We can now sketch a fairly convincing ‘bottom up’ account of one important learning task of the brain – learning to read. Cognitive reading skills appear to depend on the quality of individuals’ low level auditory and visual sensory processing. These processes make use of the links between the visual and language areas of the cerebral cortex to piggyback on the neurological apparatus that evolved for speaking, in order to associate the visual form of words with their spoken counterparts for reading.

The eyes and attention scan each word to identify its letters and their order. The visual control of these attentional and eye movements is mainly mediated by the magnocellular subcomponent of the visual system that is specialised for timing visual events. Hence, this system can detect any unwanted motion of the eyes and thus enable the ocular motor system to correct them. Impaired development of the visual magnocellular system is associated with unsteady fixation on words during reading, hence visual confusion and slow reading progress.

Analogous processes seem to be important for hearing. Unfamiliar words are read by translating the letters into their sounds, then assembling them into the auditory form of the word which gives its meaning. The distinctions between different letter sounds are conveyed by changes in the frequency and amplitude of the acoustic speech signal. These are picked up by large auditory neurones specialised for sensing auditory transients. Hence, people with high auditory transient sensitivity find it easy to acquire phonological skill, whereas poor readers tend to have low sensitivity to these acoustic transients and end up with poor phonological skills.

Thus, both visual and auditory transient sensitivity, and hence orthographic and phonological skills, are mediated by magnocellular systems in the brain that are specialised for tracking temporal changes. Ultimately therefore, the acquisition of reading skills depends on genetic and environmental influences over the development of magnocellular neurones.

Understanding the neurobiological basis of reading problems is enabling teachers to really help children who are making slow reading progress. Armed with knowledge of how auditory and visual transient sensitivity determines the development of reading skills, and of the profile of a particular pupil in each of these areas, teachers can design programmes targeted to each individual’s strengths and weaknesses. Taking advantage of the incredible plasticity of the developing brain, we now know that cognitive weaknesses can be remediated and improved by appropriate training. Thus, our increasing understanding of the neuroscience behind cognitive processes is already beginning to benefit teachers in their classrooms, directly and practically, to help children acquire the literacy skills required in modern life. And this understanding can only accelerate in the future.
The human brain

The human brain is the largest organised structure in the Universe – an extravagant claim, but true. It contains 100,000 million \((10^{11})\) neurones and ten times that number of supporting, ‘glial’, cells. Each neurone makes on average 100,000 connections with other neurones. In other words there are 1,000 million million connections in the brain \((10^{16})\). This means that the brain can make more possible combinations of connections than there are particles in the whole Universe. So, the human brain is indeed the largest organised structure in the Universe. Yet, all these connections are fundamentally organised to perform only the 3 vital behavioural functions of all animals: finding food and water, self-preservation and procreation.

The organisation of the brain arises from interaction of a genetic blueprint with environmental influences, not only during development, but throughout all the experiences of life. The most basic biochemical processes, such as control of protein synthesis, are dominated by the genes, but higher cognitive functions such as speech, language, visuospatial ability and intelligence result from a roughly equal mix of genetic and intrauterine prenatal and postnatal environmental influences throughout life. The most important environmental contributions to brain development are the mother’s and infant’s nutrition, home environment and later education.

In the last 20 years neuroscience has advanced so rapidly that, despite some doubting Thomases, we are beginning to see examples where neuroscience discoveries have really changed educational practice, though not always based on a complete understanding of the science. In particular we are now beginning to see how individual differences in higher cognitive functions arise, so that we can start to develop educational programmes that target each child’s strengths and weaknesses on an individual basis. Recent advances in understanding the biological basis of memory and learning and how they impact on higher cognitive functions are slowly leading to individually targeted ways of improving them.

It must be admitted, however, that there are not many examples of this enlightened approach yet. Too often, educationalists assume that one size fits all, and individual learning styles are not properly catered for. And there is already a backlash from people who pedantically pick on the over-simplifications in programmes like ‘Brain Gym’, without acknowledging that, despite their excesses, they are basically founded on recent advances in understanding the neurobiology of learning.

Unfortunately, although it is clear that there are clear structural brain differences between individuals, these do not automatically tell us what functional differences they represent, nor lead in any simple way to strategies that will exploit them positively. Moreover, the stage at which they develop does not necessarily tell us when is the optimum time to introduce particular kinds of education for different individuals. Likewise, knowing the biochemical mechanisms that alter the strength of nerve connections that underlies learning does not tell us directly how to help children learn.

Nevertheless, I believe there are grounds for optimism. The new techniques that enable us to look at functional differences between individuals already offer the hope that we should soon be able to adapt our teaching methods to match the learning needs of individual children. In this chapter, therefore, I will briefly describe the structural and functional changes that occur in the brain during development, and how understanding how these vary between individuals will enable us to target educational approaches to individual learning needs. Finally, I will show, using the particular example of reading, how understanding individuals’ different brain strengths and weaknesses is already being exploited to improve the teaching of reading. This shows how potentially we will be able to target our educational methods to individual children’s functional brain differences, hence match them to individuals’ different learning needs.

