

# **Biofutures – a selective review of biological discovery prospects and education to 2025**

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## **Introduction**

Thinking about biology and education is tricky because considering the two together involves causes and consequences at many different levels – from molecules to minds. At the moment, we understand the former much better than the latter, and we expect this to remain the case for the next decades. The difficulties, and possibilities, can be appreciated by beginning with molecules, and working up the levels of organisation.

**Keywords:** genes, biology, brain, genome

## **Genes to genomes**

The 20<sup>th</sup> century has been called the “century of the gene”. The designation is unusually neat. The idea of the gene – as a kind of elementary particle of inheritance – was defined roughly at the start of the century, and the structure of the genetic material DNA figured out almost exactly half way through.

You could say the second half of the century was then taken up with exploring the action of genes at the smallest level – a molecular biology in the real sense of the word. The molecules involved are an intricately poised set of micro-machines – or to be more up to date, nanomachines - information carriers, and links between them. They have all co-evolved in ways we are still elucidating, but their elementary workings were first puzzled out in simple bacteria. The eukaryotic (that is, nucleated) cells of more complex organisms mainly use the same mechanisms, but with many additional refinements.

In the last couple of decades of the century, it became possible to think about achieving a complete knowledge not just of how genes work in general, but of all the genes of an organism. And, astoundingly, that has now been done – beginning with the tiniest viruses and working up to the total complement of genes and DNA of the organism we are most interested in, the human genome. (Genes and DNA are not quite the same, as only a small proportion of our DNA sequence encodes working genes. The functions of all the rest are still being worked out.)

## The century of the genome?

The completion of the human genome sequence was a landmark. Three billion bases (the paired chemical units whose order preserves genetic information in the DNA double helix) are now catalogued in the databases. Among them are stretches of DNA which encode around 25,000 genes. Keep that number in mind.

However, this notable success has also proved disappointing in some respects. There were great expectations that generating the whole sequence would somehow lay out the secrets of life, and at least cure cancer and shed light on many other conditions. These were partly built up by the biologists' campaign rhetoric which was needed to get the genome project funded. They were also partly due to a slightly exaggerated idea of genes' importance which grew up during the post-DNA decades when they were such a focus for brilliantly successful research. And there was a tendency to argue that, since the last fifty years of biology have been so astonishingly productive, the next fifty will be equally spectacular. Maybe so, but the new problem of making sense of all the information in the genome, and how it is used, has occasioned a new realism, and given rise to new programmes of work<sup>1</sup>.

Simply recording the strings of text which make up the "book of life" does not reveal all the keys to how it is read. To change metaphors, the gene catalogue is a parts list, not an assembly guide. But even that is too static. Genomes, and even to some extent genes, are dynamic entities. The text is extensively annotated, with chemical tags joined directly to the DNA and with more ephemeral signal molecules which embrace and release particular genetic sequences and affect their activity. Most, perhaps all, human genes which code for proteins, it turns out, produce more than one product, according to circumstance. Many genes, and many portions of what used to be dismissively termed "junk" DNA, regulate expression of other genes<sup>2</sup>. Considering the genome, in short, reveals new layers of complexity.

The outcome is a recognition that we are entering a new era of "post-genomic" biology, but what that means is more clearly defined in terms of what it is not than what it will actually be like. It will depart from the largely reductionist, and in their time very fruitful, assumptions which underpinned the early days of molecular genetics and the planning of the genome projects. Genes seen as unchanging, crystalline information stores, which can be unplugged and swapped around like Lego bricks, are a less dominant image now. Defined DNA sequences are still a source of fundamental information – and the key to inheritance. But the use of that information is seen in context, and feeds into complex, shifting circuits and networks. Our ability to extract that information is now impressive. Making sense of it is harder<sup>3</sup>.

