SCHIZOPHRENIA: A NEW THEORY

Derek Goldsmith

Email: derekgoldsmith@aol.com

Introduction

Schizophrenia remains one of the great unsolved problems of modern psychiatry. Great progress has been made in understanding other mental disorders, such as phobias and autism, but schizophrenia remains obscure. Experts disagree about even the fundamental questions. Is it one disorder or many? How should we characterise the underlying deficits in cognitive terms? What are the causal factors responsible for the disorder?

Some have despaired of ever finding a good theory of schizophrenia (Bentall 1998). I think this is unduly pessimistic. The current state of confusion is due, I claim, to the fact that psychiatrists have not taken a sufficiently Darwinian approach to the mind. In this essay, I propose a theory that explains schizophrenia based on Darwinian lines. I argue that schizophrenia is the price that humans have paid for their survival in a hostile environment where predators were a major selection pressure. Schizophrenia, I claim, results from a malfunction in a mechanism I call 'the frozen state' which evolved to help our ancestors avoid attracting the attention of predators. I have called my theory the 'frozen state theory', for want of a better name. I could have called it the 'startle-freezing theory', as there are clear links between my concept of the frozen state and the common psychological notion of the startle mechanism. These links are explained in section 3, where I explain how the startle reaction is what triggers the frozen state.

Darwin himself did not propose any ideas about schizophrenia ¬ the term itself was not coined until after his death, and the psychoses were even less well understood then than they are today. However, of the hundreds of photographs which Darwin collected in preparing his book on The Expression of the Emotions in Man and Animals (1872), most are of mental patients. These photographs testify to Darwin's interest in the relationship between facial expression and mental illness. Although the connection between these two phenomena are not explored in the text of the book, Darwin wrote about it in detail in his correspondence with Sir James Crichton Browne, one of the most distinguished psychiatrists of the nineteenth century. The theory put forward in this essay, which also focuses on the relationship between mental illness and the facial expression of emotion, can thus lay some claim to continuing some of the work initiated by Darwin.

Darwinian approaches to mental disorder are beginning to gain acceptance in some quarters (Baron-Cohen 1997), but they are far from becoming mainstream. Most psychiatrists continue to work in blissful ignorance of evolutionary theory. This is a terrible shame, as Darwinian theory holds out the promise of an integrated, multidisciplinary approach to mental illness. The evolutionary psychopathologist has a firm theoretical basis with which to integrate the mass of data emerging from neuroscience, cognitive and developmental psychology, evolutionary biology - as well as genetics, anthropology and linguistics. The theory proposed here draws on research in all these disciplines, and should therefore also prove of interest to workers in all these fields as well as to psychiatrists.

Another weak point in contemporary psychiatry is its poor understanding of the emotions. Despite their fundamental role in many mental disorders, the emotions are still ignored or down-played by many psychiatrists. Even when the emotions are given centre stage, they are not well understood. This is especially true in recent theories about schizophrenia, which tend to see it as an almost entirely cognitive disorder with biological origins, in which emotions play at most a minor role. In this essay, I propose a theory of schizophrenia in which the emotions play a fundamental role.

My ideas about schizophrenia arose originally from my experience of caring for a person I knew who developed the disorder. This was some time ago, before the new neuroleptic drugs made schizophrenia a much more treatable condition. In those days, the psychiatric wards were filled with schizophrenics and there seemed to be little that doctors could do for them except look after their daily needs. The sight of so much pain on the faces of those patients with no effective treatment to help them caused a profound effect on me as a young man. Ever since then, the problem of schizophrenia has weighed on my mind, and I have sought ways to understand it in the hope that, some day, an effective means of prevention may be found.

I hope that my views in this essay will not offend those who suffer from schizophrenia or those who care for them. The ideas proposed here are offered as suggestions for future research, not as dogma. Perhaps there could even be some useful feedback from sufferers, who may recognise some of the themes touched on here from their own experience.

My training is not in psychology or psychiatry, but engineering. This background might strike some as rather odd for someone who wishes to solve the riddle of schizophrenia. There are, however, distinct advantages in thinking about the mind from an engineer's point of view. The engineer naturally adopts what Daniel Dennett has termed 'the design stance'. When looking at any problem, even a psychological one, I ask what mechanism has gone wrong, and why. I ask about the normal function of the mechanism, and how its design features also put it at risk of malfunctioning in some ways rather than others. Designs are always flawed in some way or other, and have to be thoroughly tested before being put into production. Natural selection can be seen as a test procedure, but one that cannot foresee all the environments in which the designs she produces will need to work.

The engineering approach is consistent with the basic methodology of neuropsychology. By looking at particular mental disorders, we will also learn a lot about how the normal brain works. This applies to schizophrenia even more than to other mental disorders.

This paper is organised as follows. In the first two sections, I summarise current thinking about fear conditioning and emotional transmission in humans. Those who are already familiar with current scientific thinking about these topics may jump straight to section 3, where I build on them by putting forward my theory of the frozen state. In section 4 I explain how the frozen state is involved in the aetiology of schizophrenia, and in section 5 I flesh out the developmental aspects of my theory further. In the final sections I explore some of the consequences of my theory for preventative measures (section 6) and research (section 7).

The meat of the theory I propose is largely in sections 3-5, but to anticipate somewhat, the main points are as follows:

1. There is an emotional state during which a person is silent, immobile and particularly suggestible. I call this 'the frozen state', though it is clearly related to the startle state and the hypnotic state.
2. The frozen state evolved as a tool to enable infant humans to avoid attracting the attention of predators and to learn about dangerous situations.
3. The capacity for the frozen state was once universal, but has been declining in frequency ever since predators ceased to represent a significant selection pressure.
4. Those people who are still highly prone to the frozen state are at the greatest risk of contracting schizophrenia.
5. The aetiology of schizophrenia involves repeated bouts of the frozen state in infancy.
6. A common reason why infants go into the frozen state is because they see ¬ or think they see ¬ an expression of fear on the face of a parent. (When I say 'think', of course, I mean unconscious mental processing. The recognition or misrecognition of facial expressions of emotion happens very quickly, way below the level of conscious awareness).
7. Some infants have a poor ability to discriminate fearful from angry faces; when such infants also have a high propensity to enter the frozen state, they can be repeatedly frozen by bouts of anger from their caregivers, which they mistake for fear.

This is, of course, rather speculative stuff. It may be that I am completely wrong. But my theory does at least have the merit of being eminently testable, and I suggest several ways of testing it in the final section.

1. Fear conditioning: the traditional view

Fear conditioning occurs when an animal learns to be afraid of new stimuli that were not previously seen as frightening. In a classic laboratory demonstration of fear conditioning, an animal such as a rat is exposed to a stimulus that does not normally induce fear (the conditioned stimulus) at the same time, or shortly before, it is exposed to a naturally frightening stimulus (the unconditioned stimulus). For example, whenever the rat goes into a particular part of its cage (which is not normally frightening), it might be exposed to a loud noise (which is naturally frightening). If this is done a few times, the rat soon learns to be afraid of going into that particular part of its cage. The conditioned stimulus (part of its cage) has become linked in the rat's mind with the unconditioned stimulus (the loud noise).

According to the traditional view, fear conditioning evolved because it allows animals to become sensitive to novel dangers (Marks 1987; LeDoux 1998). Natural selection is thought to build some knowledge of environmental dangers into the neonate's brain, so that organisms are born with this knowledge and do not have to acquire it by experience. But it takes natural selection many generations to accomplish this engineering feat, so innate knowledge of dangerous objects is only possible for those objects that are consistently dangerous for many generations. There are some objects that fall into this category for most species, so natural selection is thought to program animals to be instinctively afraid of some things. This is supposed to explain why some things are inherently frightening, like loud noises, and why one doesn't have to learn to be afraid of such things. It is clear, however, that not all dangerous things are such a regular part of the environment. An animal that relied completely on innate knowledge for recognising danger would not be capable of reacting to novel threats. This is where fear conditioning is thought to come in. As well as building in some knowledge of dangerous objects at the beginning, natural selection is supposed to have endowed some

animals with a mechanism that enables them to learn about new dangers. By allowing animals to pair previously neutral events and objects with innately frightening events and objects, this mechanism is believed to permit new fears to be acquired.

In solitary animals, new fears can only be acquired by fear conditioning when the organism comes into direct contact with the novel danger. In social species, however, animals can learn new fears indirectly, by acquiring them from other conspecifics. So long as there is a signal that functions as an unconditioned fearful stimulus, the theory goes, one animal can pass on its fears to another by emitting the signal in the presence of the frightening object. If a herd of pigs, for example, is kept in a field surrounded by an electric fence, it is not necessary for each individual pig to receive a nasty shock before it learns to avoid the wire. It is enough for one pig to receive the shock and squeal in fear and pain for the other pigs also to become afraid of the fence. In this example, the squeal is the unconditioned stimulus that functions as a signal transmitting information to the fear system of the other pigs.

In social species such as pigs, then, emotions are doubly useful. Not only do the internal feelings and the bodily changes of emotion cause the organism to pursue or avoid particular courses of action. In addition, the external expressions of emotion provide information to others, allowing them to learn by example. To sum up; emotions both transmit and motivate. We could call this 'Ekman's law', as Paul Ekman has consistently emphasised these twin functions of emotions.

For those species in which parents provide sustained postnatal care for their offspring, the transmission of fear is a particularly important part of such care. In such species, infants tend to be particularly sensitive to the signals of fear emitted by their parents, because these signals are likely to provide a particularly honest source of information about threats in the environment because they are uttered only when some genuine danger is present (they are 'in context'). The high degree of genetic relatedness between parents and offspring mean that parents have the least incentive to mislead their offspring about what is dangerous and what is not. This is why natural selection has designed the minds of young offspring in such species to be particularly malleable by their parents. Adult expressions of fear function as an unconditioned stimulus; infants are born with an innate predisposition to pay attention to this signal and to become frightened of the thing that apparently caused it.