Brain information processing

Our \(10^{11}\) nerve cells gather information from sensory receptors and from other neurones through their highly branching ‘dendrites’. These signals are then integrated in the nerve cell body, and the outcome of
their processing is delivered to the next neurone in the chain via their elongated ‘axons’. Those from the toes are as long as 2 metres, and the messages are conveyed electrically by trains of 0.1 volt action potentials, also known as impulses or ‘spikes’. These travel along the axon at speeds of 1–100 metres per second. So they’re not that fast compared with electricity, which travels along a wire at the speed of light (nearly 300 million metres per second!); pain signals can take two seconds to get from the toe to the brain.

At the axon terminal, transmission to the next nerve cell is no longer electrical, but chemical at ‘synapses’. Each action potential causes the release of a chemical transmitter that diffuses across the synaptic cleft, binds to a receptor molecule on the next neurone and causes the electrical activity of that neurone to change. Either it is excited, making it more likely to fire impulses and continue the message onwards, or it is inhibited, making it less likely. Thus, the interaction of excitatory and inhibitory inputs to each nerve cell means that each acts as a little computer, weighing up the strength of its various inputs to decide whether the message should be passed on.

Thus, the strength of individual synapses is of crucial significance. From the point of view of learning, their most important feature is that this strength can be changed. The amount of transmitter released can be increased or decreased, and the number of receptors on the next neurone can also be increased or decreased. This is called synaptic modulation and it underlies ‘neural plasticity’.

Such synaptic modulation involves two basic processes, known as long-term potentiation (LTP) and long-term depression (LTD). LTP facilitates synapses between two neurones. If one axon firing is associated with the next neurone discharging an impulse, then subsequently that synapse is slightly strengthened (‘cells that fire together, wire together’), whereas, if it does not cause an impulse, that synapse is slightly weakened. Millions of these interactions modulate networks to mediate the acquisition of skills and to represent events and knowledge stored in memory. LTD is equally important to remove unwanted and unuseful associations.

LTP and LTD have been most studied in two ‘model’ systems in animals. The hippocampus is the brain structure that plays a crucial role in memory of events and laying down knowledge, ‘episodic’ or associative memory. Evolutionarily it developed in close association with our chemical senses of taste and smell for remembering where to find food. It is therefore relatively enlarged in food-hoarding birds and rodents and it has proved ideal for investigating LTP. Here, short-term memory of, for example, the new associations required to remember where a food cache is hidden is mediated by ‘theta’ oscillations in the hippocampal network. If these are judged worthy of retention because they led to a positive, or on the contrary, a dangerous outcome, they are then exported so that synaptic strengths are strengthened accordingly in the cortical areas that were involved in the initial experience in order to represent it in long-term memory. Such new associations are often facilitated by the generation of wholly new neurones, though at a much slower pace than during early development of the brain.

LTD has been mainly studied in the cerebellum. The main function of this ‘little brain’, which is situated behind and below the cerebral hemispheres, is to mediate another important form of learning, ‘procedural’ learning, which is the acquisition of automatic skills for the smooth coordination and optimisation of movement. If a movement is poorly coordinated, the cerebellar synapses that mediated the defective movement are weakened by LTD, but strengthened if the movement was coordinated and skilful. Both LTP and LTD occur throughout the whole brain, however, particularly in the association areas of the cerebral cortex, which are the areas that mediate the cognitive functions that interest educationalists most.

Synapses can not only change their strength, but they can also switch to new positions; in addition, new branches can sprout from dendrites and axons as well. During learning, synaptic spines on neuronal processes seem to search around randomly to find useful contacts. Once found, these consolidate and stabilise, embodying the learnt material.

No one synapse represents a single memory, ‘an engram’, therefore. Each memory is represented by slight alterations to literally millions of synapses linking a whole network of hundreds of thousands of nerve
cells. For example, written words are represented over a very extensive network of cortical areas distributed over the whole of the left cerebral hemisphere and involving the right hemisphere as well. Not only are representations of its sound, its visual written form and its meaning included in the network, but importantly also how you would speak the word, together with how you would act it, are all involved. The network includes the planum temporale in the left temporal lobe, the fusiform gyrus in the left occipital lobe, the angular and supramarginal gyri in the left posterior parietal lobe, together with the motor speech area in the inferior frontal gyrus and even the motor cortex in the precentral gyrus of the frontal lobe. All these areas and several million synapses are therefore involved in representing just one word, and these overlap extensively with the representation of other words.

**Development**

The most rapid period of brain development occurs *in utero* from the sixth week to the sixth month of pregnancy. During this time, a million million (10^{12}) new neurones are generated; an astonishing 250,000 new neurones are added every minute! However, only 10 per cent (still 10^{11}) of these neurones survive to adulthood. The other 90 per cent are programmed to self destruct (‘apoptose’) if they fail in a lethal competition with other neurones to make useful functional connections. This is an example of another general principle of brain function that persists throughout life: ‘use it or lose it’. The main function of each neurone is to communicate with other neurones via synapses made with its dendrites and axons, either close by or at greater distances. But, unless a connection or contact turns out to serve a functional purpose in representing the outside world or organising movements, it will lose out in the cut-throat competition to survive; and it will be removed.

Thus, the connections that grow between neurones only survive if they perform a useful service. Genetic control only specifies the general ground plan of the brain and, even at this early stage, which particular neurones survive are selected by current environmental influences according to how effective each neurone is in communicating with others. If the connecting synapses between neurones give rise to correlated electrical activity between them, this enables them to switch on the synthesis of essential growth factors. These rescue the neurone from self-destruction, consolidate and strengthen existing synapses and even promote sprouting of new axon terminals to make more synapses with neighbouring neurones.

