Do we learn to be good?

Whether moral sense can be innate has been questioned, says
s ananthanarayanan

IN 2007, the journal *Nature* carried a report of experiments conducted at Yale University to prove that babies as young as six-10 months old could make social judgments and choices based on the behaviour of individuals towards other people. A group in New Zealand's University of Otago has recently concluded that the experiments were flawed and they showed that the infants' preference was for "interesting events" rather than "evaluation of individuals". The Otago study has been reported in *Plos One*, an international peer-reviewed journal.

The Yale experiment was to present to babies a model of a person, a wooden figure with eyes, trying to climb over a hill. After a few attempts, the figure encountered another distinct figure who was either a "helper" or a "hinderer", who either helped the figure reach the top or pushed the figure down.

When the babies had seen this sequence a few times and had registered various roles, their "looking time", when the figure approached either the helper or the hinderer, was measured. Length of "looking" indicates that what is seen is "surprising" rather than "expected" — and it was found that the babies "looked" longer when the figure approached the hinderer, a case of surprising behaviour.

The next trial was to see how the babies themselves evaluated the helpers and hinderers – that is, had their observation of how these personages acted with a third person (the climber figure) affected their own preference? The test was simply to allow the babies to choose either the helper or the hinderer when both were offered. "The babies robustly chose the helper," says the Yale University paper. Further experiments then showed that the preference registered only when the climber was actually trying to reach the top – and not when the climber was "depersonalised" by covering the eyes and did not move on its own — and the other two figures simply pushed the climber "up" or "down", without social interaction.

And yet another experiment showed that if there was third, extra figure who neither helped nor hindered, then this figure was still preferred over the hinderer – suggesting a poor "moral" value associated with the hinderer.

There is evidence to show that the complex social organisation of humans in groups is brought about by training and "reward or penalty", as opposed to the simpler order in ant colonies or wolf packs, which appears to be genetically "wired in". In the case of the Yale experiment, the participants were just coloured wooden objects that did not threaten or reward. In this context, that babies at just six months of age showed a preference for "helpers" strongly suggested that "social evaluation", on the basis of which further socialisation can be built, was also genetic and innate in humans, too.

New research carried out by a team led by Dr Damien Scarf (left) at New Zealand's University of Otago is casting doubt on a landmark US study that suggested infants as young as six months old possess an innate moral compass that allows them to evaluate individuals as "good" or "bad".

The Otago study

The group at the New Zealand university examined the notion that moral sense was innate as they saw implications of this idea for the human moral system and the dynamics of the development of social structures. But while watching videos of the Yale experiments, the Otago viewers discovered a pair of perceptual elements in the experiment that could be the driver of the babies' preference, rather than social evaluation. "On the help and hinder trials, the toys collided with one another, an event we thought infants may not like. Furthermore, only on the help trials, the climber bounced up and down at the top of a hill, an event we thought infants may enjoy," says Dr Damien Scarf, lead author of the paper in *Plos One*.

The researchers then carried out experiments with a manipulation of the colliding and bouncing events and found that the preference for the helper over the hinder disappeared once these events were eliminated or reversed. "For example, when we had the climber bounce at the bottom of the hill, but not at the top of the hill, infants preferred the hinderer, that is, the one that pushed the climber down the hill. If the social evaluation hypothesis was correct, we should have seen a clear preference for the helper, irrespective of the location of the bounce, because the helper always helped the climber achieve its goal of reaching the top of the hill," says Dr Scarf.

Although the Yale researchers had followed up the study and appear to have collected more proof for the concept of innate social evaluation, the Otago group thinks these could also be explained based on simple association, as in the case of the preference for the helper. "… While we accept it is not easy to develop paradigms that perfectly match up the perceptual attributes of the helper and hinderer events, we still think there is room for improvement," says Dr Scarf.