Experiments in the laboratory have confirmed the power of adult influence in creating fears in the minds of young conspecifics. In one experiment, rhesus macaques reared in a laboratory were unafraid of snakes when they were first exposed to them. However, after watching a film of another monkey reacting to a snake with fear, they too began to be afraid of snakes. We can assume that most of this vital information about predators is normally transmitted from parent to offspring.

2. Emotional transmission in humans

The phenomenon of parents passing on their fears to their offspring can be observed in humans. Children who see that their parents are afraid of bathing in a particular river can infer that the river is dangerous without having to test it out for themselves. This is common knowledge; what I wish to emphasise is that, unlike monkeys, humans rely much more on facial expression to pass on their fears to their offspring. When a monkey is afraid, it communicates this to other monkeys by grimacing and screaming. Humans scream too, but if this vocal signal is not

accompanied by the relevant facial expression, it may be interpreted as a signal of something other than fear. The importance of facial expression in transmitting human emotions arises from the unique face-to-face posture that human mothers adopt when suckling their young. Chimpanzees suckle their young in a similar position, but they do not engage in prolonged mutual gazing like human mothers and infants do.

Human parents also have access to another signaling system altogether – language. In the first year or so of the child's life, the non-verbal signals such as facial expression are the only means of passing on fears to offspring. But as the child learns to understand speech, the parent can pass on fears simply by issuing a verbal warning. Children are programmed to take such warnings seriously when issued by their caregivers. Those children that did not heed their parent's verbal warnings were presumably eliminated by natural selection. However, I think that fears acquired by verbal means are very different from fears acquired by non-verbal, emotional means. The former are acquired later, are conscious, and can be modified by later experience. Fears acquired by emotional transmission, however, are unconscious and permanent.

This idea is in agreement with recent developments in neuroscience. In particular, neuroscientists have begun to accumulate evidence that suggests that the emotional system in the brain is relatively independent of the system dedicated to verbal reasoning. This idea goes back at least as far as Paul MacLean, the American neuroscientist who introduced the term 'limbic system' in 1952 to refer to a set of neural structures involved in emotional processing.

More recently, other neuroscientists such as Joesph LeDoux have taken issue with some of the details of MacLean's theory. Even LeDoux, however, gives MacLean the credit for the idea that verbal reasoning and emotion are processed by different neural structures:

> Like MacLean, and unlike many contemporary cognitive and social constructivist theorists, I believe that it is essential that the emotional brain be viewed from an evolutionary perspective. I am very fond of his idea that the emotional brain and the 'word brain' might be operating in parallel but using different codes and thus are not necessarily able to communicate with each other. And I also think that his idea that some psychiatric problems might represent the operation of the emotional brain independent of the 'word brain' is on the mark.
>
> (LeDoux 1998: 99)

According to the current orthodoxy, verbal reasoning is processed largely in the prefrontal cortex, while emotional memories are laid down in subcortical structures known as the limbic system. A key component in the limbic system is a small almond-shaped structure called the amygdala, which LeDoux has shown to be especially important in processing fear.

Research by Ray Dolan and colleagues at the Institute for Cognitive Neuroscience in London has confirmed the functional independence of the emotional memory system from conscious reasoning. In one experiment, they showed two slides of angry faces to people. While one of the slides was shown, a burst of unpleasant 'white noise' was played, thus ensuring that the memory of this face would be unpleasant. The other angry face was presented without any accompanying sound. In the next part of the experiment, one of the slides was flashed up very quickly, immediately followed by a slide of an expressionless face. This technique is called 'backward masking' because perception of the second slide masks the perception of the first. When asked what they saw, subjects reported seeing the second slide, but not the first. Even

though they did not report seeing the first slide, the subjects must have perceived it at some unconscious level, because their brain activity was different depending on whether the first slide was the one that was associated with the unpleasant noise or not. The main brain region associated with the unconscious recognition of the negatively-charged face was the right amygdala (Morris, Ohman et al. 1998).

Dolan's study not only confirms the functional independence of the emotional memory system from conscious reasoning; it also supports my contention that the perception of facial expression is particularly important in the rapid formation of emotional memories in humans. Exact figures are not known, but it is safe to say that a surprisingly large proportion of our neural machinery is involved in processing information about facial expression. This reflects the fundamental importance of the nonverbal, emotional signalling system as opposed to the verbal one. Not only are emotional signals, and emotional memories, much more powerful than verbal ones, but the verbal ones cannot even get off the ground without an emotional foundation to build on.

In comparing and contrasting emotions with language, I have emphasised the signaling function of emotions: this is the first half of Ekman's law - emotions transmit. But emotions are not just signals; they are also important motivational states. Emotions have a dual role - they transmit and motivate - and we must not let the attention given to the former lead us to forget the importance of the latter. Emotions provide the force, the energy and the drive behind everything we do. Without emotions, in other words, we would literally do nothing. No emotion, no motion.

The two roles that emotions play interact via a bodily feedback system. It is not just the case that internal motivational states cause facial and other bodily expressions that serve as signals; the bodily expressions also cause internal motivational states. This two-way causal relation allows for positive and negative feedback effects. When the feedback is positive, the system becomes an amplifier, which can be seen when people 'work themselves up' into a florid emotional state simply by indulging themselves in free emotional expression.

The existence of this feedback loop between internal motivational states and feelings on the one hand, and external expressions on the other, has been noted by a number of emotion researchers, but very few have bothered to ask why humans are designed in this way. This is exactly the kind of question that a Darwinian should ask. For any feature of any organism, an evolutionary perspective can add new light by asking whether it is a design-feature (an adaptation), or a by-product of another design-feature, or just random noise. Perhaps by ignoring the functional question, most emotion-researchers have assumed that the feedback loop between feeling and expression is merely a by-product, or just noise. This seems unlikely to me, however. A feedback loop is a complex thing, and one would not expect it to arise by chance. Also, positive feedback loops (amplifiers) are integral elements in transmitters, and the expression of emotion is all about transmission. Amplifiers stabilise the signal emitted by transmitters by eliminating background noise; perhaps the feeling-expression loop in humans performs the same function. Or perhaps it is but part of a bigger feedback loop between humans, enabling rapport and emotional synchronisation between distinct individuals. These are just initial hypotheses, and need testing. Whether or not these particular hypotheses are correct is, however, less important than the general point that we need to explore the evolutionary rationale for the feedback loop between feeling and its expression.

3. The frozen state

I think the classical view of fear conditioning in and emotional transmission in humans, as described above, is incomplete. In particular, more can be said about the way that fear conditioning and emotional transmission play an important role in the aetiology of psychosis. This is the burden of my new theory, which I call the 'frozen-state theory'.

The frozen state is the name I give to a psychological state during which a person is silent, and immobile unless told to move. Their conscious mental activity is radically altered, and they become very subservient and easy to influence

What evidence is there that the frozen state exists? Several kinds of data are relevant. First, the research on hypnosis is relevant, since the hypnotic state is very similar to the frozen state.[1] Second, anthropological studies of 'culture-bound' syndromes are also suggestive. In this section, I build on these sources of evidence to construct an outline of the frozen state, its evolutionary origins, and its possible malfunctions.

The research on hypnosis includes studies of hypnosis in humans and in other animals. I look at hypnosis in humans in more detail in the following section; here, I will restrict myself to a brief discussion of so-called 'animal hypnosis'. I have put the last term in scare-quotes because the connection between the 'tonic immobility' and 'death feigning' observed in some animals, and hypnotic states in humans, is a matter of some dispute (Gallup 1974). Some animals go completely rigid when escape from a predator is impossible, in a last-ditch attempt to avoid death by faking it. In the past few decades there has been some speculation about whether this primitive escape response may be the evolutionary precursor of the states of paralysis and dissociation that people sometimes show during and after trauma, and also of human catalepsy and catatonia (Gallup and Maser 1977). Opinion is still divided on this matter; my theory plumps clearly for the view that tonic immobility is indeed the precursor for dissociative and psychotic states in humans.

I propose that the frozen state evolved as an infantile adaptation in mammal species where the young are not fully mobile for some sustained period. It would not be needed so much by infant mammals that are able to flee predators very shortly after birth. Horses, for example, can stand up and run within a few hours of birth, and consequently they do not have the frozen state. The frozen state is only necessary for those species in which the young are immobile for some extended period of time after birth. Being unable to flee from danger, their only option when a predator approaches is to freeze so as not to attract the predator's attention by moving. In rats, for example, the pups are relatively immobile for several weeks. When a mother rat detects danger, she emits a brief high-pitched vocal signal (60Hz – a frequency too high for cats to hear) which startles her pups into a state of silent immobility. This is clearly useful when predators are around. By freezing in response to the mother's call, the pup avoids making any sounds and thus avoid attracting the predator's attention. During the frozen state the pup's brain is also highly plastic, which enables important cues about potentially dangerous situations to be impressed indelibly on its memory very quickly. This 'one-shot' learning is also of clear adaptive value in preparing the infant rat for its future life in a dangerous world.

[1] The significance of the frozen state in the aetiology of schizophrenia may not have become apparent to me had it not been for my early acquaintance with hypnosis. By the age of 20 I had become an experienced hypnotist, and I continue to be very interested in hypnotic phenomena. Further research into the hypnotic state will throw more light on the origins of schizophrenia.

I think the frozen state was a key adaptation for early humans too. Like rats, early humans were often unable to pick up their children and run when confronted by a predator. They needed to freeze rather than flee, and parents needed some way of making their infants freeze too. The frozen state was the answer that natural selection provided to this problem.