The other great principle of brain development is therefore: ‘neurones that fire together, wire together’. Only the most successful neurones and synapses that interconnect usefully survive this lethal competition.

**Postnatal growth**

At birth the brain contains almost all the neurones that it ever will. At this time the baby’s brain is only one-quarter the size of an adult’s. Even giving birth to a brain that size stresses the female pelvis greatly. After birth, the brain grows another four-fold in size, not by producing more neurones, but by further division of supporting ‘glial’ cells and also, crucially, in the first few years by increasing the number of connections between neurones. This huge increase in connectivity is the main reason why our brains are so much more powerful than those of our closest primate relatives, the chimpanzees. Their brains are only 70 per cent smaller in size than ours and they have almost the same number of neurones; but they only have about a tenth of the number of interconnecting synapses that we enjoy.

The peak number of these connections is reached between one and three years after birth. It has therefore been suggested that it is during this very early period when these connections are forming that education should be most concentrated. Some have even wondered whether there is any point at all in education after this time! This growth in connections in infancy is also adduced to explain the ‘Mozart effect’, that playing Mozart to baby in the cradle increases her intelligence.
But these ideas are based on a misunderstanding. The crucial period for development of the higher functions of the brain occurs, not during, but after this period. In the visual system, for example, 50 per cent of the connections formed in the first three years of life are pruned out over the next ten years. This process remodels the brain in response to external stimuli. It follows the same general principles of ‘using it or losing it’ and ‘firing together, wiring together’ as in utero, but now the correlated activity between neurones is generated by external stimuli. Synapses are only retained and strengthened if they achieve correlated activity of both pre- and post-synaptic neurones. And this will only occur if they respond to visual features that are seen in the outside world sufficiently often, and are therefore ecologically relevant. If a feature is seen often enough, this will generate enough correlated electrical activity to allow the neurones to synthesise synapse-saving growth factors. Thus synapses and connections that correspond to salient external features are reinforced, whereas those that do not, regress. Thus the network comes to ‘represent’ in the brain visual features that are encountered sufficiently often in the outside world. This mechanism explains why it is so important to provide lots of visually stimulating objects in the environment. It was shown 30 years ago that the visual cortex of rats reared in a plain cage without any interesting objects retained 50 per cent less synapses than their genetically identical litter mates reared in the same type of cage, but enriched with other rats and many pipes, tubes, boxes and hoops to play in and explore.

Note that this process of selective pruning of connections continues throughout life; not just until puberty, although the highest rate of pruning does occur early in puberty. No wonder puberty is so stressful for all concerned! But what this long period means is that exposure to environmental influences, such as education, is going to strongly influence which synapses are pruned. Thus, all the years of education have important effects on the very structure of the brain. But we now know that restructuring the architecture of synapses in the brain in this way continues throughout life.

**Learning and memory**

Synaptic remodelling occurs throughout life because the mechanism by which we learn and lay down memories is no different in principle from the developmental processes that have been going on in childhood. There is no clear biological distinction between how the brain develops and how learning experiences throughout later life modify its structure. Whereas development of the brain in utero mainly involves culling of neurones, and in childhood it mainly involves pruning of excess connections, the laying down of memories later mainly involves adjusting the strength and numbers of the synapses that have already been formed.

**Active learning**

One important lesson that has emerged from study of these networks is that they always involve motor as well as sensory areas of the cortex. The rats mentioned earlier that were brought up in an enriched environment, and grew and retained more synapses in their visual cortex, not only explored the tubes, pipes and boxes with their eyes, but also they ran amongst them and poked their noses into them. In a classic experiment, pairs of kittens were reared in such a way as to compare the effects of active and passive visual experience. The passive kitten was confined to a basket hanging from the end of a pole, so that it could not move much, but it could see everything around it. The other kitten was harnessed to the other end of the pole so that it had almost exactly the same visual experiences, but it actively pulled the passive one round in a circle, so that active movements of his head and body could be integrated with what his eyes were seeing. After a few weeks, the visual skills of the pair were compared. Despite almost identical visual experiences the performance of the passive kitten was very severely impaired in a variety of visual tasks compared with that of the active one.
These results transfer directly to the teaching situation. Children made to sit and listen passively to teacher without active involvement in teaching themselves by play have consistently been shown to lag behind children who are encouraged to actively find things out for themselves. Learning by doing turns out to be a much more effective teaching technique than learning by passive listening or viewing and this can be traced directly back to the active participation of motor areas in the memory networks of the cerebral cortex. These areas will be much more effectively activated by acting out the motor component of the memory than by passive listening or viewing. This is because the ‘semantic networks’ that represent our knowledge and memories comprise not only the sensory inputs that initiated them, but also the motor outputs they lead to. If I hear the word ‘foot’, not only are my verbal auditory and visual lexicons activated in my left hemisphere, but also to some extent the area in the motor cortex that controls actually moving the foot.