## Genome to cell

There seems general agreement that post-genomic biology will be systems biology. Again, it is unclear just what the term will mean. It describes an aim: to understand an organism as a total system, or a system of systems<sup>4</sup>. The smallest system is thus a single cell. Sometimes, in bacteria or yeast, the cell is an organism in its own right. New

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<sup>1</sup> For a historically expansive view of this transition, see Woese, 2004

<sup>2</sup> For recent (but, inevitably, already out of date) commentaries, see Pearson, 2004; Amaral, 2008.

<sup>3</sup> For one view of the way forward, from a leading cell biologist and research strategist, see Nurse, 2008.

<sup>4</sup> See Westerhoff, 2004; Endy, 2005, and, more comprehensively, Academy of Medical Sciences, 2007.

ways are emerging which take the measure of the entire cellular system. New coinages go with them. After the genome comes the transcriptome, the inventory of all the bits of DNA which are being read at any moment and used to make the message carrying nucleic acid RNA. Then there is the proteome, the catalogue of all the different proteins being made, and – more comprehensively still – the metabolome, the complete list of all the chemicals the cell is using. The most comprehensive of all would be the physiome, which includes all the above<sup>5</sup>.

The next level is to sketch the circuit diagrams which link all these. Again, the circuits, or networks, may be genetic – sets of genes, which influence each other – or metabolic – chemical pathways linked by stepwise synthesis or degradation, controlled by enzyme proteins which are themselves subject to regulation. In between are cellular signalling chemicals, usually small molecules, made under genetic control but also responding to external stimuli. One approach to systems biology is to build computer models which bring together everything which is known about these kinds of connections, and which reproduce key features and responses of actual cells. From this point of view, yeast is the best-modelled organism so far.

Along with this effort there is also much work on so-called synthetic biology. Like systems biology, this is as much an aspiration as a reality. In one sense it is an extension of existing genetic engineering, now a well-developed set of techniques in most organisms. Although genes are not just simple plug-in modules, they can often be treated as such in simple cases – and with skilled use of suitable enzymes they can be unplugged and, with suitable modifications, plugged into new locations. They may even work in completely different contexts. Thus, in a recent feat which has been used as an emblem of one kind of synthetic biology, the complete set of genes needed to make a precursor of the plant-derived substance artemisinin, an anti-malarial drug, has been stitched into a strain of yeast, which duly manufactures the alien chemical<sup>6</sup>.

As well as moving around existing genes, other synthetic biology efforts involve synthesising new ones, designing parts of possible biological systems which would work to produce a desired result, and trying to define the “minimal” genome – perhaps for an organism which could then support an additional suite of artificial genes. At the moment, this is fairly low-level, if fascinating, stuff, and a long way from building organisms to order. It does presage enormous possibilities – not least a culture of open-source biotech and biohacking. In the medium-term, while one may expect to see some genetically-engineered pets making headlines, the main action will be in bacteria. The industrial and technical potential, for pharmaceuticals, fuels, and, alarming to some, weapons, is impressive. It is unlikely, though, that any of this will be directly applicable to humans in the next few decades, so it is not discussed further here.

## Cell to organism

The workhorse of systems biology and, on occasion, synthetic biology – yeast - is at least one of the more complex of the two main types of cell. But complex organisms, of course, are enormous assemblies of cells, which can give rise to many more interactions. A grown human has at least ten trillion cells, of 200 different types. In various combinations they build all the different tissues and organs. More ambitious systems modellers are trying to tackle individual organs – the heart, say, or the liver. This involves emergent properties of collections of cells, like regulation of heartbeat, which is achieved through a subtle combination of electrical and chemical controls. Systems

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<sup>5</sup> See Hunter et al, 2002, for the original outline of the physiome project.

<sup>6</sup> As described in Keasling et al, 2006.

biology started out in the 1960s with mathematical modelling of cardiac cells. Work to integrate understanding of cardiac muscle rhythm, from genetic influences to the electrophysiology of the whole organ, is now far advanced, but not yet complete. And modelling an entire organism, whose tissues and organs influence one another – via hormone action, for example – is another level of difficulty altogether.