We may suppose that the following scenario was a frequent one in the life of an early human. First, someone would spot a predator, and alert the other members of the group. Any mothers with young children would then covertly make the facial expression of fear, and this would send their infants into the frozen state. This all happened automatically and unconsciously, of course. The infants would remain silent and motionless until the predator passed. The adults would too, but out of conscious choice, not because they were silenced by being frozen. Then, when the predator had passed by, there would be a general sigh of relief, accompanied by laughter. And this laughter would release the children from the frozen state. According to the frozen-state theory, laughter evolved as a signal of 'no danger' to unfreeze young children. The theory thus sheds important light on the relationship between fear and happiness, two emotions which share much of the same neural machinery (including the amygdala).

For such a situation to have constituted a selection pressure leading to the evolution of an adaptation, it must have recurred with considerable frequency. The stone-age must, in other words, have been a very dangerous place, with large predators constantly roaming around near groups of early humans. This view of life on the African savannah is not so popular as it used to be, however. The growing emphasis in recent years on the role of the social environment in human evolution has led to a corresponding de-emphasis on the role of the nonhuman environment. But large predators did not grow less ferocious as the size and complexity of human social groups increased. Indeed, the continuing threat of predation was probably a main cause of the tendency to live in larger groups.

Not all contemporary evolutionists downplay the continuing importance of the threat posed by predators in human evolution. Antti Revonsuo, of the University of Turku in Finland, argues that 'the ancestral environment in which the human brain evolved included frequent dangerous events that constituted extreme threats to human reproductive success' (Revonsuo 2000). Revonsuo goes on to argue that dreaming is a adaptation that evolved to help our ancestors deal with the problem of predation by enabling them to rehearse predator-avoidance in simulation. I find this argument convincing, but it does not exclude the possibility that there may have been other adaptations too for dealing with the problem of predators. The frozen state, I claim, is just such an adaptation.

Let us turn now from the phenomenon of animal hypnosis to the second source of evidence for the frozen state: that from anthropology. Some of the so-called 'culture-bound' syndromes are particularly suggestive. Latah, for example, is a psychological phenomenon apparently found only in Malaysia. It only affects a minority of the population, yet these unfortunates are clearly recognisable to their peers. The person with latah is particularly susceptible to being startled, and react in extreme ways; they freeze, and become particularly suggestible. If given verbal commands, they will obey them even if it leads them to do things that are taboo, such as disrobing in public. In the absence of verbal commands, they may imitate the actions of the person in front of them. According to one report, a woman with latah threw her baby in the air when she was startled, in imitation of the person in front of her, who had thrown up some logs he was carrying just after she was startled (Simons 1996).

It is a common game in Malaysia to startle these people deliberately. People will often creep up on someone with latah and jab them in the ribs to startle them, and observe the funny consequences. The laughter that accompanies this is, on my theory, the signal that releases them from the frozen state and returns the person to normality. The Malay culture has thus found a way of exploiting the frozen state to produce amusement without causing too much damage to the victims, since those with latah are never allowed to remain in the frozen state for very long. The dangers of remaining in the frozen state for long periods of time will be explained later. For the moment, however, it is sufficient to note that that at some point in the history of the human race, the frozen state became maladaptive, and began to be selected out. It became maladaptive because, as the environment became safer, it became more and more common for the one-shot learning mechanism to lead young children to form inappropriate fears ¬ fears of things that are not really dangerous. These fears are relatively impervious to later correction because the emotional memories laid down while human infants are in the frozen state are processed and stored in the amygdala, which is independent of the verbal reasoning system. Later in life, when the child is able to speak and to understand the speech of others, the emotional memories remain relatively untouched. If these two memory systems contain similar information, all is well. But if the two systems contain conflicting information, a possible danger presents itself. For then it may happen that an emotional memory (laid down in early childhood) which points in one direction is activated at the same time as a verbal memory (laid down later on) which points in the opposite direction. In such a state of internal conflict, the mind can become confused, not knowing which information to prefer. A mind with such conflicting information, in which the emotional memories are at odds with the verbal ones, is emotionally unintelligent. An integrated mind, on the other hand, is what people really mean to refer to when they use that vague expression, 'emotional intelligence' (or EQ).

How can it happen that conflicting information is laid down in the same brain? If all information in the brain was accurate, this could not happen, as there can be no contradiction between pieces of accurate information. So, in cases of conflict, some information must be inaccurate. The hypothesis to be advanced here is that the inaccurate information will usually be the information stored permanently in the emotional memory. But how can such inaccuracies find their way there?

The answer to this question is also to be found by examining the nature of the frozen state. The frozen state is induced in infant mammals by some signal given out by the caregiver, usually the mother. In rats, as we have already seen, this signal is a vocal one – a high-pitched squeak emitted by the mother when danger is detected. In humans, however, the signal is predominantly a visual one; it is a facial expression. In particular, it is an expression of fear on the mother's face.

Like all the basic emotions (anger, fear, disgust, surprise, joy, sadness – see Figure 1), fear both transmits and motivates (according to Paul Ekman, anger is the only exception, since this emotion only motivates, but does not transmit). Fear transmits in the sense that, when one animal senses that another animal is afraid, it too becomes afraid. In human infants, however, the transmission of fear involves more than just becoming afraid. When a human infant sees an expression of fear on its mother's face, it not only becomes afraid, but also goes into the frozen state. Just as the high-pitched squeak is the signal that mother rats use to freeze their pups, so the facial expression of fear is the signal that human mothers use to freeze their infants.

At a very early age, however, human infants find it difficult to discriminate the facial expression of fear from the facial expression of anger. It can therefore happen that, when a

human mother becomes angry, her facial expression is mistaken by her infant child for an expression of fear. Because the infant sees, not an expression of anger but one of fear, it goes into a frozen state. During this frozen state, its brain becomes particularly receptive to information about potential threats in the environment. But in this case, there aren't any real threats there. The mother is not afraid, because there is nothing frightening around. But the infant thinks she is afraid, goes into the frozen state, and deep emotional memories are laid down connecting this harmless situation with the idea of danger. The expression of fear which the infant perceives (mistakenly) on the mother's face is out of context; there is nothing objectively dangerous in the environment. But the infant does not know this; it assumes that something in the current environment is, in fact, dangerous, and acquires a needless fear. A false connection is formed in the infant's emotional memory system. This is how inaccurate information can be laid down in the emotional memory, information that can later conflict with the more accurate information acquired later in life which is stored in verbal memory.[2]

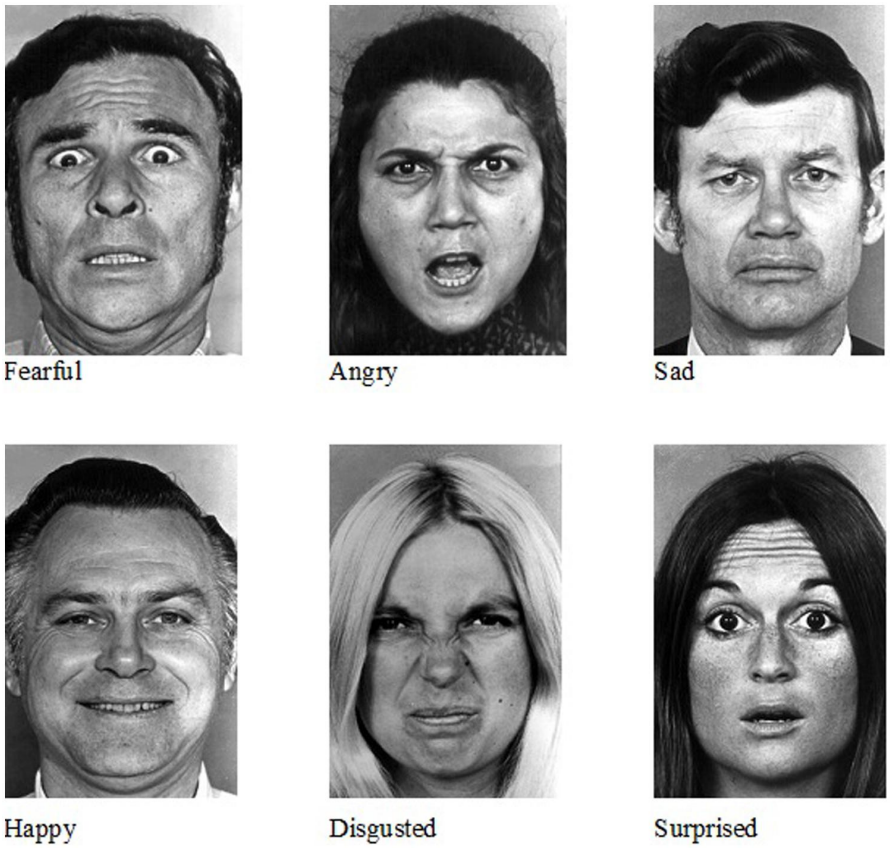

Figure 1: Facial expressions associated with the six basic emotions

[2] Some readers may have noted by now the similarities between my theory of the frozen state and the 'double-bind theory' put forward by Gregory Bateson. Both theories emphasise the destabilising effects of contradictory information in the mind, particularly in predisposing the person to schizophrenia, and both theories attribute the presence of this contradictory information to confusing signals given out by the parents in infancy. It is also interesting to note that Bateson drew much of the evidence for his theory from anthropological research on certain culture-bound syndromes (Bateson 1979). Bateson's theory is no longer taken seriously by psychiatrists, but this does not mean that my theory should be written off too. Besides the similarities, there are also important differences between my theory and that of Bateson. In particular, I think that the signals given out by the parent are confusing because of perceptual discrimination failures on the part of the infant, rather than because of inconsistencies on the part of the parent, as Bateson supposed. Also, Bateson did not bother to construct a plausible evolutionary scenario for his theory.

This situation, in which the infant mistakes the mother's anger for fear, and is thus sent into the frozen-state when there is no real danger around, thereby acquiring needless fears, may inadvertently be encouraged by mothers who see it as a way of getting their children to go to sleep. If mothers find that their children react to anger by freezing, as some do, then these mothers may acquire the habit of getting angry with their children at bedtime. The child does not, in a such a case, go to sleep in the normal way; it goes into a protracted frozen state, which lasts potentially all night. The greater duration of this frozen state means that it is much more dangerous; there is so much more time for inaccurate emotional memories to be laid down, false fears that are out of context.