**Brain plasticity**

The combination of growth and alteration of neural synaptic connections with modulation of their strength underlies what is called neural ‘plasticity’. This causes environmental influences and experiences to be captured and represented in the detailed, microscopic, ‘ultra’, structure of the brain. Thus, despite having the same genes and usually very similar upbringing, the brains of adult identical twins can turn out very differently. Your brain is the sum of not only your genetic rough draft, but also all your perceptions, experiences, thoughts, feelings and actions throughout your whole life. It is thoroughly unique.

With modern technology, these plastic processes can be demonstrated in individuals. For example, London taxi drivers have to learn ‘The Knowledge’, i.e. where all the streets in London are. This kind of learning calls particularly on the associative powers of the right hippocampus. When the size of the hippocampus was compared between taxi drivers and drivers with a similar background but without The Knowledge, the taxi drivers were found to have larger ones on the right. Of course, they’re very keen to tell you this!

One can even show very rapid changes in the representations in the cerebral cortex. A group of students who had never learnt to play the piano were taught to practise five finger exercises on the piano for 20 minutes a day for a week. A similar group were asked to look at the piano and waggle their fingers randomly for the same time. The researchers measured the size of the representation of each person’s finger movements in their motor cortex. They found that in those who had done the 5-finger exercises the representation had tripled in size, whereas the controls’ did not. In fact, in the practisers their representation had begun to increase within a few hours of starting the practice. What was even more remarkable was that another group of students were asked not to practise the exercises, but to carefully imagine they were doing them for the same 20 minutes per day. Their finger representations also increased dramatically in size. Clearly, thinking about doing something is almost as potent for increasing skill as doing it.

Another most striking example of plasticity is demonstrated by the responses of the brain to injury. Immediately after a stroke, which is caused by blockage of the blood supply to a part of the brain, this area is completely destroyed. For example, a patient may be immediately paralysed and incapable of speech. But, very often, in the weeks and months that follow s/he will recover almost all these functions. Modern functional imaging techniques have shown that this occurs because neighbouring and connected parts of the brain are able to take over the functions of the damaged area. This return of function is mediated by unmasking and strengthening latent, very weak, pre-existing synaptic connections and also by the sprouting of new connections to surviving structures. This plasticity in the face of injury makes use of the same underlying mechanisms that mediate the learning of new memories and it demonstrates that they are still potentially powerful even in adults. Who knows what talents we could unleash if we could learn how to exploit them properly for educational purposes?
Neuroscience in the classroom?

Studying the biological basis of memory has generated several Nobel prizes and we now understand the underlying mechanisms reasonably well. The details need not concern us here. But, has this helped education? has our hard-won understanding contributed materially to educational practice? The answer is, not much yet. So far, most of our knowledge is too basic to be transferred directly to the classroom because the cognitive functions that educationalists are interested in are so many levels higher than what goes on at each synapse. Thus, for example, drugs that affect these processes have far too extensive effects to be any good for selectively improving just the cognitive functions desired. Nevertheless, many people are experimenting with drugs like Modafinil and Ritalin to improve cognitive function by altering synaptic function. The improvements are modest at best, but there is no doubt that there will be significant advances in this area in the next few years.

One advance in education that all this new knowledge has provided is settlement of the old 'nature v. nurture' argument. Not only do we now know that both are equally important for the development of the brain, but also it is now clear that it is simply not possible to determine what component of personality, intelligence or athletic talent is genetically innate and what is dependent upon upbringing and environment. Theoretically, it is still possible to conceive them as being separable and to statistically apportion their contribution to individual differences, for example by means of twin studies. But it is quite clear that they are so inextricably interlinked when one gets to the level of cognitive differences that it is utterly impossible to separate their contributions to any of the mechanisms that are of interest to education, e.g. how people differ in personality, intelligence, etc.

This conclusion is heartening because it supports neither the right-wing view that justifies the status quo by claiming that all individual differences are hereditary, preordained and immutable, nor the left-wing view that genetics plays no part in individual differences, so that they can all be eliminated by sufficiently radical teaching changes. The strong influence of environment means that genetics need not consign people to an immutable fate, as many people seem to fear. Properly targeted education and remediation ought to be able to compensate for much hereditary weakness, as I shall show later in the case of reading problems.

Structural changes in the brain continue in response to experience throughout life, though at a diminishing rate after puberty. What this implies therefore, is that education really does matter a great deal, because it actually helps to determine the structure of the pupil's brain. Each thing a child learns alters his brain just a little bit. Therefore we should worry even more about the one in five people who reach adulthood and say that they gained nothing from their education. We should think hard about this indictment of modern schools. Why do current educational practices serve so many people so poorly?

Individual differences

Another thing that neuroscience has contributed to education is to emphasise how important the differences between individual 'normal' brains are. We always knew this at a psychological and personality level. Now we know it at the level of brain structure and function as well. Modern magnetic resonance imaging has shown, for example, that the anatomical structure of people's brains differs from each other, even more than the differences between their faces. Even very basic structures in the brain, such as the brain fissure where the primary visual cortex is situated, can differ from individual to individual in its position with respect to the centre of the back of the skull by as much as 1cm in either direction.

Functional magnetic resonance imaging has made this even clearer, and shown that experience changes brain structure as well. For example, if you learn two languages simultaneously as a child, the area in the temporal cortex that is activated for one language is indistinguishable from that employed by the other. But, if you learn one in childhood, but the other, just as fluently, later in life, the two activate clearly different, though adjacent, regions.
Large differences can also be seen when testing people’s sensory sensitivities. For example, although people’s visual acuity varies only over a narrow three-fold range, their sensitivity to a slightly higher level function, visual motion, varies over a ten-fold range. These individual differences then impact on much higher cognitive levels, for example, their reading or visual spatial abilities, as we shall see.