Review

Reviewing the two papers, it does seem that the Yale study, even with instances of "bouncing", has been fairly conducted. The nature of social interaction has to be a value judgment of something "preferred". A climber would prefer a helper only if the climber actually liked reaching the top of the hill. That "the climber's goal was to reach the top" is an assumption of Dr Scarf. The babies cannot have an innate preference for "up" over "down". They evaluate the role of the helper/hinderer based on what the climber seems to consider help or hindrance – as shown by "bouncing". In real social interaction, too, we may seek out hinderers over helpers, if people prefer pain over pleasure.

But the Otago study presents questions that affect an idea of importance. "I look forward to future studies on the topic of moral nativism and hope our study stimulates some discussion," says Dr Scarf.

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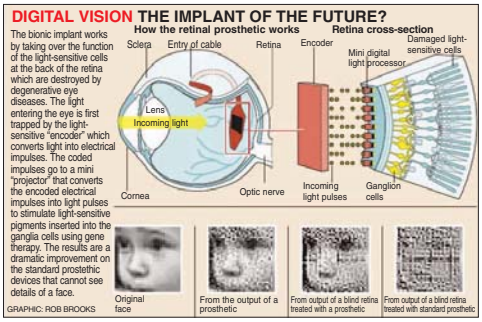
'Bionic' implants could help blind see

Scientists claim tests on mice have produced radical improvements to restoring vision.
steve connor **reports**

A BREAKTHROUGH in understanding how the eye sends visual information to the brain could soon lead to "bionic" implants that restore almost perfect vision to millions of blind people. Researchers have cracked the neural "code" used to shuttle images from the eye's retina to the visual centres of the brain and have incorporated this code into a microchip that can be inserted into the eye.

Tests on the retinas of blind mice have radically improved their vision compared to existing microchips. The scientists said they had also cracked the code for monkey vision, which is essentially the same as that used in humans. They envisage being able to construct futuristic visors for the blind, similar to those used in *Star Trek*, to enhance the visual abilities of the 25 million people in the world suffering from conditions such as macular degeneration and *retinitis pigmentosa*, which cause the loss of light-sensitive cells in the retina.

Sheila Nirenberg, a neuroscientist at Weill Cornell Medical College in New York, said that the advance was a radical improvement on existing attempts to insert bionic eye implants which had only had limited success in restoring vision to the blind. "It's an exciting time. We can make blind mouse retinas see, and we're moving as fast as we can to do the same in humans. This is the first prosthetic that has the potential to provide normal or near-normal vision, because it incorporates the code," she said.



Existing prosthetic devices used to enhance vision are based on tiny light-sensitive electrodes that simulate nerve cells within the eye to compensate for the loss of the natural light-sensitive cells of the retina, the cones and rods. However, these prototype devices, when tested on patients, only manage to produce spots of light or high-contrast edges. Patients are unable to discern the details of a face, for instance.

Scientists have tried to compensate for this technical limitation by increasing the density of electrodes in the implant. But Dr Nirenberg's team used an additional approach by incorporating an intelligent "encoder" that sits between the incoming light and electrode stimulators.

It is this encoder that can modify the stimulation of the nerves leading from the retina to the brain in a way that accurately reflects the natural visual process of the retina, the scientists say in their study published in the journal, *Proceedings of the National Academy of Sciences*.

The key to the success was the discovery that the light-sensitive cells of the retina used a type of code, or set of equations, to convert light into the electrical pulses sent to the brain via nerves cells, or ganglia, within the retina, Dr Nirenberg explained. "Not only is it necessary to stimulate large numbers of cells, but they also have to be stimulated with the right code – the code the retina normally uses to communicate with the brain," she said. "People had been trying to find the code that does this for simple stimuli, but we knew it had to be generalisable, so it would work for anything – faces, landscapes – anything a person sees."

The encoder consists of a microchip that converts incoming images into streams of electrical pulses. A mini "projector" within the encoder then converts these electrical pulses back into a pattern of light impulses that are used to stimulate light-sensitive proteins within the ganglia cells of the retina. A gene therapy technique is used to insert these light-sensitive proteins in to the mouse ganglia, which would also need to be used if human patients are to benefit from the technique, the scientists said.

