting to show differences in heart rate variation in depressed individuals versus non depressed individuals following heart attack concluded that there was no difference.³¹

Although we know that depression is associated with immune and other internal physiological changes, a neat conclusion is provided by Michael Irwin: 'translation of these observations into clinical and disease-specific outcomes remains incomplete'.¹⁹ So, with the current state of knowledge, a *causal* link between depression and physical disease cannot be made; there is merely an association. The 'depression-as-adaptation' idea is not under threat from the current evidence because one cannot conclude from it that depression causes disease.

Some important assumptions about depression

- 1 Depression is the product of nature and nurture, not one or the other. In most people it is triggered by stressful life events.
- 2 We all have the biological capacity for depression, to varying degrees. This is due to lots of genes, each of small effect, distributed over the whole population, acting together to cause susceptibility to depression. In other words, your genetic vulnerability depends on how many of these common genes (or types of gene variants) you have inherited from your parents. This is the lottery of meiosis following conception – the random jumbling of male and female deoxyribonucleic acid (DNA). The risk depends on how many relevant genes each of your parents are carrying, but you could be lucky and end up with very few. The picture is complicated because some common genes take more than one form, each carrying different risks for depression, and different genes may interact – they might act against each other or with each other. Hence, apart from inheriting more genes in total, you may inherit more potent versions of genes, or more genes that work together as a team to cause depression than your sibling. A good example of a gene that has forms of different potency is the 'serotonin transporter gene', which comes in short or long forms (s and l). It controls the turnover of the brain chemical serotonin, which is critical in the control of our moods. One large study, carried out on over 5000 children and young adults in Dunedin, New Zealand, demonstrated that, in those individuals who had experienced 4 or more adverse life events, their risk of depression was greatest in the s + s combination (an s inherited from both parents; occurring in 17% of the population), and smallest for l + l (an l from both parents; 30% of the population), with s + l falling in-between (s from one parent, l from the other).³² The ss and sl forms make up 47% of the population.

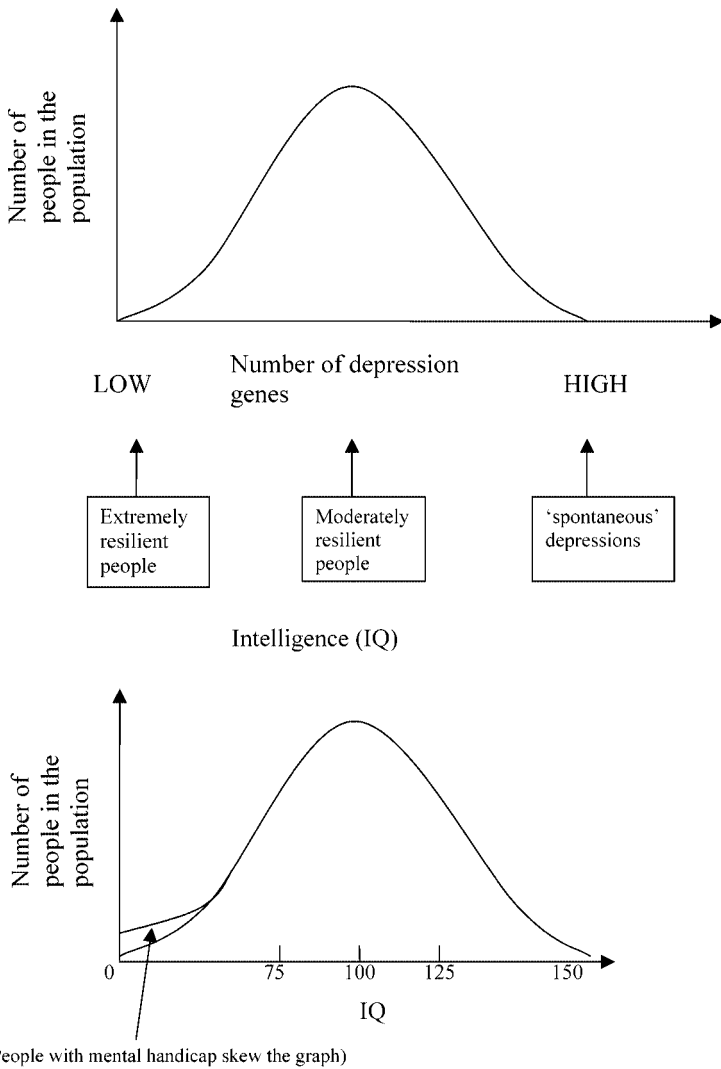


Figure 2 The genetic vulnerability to depression

3 However, the serotonin transporter gene is just one gene that is presumed to have an effect on depression. There will be many genes underlying our capacity for depression, each of small effect. I have assumed that genetic vulnerability is distributed across the

population like a bell curve, depending on the number and potency of genes inherited. The genes coding for IQ are a good analogy: most of us are of average intelligence but there will be a few geniuses and a few people who have significant learning difficulties. There is a tendency towards the mean. Similarly, I have assumed that most people will have *moderate* vulnerability to depression under stress; there will be a few people who have few or no depression genes and are highly resilient to stress; and there will also be rare individuals who have so many depression genes that they become depressed apparently 'spontaneously' – the so called 'endogenous depressives' (see Figure 2). The severity of depression that people experience is continuously variable, and I would expect that this is also under similar genetic control (when keeping the effects of the environment constant).

- 4 Psychological vulnerability – due to upbringing and adverse life experiences prior to the current crisis – acts in addition to the genetic vulnerability, and contributes to the risk of getting depressed in the face of stress occurring later on. The effects of upbringing and life difficulty explain why, in two identical twins, one can suffer depression and the other not (studies show that identical twins will both develop depression about 60% of the time, but that leaves 40% who do not).³³

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