Another much more worrying example is that the development of the frontal lobe, which is where our social behaviour is controlled, is irretrievably damaged by adverse circumstances early in life. Rumanian orphans, brought up with no love, little care and appalling nutrition had impaired development of the brain, particularly in the frontal lobes, and this is associated with lasting difficulties with social adjustment and behaviour. Likewise, prisoners in gaol for violent offences have been found to have less prefrontal cortical activity in social situations, and this can very often be correlated with the nutritional, physical and emotional deprivation they suffered in childhood. Their callous unemotional treatment of others is a direct consequence of their childhood physical and emotional needs having been completely unmet, or, indeed, often met by violence.

**Hemispheric specialisation**

An important source of differences between individuals that is of potentially great relevance to education, is the degree to which the two sides of the brain are specialised to perform different functions. In 97 per cent of people, including two-thirds of left-handers, the left hemisphere is relatively much more specialised for speech and language whereas the right is more important for visuospatial analysis and emotional expression. These differences are not absolute; the right hemisphere is important for some aspects of language, such as its emotional tone, and the left hemisphere plays a part in some visuospatial functions, such as helping to determine the relative position of letters in a word.

Now we know from magnetic resonance imaging in live humans that these functional differences are associated with structural differences between the hemispheres. Most brains are twisted anticlockwise as seen from the top, so that the front of the right hemisphere protrudes further forwards than the left, whereas the back of the occipital lobe in the left hemisphere sticks out backwards more than the right. Thus, the right frontal lobe is larger than the left but the left temporal lobe is larger than the right.

**Left hemisphere language**

The twist seems to be mainly caused by the language comprehension area, known as the planum temporale, being much larger on the left. The degree to which the left planum temporale is larger on the left tends to correlate with individuals’ language abilities, suggesting that this structural difference somehow underlies different degrees of language skill. Recently it has become possible, using magnetic resonance ‘diffusion tensor’ imaging, to measure in living subjects the thickness of the nerve fibres joining different parts of the brain to one another. Their thickness tells us how rapidly they can conduct messages between them because fibre diameter determines conduction velocity. It turns out not only that the fibres are larger in the left hemisphere, but also that the diameter of these fibres in individuals correlates with that person’s language skills. The larger they are, the faster can their owner find words to describe a situation, or learn new words in a foreign language. Excitingly it has recently been discovered that successful improvement of language or literacy skills by training in children with difficulties in these areas is associated with increasing thickness of fibres in the arcuate fasciculus which links language-receiving areas in the back of the brain with speech motor areas in the front of the brain.

The situation is complicated by sex differences. Females tend to have less marked hemispheric specialisation, with more cross-talk between the left and right temporal lobes. This explains why females have, on average, superior language skills to males and why they are less likely to become aphasic (losing language skills) after damage to the left hemisphere. But it means that the correlation between language skills and the
ratio of left to right planum temporale size and left-sided fibre diameter is much weaker in females than in males.

**Right hemisphere**

As mentioned earlier, the right hemisphere does play some parts in language. It is now clear that important non-verbal communication aspects are the responsibility of the right side. The right hemisphere seems to pick up the emotional valence of a sentence by analysing not the detailed phonology of its constituent words, which is the job of the left hemisphere, but its overall intonational shape and prosody. This function of sensing emotional signals is not confined to language, but extends also to sensitivity to the emotional content of music, facial expressions and even colours and odours.

The emotional skills of the right hemisphere derive from fundamental differences in its structure and operation that are not yet fully understood. It seems to be adapted in some way to capture holistic, rather than sequential, detail. This explains its visuospatial dominance. It provides us with a view not of the fine ordering of visual detail, but of the overall layout of a whole visual scene or of a piece of music, together with the relationships of large chunks with each other. But this aspect of hemispheric specialisation has been far less studied than language.

One must not think of hemispheric specialisation as being absolute. Both hemispheres play important parts in every kind of sensorimotor processing. But the left hemisphere contributes sequencing of fine detail in time and space. This is most suited to communication by ordered gestures, speech and writing. In contrast the right hemisphere provides a larger holistic overview that is suited to visuospatial and emotional processing and non-verbal communication. To the extent that the degree of specialisation varies in different individuals, this is another parameter that ought to be taken into account when considering different individuals’ learning needs and designing educational programmes for each of them.

**Reading**

I now want to turn to a particular example where neuroscience has begun to elucidate a higher cognitive process and thereby has begun to impact on techniques to improve its teaching. Some 10 per cent of children have serious difficulties learning to read and are defined as ‘dyslexic’. But a further 10 per cent barely learn to read because they receive so little support either at home or at school. So, 20 per cent of young people leave school functionally illiterate (Moser report, 1999). These are the one in five adults who say they gained almost nothing from their 10 years of schooling. This illiteracy has far-reaching effects; a child’s early loss of self-esteem leads to heart-rending misery and despair, or to frustration, anger and violence. A figure of 75 per cent of imprisoned criminals are illiterate. Failure to learn to read is thus a major cause of psychological, social and economic problems, and it is an indictment of our educational systems that it remains so common.