This is a particularly modern problem. During most of our evolutionary history, the frozen state was generally a short-lived phenomenon, lasting a few minutes at a time. A predator appeared one moment, and infants went into the frozen state for a few minutes, until the predator had passed and the resulting relaxation, smiling and laughter released the infants from their frozen state. In the modern environment, however, when mothers put their children to sleep by getting cross with them and sending them into the frozen state, this state lasts much longer, since the children are not unfrozen by ensuing laughter or even smiles. They are allowed to remain in the frozen state until their natural body clock re-awakens them as it would do from normal sleep. During this prolonged frozen-state, the infant brain acquires many false fears, learning to be frightened of cues in the environment that are not associated with any real danger. Also, some important aspects of brain development occur only during REM sleep; if the frozen state inhibits REM sleep, it follows that children who are regularly 'sent to sleep' by being frozen may exhibit developmental delays. Clearly, the frozen state is no longer adaptive today.

The current maladaptive nature of the frozen state is due precisely to those features which made the frozen state so adaptive for humans in the past. Although the frozen state has been a useful adaptation in many other mammal species, its effects in human development were always much more powerful. This is because the things that set humans apart from other mammals, and from other apes in particular, interact in a multiplicative way with the frozen state. For one thing, the greater neural plasticity of humans means that individual emotional learning plays a far greater role in human development than in that of other apes. Since one function of the frozen state is to act as a kind of high-speed permanent learning mechanism in infancy, it follows that this state plays a more decisive role in shaping the human brain than it does in shaping the brains of other apes.

Also, the period during which infants are dependent on their mother's care is so much longer than in other mammal species. In addition to the lengthy period of dependency, there is also the fact that human babies are born premature. In other words, compared to those of other apes, human brains are relatively unformed at birth. In fact, it is not until the human child is 12 months old that its brain reaches a state of maturity similar to that which a chimpanzee brain reaches at birth. In other words, a human pregnancy would have to be 21 months long in order to produce a baby whose brain was as developed at birth as the brain of a baby chimp.

The greater period of dependency in the human infant, combined with its highly plastic brain, provided a double-whammy of selection pressures that caused the frozen state to become a much more powerful force in human development than in the development of other mammals. The prolonged period of dependency on parental protection means that the frozen state probably occurs much more often in human infancy than in the infancy of other mammals. And the fact that the brain of the human infant is so under-developed means that, during the first

year or so of life, the frozen state tends to leave even more powerful memories in the human brain than it does in other species.

The fact that the frozen state is no longer adaptive means that it is almost certainly 'evolving out'. In other words, the genes that code for freezing are being eliminated from the human gene pool by natural selection. And the powerful effects of the frozen state in humans means that the selection pressure against it now that it has become maladaptive is just as intense as the selection pressure for it when it was adaptive. The exact proportion of the population who are highly susceptible to freezing is a matter for future research. However, if the susceptibility to freezing is what underlies the susceptibility to hypnosis, we may derive an estimate for the former from estimates of the latter. Piccione et. al. provide evidence which supports the view that hypnotic susceptibility is a stable personality trait, and that the same proportion of the population ¬ about 15 per cent ¬ all over the world is highly susceptible (Piccione, Hilgard et al. 1989). They also provide evidence that this personality trait is heritable, which is consistent with my contention that the frozen state has a genetic basis and is evolving out of the human race. Clearly, this needs further research. The normal methods of research in behavioural genetics ¬ twin studies, adoption studies, and linkage analysis ¬ could be used to test my hypothesis further.

4. Psychosis and hypnosis

In the previous section, I set out my theory of the frozen state, and conjectured how it might now be more of a problem than a benefit. The change in our environment - most notably, the absence of predators - means that the frozen state is no longer adaptive, and is therefore evolving out of the human race. Those who are still susceptible, however, are at risk of developing various problems. In this section, I argue that one such problem is schizophrenia.
If I am right, then the group of people who are most highly susceptible to freezing are also the group who are most at risk for developing schizophrenia. If freezing is the basis for hypnosis, as I also claim, then schizophrenics should be easier to hypnotise than normal people.

I am aware that this view flies in the face of current thinking. According to the orthodox view, schizophrenics are notoriously hard to hypnotise. Further research is needed to test this view, as it seems to ignore the striking similarities between the hypnotic state and the delusional one. Freud himself noted these similarities. According to Freud's theory of psychosis, the schizophrenic is particularly insensitive to the evidence of his senses. Instead of modifying his beliefs to bring them into line with what he sees and hears, he modifies what he sees and hears to bring it into line with his beliefs, and thereby his actions. In other words, he allows top-down influences to dominate bottom-up processes. This is exactly what hypnotic suggestion involves too, as is clear from the positive and negative hallucinations which can be hypnotically induced. Other researchers have also highlighted features of hypnosis and schizophrenia that are remarkably similar. Eva Bányai has shown that a common element of the many different hypnosis induction techniques is that the subject's attention is diverted away from external stimuli towards his or her own inner mental processes (Banyai 2000). This 'hyper-reflexivity' is, according to Louis Sass, exactly what characterises schizophrenia (Sass 1999). Combining Bányai's theory of hypnosis with Sass's theory of schizophrenia, then, yields a principled account of why we should expect schizophrenics to be more suggestible, not less. On purely theoretical grounds, then, the jury is still out about the relationship between hypnosis and psychosis; we need more empirical research to settle this matter.

Perhaps the apparent contradiction between the theory (which predicts that schizophrenics are easier to hypnotise than normals) and the current orthodoxy (which finds that schizophrenics are harder to hypnotise) can be resolved in the following way. My theory predicts that children who later develop schizophrenia are very susceptible to the frozen state when very young, but the developmental impairments that result from this repeated occurence of the frozen state make it more difficult for the person to enter the hypnotic state when they are older. These developmental impairments include increased distractibility, limitations in processing capacity, loose associations, and so on. All these impairments will make it more difficult to be hypnotised.

I have pointed to similarities between Banyai's theory of hypnosis and my own, but there are also differences. Perhaps the biggest difference between my theory of the frozen state and Bányai's theory of hypnosis concerns the ways in which the state of high suggestibility can be induced. Bányai stresses the need to divert the subject's attention away from external stimuli, while I emphasise the role of shocking or startling the subject, principally by means of a suddenly perceived facial expression of fear. On the other hand, this difference should not lead us to neglect the similarities. Neither Bányai nor I place much importance on verbal means of inducing high suggestibility. While not denying that words can be used for such purposes, we both assume that they are not necessary. People can be frozen/hypnotised by purely nonverbal means; many tribal peoples use dancing and other bodily techniques to induce trance states that are akin to hypnosis. This is consistent with my view that the frozen state evolved before language. The use of words to hypnotise people today is a relatively recent invention. Like jokes, which use linguistic technology to tap into a pre-linguistic capacity for humour, verbal methods of hypnosis use a later innovation (language) to manipulate an earlier adaptation (the capacity to enter the frozen state).

Bányai's discussion of hypnosis has some resemblance to my theory of the frozen state. Both of us see this state of hyper-suggestibility as an adaptation designed by natural selection, though we disagree about the precise function for which it evolved. Bányai thinks it evolved to promote group cohesion in early humans and especially to promote intimate co-operative dyadic relationships by regulating tension and input in both participants of the interaction (Banyai 2000: 10). I think that the frozen state evolved as a high-speed learning mechanism to help parents protect their offspring by passing on information about dangers in the environment. Both of us, however, agree that at some point in the evolution of early humans there was some strong selective advantage attached to an altered state of consciousness in which people were much more easily influenced than normal, perhaps because they bond very closely with those who are influencing them.

According to the frozen-state theory, then, schizophrenics are much easier to influence than other people. The group of people who are easy to hypnotise is exactly the same as the group of people who are likely to develop schizophrenia.

5. Stages of life

One difference between the various theories of hypnosis, such as those of Freud and Bányai, and my theory of the frozen state is that the former concentrate on adult suggestibility and the latter is concerned with suggestibility in infants. This particular difference, however, is perhaps not as great as it seems, for, while the theory of the frozen state is concerned first and foremost with a state that exists only in infants, it also hypothesises a link with adult suggestibility. To

understand this link, it is necessary to set the frozen state in the context of the life history of the human animal.

The frozen state, as I have already noted, is an adaptation to the particular demands of early childhood. At this time in their life, our stone-age ancestors benefited from being frozen by their parents in situations of danger so that they would (i) not attract the attention of passing predators by being silent and immobile, and (ii) learn quickly the environmental cues indicating immanent danger by means of a highly sensitive one-shot learning mechanism and by being in a state of high suggestibility. By the time they were three years old, they would be more mobile and so would be able to flee from predators with their parents, so the frozen state was no longer useful. By the age of three they would also have been exposed to enough dangerous situations for them to have acquired all or most of the necessary fears to help them avoid such dangers in later life; by this age, then, the frozen state would also have outlived its usefulness as a learning mechanism. Thus, after the age of three or four (the age of infantile amnesia, when conscious access to memories acquired before this age is no longer possible), the frozen state would cease to be part of the child's behavioural repertoire. Just as milk teeth are an adaptation specific to childhood that naturally disappear when the child becomes able to digest an adult diet, so the frozen state is eliminated when the child becomes able to escape from predators on its own.

In fact, the frozen state does not always disappear as it should. In those people who have been repeatedly frozen in infancy, especially those who have been frozen for long periods at a time, it may persist into adulthood. The repeated activation of the frozen state in infancy seems to embed this state deeper into the child's behavioural repertoire in a way that resembles the increased susceptibility to stress of those who are repeatedly shocked. The same phenomenon is observed in Malaysia with those who have latah. The difference between those children who are more easily startled and those who are not is probably quite small to begin with, but the process whereby the former group is singled out for deliberate and repeated shocks amplifies this small difference massively. By adulthood, the difference is so large that those with latah appear to be a completely separate category of person. In the same way, the small differences between those who are more or less susceptible to the frozen state in infancy is amplified by positive feedback during childhood into a qualitative difference between the pathological and the normal in adulthood.