## **And finally ... the brain**

Modelling an entire organism would include the nervous system. But some nervous systems are more elaborate than others. The most complex, and most versatile is the one which centres on a human brain. That versatility arises, somehow, from the way the brain's 100 billion neurons are selected during development – during which many neurons die – and, even more, from how the connections *between* them are selected. No-one knows how many neural connections there are in a human brain, but each neuron has between 1,000 and 10,000 links (synapses) to other neurons. Do the maths, and that means an adult human brain may have 1,000,000,000,000,000 intercellular connections. Among other things, this means that ideas about genes determining much about the details of individual brains are implausible. A few tens of thousands of genes can shape the overall structure of neurons, their patterns of development, and the ways they find their way to the right location in the growing brain and decide which other neurons to build connections with. But the scope for hard wiring is a lot smaller than would be needed for much in the way of direct links between genes and complex behaviours.

How such behaviour actually emerges is unlikely to be elucidated in detail any time soon. Computer models can now simulate networks of a few thousand neurons. But study of real neural circuits in the lab still most often focuses on little bits of the invertebrate neural system which have a few dozen neurons. Understanding how the whole brain develops in any kind of detail is thus a long way off. Although neuroscience has made many intriguing advances in recent decades, and many startling new techniques are adding to the store of useful observations about how brains work, we understand rather little about most higher brain functions.

## **Biology and education**

Education, the process by which a human being becomes a fully-functioning member of their culture, is obviously underpinned by our biology. Will better knowledge of biology shed light on the process? That would presumably mean understanding learning, a feature of brains. So whatever other factors in our biology may act on the path to learning – whether genetic, hormonal, metabolic, even dietary – they have their effect in the brain. Whatever the ultimate cause, if there is a single cause, the effects of any of these factors will involve all the levels from the molecular to the fully integrated nervous system.

This suggests caution about linking biology to education. But it need not mean that, because neither the workings of the brain nor all the links between, say, genes and brains are understood, that near future research will not be relevant to education. It does mean that it is more likely to yield hints, and possibilities for indirect intervention, than well worked out paths to improvement. It is not difficult to affect the brain. The tricky thing is to affect it in the way, and only the way you want. We already have a collection of somewhat blunt instruments. Some, like ECT, are not too distant from trying to bring a radio back on station by thumping it. Others, like a double espresso, are perhaps more akin to trying to improve the performance of your hard drive by dousing it in WD40. And of course there are a range of surgical techniques for excision or ablation

of troublesome bits of the most complex organ. With that starting point, how hard can it be to do better?

