

Book review

perspective and Deborah Bowman, a well known ethicist interested in primary care. Our aim is to bring together all those who are interested in the values as well as the science of general practice, whether as clinicians, managers, teachers or researchers. Which means pretty well all of us.

For further details and to register interest for the meeting, email sophie.baettig@rsm.ac.uk.

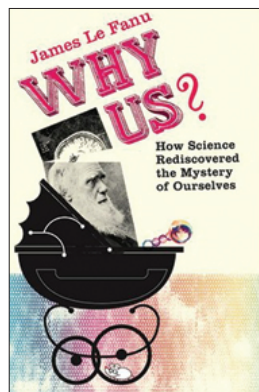
**Andrew Papanikitas and
Peter Toon**

DOI: 10.3399/bjgp10X539425

WHY US? HOW SCIENCE REDISCOVERED THE MYSTERY OF OURSELVES JAMES LE FANU

Harper Press, 2009

HB, 303pp, £18.99, 9780007120277



James Le Fanu's new book sets out to argue two things. We can't, he says, simply be the chance products of a materialist evolutionary process; and, however much we may learn about ourselves as machines, we could never learn enough to account for the awe-inspiring complexity of our nature.

The argument centres on essentially two phenomena. One is the discrepancy between our ever more detailed knowledge of the human brain and the failure of this knowledge to tell us anything much about the mind. It's true that, as he says, the complexity of experience cannot be reduced to a few patches of neural tissue lighting up on a scan. But nonetheless, such patches do tell us at least something — pretty crude though it may be — about the mind of the person whose brain we are inspecting. Where I agree, is that it still tells us nothing whatever about what it is to have a mind at all. To expect amassing scientific information ever to do so is a basic philosophical error, a category mistake.

The other phenomenon on which Le Fanu focuses, and to me this is both the more interesting and the more controversial part of his argument, is the

apparent 'decoding' of the human genome. This mighty project, which came in ahead of schedule, and was confidently expected by many to be the key that unlocked the secrets that would enable us to treat and even eliminate disease, has been a resounding disappointment. The clinical rewards have been so far meagre, to say the least. But Le Fanu goes further. The information in the DNA sequences is simply not enough — never could be enough — to explain the complex structure and functioning of the pieces of work it codes for: and what a piece of work is man! In arguing this, he adduces information that will surely prove fascinating to many readers.

We have 26 000 genes. But a blind, millimetre-long roundworm with only 959 cells in total already has over 19 000. Then there is the sheer extent of the problem of 'junk' DNA. The human genome contains so much data that, it has been calculated, it would fill 43 volumes of Webster's International Dictionary. But it is as if no less than 42 of them contained no genetic information at all, consisting of tens of thousands of repetitions of just one letter of the genetic code. What even the remaining bits do, it seems, is hard to predict. The same genetic disease can be caused by different mutations in several different genes, and conversely, several different diseases can stem from mutations in a single gene. The selfsame gene may be involved in the production of the eyes, nose, brain, pituitary, gut or pancreas, but along with thousands of other genes, and depending on precisely which other genes are involved.

It seems that 'context is all'. Philip Gell, Professor of Genetics at Birmingham, writes: 'The heart of the problem lies in the fact that we are dealing not with a chain of causation but with a network',¹ a bit like a spider's web in which a perturbation at any point of the web changes the tension of every fibre in it. And this does have implications for the classical theory of natural selection, since, as Le Fanu says, given this

interdependency where a change in one element changes all the others, and given the complexity that means that it takes 6000 genes to build a fly's heart, 'what chance was there that a "random mutation" in any one of them might generate a beneficial variation in favour of the heart's further perfection'? At least a rather smaller chance than we had been working with until now.

How do genes know how exactly to work in 'this' context? How do they know what 'this' context is? The answer was posited to lie in 'master' genes, which orchestrated the others. But unfortunately 'precisely the same "master" genes mastermind the three-dimensional structures of all living things: frogs, mice, even humans'.