I will return to the subject of psychopathology shortly, but first let us resume our account of the normal development of the fear mechanism in our stone-age ancestors. After the age of three, when the frozen-state would normally cease to be part of his behavioural repertoire, the stone-age child would have spent the following years practising the knowledge that he had acquired in the first years of life. This process of consolidation would have drawn on two principle tools: language (or, in early stages of human evolution, some kind of 'proto-language'), and play. I will briefly examine each of these in turn.

Language and emotions, as has already been noted, are two relatively independent systems of communication that operate in parallel in the adult. The infant, of course, does not have access to the language system, so in the first years of life all the information that is stored in memory comes in via the emotional system. This is why the neural machinery for reading the facial expressions of emotion are so important at this stage. I hypothesise that when the child begins to acquire language, part of this neural machinery is hijacked for language comprehension. This is one of those cases in which ontogeny seems to recapitulate phylogeny; in the evolution of the human species, the emergence of language was achieved by re-allocating neural resources that had previously been used exclusively for emotional communication.

If language acquisition does proceed by co-opting some of the neural machinery previously dedicated to decoding facial expressions of emotion, healthy development of the emotion detection system may be an essential pre-requisite for proper language acquisition. Conversely, those with early damage to the emotion detection system may have consequent difficulties in language comprehension. The critical period for emotional development, in other words, will also be a critical period for language acquisition. Clearly, this is an empirical claim which can only be assessed by appropriate research.

Language (or proto-language) was not the only tool which the stone-age child would have used to consolidate the emotional learning of infancy. There was another tool; play. Play is an important learning device for many mammals. It allows young animals to hone some of the complex skills they will need in later life by rehearsing them in a safe environment. Play-fighting and chasing games can be observed not only in human children but also in young cats, dogs and primates. The play need not be real; in humans, it can also be virtual. According to Revonsuo, dreaming is a kind of virtual-reality simulation of real dangers which evolved to allow our ancestors to rehearse the complex skills needed for predator avoidance (Revonsuo 2000).

If play is a way of rehearsing the complex skills required for fighting and for evading predators in a safe environment, some signal is required in order to distinguish such simulations from the real situations that they help to prepare for. I think that laughter may have evolved in humans partly to serve as such a signal. From an evolutionary point of view, then, laughter is a 'no threat' signal. This is why it can also serve as the trigger to release infants from the frozen state; it tells them that there is no longer any threat, and thus no longer necessary to remain silent and immobile.

When laughter ceases, on the other hand, it can indicate that the situation has turned serious. In the case of play fighting, for example, children may cease laughing and start crying if one of them goes a bit too far, and the fight turns ugly. This is usually enough to make the other children stop the game. The absence of the no-threat signal tells them to treat the situation as a real one, and their violence-inhibition mechanism, which prevents them from harming friends, is activated. Some children, however, carry on regardless in such situations. They seem to lack a properly functioning violence-inhibition mechanism. According to James Blair, this is the central cognitive defect in psychopathy (Blair 1995). Psychopaths are people who have not acquired in infancy the socially correct emotional responses to no-threat signals.

Blair's research also suggests that psychopaths have difficulty in discerning people's emotions from their facial expressions. In particular, they are unable to discern fear or sadness (Blair, Colledge et al. forthcoming). On my theory, those at risk of developing schizophrenia also have difficulty in recognising emotions, but the emotion that such people are unable to discern is anger (which they mistake for fear). This may be enough to distinguish between these two abnormal developmental courses: that which leads to psychopathy, and that which leads to schizophrenia. In particular, this difference means that the frozen state does not play a role in the aetiology of psychopathy. Nevertheless, this difference should not lead us to overlook the similarities between these two conditions. I suggest that both developmental outcomes are abnormal conditions resulting in part from emotional mis-communication early in life. In both cases, inaccurate information goes into the emotional memory in infancy as a result of misreading the emotional expressions on the face of the teacher. Whether or not these suggestions turn out to be correct, however, is less important than the general point that our

knowledge of the mind would be greatly advanced by research into the similarities and differences between psychopathy and schizophrenia.

To sum up: repeated mistaken interpretations, in which the expressions of disgust, sadness or anger are perceived as expressions of fear, may cause permanent damage. Infants the infant mistakenly read anger as fear, they may develop schizophrenia. If they misread sadness as fear, they may develop psychopathy. And if they read disgust as fear, they may develop autism. Adolphs has argued that perceptions of the expression of disgust tend to cause certain kinds of brain activity to shut down. All these processes need to be investigated in much greater detail. Let us return now to the subject of laughter. I have already sketched an account of how laughter might have evolved as a 'no-threat' signal. It is a short step from here to the hypothesis that, once laughter had evolved for this purpose, it then became possible to use laughter as a counter-dominant strategy. When a group of subordinates laugh at a dominant member of the group, they make it clear that they do not find him intimidating. Since intimidation is an important element in maintaining dominance, when a subordinate laughs at a dominant conspecific he is issuing an explicit challenge. A single subordinate who laughed alone at a dominant conspecific could be easily punished, but a group of subordinates laughing together are much harder to stop. Hence the infectious, contagious quality of laughter may have evolved as a way for subordinates to cut their leaders down to size. The medieval institution of the court jester seems to be an implicit recognition of this function of laughter.

Other animals have other ways of signaling the absence of threat, but laughter is uniquely human. Laughter involves coordinated pattern of activity in the respiratory muscles and vocal chords of a similar degree of complexity to that required for speech production, so it is not surprising that it is found only in the one species that can talk. The complexity of laughter also fits it to be a signal of infant vitality; it is hard to produce and so serves as an honest signal of fitness. So laughter probably evolved to have two functions; to signal infant vitality, and to signal the absence of threat. This kind of evolutionary story is much more informative than merely saying that laughter is a signal indicating a state of happiness; after all, we do not always laugh when we are happy.

Neither of these functions requires a sense of humour, so laughter probably emerged before humour. The reason why humans evolved a sense of humour is still a mystery, but Geoffrey Miller has suggested that it is a sexually-selected fitness indicator signaling high intelligence (Miller 2000). Miller's hypothesis presupposes that there is some variable (or set of variables) that reliably distinguishes funny situations from unfunny ones, and that this variable cannot be tracked without a high degree of intelligence. Whatever the evolutionary purpose of the sense of humour may be, it clearly required a signal to indicate its presence, and the pre-existing capacity for laughter was co-opted to serve as such a signal. For an infant to demonstrate that it is fit and healthy, the only criterion it need take into account when 'deciding' to laugh is whether or not there is an external threat; for an adult to demonstrate that he has a sense of humour, however, he must take many more things into account. He must laugh in all and only those situations that are funny, which means that he must be sensitive to all the subtle variables that affect the humour value of a situation.

The two relatively recent tools for consolidating early emotional learning ¬ language and play come together in the even more recent art of telling jokes. Jokes are a kind of technology by which language taps into the humour system, which we may suppose evolved prior to language (as is suggested by the fact that there are clearly nonverbal types of comedy, such as slapstick). In this context, it is interesting to note that some research suggests that schizophrenics are bad

at getting jokes. This may be explicable on the theory proposed here along the following lines. Schizophrenia is caused, in part, by frequent and prolonged bouts of the frozen state in infancy. This leads to disorder in the emotional memory, in the way already explained, and since play (including jokes) is a way of consolidating a healthy emotional memory, it is likely that play will have a different effect on someone with an unhealthy emotional memory. Furthermore, a common reason for the abnormal prolongation of the frozen state is that the infant is not unfrozen by hearing laughter. The people who are most at risk from schizophrenia, therefore, will also be likely to have been exposed to less laughter, and so less able to cope with jokes, or at least less capable of laughing. The frequent grimaces made by some schizophrenics may be a failed attempt at laughing, in which case they may not suffer from any inability to get jokes but merely from an inability to laugh. Alternatively, and perhaps more plausibly, these grimaces are misguided attempts by the schizophrenic to elicit laughter from others. This would show an implicit recognition by the schizophrenic that laughter is the trigger that can 'un-freeze' a frozen infant. The grimacing schizophrenic is thus begging to be released from the frozen state in which he spent too much of his childhood.

The next big turning point in development would come at puberty, which marks the transition from child to potential parent, from pupil to teacher. The time for acquiring and consolidating knowledge of danger comes to an end at this point, and the time for passing that knowledge on to the next generation begins. Emotional expressions cease to be things that one must read in one's parents and start to be things that one must use to pass on information to one's children. This is, indeed, one of the key responsibilities of parenthood: the education of one's children by the intelligent use of facial expression to aid emotional development. This responsibility is not just a feature of modern parenthood, however; it was even more important in ancestral times than it is today, when the burden of parenting is partially offloaded onto a host of social institutions such as schools and health services.

The transition from pupil to teacher, from child to adult, is an enormous challenge, and one that can only be met adequately if one has been properly prepared during childhood. Those children who have not received the right kind of emotional stimuli (i.e. stimuli that are genuinely dangerous) during infancy will find it much harder to make the transition. Since the frozen state is a particularly heightened state of emotional learning, it is likely that a high proportion of the difficulties in making the transition from childhood to adulthood at puberty can be traced back to inaccurate information acquired during this state in infancy.

At puberty, then, the emotional memory is re-configured so that we may transmit the information stored there during infancy and childhood. If the emotional memory has been poorly set up, it cannot be re-configured properly, and puberty will then mark the beginning of serious problems. My hypothesis is that schizophrenia is simply the most extreme of these problems.

