Evolution's 'missing link': a hypothesis upon neural plasticity, prefrontal working memory and the origins of modern cognition

email: John R. Skoyles

Visit John Skoyles' Webpage

Centre for Philosophy of Social and Natural Sciences, London Schoolo of Economics, London, WC2A 2AE

Medical Hypotheses (1997) 48,499-501

Date received 26 April 1996 Date accepted 17 May 1996

Abstract - Many activities such as reading, mathematics and chess depend upon cognitive processes which arose after our evolution. Why could they arise if not evolved? I argue four things fortuitously came together to make our nonevolved cognitive skills possible: (i) neural plasticity; (ii) large functionally uncommitted prefrontal, temporal and parietal cerebral cortices; (iii) the ability of their neural circuits (due to neural plasticity), if trained, to take on novel symbolic and nonsymbolic skills; and (iv) a large prefrontal cortex which could use its working memory as a tuition management sketch pad in which to train them. Pre-evolved for other reasons, these four (together with invented symbolic systems and technology) together enable modern humans to 'upgrade' our already evolved cognitive skills to do new and nonevolved things.

The problem of modern cognition

Most of us can read, spelt, solve maths and chess problems and appreciate, if not play, diatonic music. Yet, these mental skills were unknown, even a few thousands or tens of thousands of years ago, by anatomically modem people. Evolution could not have anticipated such skills, so why can our brains perform them? This briefly is the sine qua non problem of modern cognition. At present we have no answer: what follows links previously unconnected areas of neuroscience to provide a solution.

Neural plasticity

Research finds that rather than being predetermined the neural networks in the brain underlying cognition are open to process new skills: 'neural plasticity'.

- In those born blind the visual cortex aids bearing (1) and braille reading (2,3).
- The functions of motor cortex destroyed by brain tumours can be taken on by other motor and nonmotor areas up to 4.2 cm distant (4).
- In amputees, remapping up to 3 cm occurs when somatosensory and motor maps invade areas which have lost their input (5).
- In ferrets, visual input experimentally redirected to the somatosensory cortex causes it to develop into 'visual cortex' (6).
-]n the blind mole rat, evolution has redirected auditory input via tie lateral geniculate nucleus to the visual cortex causing it to function as an 'auditory cortex' (7).

It might be objected that the above examples concern shifts of function rather than innovation. Neural plasticity should, however, give neural networks the potential to do novel things. First, neuroscience finds that the neurocircuitry of the cortex is nonspecific, with its functionality determined by its inputs and outputs rather than intrinsic connections (8). There is no reason therefore why such circuits should be functionally limited. Second, a shift is a novel function at least evolutionarily for the area into which it has 'moved' - evolution did not evolve the visual cortex to hear (a shift of function) as much as it did not to read (a novel one). Third, modern brains perform evolutionarily novel skills - such as your reading this - which requires some kind of ability for our brains to do new things.

The five above-mentioned examples of neural plasticity concern areas of the brain which are 'hardwired' with direct sensory input or motor output. The important parts of the human cerebral cortex, however, consist of association areas in the parietal, temporal and prefrontal lobes which are free of such input and output constraints - it is therefore likely that they have, if anything, greater flexibility to take on new functions. This matters: human evolution increased the size of the brain by expanding such association areas but not our primary sensory and motor areas, which remain no larger than would be expected in an ape of our body-size (9). The human brain has, as a result, the cortical space for doing new tasks.

The need for a tuition manager

The existence of neural plasticity, however, will not, of itself, lead to new skills - they must be learnt. For this to happen the brain must be able to train novel functions and skills upon its neural networks. what might be the preconditions for this?

We can gain some clues by looking at how computer neural networks learn. Basically, new functions are learnt by tuition using the presentation of learning inputs in the form of exemplars and correction feedback (10). The information these provide is converted into patterns of network node strengths. The functional abilities of a neural network derive from this information transfer during learning.

An important precondition for successful network learning will, as a result, be the existence in the brain of an efficient and an effective means to train its networks with exemplars and correction feedback information. The brain thus needs some kind of manager to optimally organize the information used to tutor its neural networks.

Such a manager will require skills such as

- temporary buffers to hold input and output information;
- the capacity for attention, to compare neural network output with desired output and to detect errors, thus enabling them to be corrected and the network's performance improved;
- the ability to do these things across sensory, motor and other cognitive modules (an example would be speech and sight during the learning of phoneme-letter symbolization); and
- the capacity, since network learning will not be spontaneous, to actively present information to networks so that their training can be controlled, directed and focused and they can pick up new desired skills.

In summary, effective and efficient network learning needs some kind of 'tuition management sketch pad' where information for learning can be temporarily held and organised.

The ability of the brain to exploit neural plasticity to learn new skills will be restricted, therefore, if processes for doing this do not exist or are limited. Thus, the rise of modern cognition depends not only upon the existence of (i) neural plasticity but also (ii) the brain possessing processes capable of acting as a 'tuition management sketch pad'.

