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Brain Research Bulletin 66 (2005) 311–316

BRAIN RESEARCH BULLETIN

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Avian and mammalian "prefrontal cortices": Limited degrees of freedom in the evolution of the neural mechanisms of goal-state maintenance

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Available online 24 February 2005

Abstract

Is it possible to produce the same cognitive function with different brain organizations? This question is approached for working memory, a cognitive entity that is equally organized in birds and mammals. The critical forebrain structure for working memory is the nidopallium caudolaterale (NCL) in birds and the prefrontal cortex (PFC) in mammals. Although both structures share a large number of neural architectural features, they are probably not homologous but represent a remarkable case of convergent evolution. In reviewing the neuronal mechanisms for working memory in birds and mammals it becomes apparent that the similarities of NCL and PFC extend from the neuronal activation patterns during memory tasks down to the biophysical mechanisms of synaptic currents. Both in mammals and birds, dopamine acts via D1-receptors to tune preactivated neurons into sustained high-frequency patterns with which goal states can be held over time until an appropriate response can be generated. The degrees of freedom to create different neural architectures to solve the problem of 'stimulus maintenance' seem to be very small.

Keywords: Dopamine; D1-receptor; Working memory; Nidopallium caudolaterale; Delay cells

1. Introduction

The evolutionary events that formed the pallial entities of avian and mammalian brains are far from being understood. The classic conceptualization of this issue was ignited more than 100 years ago in Germany by Ludwig Edinger. According to his formulation, vertebrate brain evolution was made of additions of new brain entities, with the mammalian neocortex being the last and most advanced step. Birds had, in his view, only a very limited cortical homologue but had instead elaborated their basal ganglia to an enormous degree [15]. This assumption was dominant for a long time until the seminal studies of Karten [26] clearly showed that most of the avian forebrain was composed of pallial entities and displayed connectivities that closely resemble the ascending sensory systems to the cortex. Although the pallial identity of most of the avian forebrain is firmly established since then, there are opposing views on homologies between birds and mammals regarding the largest neural entity of the avian forebrain, which is the dorsal ventricular ridge (DVR). According to some authors, the avian DVR might be homologue to the claustrum/amygdala [38], while others assume a homology with the temporal neocortex of mammals [5,40].

Regardless which of these two hypothesizes will turn out to be correct, the DVR of modern birds displays so many unique and possibly derived features, that it probably changed its original architecture during evolution to a large extent, whatever the homologous counterparts in terms of phyletic continuity in the mammalian brain are. To understand the evolutionary mechanisms that guided these changes, it is important to study the interplay between brain and behavior. Behavior defines the frontier along which each organism interacts with evolutionary selection pressure. Therefore, neural architectures are shaped during evolution to produce certain behavioral traits that are required to stand the race for fitness. If species from different lineages are faced with the same selection pressure they might react with the same solution at the

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doi:10.1016/j.brainresbull.2005.02.004

behavioral level. But are these similar behavioral repertoires produced by similar neural entities or are completely different neural architectures capable to come up with the same behavior? To put this question in a more specific way, what are the degrees of freedom in producing the same behavioral repertoire with different brains?

To study this question, I will concentrate on a clearly defined cognitive module—working memory—and on a forebrain area—the prefrontal cortex—that is associated with it. Working memory is defined as a cognitive mechanism that holds currently attended information of any modality online and manipulates it according to the contextual needs of the moment [1]. According to a wealth of studies, working memory seems to be identical in birds and mammals [30,51]. Working memory was even defined parallel and rather independently in pigeons and humans [22], and according to Becker and Morris [3], the non-human [22] and the human [1] definition of working memory only differ with respect to the presence of a language component in humans. Two properties are at the heart of working memory: (1) maintenance of information (a function that is largely identical with the older conception of short term memory), and (2) manipulation of information according to a time sequence. In mammals, the prefrontal cortex (PFC) creates both aspects of working memory, albeit in concert with various other brain structures [39]. Numerous behavioral and electrophysiological studies show the nidopallium caudolaterale (NCL; the nomenclature of this paper follows [41]) of birds to be of prime importance to generate working memory in birds. This similarity of PFC and NCL was first pointed out by Ivan Divac and coworkers [10,34]. Since then, numerous similarities at the behavioral, physiological, anatomical and biochemical level between NCL and PFC could be shown [7,8,24,28] (Fig. 1). However, there are strong topographical and genetic arguments that make it likely that these two brain entities are not homologous in terms of their phyletic continuity [31,38]. Thus, the capability of PFC and NCL to generate the same kind of cognitive operations probably represent a case of evolutionary convergence (homoplasy). By studying the cellular mechanisms of working memory in PFC and NCL it should therefore be possible to analyze if different neural solutions exist for a single functional problem, or if mammals and birds converged onto the same device. To do so, I will mostly concentrate on the information maintenance function of the PFC and will show that the cellular processes generating this function are largely identical for PFC and NCL (Table 1).

