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paternally expressed genes), and for the remainder, it is the copy from mum (maternally expressed genes). Why these unusual genes exist is the subject of much debate, since damage to the single active copy could potentially have devastating consequences for the organism. Consequently there has been considerable debate about why imprinting exists. The most widely accepted theory of imprinted gene evolution (the 'conflict theory') proposes that the phenomenon arose because mum and dad disagree with regard to supplying their shared offspring with resources. In most mammals (including humans sometimes) fathers tend to mate with one female and then move on - according to the theory, genes inherited from dad (paternally expressed genes) would promote offspring growth and the offspring's demand for maternal nutrients (even at the expense of the mother's future reproductive capacity). In contrast, as mothers are equally related to their offspring and want to retain their reproductive capability, the genes they pass on would be expected to counter these paternal demands by restricting offspring demands and growth.

If your parents can't agree on how big and how demanding you should be, perhaps they also disagree on other matters. As imprinted genes are highly active in the brain an obvious question that follows is 'Are paternally and maternally expressed genes also at odds with regard to brain development and behaviour?' Ingenious studies in mutant mice have already provided some evidence that the two groups of genes may act differently in the offspring's brain: cells containing two copies of maternal genes, but no paternal genes, accumulate in areas of the brain responsible for complex thought - therefore it is possible that mum's influence is on how clever you are. Certainly, the fact that a lack of maternal gene expression in the rare human disorder Angelman syndrome is associated with mental retardation seems to support this idea. So, if mum wants you to be more intelligent, what are dad's priorities? In mice, cells containing two copies of paternal genes but no maternal contribution tend to accumulate in the hypothalamus, a brain region important in basic functions such as temperature regulation and feeding. Convincing evidence that paternally expressed genes are important in this latter process has come from studies in humans with the rare behavioural disorder Prader-Willi syndrome (PWS): PWS subjects lack certain key paternally expressed genes and as a consequence have hypothalamic deformities and fail to suckle properly as babies. Around weaning these subjects undergo an incredible change, developing an insatiable appetite and a compulsive desire to obtain food, which in some cases stretches to retrieving discarded food from dustbins and even eating frozen raw meat! Incidentally, a third major role of the hypothalamus is in modulating sex drive and sexual function - it may be our dad's genes that are to blame for making us men think about sex every six seconds!

One aim of my work is to clarify the extent to which conflict in the brain exists and to determine what the particular substrates of the dispute may be, with a view to understanding why imprinting has evolved and why it has been preserved. Imprinted gene malfunction has been associated with vulnerability to a wide variety of common mental disorders arising from abnormal brain development including autism, schizophrenia and depression. Therefore, we expect that investigations into imprinted gene action in the brain will provide insights into the pathways disrupted in such disorders. Ultimately, whilst the battle of the brain might still rage, at least we will be forearmed with a knowledge of the paternal and maternal strategic plans.

William Davies graduated with a degree in Biochemistry from Bath University, and subsequently gained a PhD in Behavioural Neuroscience from Cambridge. He currently works as a Postdoctoral Researcher at The Babraham Institute, Cambridge where his primary research interests are in the effects of X-linked imprinted genes (and X-linked gene dosage) on brain development and behaviour. Dr. Davies' research is likely to provide insights into why, and how, male and female brains develop differently, and into why certain neuropsychiatric disorders affect the two sexes differently.

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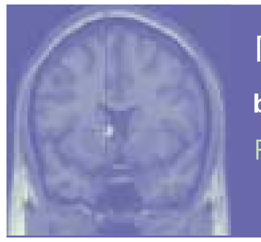

MY MONEY ON MY MIND

by Martin O'Neill

Runner up - Researcher Category

Everyday we are faced with a bundle of options which we have to choose between, how do we decide which is the most lucrative? This is at the crux of human and animal behaviour that has baffled scientists for generations, but the burgeoning field of neuroeconomics hopes to shed some light on the matter.

