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Reviews

On the Neural Bases and Evolution of Free Will

Freedom and Neurobiology: Reflections on Free Will, Language, and Political Power. By John R. Searle (New York: Columbia University Press, 2007), 113 pp. $24.50/£16.00cloth.

Philip Lieberman

John Searle in *Freedom and Neurobiology* performs a signal service in attempting to address the neural bases of free will. Searle's essays on language have pointed out many of the elements of language use that have eluded the Chomskian school, and in these essays he directs our attention to the question of what aspect of the human brain confers free will. Searle assumes that free will exists—that human beings generally have a choice in deciding on a course of action or thought process. However, though Searle's hypothesis is consistent with the findings of neurophysiological research, it offers few insights on the neural mechanisms that might confer free will. Searle claims that free will results from the "behavior of the neurons" over the course of time (65). His model of the brain's actions, though it is really a stretch to call it a model, appears to be a sort of finite state automation where a transition occurs from S1 to S2 with a "gap" between the two states in which "the brain is such that the conscious self is able to make and carry out decisions in the gap, where neither decision or action is determined in advance" (73).

It is difficult to see how Searle's views could advance our understanding of the nature of free will. All brains have neurons. Does a fruit fly possess free will when it moves from S1 to S2 as it feeds? How about dogs and cats? We can all agree that humans possess the *capacity* for free will. We also can agree that the neurons of their brains must carry out the operations that confer this capacity. However, the neurons of our brain also regulate breathing, the pumping action of our hearts, our temperature and a host of other functions that clearly have nothing whatsoever to do with the capacity for free will. Searle in fact seems to take note of the scope of the problem and the tenuous nature of his proposal when he states: "We don't know enough about how the brain works, specifically how it produces consciousness, which it definitely does, and how it gives us the experience of free will which it definitely does" (31).

Searle's extended essay perhaps should be regarded as a statement of a belief that the neurophysiology of the brain in some manner explains how humans can exercise free will. I do not demur to his appraisal of the present imperfect state of knowledge of the neurophysiology of cognition, free will, and consciousness. However, I think that we can begin to approach the questions at least of cognition and free will. My proposal here is that humans have an inherent neural capacity for free will that derives from the cognitive capacity that also confers human linguistic ability. My argument will depart from that of a philosophical enquiry or a religious tract. I shall instead present a theory for the evolution of the neural substrate that allows humans to exercise free will that invokes the principles and findings of neurophysiological research and evolutionary biology.

Free will first must be recognized as *a capacity that can be expressed in an appropriate setting*. The historical record should convince the skeptic. Although the thoughts of free thinkers might deviate from Calvin's dogma,

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while Calvin ruled Geneva, overt expression was risky. In recent memory, the thoughts of multitudes were channeled through Nazi, Marxist, and Maoist indoctrination. In short, an appropriate environment–a social order that allows the exercise of free will–must exist for this capacity to be expressed. But free will is a capacity that can be expressed, and it is almost a certainty that ants or bees lack this capacity. It is doubtful whether a turtle has this capacity. So in accounting for the nature of the neural bases of free will in humans, we must take into account their evolution. In this, I follow Theodosius Dobzansky (1973) view that "Nothing in biology makes sense except in the light of evolution." My proposal, which may at first seem improbable, is that the neural substrate that grants the capacity for free will ultimately derives from brain mechanisms that regulate motor control in other species.

Neural Circuits

Over the past two decades, it has become evident that in complex brains including our own, thought processes, motor activity, and emotion and affect are not regulated by discrete neural "organs" or "modules." Instead, complex aspects of behavior are regulated by means of neural circuits that link activity in different regions of the brain. There is, for example, no local discrete part of the brain that, in itself, regulates language, or even a component of the elements that linguists believe to constitute our linguistic capacity. The nineteenth-century theory in which Broca's and Wernicke's cortical areas constitute the language organs of the human brain is simply wrong. Studies of aphasia, the permanent loss of language that was attributed to damage to these areas, does not occur unless the subcortical structures that are key elements of a class of neural circuits that regulate language and speech are damaged.

The operations performed in the human brain can be described at two levels. A particular discrete structure or region of the neocortex can support groups of neurons (the term "population" is often used to designate these groups of neurons), each of which performs some local operation such as processing colors or shapes. Other structures and cortical regions perform local operations such as regulating aspects of motor control or holding information in short-term (working) memory, etc. However, a discrete structure or cortical area usually does not by itself regulate a complete complex behavior. Each neural structure instead may support many groups of neurons that carry out a similar local operation. Each neuronal population is linked to or "projects" to an anatomically distinct neuronal population in another region of the brain. A series of linked neuronal populations forms a neural "circuit." The circuit constitutes the neural basis of an observable aspect of behavior, such as walking, running, talking, dancing, or processing the syntax of a sentence. Moreover, within a given neural structure, distinct anatomically segregated neuronal populations may occur, each of which projects to neurons in different brain structures, forming multiple circuits that individually regulate some other behavior. Therefore, a given neural structure may be involved in different aspects of behavior, each regulated by a different circuit.