Another reason why the transition from childhood to adulthood is difficult for the schizophrenic is that the powerful bond set up between the schizophrenic and his parents in infancy. This bond is much stronger than normal, due to the repeated bouts of the frozen state which have been experienced. It is thus much harder for the schizophrenic to break these bonds and become an adult in his own right, an autonomous individual free from parental control.

One of the most puzzling aspects of schizophrenia is its age of onset. Unlike other mental disorders, which often show first up in childhood, schizophrenia does not usually make its first appearance until after puberty. This aspect of schizophrenia is, however, more explicable on

my theory. I think that schizophrenia is simply a particularly extreme result of failing to make an adequate transition from childhood to adulthood, of the inability to deal with the responsibilities imposed by potential parenthood. When a person has been fed inaccurate emotional information in infancy, he is unable to cope with the adult role of transmitting accurate emotional information to children.

The link between the onset of schizophrenia and puberty is not obvious today, since puberty now occurs around the ages of 12 to 13. This, however, is an artefact of modern civilisation, in which various circumstances such as improved nutrition have brought the age of puberty forward. In the stone-age, our human ancestors probably experienced puberty much later, around the age of 18 or 19. This is very close to the average age of onset of schizophrenia today.[3] The change in human environments since the stone-age has brought the age of puberty forward, while the timing of the brain maturation associated with the demands of parental responsibility has been unaffected. The mismatch between today's world and our environment of evolutionary adaptedness thus hides the fundamental link between the transition to potential parenthood and the onset of schizophrenia.

The difference in age of onset for males and females is another mystery that becomes explicable only when we take an evolutionary view. In the stone-age, males would have begun to go out hunting around the age of 17, and needed to express fear by facial expression to warn other males of possible dangers such as approaching predators. This additional demand of adulthood in males, not faced by females, would have brought the age at which they needed to become transmitters of emotional information slightly earlier relative to females, in whom the need to transmit emotional information was linked exclusively with motherhood. Stone-age females would typically not have borne their first child until their early twenties.

The mismatch today between the age of puberty and the maturation of the brain required for good parenting increases the risk of children acquiring inaccurate emotional information. Females can now give birth to children in their teens, long before their brain has matured sufficiently to allow them to transmit emotional information accurately to their offspring. The children of teenage mothers are therefore at much higher risk of being exposed to confusing facial expressions of emotion from their mothers than children of older mothers.

The idea that the onset of schizophrenia is precipitated by the inability to deal with the responsibilities imposed by potential parenthood is supported by some anecdotal evidence that actual parenthood can worsen schizophrenic symptoms. This anecdotal evidence comes from both my own observations, and from various case-studies of schizophrenia. In the autobiography of the famous nineteenth-century schizophrenic, Daniel Paul Schreber, for example, one of the episodes is precipitated by the news that Schreber's wife is pregnant. Schreber's case supports my theory in another way. When he was a child, his father (who was, paradoxically, an educational reformer) treated him with great cruelty, forcing him to spend hours locked in strange contraptions with purportedly educational aims. It is not hard to guess at the confusion this must have caused to the young Schreber. Here was a supposedly loving father locking him up in what resembled torture-devices all for the sake of 'improving' his son'.

[3] The average age of onset for schizophrenia is 26 for males and 30 for females. This is, however, the median age of onset. A brief glance at graphs showing the age distribution at onset of the prepsychotic (prodromal) phase of schizophrenia shows a peak at much younger ages - around 19-20, in fact, exactly in line with my theory (Hafner, Maurer et al. 1995). When using numerical data, it is important to look at the shape of the graph, and not just take isolated figures!

The fact that Schreber's childhood was full of such mixed messages lends support to my theory that when a person has been fed inaccurate emotional information in infancy, he is unable to make the transition to the adult role of transmitting accurate emotional information to children.

6. Education and parenting

In the previous three sections, I have set out the main points of my theory of emotional learning. In the following section, I explore some of the implications of my theory for scientific research and ask what experiments could be done to test it. Before that, however, I wish to look at some more practical applications of my theory in the field of education and parenting.

Throughout the twentieth century, psychologists largely ignored the role of the emotions in education. The emphasis on IQ was accompanied by a corresponding neglect of EQ. The result was a very impoverished view of how students learn, and a disastrous exam-oriented way of assessing job candidates. As Daniel Goleman has shown in his books on emotional intelligence, IQ explains surprisingly little of achievement at work or in life. When IQ test scores are correlated with how well people perform in their careers, it turns out that IQ only accounts for between 4 and 25 per cent of the variation. In other words, IQ leaves up to 96 per cent of job success unexplained. Emotional competencies, on the other hand, matter at least twice as much (Goleman 1998: 19, 31).

Thanks to people like Goleman, psychologists are now beginning to wake up to the importance of emotional factors in learning. But the growing recognition of the need for EQ needs to be rapidly translated into real changes in education if we are to repair the damage done by a century of over-emphasis on IQ. Children are getting intellectually smarter but emotionally dumber. Since 1918, the average IQ score in the United States has risen 24 points, and there has been a similar rise in other developed countries. But emotional intelligence has not seen a parallel increase; if anything, it has experienced a steady decline. One large survey of American children showed that children in the late 1980s were much more emotionally troubled than a comparable group of children in the mid 1970s (Achenbach and Howell 1989). According to one of the authors of this study, the decline in children's emotional intelligence is a worldwide phenomenon (Goleman 1998 12). Among the consequences of this decline are growing rates among teenagers of depression, eating disorders, drug abuse, crime and unwanted pregnancies. Some particularly dramatic cases have come to symbolise the increasing alienation of young people. In Britain, there was the murder a few years ago of a toddler, Jamie Bulger, by two pre-teen boys. In the United States there was the mass killing of students at Colombine High School by two disaffected teenage students. In both of these cases, it later emerged that violent forms of entertainment had been freely available to the perpetrators of the crime. In the Bulger case, the parents of the two young murderers often watched violent videos on the family television while their children were in the room. The fact that the parents enjoyed watching these films, and communicated this enjoyment to the children by laughing during violent scenes, almost certainly helped to forge a link in the children's minds between violence and fun. In the US case, the two teenage killers spent several hours each day playing violent videogames in which they were rewarded for the most deadly types of attack, such as gunshots aimed directly at the target's head. The parents played a role here too, by allowing their children to play such games, and thus implicitly endorsing them.

Clearly, parents play a fundamental role in the emotional development of the child. We cannot, therefore, place all the blame for declining EQ on poor education. Conversely, the measures

we take to remedy the problem cannot be of an entirely pedagogical nature. We must also take steps to improve the quality of parenting. This is where my theory can contribute some practical suggestions.

One bone of contention that periodically reappears in debates about parenting is the question of whether or not parents should slap their children. Many people have strong opinions on this matter, but arguments to support them are thin on the ground. Unless we have good theories to decide the question, backed up by empirical evidence, we will be left trading intuitions. My theory suggests that surprise slapping will almost always be a bad thing. This is because such slapping may be another trigger that can send a child into the frozen state. When a parent slaps a child, it shocks the child and activates the startle response. The child turns his attention to the parent's face, and whatever emotional expression is on that face goes straight into the child's emotional memory. As I have already explained, if the information transmitted to the child while it is in this extremely suggestible state is out of kilter with the objective nature of the situation, the child will end up with errors in its emotional memory. If, for example, the child sees an expression of fear on the parent's face (which may itself be an error, caused by misinterpreting the expression of anger), he may acquire irrational fears of a harmless situation. If the child is younger than three, the damage done to the emotional system by such inaccurate information is permanent, as the emotional learning that occurs before the age of three is indelible. Even though the child may be taught the correct information by verbal means later on in life, this will not eradicate the earlier incorrect information. Rather, both forms of memory, emotional and verbal, will come to co-exist in the child's mind, leading to internal contradictions.

My theory suggests that it is surprise slapping, rather than slapping per se, that is most dangerous. A simple lesson to be derived from my theory, then, is that if parents use some form of physical punishment such as slapping to teach their children how to behave, they should always give a verbal warning first. If parents warn their children that they will be slapped if they persist in the unwanted behaviour, and the child then persists regardless of the warning, the slap will not come as such a surprise, and so will be less likely to trigger the frozen state, other things being equal.

Of course, these ideas about slapping need to be tested before it can be used to settle debates about the dangers of slapping children. However, there are obvious ethical problems which might make research in this area out of bounds. Scientists researching the dangers of slapping would probably have to use questionnaires rather than direct observation, with a consequent loss of scientific rigour. However, the well-known methodological difficulties involved in using questionnaires to collect good data are not so severe as to render such research entirely invalid. If properly designed, a research program that used questionnaires to assess the effects of slapping on emotional memory and educational performance would be one way to go about testing part of my theory. In the next section, I explore some further suggestions as to how scientific research could put my theory to the test.

7. Suggestions for future research

My theory makes a number of predictions which could be tested by some fairly simple research. One such prediction is that there is a correlation between poor ability to distinguish between angry and fearful faces and increased risk for schizophrenia.

One simple experimental design to test this prediction would be to test a group of schizophrenics and test their ability to discriminate angry and fearful faces. Their performance would then be compared to that of a matched control group. If the schizophrenics performed significantly worse than the controls, this would be consistent with my theory.

It would be better, of course, to employ a prospective design, in which children were tested for their ability to discriminate fearful faces from angry ones, and then tracked to see which ones developed schizophrenia. But this way of doing things would take much longer, involve many more subjects, and cost a lot more. As my hypothesis is rather speculative, it would be better to run the first kind of test first, and set up a prospective study only if it passed the first test.