2. The cellular machinery of working memory in mammals and birds

PFC neurons show elevated sustained activity levels while holding active an internal representation of the relevant stimulus during its physical absence to guide a forthcoming response [17]. Persistent delay activity very likely encodes a previously presented cue, a forthcoming response or an expected choice situation [32]. If the persistent activity is disrupted by different means, the animal is likely to make an error [18]. Thus, the maintenance of elevated firing rates in specific subpopulations of prefrontal neurons probably constitutes the cellular correlate of the short term memory component of working memory. Similarly, single unit recordings from the NCL of awake pigeons that participated in delayed Go/No-Go tasks revealed a class of neurons that showed elevated activity levels during the delay period [8]. Firing patterns of these neurons were clearly related to the success rate of maintaining the relevant event to guide the subsequent choice behavior. Similarly, Kalt et al. [23] had shown that the ability of NCL-neurons to differentiate between the Go- and the No-Go-stimulus correlates with the overall discrimination performance of the animal. Thus, delay units in the avian NCL show the same functional characteristics as those in primates.

Kröner et al. [27] described in the chick's NCL a neuron type (type II) with an initial tonic firing and a relatively hyperpolarized action potential threshold. This may indicate that the firing of type II cells is readily elicited by weak excitatory inputs. The phasic–tonic firing pattern elicited with large depolarizing currents also makes it likely that type II neurons respond strongly but transiently to a brief input, yet produce a

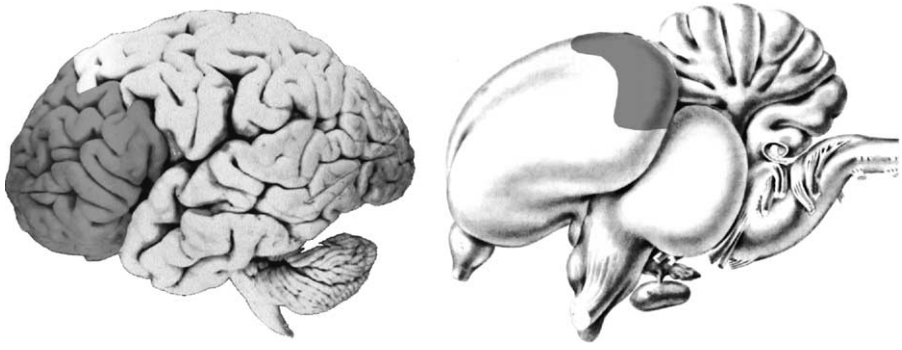

Fig. 1. Maximal outline (gray overlay) of the prefrontal cortex in the human brain (left) and the nidopallium caudolaterale in the pigeon brain (right). The brains are not depicted to scale. The nidopallium caudolaterale is shown as if being visible from the outside, although it is, in fact, encapsulated by a thin tissue lamina of the parahippocampal area.

Table 1
Several aspects of the neuronal realization of short term memory in the prefrontal cortex of mammals and the nidopallium caudolaterale in birds

		Mammals	Birds
Behavioral ⟷ Cellular	Prefrontal lesions disrupt the performance of animals in delay-task	11	7
	Prefrontal neurons display sustained activity levels during delay periods	17	8
	Prefrontal areas are characterized by a high density of dopaminergic fibers and D1-receptors	18	13
	Delay tasks are accompanied by an increase of extracellular DA in the prefrontal area	36	26
	Blockade of prefrontal D1-receptors disrupt delay performance	43	22
	Prefrontal DA-system favors diffusion mediated volume transmission	46	2
	DA increases the firing frequency of preactivated neurons via an activation of D1-receptors	50	21

The functions are ordered from more behavioral to more cellular ones in a descending sequence. The numbers in the two right-sided rows correspond to publications listed in the references.

sustained response to a prolonged input; a pattern that favors the augmentation of synaptic connections [46]. Functionally, the ability to generate a tonic firing mode could enable type II cells to retain information of their input for a short time period as required for sustained elevated firing bouts during short term memory episodes.

In mammals, delay-type neurons can also be found outside the PFC. Sustained, memory-related delay activity is observed in many brain areas, including parietal and inferotemporal cortex [18]. However, delay activity is more prominent in the PFC than in other areas, and also more robust to interfering stimuli [33]. Consequently, PFC-lesions always disrupt delay-task performance [11], while lesions of other cortical areas produce, if at all, less prominent delay deficits [37]. Similarly, NCL-lesions in pigeons disrupt delay performance in delayed alternation tasks [19,34]. Unfortunately, delayed alternation experiments always require the animal to make a spatial working memory decision. Since it is known that the prefrontal cortex of rodents seems to be especially tuned to spatial processes [36], it is important to clarify that the deficits in delayed alternation-tasks of NCL-lesioned pigeons are due to the memory and not due to the spatial component of this task. To this end, Diekamp et al. [7] devised a matching-to-sample task that can not be coded in spatial terms and showed that NCL-lesions in pigeons resulted in working memory deficits. Additionally, the volume of tissue loss within NCL correlated significantly with memory loss within the task.