Measures to improve our understanding of the reading process and how to improve it should therefore receive much greater support than they currently enjoy. I study eye movement disorders, not only in dyslexics, but also in diseases such as Parkinson’s disease. Parkinson’s affects 1 per cent of people over 65, i.e. for perhaps 10 years; dyslexia causes misery to 10 per cent of children for their whole lives. Yet, I’ve always found it easier to raise money for my research on Parkinson’s disease, and it receives roughly 100 times the funds that dyslexia does.

**Orthographic and phonological analysis**

Reading is probably the most complicated skill that most of us ever acquire. It is difficult because it requires the analysis of small, visually sparsely detailed, letters and their order, their conversion into sounds, and then association with the word’s meaning, all this at the rate of two to three words a second. The process begins...
with vision. The visual system scans the print in order to put letters and words in their correct order and thus to identify them. For familiar words we recognise the whole word at once and this allows us to grasp its meaning straight away from its visual form; this is the ‘lexical/orthographic’ route for reading.

But, for unfamiliar words, and remember that most words are unfamiliar to a child learning to read, a further phonological process is required. Each letter has to be converted into the sound that it stands for; then the auditory sequence has to be blended together to give its spoken form, thence its meaning. The visual orthographic route is clearly faster, but it will only work for words that are already in our ‘sight vocabulary’. The auditory/phonological route, although slower, will work for any regular word, however unfamiliar. Clearly, the whole word route will mainly rely on visual orthographic processing, whereas the phonological route will also require accurate auditory processing.

**Visual/orthographic analysis**

Although the spatial ordering of letter features and letters in a word sounds a simple process, in reality it requires a highly complex series of visuomotor operations. This is because only the centre of gaze has sufficiently high acuity to analyse print, so that we can only identify about seven letters accurately during each fixation of the eyes. Each fixation lasts for only about one-quarter of a second. The eyes then have to move along the line of print to precisely fixate the next word, one at a time. It is therefore particularly important that the eyes move accurately to each fixation point and remain stationary there whilst the letters are being taken in. In fact, poor readers have little difficulty identifying separate letters; neither their visual acuity nor their visual memory for individual letters is usually much impaired. Instead, their main visual problem is usually ocular motor; in particular, they are poor at keeping the eyes and attention steadily fixated on each word. This unsteady control often leads the letters to appear to move around, so very characteristically dyslexics confuse letter order.

The stability of ocular motor control depends upon the quality of the control signals that are fed back from the eyes. Any unwanted movements tend to cause images to slip off the high acuity centre of the retina. Normally the visual motion generated by this image slip is fed back to the ocular motor control system very rapidly; this prompts the reversal of the eye movements that caused it and brings the eyes back on target. Hence, high sensitivity to such visual motion is essential for steady binocular fixation. We have found that many children with problems learning to read have low visual motion sensitivity and that this often results in very unsteady fixation, ‘wobbly eyes’. This can cause letters to appear to move around and cross over each other when the lines of sight of the two eyes cross over each other. These children complain that the letters seem to blur and move over each other when they are trying to read.

We have therefore been measuring the quality of individuals’ visual motion signalling systems to attempt to trace the connection between this and their reading. Visual motion is detected by a subcomponent of the visual system termed the magnocellular system: magnocellular because the nerve cells involved are exceptionally large, hence specialised for rapid responses to time transient events in the visual world. Thus, it is particularly sensitive to visual motion. The sensitivity of individuals’ visual magnocellular system can be measured relatively easily to see whether this relates to reading skill.

We have therefore made use of a technique developed for assessing motion sensitivity in the early stages of the visual system in animals, using simple stimuli that have nothing to do with reading. We display a field of bright dots moving around randomly on a dark background, so that the display looks like an un-tuned TV receiver. A proportion of the dots is then moved all together in the same direction so that they look like a cloud of snowflakes blown by the wind. By reducing the proportion that move together instead of randomly, until the subject can no longer see any coherent motion in the cloud, we can measure individuals’ sensitivity to visual motion; this indexes the basic sensitivity of their visual magnocellular, transient, system.

We can then compare this with their visual reading skill. We assess this by measuring their ability to spell irregular words like ‘yacht’ or to spell homophones – which is correct, ‘rane’ or ‘rain’? Neither of these
tasks can be solved by sounding out the words; the correct ‘orthographic’ visual form of the word has to be remembered. We found, as expected, that there is indeed a good correlation between individual subjects’ motion sensitivity measured in this way and their visual orthographic reading skills. Thus, we have been able to show that a very basic low-level sensory function, visual motion sensitivity, plays an important role in determining how well individuals can acquire the much higher-level, orthographic, cognitive skills required for reading. This has turned out to be true for everyone: children and adults, good and poor readers.

However these correlations are not huge and, of course, correlation does not prove causation. Probably visual motion sensitivity is only one of several indirect influences contributing to how well orthographic reading skills develop. We need to fill in the gaps. Therefore we have tested and confirmed that visual motion sensitivity predicts subjects’ ability to fixate steadily on non-reading small targets, that individual differences in fixation stability correlate with the ability to order non-letter symbols correctly in a sequence, and finally that symbol-ordering skill predicts orthographic ability.