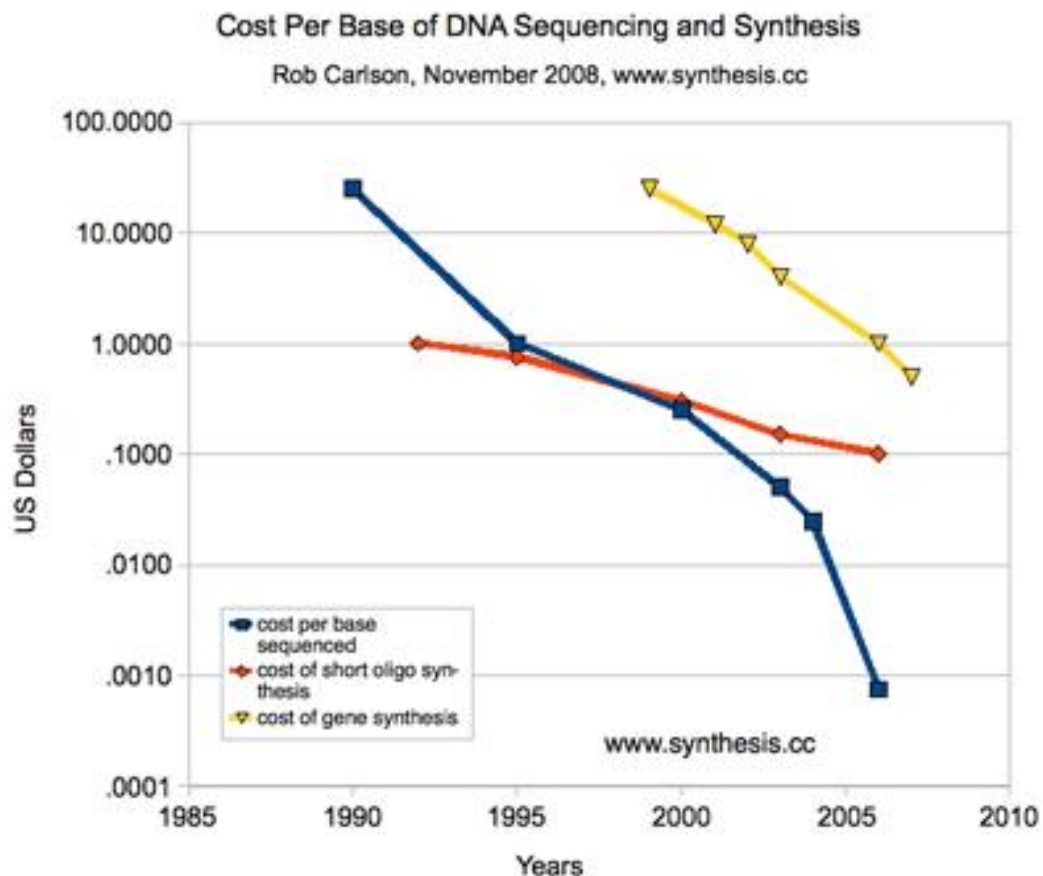
## Future prospects

### Diagnosis

Having moved up to brains, let us retrace our steps and go back to genes. The human genome project was about technology as well as science – a distinction which is becoming harder to draw in any case. Sequence one genome, sequence them all. And not just all species, but all individuals of a species, as often as you want.

That is already possible. The easy prediction is that it will become practical as well. As with Moore’s “law” which charts the exponential increase in the number of transistors in a single integrated circuit over the last five decades, DNA sequencing has been getting steadily faster and cheaper for decades. In that light, the genome project was one portion of a fairly steady trajectory. The same is true for the complementary operation, artificial DNA synthesis.

Figure 1: Cost per Base of DNA Sequencing and Synthesis



The sequencing trend is a powerful indicator that one aspect of near future biology will be diagnostic. The thousand dollar genome is now on the horizon. Another tenfold improvement would bring a complete genome sequence within the realm of routinely affordable medical tests. There are already microarray techniques on offer – using many RNA fragments of known sequence to detect the presence of particular stretches of DNA – which can test for hundreds of thousands of markers. These are usually single

nucleotide polymorphisms (SNPs, or “snips”) – locations where the genome differs in different individuals by substitution of a nucleotide base for one of the other three. They are identified and mapped by a process which is indifferent to whether the base in question is part of a functional gene, but they act as markers for a gene or genes which are nearby and are inherited together with the base location in question. Existing genome surveys are typically a mixture of checking for a large number of SNPs and a smaller number of well-characterised genetic variants. But these are technicalities. The point is that they are a definite step on the path to “personal genomics”<sup>7</sup>.

Getting useful information out of a personal genome will also depend on providing interpretative software which is usable by the owner of the genome. But the general idea is already being exploited as a sales pitch. The old image of genes as powerful determinants of outcomes is a good fit with the marketing hype of people who deal in commercial clairvoyance. The (justifiably) high-tech sheen of genetic testing is initially convincing, and goes along with a rhetoric of empowerment through knowledge. The current offer is typically to test your DNA sample (a cheek scraping or just a little saliva suffices) for a suite of gene variants held to affect probability of a range of medical conditions such as heart disease or Alzheimer’s. In the first case, knowing you have a high risk of heart disease might help you follow all the good advice about diet and exercise, or begin taking a daily dose of cholesterol-lowering drugs, before the damage is done. In the second, well, at least you or your family would have an incentive to deal with the bureaucracy required to instate an enduring power of attorney before cognitive deterioration sets in.