Neuroeconomics is a relatively new field of research with the mission of understanding human and animal behaviour by combining the principles of behavioural neuroscience (the study of the brain and behaviour) and economics (the study of financial behaviour). This field is an intriguing area at the moment as neuroscientists, including my colleagues and I, attempt to get our heads round economical concepts such as utility theory. Meanwhile economists try to digest the possibility that a single neuron in the brain, a unit so small that 10,000,000,000,000 fit into every human brain, may be capable of processing the type of information contained within complex economical theories.

The defining crossroads where these two fields meet is the study of decision-making. Everyday we encounter many types of decisions, ranging from the mundane to the profound. Whether one should buy a lottery ticket or a bar of chocolate is an example of the former. Embedded within such decisions are varying degrees of uncertainty and as a consequence one must choose between certain and uncertain options. A chocolate bar and a lottery ticket may cost roughly the same but purchasing a chocolate bar provides a certain outcome that is satisfying. The lottery ticket, on the other hand, has an unknown outcome that ranges from a loss (of the money spent on the ticket) to a monumentous gain.

By realising that most decisions involve some sort of currency, in other words something must be gained or lost from most decisions, us neuroscientists are now borrowing ideas from the economists to try and understand how the brain makes decisions. Our research, which is a combined effort between neuroscience and economic specialists, is attempting to explain many types of behaviour ranging from the everyday, like our lottery example, to the dysfunctional, such as pathologic gambling and drug addiction.

By using advanced technological tools available to the contemporary neuroscientist the search is on to find areas of the brain that participate in economic decision-making. Aside from generating basic knowledge of how systems in the brain function, the raison d'être is an attempt to understand what goes wrong with these systems in life-debilitating conditions such as drug and gambling addiction.

My colleagues at the University of Cambridge, working under the guidance of Professor Wolfram Schultz, are currently using a technique known as functional magnetic resonance imaging (fMRI) to investigate which parts of the brain are activated when human subjects are engaged in a task where they have to choose between two options. The choices offered are either a fixed amount of money (safe option) or a gamble (risky option). If the gamble is chosen, the subject either receives a larger or smaller amount of money than the certain option. Figure 1 shows an image generated from an fMRI scan while a subject performed the task.

This is a view of the subject's head face forward. The fMRI technology allows images to be taken throughout the brain. The white area, where the lines cross, signifies that a greater amount of activity occurred in this area compared to the rest of the brain. This particular scan was taken while the subject selected the risky choice. George Christopoulos, a PhD student with a Master's Degree in Economics Psychology, describes preliminary data from this study: 'An area of the brain known as the striatum appears to become active when subjects make risky decisions. Importantly, this area did not appear to be as active when the subject made safe choices'. Another research group at the Experimental Psychology Department in Cambridge, under the guidance of Professor Barbara Sahakian, is investigating the behaviour of drug addicts in risky decision-making.

The hope is that by combining the findings from both research groups, in the not too distant future therapies may be developed that target specific brain systems in the treatment of addictive, risk-seeking behaviours.

Martin O'Neill is currently in his first postdoctoral position at Cambridge University researching mechanisms of reward processing with Professor Wolfram Schultz. Martin first became interested in neuroscience while studying for a degree in psychology at Glasgow University. Thereafter, he pursued a PhD in behavioural neuroscience at St. Andrews University with Professor Verity Brown. This was an ideal opportunity for Martin to combine his main interests of psychology, neuroscience and pharmacology. To further extend his technical skills and knowledge of psychological processes, Martin reasoned that learning neurophysiological techniques and applying these to the research of reward processes was the next logical step.

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Figure: An fMRI scan of a human subject, which was taken while the subject chose between risky and safe choices. This shows the brain activity when the risky option was selected. The white region where the blue lines cross shows an in increase in activity in an area of the brain known as the striatum.