But all these are correlations; they do not prove causation. The best way to prove that one thing causes another is to see whether changing one changes the other. Therefore, we have investigated whether improving eye control helps children with reading difficulties to learn to read. There are several ways of doing this depending on the child’s particular pattern of problems. But in all instances we have found that, if we can improve the steadiness with which children can fixate on the words they are trying to read, their reading improves greatly thereafter.

Indeed, we can often increase children’s reading progress dramatically. If poor readers receive no special help at all, their reading tends to regress with respect to their age, so that on average in six months their reading age will only increase by three months. But, we have been able to show that, after successful treatment designed to improve children’s binocular control, their reading leaps ahead, increasing on average 12 months in six months, so that many catch up with their peers in two years.

Thus, our intervention studies have suggested strongly that improving binocular control improves reading in many dyslexics. Since binocular stability depends on people’s visual motion sensitivity, which is an index of their visual magnocellular performance, these results support the hypothesis that visual magnocellular function helps to determine how well children can develop their orthographic reading skills through its effect on binocular stability. Likewise, it means that targeting poor binocular control can help children to avoid reading failure.

**Auditory/phonological skill**

But visual analysis alone is not sufficient for reading unfamiliar words; as we have seen, we need to use the phonological route. A child confronted with the word ‘bad’ will need to sound out the three letters separately and then blend them together in order to recognise the word. These different letter sounds, ‘phonemes’, are distinguished in the speech acoustic signal by our auditory system, by tracking the changes in frequency and amplitude that characterise them. For instance, the only difference between /b/ and /d/ is that /b/ has upward frequency shifts at the onset of the sound, whereas /d/ has downwards ones.

Since these transient cues are so important for identifying phonemes, we have a specialised auditory transient processing system, analogous to the visual one, for identifying them. Hence, this plays a crucial part in the development of phonological skill. Not only is it analogous to the visual magnocellular transient system, but also auditory transient processing seems to be mediated by a system of large magnocellular auditory neurones.

As for the visual system, we can measure subjects’ basic sensitivity to acoustic frequency and amplitude transients using much simpler stimuli than speech. In this case we play a warbling sound. The warble is produced by regularly increasing and then decreasing the frequency of a tone; this is called frequency modulation (FM). We can then reduce the frequency change, the degree of warble, until the subject can
no longer distinguish it from a pure tone. Again we have done this in good and poor readers, in both adults and children. As expected, we found that individuals’ sensitivity to warble correlates highly with their phonological ability.

We can assess children’s phonological ability by measuring the ability to make spoonerisms, such as converting ‘car park’ into ‘par cark’, and also by assessing their reading of nonsense words. Spoonerisms require the subject to break down the words into their constituent sounds and then to exchange the initial phonemes. Nonsense words like ‘tegwop’ can be read perfectly well and quickly if the subject is skilled at applying the letter/sound rules, even though they are unfamiliar. Both tasks therefore depend on phonological skill. Again, therefore, we’ve been able to show that a very basic low-level sensory process, in this case auditory FM detection, plays an important part in determining how well individuals can develop the much higher cognitive phonological skills required for reading.

**Magnocellular systems**

Thus, our research has demonstrated that visual transient processing is very important for the development of orthographic skill, whereas auditory transient sensitivity plays a large part in the development of phonological reading skill. Both these sensory processes seem to be mediated by large magnocellular neurones that are specialised for timing transients. Magnocells are found throughout the brain and they are important for transient processing, not only for vision and audition, but also in the cutaneous, muscle, attentional, memory and movement systems. Moreover, development of these neurones seems to be impaired in extremely poor readers, such as developmental dyslexics, so that many dyslexics display a plethora of other symptoms, such as inattention and incoordination, as well as having reading difficulties. When we compared individuals’ visual, auditory and motor transient performance we found that they all tended to be closely correlated. This immediately suggests that the development of magnocellular systems throughout the brain may be under some sort of common control and that their development may be impaired in poor readers.

**Genetics**

One very likely candidate for such control is genetic; for the development of magnocellular neurones throughout the brain is of course initially under genetic control. Thus, reading ability is partially genetically determined, and this explains why reading problems tend to run so strongly in families. The genes concerned are located on the 23 pairs of chromosomes situated in the nucleus of every cell in the human body. The genes direct the synthesis of proteins on which life depends. Several genes are likely to be involved in any cognitive skill as complex as reading. We have been studying families with at least one dyslexic child, looking for linkage of reading ability to particular chromosomal sites and we have been able to show that reading ability links to several sites on at least chromosomes 1, 2, 3, 6, 15 and 18.

The strongest linkage evidence so far connects reading ability with sites on the short arm of chromosome 6. This site has now been confirmed in at least seven different samples from all over the world. It was at first argued that the strongest loading was for phonological skill, therefore that the C6 site was selective for phonology. But it turned out that real word reading, orthographic skill, spelling and attention also link to the same site, and this was confirmed by principal component and multivariate analysis. These results suggest the possibility that the gene or genes concerned on C6 may affect the development of all magnocellular nerve cells throughout the nervous system, not just for reading, since these play a part in so many processes.