Prefrontal cortex

The prefrontal cortex recently has been found to have the skills, 'working memory'. required for acting as a tuition management sketch pad (11). Working memory combines (a) the short-term buffering online of information suitable for holding associations, examples and feedback, (b) the attentional and organizational skills which can monitor their use in learning, and (c) the ability to focus neural processing in terms of relevant on-going goals.

Evidence directly supports the specific involvement of the prefrontal cortex in training: functional brain imaging finds that the prefrontal cortex is needed in learning skills, but not afterwards when they become routinized (12). Moreover, lesion research finds that prefrontal cortex involvement is necessary during acquisition of symbol-like conditional associations, but not for their later use (13).

Modern nonevolved cognition, I therefore suggest, arose from the fortuitous coming together: (i) of a brain evolved (perhaps from earliest vertebrates) to be neurally plastic, and (ii) the rise in hominid evolution of an enlarged prefrontal cortex with the tuition management skills able to rework neural plasticity to perform new, nonevolved cognitive skills. Neural plasticity and the prefrontal cortex, in sum, constitute the sine qua non precondition for our modern mental skills.

Four comments need to be made.

First, this is a theory about what in the human brain permits new skills, not why they arose' Modern cognition is also the product of nonneurological phenomena such as appropriate technologies, symbolic systems and circumstances which encourage and support their acquisition. For instance, in the case of reading, the existence of pens and paper, alphabets and logographs, schools and a literate civilization. Neurology does not explain why they exist, nor why they have only arisen in recent millennia.

Second, if modem cognition is due to neural plasticity upgraded in function by the prefrontal cortex, then deficits in the ability of the prefrontal cortex to manage learning might be a source of developmental problems such as dyslexia and developmental acalculia. Conversely, unusual mental skills might arise from superior prefrontal linkage to those parietal and temporal areas optimal for their development and processing. Such possibilities are readily testable given recent developments in PET and functional MRI imaging.

Third, it cannot be ruled out that the expansion, in human evolution, of the prefrontal cortex and the other association areas might have occurred in part to exploit neural plasticity to create novel competence in regard to dexterity and speech. However, even if evolved for this, nothing stopped the human species later developing technologies, symbolic systems and circumstances letting the prefrontal cortex exploit them further.

Fourth, a puzzle faced by science is why the human mind is so qualitatively different from the ape one in spite of human and ape brains being made of the same components with differences only in relative proportion and quantity. How could old components lead to something new? There has been therefore a need to find a 'missing-link'. The above theory suggests a new direction for answering this problem: rather than some unknown fossil, the 'missing-link' lies in the combined potential of neural plasticity and prefrontal working memory of the human brain to refashion old neural networks to do novel and nonevolved things.

References.

1. Kujala T, Huotilainen M, Sinkkonen J et al. Visual cortex activation in blind humans during sound discrimination *Neurosci Let* 1995; 183: 143-146.

2. Uhl F, Franzen P, Lindinger G, Lang W, Deecke L. On the functionality of the visually deprived occipital cortex in early blind persons. *Neurosci Let* 1991; 124: 256-259.

3. Sadato N, Pascual-Leone A, Grafman J. et al. Activation of the primary visual cortex by Braille reading in blind subjects. *Nature* 1996; 380: 526-528.

4. Seitz R, Huang Y, Knorr U, Tellmann L, Herzog H. Freund H.- J. Large-scale plasticity of the human motor cortex. *NeuroReport* 1995; 6: 742-744.

5. Yang TT, Gallen CC, Ramachandran VS, Cobb S, Schwartz BJ. Bloom FE. Noninvasive detection of cerebral plasticity in adult human somatosensory cortex. *NeuroReport* 1994; 5: 701- 704.

6. Roe A, Pallas S, Kwon Y, Sur M. Visual projections routed to the auditory pathway in Ferrets: Receptive fields of visual neurons in primary auditory cortex. *J Neurosci* 1991; 12: 3651-3664.

7. Doron N, Wollberg Z. Cross-modal neuroplasticity in the blind mole rat *Spalaz ehrenbergi*. *NeuroReport* 1994; 5: 2697-2701.

8. O'Leary DD, Schlaggar BL, Tuttle R. Specification of neocortical areas and thalamocortical connections. *Ann Rev Neurosci* 1994; 17: 419-439.

9. Deacon T. Fallacies of progression in theories of brain-size evolution. *Intern J Primatol* 1990; 11: 193-236.

10. Skoyles JR. Training the brain using neural-network models. *Nature* 1988; 333: 401.

11. Goldman-Rakic PS. Cellular basis of working memory. *Neuron* 1995; 14: 477-485.

12. Raichle ME, Fiez, JA, Videen TO et al. Practice-related changes in human brain functional anatomy during nonmotor learning. *Cerebral Cortex* 1994; 4: 8-26.

13. Petrides M. Nonspatial conditional learning impaired in patients with unilateral frontal but not unilateral temporal lobe excisions. *Neuropsychologia* 1990; 28: 137-149.