Cortical-Striatal-Cortical Circuits

The class of neural circuits that confer free will involves linkages between areas of the cortex and the subcortical basal ganglia. The basal ganglia are subcortical structures located deep within the brain. They can be traced back to animals similar to present day frogs. The striatal component of the basal ganglia includes the caudate nucleus and the putamen and globus pallidus. The putamen receives sensory inputs from most parts of the brain. The globus pallidus is an output structure receiving inputs from the putamen and caudate nucleus. These neural structures are interconnected and form a system with close connections to different regions of the cortex through other subcortical structures *and cortex*. Disruptions in *seemingly unrelated* behavior, such as obsessive-compulsive disorder, schizophrenia, and Parkinson's Disease, derive from the impairment of neural circuits linking cortical areas with the basal ganglia. Cognitive deficits usually attributed to frontal lobe cortical dysfunction can be observed in patients having damage to basal ganglia. *The particular cognitive act germane to free will is the ability to change the direction of a thought process.*

THE BASAL GANGLIA—THE ENGINE OF FREE WILL

Research focused on the role of the basal ganglia in Parkinson's disease has established the role of the basal ganglia that pertains to free will. The primary signs of Parkinson's disease involve motor impairment. Tremor occurs, movements slow down, balance and upright locomotion are impaired. These deficits reflect the breakdown of the basal ganglia sequencing engine. Studies of basal ganglia activity in humans and other species show that they call out a sequence of stored motor submovements in other parts of the brain and connect the series of *pattern generators* that constitute the submovement instruction sets to regions of the motor cortex that ultimately result in muscle activity. The basal ganglia in these routine motor tasks thus constitute a sequencing engine. However, the basal ganglia perform another motor function. When incoming sensory information suggests that a change in the course of an ongoing motor act is in the best interest of an animal, the basal ganglia terminate the ongoing activity and another, more appropriate motor act is initiated. The basal ganglia thus play a key factor in *adaptive* motor control.

It is clear that the basal ganglia play a similar cognitive role. Tracer studies reveal connections between the basal ganglia and prefrontal cortical areas implicated in cognition. The findings of studies employing functional magnetic resonance imaging (fMRI) of cognitively intact human subjects, and behavioral studies of instances in which basal ganglia operations are degraded by Parkinson's disease, oxygen deficits, strokes and genetic disorders, demonstrate one of the basal ganglia's cognitive roles—freely changing the direction of a thought process. In limiting conditions such as Obsessive-Compulsive Disorder a person is unable to escape from repetitively performing the same act over and over. This aspect of basal ganglia function may provide the bases for the productivity of human language. We can form a potentially infinite number of words from a finite number of phonetic contrasts. We can as well form a potentially infinite number of sentences from a large, but finite number of words and grammatical processes. And we have the potential for free will.

Any individual, in a given cultural setting, can express the capability to exercise free will that this neural capacity allows. How, though, might the neural capacity for free-will have evolved? Examination of the syndromes associated with a breakdown in basal ganglia operations and comparative studies of our non-human primate cousins suggests that upright bipedal locomotion and the concomitant necessity for upright balance was the factor that initially provided a selective advantage for enhancing basal ganglia functions in the realm of motor control. Running, a later hominid specialization, may have enhanced this process. And chance events, which, as Darwin noted, result in an organ adapted for one function taking on new roles, appear to have set the path towards cognitive operations involving *freely made* decisions—and hence involving free will. The demands of speech and language are interwoven with this last phase of the evolution of the neural substrate that yields free will. Chimpanzees, for example, cannot talk. Although they lack the human speech anatomy, they can produce somewhat less intelligible speech subject to the constraints imposed by their vocal anatomy. However, their vocalizations are stereotyped—bound to particular situations. The FOXP2 gene, mistakenly identified as a language gene by some linguists, determines the embryonic development of the basal ganglia and other subcortical structures that support the neural circuits regulating speech production, comprehending syntax and the cognitive flexibility that underlies the human capacity for free will. The human speech anatomy dates back to 50,000 years ago when human artifacts first appeared. The human speech anatomy results in increased risk for choking to death on food lodged in the larynx and infection from impacted third molars. There would have been no reason for retaining tongues that had descended into the throat, carrying the larynx down to a position in which choking could readily occur, unless the neural substrate were present that allows us to freely reiterate the motor pattern generators that underlie speech. And that neural substrate yields cognitive flexibility—the ability to break away from predetermined actions and beliefs—that is, the capacity for free will.[1]

Note

1. For details on basal ganglia operations in motor control, syntax and cognition, see Philip Lieberman, *Toward an Evolutionary Biology of Language*, (Boston, MA: Harvard University Press 2006), and "On the Evolution of Human Speech: Its Anatomical and Neural Bases," *Current Anthropology*, 48 (2007): 39–66.