One problem in the first kind of test might be the confounding variables introduced by drug treatment. The powerful neuroleptics that are prescribed for the treatment of schizophrenia today might interfere with face recognition themselves. If this were the case, the lower performance of schizophrenics on the emotion recognition task would not provide any support for my theory, since it could be due entirely to the effect of the drugs they are taking. Perhaps we could get round this problem by testing schizophrenics who are no longer taking these drugs, but what if a single course of neuroleptics permanently impairs the ability to recognise facial expressions of emotion? This seems unlikely, but it cannot be ruled out altogether. The only way to get round this problem completely would be to find “neuroleptically naïve” schizophrenics, who had never been treated with these drugs. This, however, is practically impossible in modern Western countries, where neuroleptics are now the first line of treatment for virtually all cases of schizophrenia.

A test to evaluate the ability to discriminate facial expressions of anger and fear could take a number of different forms. One possibility would be to take series of so-called 'Ekman faces'. These are photographs of faces whose expressions have been modified so that they conform as closely as possible to the prototype of each of the six 'basic emotions' identified by Paul Ekman: happiness, sadness, anger, fear, surprise and disgust. The photographs could be shown to the subjects in pairs, for varying amounts of time, and the subject asked to name the emotion for each. Or perhaps the Ekman faces could be shown in pairs with the name of the emotion underneath. Sometimes the labels would be correct, and sometimes they would be swapped around, with each photo labelled with the emotion word appropriate to the other. Subjects would have to discriminate, as quickly as possible, between congruent and non-congruent labels. Both accuracy rates and response times would be relevant data for assessing performance.

Another way of testing the ability to discriminate fearful faces from angry ones would be to take the Ekman faces for fear and anger and use image-morphing software to produce a series of intermediate faces. By asking subjects to choose, say, the more fearful face, out of pairs of randomly chosen faces from this series, one could measure the minimal distance for accurate discrimination. Presumably, the shorter this minimal distance was, the better the ability to discriminate fearful faces from angry ones would be. Here again one could also measure response times.

These experiments are similar to experiments recently conducted by James Blair on emotional recognition in psychopaths. As noted earlier, Blair found that psychopaths were significantly worse than normals at recognising the facial expressions of fear and sadness (Blair, Colledge et al. forthcoming). If my hypothesis is correct, and schizophrenics also show a deficit in the capacity to recognise fear, this would raise interesting questions about possible links between

psychopathy and schizophrenia, which have, until now, been thought of as completely unrelated disorders. Perhaps some of the same brain regions are affected in both disorders, or perhaps there are similarities in their aetiology. These are questions for further research.

Another prediction that follows from my theory, which would also be fairly easy to test is that there is a correlation between high suggestibility and increased risk for schizophrenia. There are already a number of established ways of measuring individual differences in suggestibility, such as the tests mentioned by Piccione et.al. for measuring hypnotic susceptibility (Piccione, Hilgard et al. 1989). The same kinds of experimental design mentioned above in respect of looking for correlations between poor ability to distinguish between angry and fearful faces and increased risk for schizophrenia. As already noted, a prospective design would produce more reliable data, but would be expensive and time-consuming to implement. A retrospective design would therefore be a good way of testing the initial plausibility of the hypothesis. If the results of such a study were consistent with the prediction, a prospective study would then be warranted.

Conclusion

In this essay I have advanced an original theory of schizophrenia, and explored some of the consequences for preventative measures and for research. Whether or not these ideas will be found wanting or confirmed will depend, of course, on how the tests turn out. Until such tests are done, we can only rely on evidence gathered for other purposes that may happen to be relevant to the hypotheses proposed here.

Any theory of schizophrenia must answer a number of fundamental questions about the disorder:

1. What are the environmental risk factors for schizophrenia?
2. Why do the genes for schizophrenia persist?
3. Why is the typical age of onset in the late teens?
4. Why are schizoprhenics more likely to be born in the Spring?
5. What causes the precursors of psychotic symptoms observed during infancy and early childhood in those at risk for schizophrenia, such as delays in the acquisition of gross motor skills and language?

My theory provides clear answers to these questions (see *Further Conclusions*, below).

There are further questions about the disorder that a complete theory of schizophrenia must also answer. These include:

6. Why do most schizophrenic patients not have a biological parent with the disorder? (Gotterman, 1991).

7. What causes the full blown psychotic symptoms observed in those suffering from schizophrenia, such as loose associations, illogical thinking and formal thought disorder?

My theory may also throw light on these questions, but the details here are less clear to me. The answers will, I am sure, have something to do with what I see as the fundamental cause of schizophrenia - the emotional miscommunication between parent and offspring early in life.

I recognise that my theory may never be properly tested. Research costs time and money, and there is not enough of either to enable every hypothesis to be investigated. Whether or not my ideas are ever put to the test will depend on how persuasive people find the arguments put forward in this little booklet. Even if my specific views on the aetiology of schizophrenia are never investigated, however, I hope that readers may come away with a greater appreciation of the general value of taking a Darwinian approach to mental disorder in general.

Further conclusions (2003)

Looking back to the time when I wrote this essay, towards the end of the year 2000, I now realise that I did not fully appreciate the importance of Pavlov's ideas about conditioning and their relevance to my theory of schizophrenia. In the time since then, it has become clearer to me that the laws of conditioning play a fundamental role in the pathogenesis of schizophrenia and the development of mental disorder in general.

It is vital to analyse the emotional communication between parent and child in terms of the theory of conditioning. The emotional expression on the face of the parent functions as an unconditioned stimulus (US), which elicits an unconditioned response (UR) on the part of the child. The context in which this unconditioned stimulus-response process occurs then becomes a conditioned stimulus (CS). Later, when the child is in a similar context, this context alone may be sufficient to trigger the same response - even in the absence of the parent. The emotional response in the child has then become a conditioned response (CR) to the context which was originally associated with the emotional expression of the parent.

To be more specific, an emotional expression of fear on the parent's face (the US) triggers fear in the child (the UR). The child learns to associate his or her fear with the situation in which this emotional communication occurs. Later, the situation on its own can function as a CS that triggers fear (now a CR) in the child, even if the parent is absent.

Similar ideas are developed in more detail by Joseph LeDoux in chapter six of his book, *The Emotional Brain* (LeDoux, 1998). The reader is also referred to the new book by Paul Ekman, *Emotions Revealed* (Ekman, 2003). In this book, Ekman explores in print some ideas that previously, to the best of my knowledge, he only discussed verbally. Some of these ideas, such as the claim that emotions serve a dual function - to transmit information to others and to motivate the self - helped to frame my thoughts when I wrote the above essay in 2000. Now that Ekman has developed these ideas in more detail in print, I hope that they inspire other researchers to think about the importance of this dual function of emotions. In particular, this approach helps to reveal the importance of accurate transmission of emotions from parents to children. It is when emotions are transmitted from parent to child in innacurate ways that complications arise.

The importance of Pavlov's theory of conditioning is also relevant to my earlier remarks on hypnosis. I now regard the carrying out of post-hypnotic suggestions as a form of conditioned response (CS), triggered by the conditioned stimulus (CS) of the suggestion itself. As I explained earlier, these linguistic forms of influence have exploited pre-linguistic mechanisms of influence.

Further clarifications (2007)

Today, seven years after I published the original version of this theory, schizophrenia is still shrouded in mystery. There remain fundamental questions that any theory of the disorder must answer:

1. What are the environmental risk factors for schizophrenia?
2. Why do the genes for schizophrenia persist?
3. Why is the typical age of onset in the late teens?
4. Why are schizoprhenics more likely to be born in the Spring?
5. What causes the precursors of psychotic symptoms observed during infancy and early childhood in those at risk for schizophrenia, such as delays in the acquisition of gross motor skills and language?

My theory provides clear answers to these questions. This new preface briefly outlines these questions and summarises my proposed answers.

1. What are the environmental risk factors for schizophrenia?

It is well known that the causes of schizophrenia are partly genetic, and partly environmental. Concordance rates between monozygotic (MZ) twins is about 50%. What this means is that the expression of the genetic predisposition is dependent on additional environmental factors. It is to be expected that geneticists will eventually identify the relevant genes. It remains for others to idenitify the relevant environmental factors. This is what my theory attempts to do.

My theory states that the crucial environmental factor in the development of schizophrenia is the failure to get enough REM sleep as an infant, when this failure is caused by being put into a "frozen state" too often - and in particular being put into the frozen state in the absence of a genuine danger.

2. Why do the genes for schizophrenia persist?

It appears that the genes for schizophrenia maintain a more or less constant prevalence in the population despite the reduced fecundity of those who suffer from the disorder (though the evidence here is consistent with the hypothesis that the genes for schizophrenia are diminishing in frequency too slowly for us to measure). Several experts have suggested that this may be because unaffected close relatives of schizophrenics have higher than average fecundity. In other words, the genes for schizophrenia would actually confer an advantage on carriers who do not develop the disorder. Yet nobody, to my knowledge, has suggested what this advantage is. My theory does.

My theory states that the advantage conferred on unaffected carriers by the genes for schizophrenia is an enhanced ability to enter the frozen state. This would have been an advantage in our evolutionary past, as I explain below.

The concept of the frozen state lies at the heart of my theory. I describe this state in some detail below, and explain its evolutionary origins, but before getting into the details I would like to emphasise an important feature of this state. As I explain below, the trigger that releases an infant from the frozen state is a signal of happiness such as laughter. When such signals are rare in the environment, it is likely that infants will remain in the frozen state for longer than

is healthy. In such circumstances, infants would be protected by the application of a method that induces happiness during early pregnancy. Such a method is described in an application for a patent that I submitted to the UK Patent Office in 2003 (see Appendix).

3. Why is the age of onset in the late teens?

Schizophrenia tends to make its first appearance in the late teens (see section four below). This is a well known phenomenon, yet nobody appears to be able to explain why it should be so.

My theory states that the major symptoms of schizophrenia occur as the young adult attempts to reconfigure its brain for the challenges of parenthood. The main challenge of parenthood is to be an emotional teacher rather than simply an emotional learner. When this reconfiguration is impeded by confusing childhood experience, schizophrenia is the result. See section 5 below.