3. The role of dopamine in working memory

In mammals, the integrity of the active short-term memory trace appears to be critically dependent on an optimal level of dopamine (DA) receptor stimulation. Both, prefrontal DA depletion [4] and blockade of D1-receptors [42] disrupt performance on delay tasks. The data for the NCL of pigeons are highly similar. Güntürkün and Durstewitz [20] tested pigeons in a 16 chambered labyrinth task with a cup being positioned in each of these chambers. Cups came in two colors, red and white, with the white ones always containing a few grains at the beginning of a session and the red ones always being empty. The animals were removed only after consuming all grains from all white cups. The pigeons quickly learned never to enter chambers with red cups. This is the reference memory part of the experiment. After finishing the grains of one of the white cups it of course made no sense to return to this specific chamber. Thus, the information on the white cups that were depleted during a single session constantly had to be updated. Additionally, this information is no longer valid as soon as the session is finished since all white cups are refilled for the next session. Therefore, information on the status of the white cups within a single session is stored in working memory. After the pigeons had acquired the task, the D1-receptor blocker SCH23390 was slowly injected into their NCL. The results clearly show that D1-receptor blockade drastically increased working memory deficits while leaving reference memory performance largely intact. Similarly, Diekamp et al. [9] revealed that D1-receptor lesions within NCL resulted in deficits in serial reversal tasks. This deficits, however, did only occur in the late reversal sessions where the animals already had developed a strategy to probe the correct stimulus of this session, to then maintain this information in their working memory. Together, these studies clearly reveal that working memory performance of the NCL is identically dependent on an activation of D1-receptors as is working memory in the mammalian PFC.

If working memory performance depends on DA-release and a subsequent activation of prefrontal D1-receptors, working memory episodes should be accompanied by an increase of extracellular DA within the PFC. The first study to approach this prediction was conducted by Watanabe et al. [47]

who studied DA efflux in the primate PFC during a delayed alternation and a subsequent visual discrimination task. Both tasks were similar to a large extent, but differed with respect to the delay component. Using in vivo microdialysis, Watanabe et al. [47] showed DA-release to occur mainly during the delayed alternation and not during the visual discrimination task. Recently, Phillips et al. [35] showed that DA efflux in the PFC of rats is increased in a phasic manner when a rat engages in search behavior for food reward on an eight arm radial maze guided by working memory. Furthermore, the magnitude of mesocortical DA-efflux was predictive of the working memory accuracy. These results clearly reveal that an increase of DA-release within the PFC accompanies cognitive events in which a goal state has to be held active in memory. Karakuyu et al. [25] used a new design to study DA-release in NCL during the short term memory component of working memory. Using in vivo microdialysis techniques, samples of extracellular fluid were collected every 20 min and analyzed for DA-concentrations. During this procedure pigeons were performing either a delayed matching-to-sample or a simultaneous matching-to-sample task. Both tasks differed only in the presence of a delay component in DMTS. The data revealed that an increase of DA-efflux within NCL was only related to this delay component. Neither slight differences in motor activity nor in reward amounts could explain variations in extracellular DA-concentration. Thus, identical to the data from the mammalian prefrontal cortex, DA-release in the avian NCL is related to episodes of holding active an internal representation of the relevant stimulus during its physical absence to guide a forthcoming response.

In mammals, dopaminergic neurons in the mesencephalon typically display a phasic burst at the onset of working memory tasks [43]. Yet although dopaminergic midbrain neurons respond only transiently to important events, DA-levels within PFC rise slowly, remain elevated for longer time periods and modulate receptors that are distant to the location of release [16]. This characteristic sluggishness of DA-levels within PFC probably plays a key role in integrating stimulus-driven input and DA-release, by allowing the latter to be less precise with respect to time and synaptic location. Neurochemical studies showed that PFC and striatum differ markedly in the regulation of extracellular DA. In the PFC, due to less extensive reuptake of extracellular DA by the DA transporter, the life time of released extracellular DA is greater and released DA can diffuse over much longer distances than in the striatum [45]. This favors a diffusion-mediated volume transmission of DA in the PFC [52]. In the striatum, however, radius and duration of DA efflux are rather minutely regulated by a highly active reuptake system. Thus, volume transmission probably represents a key feature enabling associative forebrain structures to integrate stimulus-driven events and DA-release [43]. An in vivo microdialysis study of the extracellular values of DA and its metabolites within the pigeon's NCL revealed that the ratio of homovanillic acid (HVA) to dihydroxyphenylacetic acid (DOPAC) was significantly greater in NCL than in the avian striatum [2]. Since an increase of HVA relative to DOPAC signals a lower reuptake by the DA transporter and a correspondingly greater proportion of extracellular DA, the avian NCL seems to utilize a volume transmission mode. Thus, the differential mode of DA-utilization in the avian NCL and striatum corresponds to those of the mammalian PFC and striatum.