HOW THE BRAIN LIT THE FUSE TO THE CAMBRIAN EXPLOSION

By Jim Stone

Highly commended

Five hundred and fifty million years ago, the number and diversity of species exploded. This continued throughout the next 45 million years of the Cambrian period, and is called the *Cambrian explosion.* Many theories have been proposed as to why the Cambrian explosion occurred. However, the fact that it began with the emergence of animals with brains that *learn* is probably more than a coincidence.

The environment changes. Not just cataclysmic changes like the meteor that destroyed the dinosaurs, but also more subtle changes. The stars slowly shift their positions; any bird that navigates by the stars had better not be born with an innate star map that only King Tutankhamen would recognise. The environment changes, and animals play catch-up. The problem is that evolution is inefficient at catching up.

Every generation is born into an environment that is different from its parents'. Sometimes this difference is trivial, sometimes a meteor falls. Whatever the difference, natural selection seeks out individuals with the optimal combination of genes for a given environment. But before such individuals are born, their genes are scattered across many individuals. It is as if every offspring represents a shuffling of the genetic cards. Shuffling combined with natural selection ensures that the optimal genes will eventually coalesce in a single individual. That individual (and its relatives who share many of its genes) will pass those genes on to many, many offspring. These optimal genes spread at an exponential rate through the population. But all this shuffling is like syrup in the engine of evolution. Fortunately, evolution found a way to make this syrup less viscous.

Imagine two populations, the *learners* and *non-learners* whose only difference is that one can learn, and one cannot. These populations inhabit similar environments, which suddenly change. In order to adapt, the non-learners have to wait while natural selection gradually accumulates the optimal genes in particular sub-populations of individuals. In this way, the slow trek up "mount improbable" proceeds. In the meantime, the learners face the same crisis.

Lets consider what happens when some learners have near-optimal genes. Such individuals can learn to change their behaviour so that it is optimal for the new environment. After learning, these individuals will be *indistinguishable* from non-learners born with optimal genes. In other words, *learning within a single life time acts as a substitute for genetic adaptation over many lifetimes*. Thus, given one individual from the learners and one from the non-learners with the same *sub-optimal* innate behaviour, *the learner has a higher fitness* once it has learned, and will (by definition) have more offspring than the corresponding non-learner. If all other things are equal then this difference in offspring number ensures that one of the learners' offspring is more likely than the non-learners' offspring to have optimal genes. Any individual facing this crisis, whether a learner or a non-learner, is like a blind man with a blunderbuss. Each individual sprays genes into the world at random, and the more offspring it has, the more likely it is that one of its offspring will have the optimal genes.

There is one remaining problem. The learners would like be born with *innate* optimal behaviour. This is because learning takes time, and time spent learning is time spent not breeding; at worst, it is time spent being eaten. In other words, learning comes at a price: that price is paid in time, and time costs fitness.

Within the learner population, learning increases fitness. This amounts to having more offspring, which increases the chances that behaviours which had to be learned by the current generation will be innate in the next. Over many generations, an increasing proportion of learner individuals in each successive generation will display optimal behaviour without *any* learning. Thus the cost of learning ensures that behaviours which once had to be learned gradually become innate. This phenomenon is known as the *Baldwin effect.*

This does not imply that learned behaviours somehow invade the genes, so that behaviours learned by one individual are innate in its offspring. The inheritance of acquired characteristics is known as Lamarckism, and has long-since been discredited. However, the fact remains that learned behaviours usually increase fitness. It is this learning-related increase in fitness, and the evolutionary blunderbuss it provides, which increases the chance that offspring have innate behaviours that had to be learned by their parents.

In conclusion, over successive generations, populations that can learn evolve optimal *innate* behaviour faster than populations that cannot learn. Animals with brains that learn must, therefore, be a prime suspect in the unsolved case of the Cambrian explosion.

Jim Stone is a Reader in the Psychology Department at the University of Sheffield. His main interests are in vision, the brain, and evolution.

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