Two of the genes on chromosome 6 that link to reading are known as ROBO and KIAA 0319. These are now known to be involved in the control of neuronal migration very early in the development of the brain. Nerve cells destined to form the six layers of the cerebral cortex are derived from stem cells lining the cerebral ventricles. These divide to form a radial glial cell that forms a long fibre that ascends to the
surface, together with the neurones themselves. The latter then migrate up the radial glial fibre to form the correct layer in the cerebral cortex. The ROBO and KIAA mutations associated with dyslexia seem to impair the synthesis of proteins that control this migration, so that the neurones tend to migrate too far and cause outgrowths (ectopias) from the cortical surface in dyslexic brains, together with similar mismigrations in magnocellular relay structures supplying visual and auditory sensory information to the cortex. These kinds of abnormal migration are characteristic of dyslexic brains examined post mortem.

These two genes are located on chromosome 6 amongst a set of 150 genes concerned with immunological regulation, known as the Major Histocompatibility (MHC) gene complex. This is the system that controls whether your immune system recognises your own cells or attacks foreign invaders. It has recently been shown that the development of magnocellular neurones is also under the control of the MHC system. It is therefore possible that, whether neurones become specialised to become magnocells, together with the quality of their eventual temporal transient processing performance, may depend on genetic control via the MHC system. Furthermore, its impaired development in dyslexics may not only explain variation in individuals’ magnocellular/transient processing functions, but also why so many dyslexics suffer from other immune dysregulation, such as asthma, eczema and allergic reactions.

In summary, the quality of magnocellular specialisation for neural timing functions may depend on genetic regulation via the MHC system, and this in turn may ultimately determine how well each individual develops her orthographic and phonological skills for reading and spelling. Thus, the well-known fact that literacy skills are inherited may be explained in part by this genetic regulation of magnocellular neuronal development.

**Nutrition**

However, I certainly do not want to leave the impression that the genes you inherit are the only factor that determines literacy or any other cognitive function. The heritability of dyslexia is only c. 50 per cent. This means that environmental factors are equally important. Many of these one can do little about in the short term, such as the quality of home life and support given by parents and the quality of teaching. But, one factor that is often overlooked, but that is turning out to be extremely important, is the quality of children’s nutrition. Our modern diet is appalling compared with only 50 years ago: too much sugar, salt and saturated fats, and too few vitamins, minerals and essential omega 3 fatty acids. These last three are all provided by a diet high in oily fish, but 75 per cent of teenagers now eat no oily fish at all. Magnocellular neurones turn out to be extremely vulnerable to lack of these essential nutrients, because they are crucial to the rapidity of their responses. So, we have shown that supplementing the diet of children with them can greatly improve attention, memory, cognitive function, even antisocial behaviour. All teachers should become aware of how important such a seemingly simple thing as diet can be to the learning process.

**Classroom applications**

I hope that this account has given readers an insight into the way in which we learn, from a neuroscience perspective, and that you agree that it provides a plausible explanation as to why some individuals have difficulties with acquiring literacy. But, does it do what I set out to do? does this knowledge actually help teachers in the classroom? Many people fear that, if a genetic account is correct, it will consign poor readers to the literary dustbin, their fate having been dealt out to them by their genetic inheritance, and that there is nothing that neuroscientists or teachers can do to alter this.

But this is very far from the truth. One of the most encouraging developments in the last ten years of neuroscience has been the finding that the brain remains so remarkably plastic and adaptable even into adulthood. Therefore, quite apart from only explaining 50 per cent of children’s differences in reading ability, genetic regulation is not anything like the death sentence that some people fear. Elucidating its
mechanism will tell us how the brains of poor readers differ from those of good readers. Far from being immutable, this knowledge will enable teachers to assess individual children’s basic sensory skills and thus show them how to develop individual training programmes that will help children to compensate for their particular weaknesses. Armed with this knowledge about individual differences, the teacher will be able to exploit the brain’s wonderful plasticity to help each child personally. As we have seen, the right treatment applied at the right time can help a child enormously.

Ultimately therefore, performing a few simple visual and auditory transient tests that do not depend on reading should enable teachers to identify their pupils’ strengths and weaknesses. They will then be able to adapt their teaching strategies to match individual children’s profiles. For most children, a standard mix of orthographic and phonological training will suffice; but, for the identified minority, special attention can then be devoted to extra training in the auditory or visual transient skills that they lack.

**Conclusions**

Thus, I believe that now neuroscientists really are beginning to be able to help teachers understand how children learn. Although the details of recent discoveries about brain development cannot yet be applied directly in the classroom, they do lead to general principles that can and should be applied. The details of how LTP & LTD contribute to the synaptic modulation that underlies learning cannot be used directly yet. But now that we understand how individual differences arise from interaction of genetic and pre- and post-natal environmental influences on brain development, we are in a much better position to exploit environmental influences, such as education and nutrition, to obviate any adverse genetic ones. Now that we know how memory networks involve synaptic linkage of all the different visual, auditory, language and movement areas associated with an idea, it is obvious that teachers should use all these multimodal relations, particularly involving the child in active finding out for themselves, to help them understand and remember the idea. Our newer understanding of hemispheric specialisation will replace the over-simplistic idea that the left hemisphere is confined to language and the right to emotion, with a more sophisticated account of how both hemispheres interact to mediate all functions. Teachers will be able to measure individual differences in the analytical performance of the left hemisphere and the holistic performance of the right and use them to optimise their teaching.

**References**


**Further Reading**