As this suggests, one consequence of personal genomics might be to increase the number of what are termed the “worried well”. Some of them might be children. Some might be parents whose children were identified as at risk of a particular condition. Suppose there actually was a gene variant which gave a clear indication of a high risk of schizophrenia, for instance. Knowing a child had it would create stresses which are just the kind a person at such risk ought to avoid.

Personal genomics will throw up concerns like this – if not necessarily that particular one. It may also have other effects on young people’s sense of identity. This will relate to their history and cultural affiliation as well as the possibility of particular abilities or disabilities. One child may rule themselves out of athletics because they do not have the best combination of genes for fast action muscle fibres. Another will be forbidden from doing athletics at all because they are at risk of sudden death from cardiomyopathy (children in Italy are already tested for this – non-genetically – before starting school sports).

But both children could also be reading their personal genome in ways which reinforce, or loosen tribal or other loyalties. The genome can be interpreted as a record of genealogy and ancestry as well as a harbinger of medical futures. And if the habit of comparing genomes down generations catches on, some will have questions to ponder about their actual paternity. Maternity, in general, is less open to doubt, but adoption and some kinds of assisted reproduction obscure maternal origins in ways which genome analysis can overcome. This is already possible through established techniques of DNA fingerprinting. The forecast is simply that this kind of information will be more generally in use.

The overall effects of this ready availability of total genetic information could go one of two ways. And it is difficult to judge which is more likely. One possibility is that, having heard for decades that genes are powerful, the full genome readout will be seen as data of great significance. That itself might induce activism (I’m going to beat the odds here) or fatalism (there’s nothing you can do so why try?).

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<sup>7</sup> See Feero, 2008.

On the other hand, it may become more apparent that genes, and certainly individual genes, only produce their effects in concert with many other factors. In a future which is likely to see much more information, from mobile phone records to medical tests and financial history, archived and cross-referenced in databases - a future, perhaps of ID cards, surveillance and biometrics - genomic information could come to be seen as just some more information, rather than crucial information.

Will there be more specific education implications when readouts of large subsets of genes of medical or neurological interest are widely and cheaply available? Similar advances can be anticipated in subtler analyses like which genes are actually active, perhaps in particular brain regions. Genetic analysis will advance in tandem with refinement of other techniques for registering brain structure and activity - such as ECG and functional magnetic resonance imaging (fMRI)<sup>8</sup>. Further ahead, this information will be fed into computer-simulations of brain regions, exemplified by the "Blue Brain" project in which an IBM supercomputer is running a simulation of a biologically realistic cortical column. So far, this models an assembly of 10,000 rat neurons, with their many millions of individual connections. Finding ways of incorporating molecular and genetic data to influence how the model neurons are wired up is one of the future ambitions of the designers. If that can be done, then it might be possible to use such a model to understand the effects of changes in genetic or chemical make-up of the simulated neurons, but this is a long-term prospect<sup>9</sup>.

Where might diagnostic work lead in the meantime? Around half of human genes are active in the brain. Variant forms of those genes will be associated with many different effects - and sometimes different genes will go with what looks like the same effect. Conditions like autism, or dyslexia, which are neurodevelopmental in origin, will be shown to arise in complex ways. Some genetic variants will make them more likely. Unravelling all of them is also likely to show that these, like many other conditions, are labels, which cover a more complex set of possibilities.

How much difference this will make is harder to say. Early and accurate diagnosis could assist timely remediation - one to one help with reading, say. A better understanding of brain development might allow some aspects of early education to be tailored more closely to the timing of crucial stages of neural wiring. This will be aided by the increasing richness of comparative genomics. Studying which genes have evolved most rapidly since humans and chimps began to diverge from their last common ancestor, for example, may yield further clues about changes in the brain which were crucial for the emergence of language and culture.