4. Why are schizophrenics more likely to be born in the Spring?

There is a so-called "seasonality effect" in both schizophrenia and psychopathy. For those born in springtime (March-May in the northern hemisphere, September-November in the southern hemisphere) there is an increase of about 10 per cent in the rate of occurrence of these disorders. Yet nobody appears to be able to explain why this is.

My theory states that schizophrenics are more likely to be born in the Spring because mothers will be more likely to have been in less happy emotional state during the winter (see page 4 of my patent application).

5. What causes the precursors of psychotic symptoms observed during infancy and early childhood in those at risk for schizophrenia, such as delays in the acquisition of gross motor skills and language?

Research shows that infants and young children at risk of developing schizophrenia show certain developmental delays. They take longer to acquire gross motor skills and language, and are also slower in acquiring fine motor skills. These delays can be regarded as precursors of the full-blown psychotic symptoms that mark the onset of the disorder later on in life. We need an explanation for these delays.

My theory states that the developmental delays evident in infants at risk of developing schizophrenia are due to loss of REM sleep in early infancy, and the consequent neurlogical damage that follows from this.

Thoughts on preventing schizophrenia (2014)

Until recently most experts assumed that humans communicate emotional states primarily through visual means (facial expressions, bodily postures, gestures, etc.). In some cases auditory cues also play a supplementary role in emotional communication, but the other senses are assumed to play no role whatsoever.

Evidence is now emerging, however, that humans may also communicate emotional states via chemical signals. Experimental work has demonstrated that fear chemosignals emitted by the

sender can generate a fearful facial expression and sensory acquisition (increased sniff magnitude and eye scanning) in the receiver. The response is specific to the signal; disgust chemosignals, for example, evoke a disgusted facial expression and sensory rejection (decreased sniff magnitude, target-detection sensitivity, and eye scanning). Researchers hypothesize that normal communication of emotion involves the simultaneous activation of visual, auditory, and olfactory channels, establishing a multilevel correspondence between sender and receiver.

These results imply the existence of neural pathways that link the olfactory system directly to the major parts of the limbic system including the amygdala. It is very likely, therefore, that the development of the limbic system during early childhood may be sensitive to olfactory input, and perhaps also to the appropriate conjunction of visual and olfactory input. Indeed, this conjunction may turn out to be the missing piece of the puzzle concerning the aetiology of schizophrenia.

So far, my theory of schizophrenia has focused entirely on visual input. In particular, I have argued that the origins of schizophrenia lie in the misrecognition of parental expressions of emotion by the infant. The caregiver may produce a facial expression of fear, but the infant misreads this expression as one of anger. This is fine as far as it goes, but it does not explain why certain infants misread facial expressions, while others interpret them accurately. The new research on the communication of emotion via chemosignals may supply such an explanation.

I hypothesize that the proper development of the fear system requires exposure to fear chemosignals during a certain critical period in infancy. This would have occurred naturally in the ancestral environment, since caregivers were often afraid (of predators, for example) in the presence of their infants. The natural response of the infant under such circumstances would have been to freeze. In the modern environment, however, it is much less common for caregivers to be frightened in the presence of their infants, and as a result fewer infants are exposed to fear chemosignals during the critical period. The neural systems responsible for fear consequently fail to develop correctly in these infants. They may freeze during the critical period, but not in response to a fearful facial expressions; instead, they may startle in response to an angry facial expression, and this causes their fear system to wire up the wrong way. From then on, the infant will tend to mistake other negative facial expressions, such as expressions of anger, sadness, or disgust, as expressions of fear. These are the infants who are at risk of developing schizophrenia in early adulthood. Infants who do not mistakenly see fear in other facial expressions during the critical period, either because their fear system has been primed by exposure to fear chemosignals, or because they are born blind, are not at risk of developing schizophrenia.

Schizophrenia is, according to my theory, a pathology of the fear system. The increasing safety of human environments over the past centuries has led to a decline in the frequency in which infants are exposed to fear chemosignals. This has in turn led the freezing response to atrophy, with the result that only a minority of infants inherit the full complement of genes for a fully functioning freeze response. It is in these genes that we should look for genetic associations with schizophrenia. In other words, it is in the genes responsible for the development of the freezing response that we will find any genes that increase the risk of developing schizophrenia.

One important implication of my hypothesis is that it suggests a way to prevent the occurrence of schizophrenia. If schizophrenia is due to the infant mistaking other facial expressions of emotion for the expression of fear, and if this is due to the lack of exposure to the fear

chemosignal during a critical period, then it follows that schizophrenia will not develop under the following conditions:

(1) If the infant is blind, it will not perceive any facial expressions of emotion on the part of the caregiver. It will therefore not misperceive any such facial expressions. It follows that congenital blindness should prevent the development of schizophrenia.

(2) If the infant is artificially exposed to the fear pheromone during some critical period (which we estimate to be the first three months of life), then its ability to recognize facial expressions of fear should be primed in advance of visual acuity and it will be protected against schizophrenia.

(3) If the infant is born without the full complement of genes required for a fully functioning freeze response, it will never freeze in response to any facial expression of emotion, whether the appropriate one (fear) or not (eg. anger, fear). It will therefore not come to associate inappropriate facial expressions with the freeze response, and will not therefore be at risk of developing schizophrenia.

There is some evidence that (1) is indeed the case; people born blind do not appear to suffer from schizophrenia. Experiments are needed to test predictions (2) and (3).

Acknowledgements

2000: I am grateful to Dr Dylan Evans for helping me to put my ideas down clearly. Dylan conducted regular interviews with me and translated my verbal musings into written text. This booklet would not have been possible without his assistance. I am also grateful to Professor Nicholas Humphrey for providing comments on a previous draft of this booklet. Since 1998, Nick has been working on a three-year research project on psychopathology at the London School of Economics that I funded. His enthusiasm and dedication has been exemplary. I am also indebted two psychiatrists, Dr Bill McIllroy and Dr Elizabeth Tilden, who taught me so much many years ago. Needless to say, the responsibility for any errors is entirely mine, as are the original hypotheses advanced here.

2007: Since the initial publication of this essay, I have funded some research at the Institute of Psychiatry. This research was carried out by Professor Mary Phillips, Dr Tamara Russell and Dr Catherine Herba. I am very grateful to them for their dedication. The research on eye-tracking of schizophrenic patients conducted by Dr Russell demonstrates that these patients do not look at faces in the normal way. In particular, they avoid looking at many of the important facial features. This would explain why they are not very good at recognising emotional expressions. In further research conducted in Australia, Dr Russell made schizophrenic patients aware of this deficit and taught them ways to compensate for it. This not only worked, but seemed to provide significant benefit the patients.

Document history

This essay was first published online and in print in October 2000. The "Further conclusions" were added in March 2003. The "Further clarifications" were added in 2007, along with some

minor revisions to the main body of the essay. The section on preventing schizophrenia was added in 2014.

Bibliography

Achenbach, T. and C. Howell (1989). "Are America's children's problems getting worse? A 13-year comparison." Journal of the American Academy of Child and Adolescent Psychiatry.

Banyai, E. I. (2000). On the adaptive nature of hypnosis: an evolutionary perspective. XXVII International Congress of Psychology, Stockholm, Sweden.

Baron-Cohen, S., Ed. (1997). The Maladapted Mind: Classic Readings in Evolutionary Psychopathology. Hove, Psychology Press.

Bateson, G. (1979). Mind and Nature. London, Wildwood House.

Bentall, R. (1998). Why there will never be a convincing theory of schizophrenia. From Brains to Consciousness? Essays on the New Sciences of the Mind. S. Rose. Harmondsworth, Penguin: 109-36.

Blair, R. J. (1995). "A cognitive developmental approach to morality: investigating the psychopath." Cognition 57: 1-29.

Blair, R. J. R., E. Colledge, et al. (forthcoming). "Children with psychopathic tendencies: selective impairment in processing sad and fearful expressions."

Ekman, P. (2003) Emotions Revealed. London, Weidenfeld & Nicholson.

Gallup, G. G. J. (1974). "Animal hypnosis: factual status of a fictional concept." Psychological Bulletin 81: 836-53.

Gallup, G. G. J. and J. D. Maser (1977). Catatonia: tonic immobility: evolutionary underpinnings of human catalepsy and catatonia. Psychopathology: Experimental Models. J. D. Maser and M. E. P. Seligman. San Francisco, W. H. Freeman : 334-57.

Goleman, D. (1998). Working with Emotional Intelligence. London, Bloomsbury.

Gottesman, H. (1991). "Schizophrenia Genesis". Freeman, New York.

Hafner, H., K. Maurer, et al. (1995). Onset and early course of schizophrenia. Search for the Causes of Schizophrenia: Volume III. H. Hafner and W. F. Gattaz. Berlin, Springer-Verlag.

LeDoux, J. (1998). The Emotional Brain: The Mysterious Underpinnings of Emotional Life. London, Weidenfeld & Nicholson.

Marks, I. (1987). Fears, Phobias, and Rituals: Panic, Anxiety and their Disorders. Oxford, Oxford University Press.

Miller, G. (2000). The Mating Mind: How Sexual Choice Shaped the Evolution of Human Nature. London, Heinemann.

Morris, J. S., A. Ohman, et al. (1998). "Conscious and unconscious emotional learning in the human amygdala." Nature 393(6684): 467-70.

Piccione, C., E. R. Hilgard, et al. (1989). "On the stability of measured hypnotizability over a 25 year period." Journal of Personality and Social Psychological Review 57: 289-95.

Revonsuo, A. (2000). "The reinterpretation of dreams: an evolutionary hypothesis of the function of dreaming." Behavioral and Brain Sciences 23(6).

Sass, L. A. (1999). Schizophrenia, self-consciousness, and the modern mind. Models of the Self. S. Gallagher and J. Shear. Thorverton, Imprint Academic : 319-41.

Simons, R. C. (1996). Boo! Culture, Experience, and the Startle Reflex. Oxford, Oxford University Press.