As Stephen Jay Gould commented, the central significance of such findings lay not in their being previously unknown, but in their being so completely unexpected.² The plot thickens when it turns out that the same gene, Pax 6, brings all eyes into existence — a fly's, a frog's, and yours, with their profoundly different construction and mode of operation. And eyes, unimaginably complex as they are, are as nothing when one considers the complexity of the development of the human brain. The fetal brain produces an average of 25 000 new neurones a minute, forming trillions of connections in a nonetheless carefully and minutely structured array of a hundred billion neurones. And by what means do the genes 'know' to adapt to the demands of neuroplasticity in the growing child, or after neuronal loss? How, above all, can they specify a cortex that will hear Beethoven, rather than see Michelangelo?

The favoured theory is that the master gene, given its environment in a certain species, switches off and on the other genes in a way that helps it produce species-specific results. But this staggeringly complex switching raises the question 'how the relevant master gene for each could have chanced on the correct sequence of switches to generate

the appropriate part?' Furthermore, as Le Fanu puts it,

'it is as if the 'idea' of the fly (or any other organism) must somehow permeate the genome that gives rise to it, for it is only through the master genes of the embryonic fly knowing it is a fly that they will activate that sequence of switches that will give rise to those appropriate structures'.

There is a chicken and egg problem here. And the importance of wholeness or reverberative interaction, rather than bottom-up causation, does not stop there, since 'the vastly more complex cell within which [the genome] is located must somehow "know" its own needs, and then determine which of those 26 000 genes at any moment are to be activated ... And the cell in issuing those instructions will in turn be influenced by the needs of the tissues and organs within which it is located, and so on all the way up the hierarchy to the organism in its entirety.'

For Le Fanu this is:

'a nail in the coffin of Darwin's proposed mechanism of natural selection acting on numerous small, random genetic mutations'.

But if the information is not given in the base sequences of the double helix, where then is it? Le Fanu's solution is to posit a formative 'life force' that we cannot know directly, but whose necessary existence we can infer from the complexity of nature. Such a force may, for all we know, exist, I agree; but to argue for it like this seems rather reminiscent of invoking a 'God of the gaps'. As Dietrich Bonhoeffer wrote:

*'how wrong it is to use God as a stop-gap for the incompleteness of our knowledge ... We are to find God in what we know, not in what we don't know.'*³

Surely these are different levels of

explanation. For my money, this is where Rupert Sheldrake's theory of morphic resonance, unjustly neglected in the past by mainstream science, but beginning to seem more compelling with greater understanding of the issues Le Fanu discusses, could well hold the key. According to this theory, there are evolving 'morphogenetic' or organisational fields which, through influence on cell membranes and microtubules, direct the forms of living things, and effectively constitute a collective 'memory' for the structures of physical organisms — as well as of thoughts, activities, and experiences.⁴ These have the advantage that their effects can be predicted and demonstrated. Certainly we can never again suppose the world to be simply mechanical, rather than organic, in structure, the whole being as much the determinant of the part, as the part is of the whole.

Iain McGilchrist

REFERENCES

1. Le Fanu J. *Why Us: How Science Rediscovered the Mystery of Ourselves*. London: HarperPress, 2009, pp: 140.
2. Le Fanu J. *Why Us: How Science Rediscovered the Mystery of Ourselves*. London: HarperPress, 2009, pp: 141.
3. Bonhoeffer D, letter to Eberhard Bethge, 29 May 1944, pages 310–312, *Letters and Papers from Prison*, ed. Bethge E, trans. Fuller RH, Touchstone, 1997; Translation of *Widerstand und Ergebung*. Munich: Christian Kaiser Verlag, 1970.
4. Sheldrake R. *Morphic Resonance: The Nature of Formative Causation*. Maine, NE: Park Street Press, 2009.

DOI: 10.3399/bjgp10X539434