However, at the moment it appears unlikely that genetic analysis alone will lead to findings which have a big impact on other, more general aspects of educational practice. Studies aimed at identifying genes which influence traits which are seated in the brain - from general intelligence to anxiety or depression - have one fairly consistent result. (Some hold that studies of most traits have the same feature.) They find, not a few genes of large effect, but many genes with a small influence<sup>10</sup>. While these undoubtedly combine to produce a substantial genetic shaping of such traits, and can provide important clues about mechanisms and avenues to explore for treatment, they will make individual prognosis hard to read. Again, any individual gene effect is heavily modulated by other influences, genetic and non-genetic. This is also the reason why the discussion of "designer babies" produced by selection during IVF and similar procedures is

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<sup>8</sup> See Miller, 2006.

<sup>9</sup> Details can be found at <http://bluebrain.epfl.ch/>

<sup>10</sup> For the story for psychiatric disease see Flint, 2008. For obesity, see Thorlieffson et al, 2008. More generally, see Maher, 2008.

misleading. Gross defects can be avoided, but selecting for other traits is likely to prove elusive.

The small number of apparent exceptions to these complexities unearthed so far turn out to be less specific in their effect than first thought. A well-known example is the FOXP2 gene, which appears to exist in altered forms which disrupt aspects of spoken language ability. However, more detailed studies – including comparisons with other species – suggest that the gene product helps regulate a suite of other genes involved with fine muscular co-ordination.

The general tendency to find a large number of genes with small, additive (or occasionally synergistic) effects also reduces the chances of radical simplification of approaches to general issues relating to health and well-being which are likely to be of concern to the education system. Two important examples are obesity and depression. The latter is widely believed to be increasing among the young, a trend sometimes seen as part of the “affluenza” thesis. The evidence for an increase in adolescent depression in recent decades turns out to be poor<sup>11</sup> – and the historical incidence in previous eras is, of course, much harder to gauge. However, depression – and even more so, suicide, is a real issue for those caring for the young. At present, it appears that mixed approaches, taking into account what young people themselves have to say about mental health, may be the way forward, rather than application of any new biological insights<sup>12</sup>.

The same is probably true of obesity, which certainly does appear to be on the rise among children and young people in affluent countries, and will compromise their later health prospects. As the recent Foresight report recognised, this is a problem which will only be affected, if at all, by policies which draw on many actors, in government and elsewhere, rather than succumbing to a magic bullet<sup>13</sup>.

## Enhancement

The advance of diagnostics via genomic readouts and functional imaging will likely be gradual, and if so could be assimilated without too much trouble by existing systems for assessment – such as those for identifying special needs. But what of the prospect for more dramatic effects on education more generally? There is much speculation about souped-up technological futures in which education is transformed. Much of it rests on the predicted convergence between biology, nanotechnology, information technology and cognitive science (bio-nano-info-cogno). Some of these visions can be ruled out for the foreseeable future. The notion that a defined body of knowledge – a new language, say – could be acquired by plugging a chip into some kind of neural interface founders on the fact that we have essentially no idea what the internally stored neural coding might be, beyond saying that memory somehow depends on patterns of firing of cells connected in the right way<sup>14</sup>.

Assume then that learning will occur, if it occurs, in brains configured as they are now. As with diagnosis, complete understanding of how they work may be far off, but is not a prerequisite for thinking about how their general performance might be improved. We do know a lot about neurons, synapses, and neurotransmitters. We will know more. How might this knowledge be applied?

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<sup>11</sup> See Paykel, 2000 and Costello et al, 2006.

<sup>12</sup> For one such recent initiative in the UK, see <http://www.right-here.org.uk>

<sup>13</sup> Foresight, 2007.

<sup>14</sup> As argued in Stix, 2008.