To test the idea, the scientists built two prosthetic devices attached to mouse retinas, one with the code and one without. The results, and those combined with experiments on laboratory mice, showed that the bionic implant enabled blind mice to see visual details, said Dr Nirenberg.

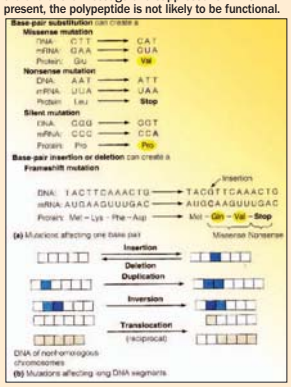
The Independent, London

Mutation primer

When we think about the potential effects, it is useful to remember that genes have important non-coding components, writes
tapan kumar maitra

IN its broadest sense, the term "mutation" refers to any change in the nucleotide sequence of a genome. Having examined the processes of transcription and translation, we can understand the effects of a number of different kinds of mutations. Limiting our discussion to protein-coding genes, let's consider some of the main types of mutations and their impact on the polypeptide encoded by the mutant gene. There are several types of mutations in which the DNA change involves only one or a few base pairs. For instance, the genetic *allele* that, when homozygous, causes sickle-cell anaemia. This allele originated from a type of mutation called base-pair substitution. In this case, an AT base pair was substituted for a TA base pair in DNA. As a result, a GUA *codon* replaces a GAA in the mRNA transcribed from the mutant allele, and in the polypeptide (b-globin) a *valine* replaces a glutamic acid. This single amino acid change, caused by a single base-pair change, is enough to change the conformation of b-globin and, in turn, the haemoglobin *tetramer*, altering the way haemoglobin molecules pack into red cells and producing abnormally shaped cells that become trapped and damaged when they pass through small blood vessels.

Such base-pair substitution is called a *missense* mutation, because the mutated codon continues to code for an amino acid — but the "wrong" one. Alternatively, base-pair substitution can create nonstop mutation by converting a normal stop codon into an amino acid codon; or conversely, it can create a nonsense mutation by converting an amino acid codon into a stop codon. In the latter case, the translation machinery will terminate the polypeptide prematurely. Unless the nonsense mutation is close to the end of the message or a suppressor tRNA is present, the polypeptide is not likely to be functional.



Nonsense, nonstop and missense codons can also arise from the base-pair insertions and deletions that cause frameshift mutations.

A single amino acid change (or even a change in several amino acids) does not always affect a protein's function in a major way. As long as the protein's three-dimensional conformation remains relatively unchanged, biological activity may be unaffected. Substitution of one amino acid for another of the same type — for example, valine for isoleucine — is especially unlikely to affect protein function. The nature of the genetic code actually minimises the effects of single base-pair alterations because many turn out to be silent mutations that change the nucleotide sequence without changing the genetic message. For example, changing the third base of a codon often produces a new codon that still codes for the same amino acid. Here, the "mutant" polypeptide is exactly the same as the wild type.

In addition to mutations affecting one or a few base pairs, some alterations involve longer stretches of DNA. A few affect genome segments so large that the DNA changes can be detected by a light microscopic examination of chromosomes. Some of these largescale mutations are created by insertions or deletions of long DNA segments, but several other mechanisms also exist. In a duplication, a section of DNA is tandemly repeated. In an inversion, a chromosome segment is cut out and reinserted in its original position but in the reverse direction. A translocation involves the movement of a DNA segment from its normal location in the genome to another place, in the same chromosome or a different one. Because these largescale mutations may or may not affect the expression of many genes, they have a wide range of phenotypic effects, from no effect at all to lethality.

When we think about the potential effects of mutations, it is useful to remember that genes have important non-coding components and that these, too, can be mutated in ways that seriously affect gene products. A mutation in a promoter, for example, can result in more or less frequent transcription of the gene. Even a mutation in an *intron* can affect the gene product in a major way if it touches a critical part of a splice-site sequence.

Finally, mutations in genes that encode regulatory proteins — that is, proteins that control the expression of other genes — can have far-reaching effects on many other proteins.

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