On a cellular level, DA has several effects on voltage-gated and synaptic currents in PFC neurons. Acting via D1-receptors, dopamine enhances a persistent Na^+ current while reducing a slowly-inactivating, voltage-gated K^+ current, thus enhancing cell excitability [49]. By reducing dendritic HVA Ca^{2+} currents, DA might additionally reduce the impact of stimuli that could interfere with working memory processing, since this input arrives in superficial cortical layers and therefore synapses at the dendritic tufts of PFC neurons [49]. DA acting via D1-receptors has furthermore a major impact on all classes of synaptic currents, enhancing both excitatory NMDA and inhibitory $GABA_A$ currents [44]. Computational models integrating these features show that D1-receptor activation within PFC results in a stabilization of pyramidal neurons either in their low activity mode or in their sustained high activity mode, while at the same time suppressing background activity [14]. In this manner, a DA-release in PFC could result in self-sustained activity being more robust to distracting stimuli and keeping the system focused on a particular goal state [12].

Güntürkün et al. [21] analyzed the effects of DA on firing behavior of chick NCL neurons recorded in vitro, using both whole-cell and perforated patch-clamp techniques to analyze the cellular DA-mechanisms in birds. They showed that DA increased the firing frequency of preactivated principal neurons via D1-receptors but not of possibly GABAergic interneurons in chicks. Similar to reports of PFC neurons [48,50], the effect of DA followed an inverted U-curve with having an optimal effect on firing mode in middle concentrations. Additionally, DA had virtually no effect on neurons that were not depolarized. Although the detailed ionic mechanisms by which DA exerts its effects are presently not known in birds, the demonstration of a TTX-sensitive inward rectification in NCL makes it likely that, similar to mammals, D1-receptor activations could shift the threshold of the persistent Na^+ current in the avian forebrain towards more hyperpolarized potentials, such that the cell becomes more excitable already at lower membrane potentials [20,29]. Taken together, these analyses show remarkable similarities between PFC and NCL with respect to the cellular mechanisms with which DA increases the firing frequency of depolarized neurons.

4. Conclusion

The question that is central to this paper is, if working memory can be generated by different neural architectures or if there is only a single neural solution for its realization. I approached this question by comparing the mechanisms with

which working memory functions are performed in the PFC of mammals and in the NCL of birds. These two brain areas are very likely not homologous in terms of their phyletic continuity but are analogous in their general function. If multiple computational solutions for working memory would exist, it is likely that we would discover at some level important differences in the neural means that give rise to this memory system between mammals and birds. For the purpose of this review I thereby concentrated on the better analyzed maintenance function, which is largely identical to the old conception of short term memory.

The present overview shows that the neural mechanisms responsible for the maintenance of information during its physical absence are remarkably identical in PFC and NCL. The prefrontal areas in both mammals and birds show identical functional organizations as revealed in behavioral experiments. Their general anatomical and neurochemical architecture is also remarkably comparable [13,28]. These similarities also extend to the cellular level, from the neuronal activation patterns during memory tasks down to the biophysical mechanisms of synaptic currents. Both in mammals and birds, dopamine acts via D1-receptors to tune preactivated neurons into a mode that enables long-lasting, tonic spiking patterns with which stimuli or goal states can be held over lengthy periods of time until an appropriate response can be generated. Therefore, I suppose that the degrees of freedom to create different neural architectures to solve the problem of 'stimulus maintenance' are very small.

It is possible that this remarkable amount of convergence is related to some specificities of the dopaminergic system. The cellular cascade that characterizes the D1-receptor is shared identically by birds and mammals [6]. Activation of D1-receptors produces an increase in the firing frequency of preactivated neurons and so a strengthening of their synaptic coupling, while at the same time reducing the activity level of background neurons. This increase of the neuronal signal-to-noise ratio is uniquely suited for working memory. It is, therefore, conceivable that the cellular effects of a dopaminergic activation of D1-receptors are phylogenetically at the heart of working memory. Both birds and mammals might have used this commonly inherited biophysical mechanism to transform different brain entities into a convergent 'prefrontal' architecture.

Acknowledgement

Supported by the Deutsche Forschungsgemeinschaft.

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