Start with synapses. These are a pretty essential part of any theory of learning and memory. They are inter-cellular junctions which modulate signals electrochemically. At the moment, we are building up knowledge of the individual parts. The molecular biology of synaptic transmission is tolerably well understood, although, as a recent review puts it "study of the mechanisms underlying plasticity [of synaptic connection] and hence memory and learning, is proving thornier than expected."

However, there could be routes to enhanced performance which do not need a fully worked out systems biology of synapses. Genomics will lead to a full inventory of neurotransmitters molecules, their multiple receptors and the enzymes which control their transport and turnover. This offers great scope for refining blunt instruments like the current generation of psychoactive drugs. The selective serotonin reuptake inhibitors – SSRIs - widely prescribed for depression, for example, are an advance on earlier medication because they only influence one transmitter. But they still increase levels of a neurotransmitter substance for which there are at least a dozen different receptors. Tuning drug design to select particular receptors or cell sub-types would enable more specific effects.

There is a range of other molecules which are being characterised and might have desirable effects in the brain. They include neurotrophic factors, which aid growth and survival of neurons. Further ahead, gene expression can, in principle, be affected by "antisense" RNA molecules which bind to particular gene sequences and block their use. However there is a major block to their application in the brain as they are large molecules and cannot pass the so-called "blood brain barrier". Indeed, many small molecules are similarly excluded from the brain. Direct administration of new drugs to the brain, and ideally to particular brain regions presents obvious difficulties and achieving it by reasonably non-invasive means will depend on a good deal more research intended, in the first place, for therapeutic application.

Assuming that hurdle can be jumped, there are some forecasters who envisage a future which offers an array of brain-active drugs which, in the right combinations, would enhance mood, attention, sensory acuity, and memory, if not yet actual understanding. Used in the right combinations, such drugs could enhance learning by making more efficient use of study time and aiding retention. They might be suitable for use by future adults, whose portfolio careers require continual development of new skills. Another more general enhancement is relevant here. Increasing lifespan appears a fairly likely possibility arising from the improvement of genetic knowledge and cell biology. It is, in many ways, a logical consequence of the medical conditions which will be a focus of research in the West<sup>15</sup>. This need not be radical life-extension. The limit of the existing human phenotype is around 120, so simply enabling some appreciable proportion of people to reach this age without falling into decrepitude would be a big change. Some people enjoying this extra life-expectancy would no doubt want continuing education to help make use of their time. This takes us beyond our timespan, though as such a change, by definition, would take a long time to work through the population.

Meantime, demand for the relatively crude cognitive enhancers which already exist suggests that such drugs will be widely sought if they become available. There are a few reservations, though. Some of the existing drugs work, to an extent - enhancing memory, for example. But more powerful effects to come could easily be a mixed blessing. Forgetting, which can also be potentially enhanced by drug use, is as important as remembering. Effective educational use would require specific, not general memory enhancement, and would need to be short term (the more effective retention, not the actual memories). Similarly, general improvement of synaptic transmission or nerve impulses *might* speed up brain processing. But one serious theory of the origins of autism suggests that it arises from enhanced brain functions, including sensory acuity,

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<sup>15</sup> On ageing, and prospects for a "cure", see Vijg, 2008.

which leads to the person blocking out some external signals as a way of coping with unbearably strong stimulation. Attempts at enhancement of a complex system which is poorly understood can easily fall foul of the annoying fact that you *can* have too much of a good thing.

There are also likely to be trade-offs between enhancements. A well-known example is a strain of mice engineered to be more receptive to a particular neurotransmitter which, among many other roles, is involved in formation of memory. Giving adult mice more of a variant of the receptor normally prevalent in babies did appear to improve their capacity for mouse-level learning. However it also made them more sensitive to pain.

As in this example, many of the changes envisaged as achievable through drugs are potentially reachable through genetic modification. As emphasised above, ways of doing this are already known, in principle. But getting reliable results, indeed any results at all, in human patients has proved extremely difficult. The chance of any non-medical gene alteration which affects brain function being proven, and agreed to be ethically acceptable for trial in humans, by 2025, appears vanishingly small.

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