ELSEVIER

Brain Research Bulletin 76 (2008) 317–323

BRAIN RESEARCH BULLETIN

www.elsevier.com/locate/brainresbull

Research report

Sustained activation and executive control in the avian prefrontal cortex

Michelle Milmine [a], Jonas Rose [b], Michael Colombo [a,*]

[a] *Department of Psychology, University of Otago, Dunedin, New Zealand*
[b] *Department of Biopsychology, Ruhr-University Bochum, Bochum, Germany*

Received 15 August 2007; received in revised form 22 November 2007; accepted 7 February 2008
Available online 3 March 2008

Abstract

We review our studies examining neural correlates of directed forgetting and executive control in the avian prefrontal cortex. One of the fundamental forms of executive control is the ability to selectively filter information, retaining that which is critical for the current purposes and discarding that which is not. In our first experiment, we trained birds on a directed-forgetting version of a delayed matching-to-sample task. Following a sample stimulus, a bird heard either a remember tone indicating that a memory test would follow, or a forget tone indicating that no memory test would be given. We found that neural activity in the avian prefrontal cortex increased when the bird was told to remember, and decreased when the bird was told to forget. Behavioral probe tests confirmed that the animals were forgetting on forget trials.

Although the sustained activation observed on remember trials and the absence of such activation on forget trials could be a code of remembering and forgetting the sample stimulus, it could also be a code of the possibility of obtaining a reward. To address this issue we conducted a second study in which we used three cues: remember, forget, and forget–reward. The forget–reward cue instructed the subject to forget the sample yet at the same time provided a free reward. Neural activity on forget–reward trials matched that on remember trials tentatively indicating that the sustained activation on remember trials might be a reward code rather than a sample stimulus code. Behavioral probe tests, however, failed to indicate that the animals were forgetting on forget–reward trials, and hence it still is possible that the sustained activation could be a code for memory of the sample stimulus.

Keywords: Sustained activation; Neural correlates of memory; Delay cells; Executive control; Prefrontal cortex; Nidopallium caudolaterale

That we remember is self evident, yet how remembering is expressed in the activity of neurons is almost completely unknown. One possible mechanism that has been proposed is that of sustained activation [4,9,20]. Sustained activation is best understood with reference to the delayed matching-to-sample (DMS) task, a classic test of short-term memory in animals [23]. At the end of an intertrial interval (ITI), a sample stimulus (e.g., a circle or a line) is presented on the center of three projectors (see Fig. 1). After the subject makes a response to the sample stimulus, the sample stimulus is turned off for a period of time (delay) during which memory for the sample stimulus is activated. At the end of the delay period, both stimuli (circle and line) appear on the side projectors (comparison). A correct response requires the subject to select the comparison stimulus that was the same as the sample stimulus. In the case of trial *n* in Fig. 1, the correct response is to press the circle stimulus, which results in a reward. Selecting the incorrect stimulus results in punishment, typically a 30 s to 1 min time-out from playing the DMS game. Following either reward or punishment there is a brief intertrial interval, and the next trial begins. A DMS session consists of a number of such trials with the circle and line stimuli each appearing equally often as the sample stimulus. The number of times that the circle and line stimuli appear as the sample stimulus, as well as the position of circle and line stimuli as comparison stimuli (e.g., circle-left/line-right or line-left/circle-right), are perfectly balanced within a session.

Humans, monkeys, rats, and birds can all be taught to play the DMS task [23]. In order to solve the DMS task, the subject must engage memory either by remembering the sample stimulus during the delay period (retrospective processing), or by

* Corresponding author.
E-mail address: colombo@psy.otago.ac.nz (M. Colombo).

0361-9230/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.brainresbull.2008.02.009

remembering, during the delay period, the comparison stimulus that it needs to respond to (prospective processing). Whether a retrospective or prospective code is adopted, the animal must remember some stimulus information, and so the question is: *How is this accomplished by the brain*? A number of investigators have found that some neurons in the brain increase their activity during the delay period, that is, show sustained delay activation [2,10,25] relative to the intertrial interval period of a DMS task. Such neurons are referred to as delay neurons, and because the increased activity occurs during the delay period when the subject should be remembering the sample stimulus, it is believed to represent a neural code of the subject remembering the sample stimulus. Examples of three delay neurons recorded from the brain of a bird playing a DMS task are shown in Fig. 2 [31]. This figure shows the baseline firing rate of the neurons during the intertrial interval period when the animal is not engaging memory, and a sustained activation during the delay period when memory is required. Activity to the sample stimulus is not a prerequisite for delay period activity to be seen. For example, neurons 1 and 3 show an increase in activity when the sample stimulus (S) is shown, whereas neuron 2 does not, yet in all three cases there is sustained activation during the delay period. We will discuss the significance of the sustained activation in the cue period, as well as this figure in general, in a later section.

Sustained activation has been noted in the brain of humans, monkeys, rats, and birds, and it is more than likely a universal feature of animals that show memory abilities. It has been

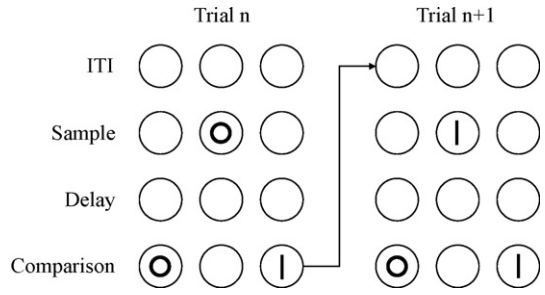

Fig. 1. Schematic diagram showing the sequence of events on two typical DMS trials. The large circles represent projectors onto which the stimuli, in this case a circle and a line, are backprojected. In front of each projector is a clear plastic key that serves as a response mechanism. Note that during both the intertrial interval (ITI) and the delay period there is nothing displayed on the projectors, but only in the case of the delay period is the subject engaging memory for the sample stimulus.

suggested that delay activity represented a neural code of memory [2,10,25], that is, sustained activation is the brain's way of remembering the sample stimulus. However, it does not have to be the case that the sustained activation is a code of the sample stimulus [4]. Indeed, there are other plausible mechanisms that could account for the increased activation during the delay period. At the simplest level, the sustained activation may reflect a general arousal code, for the delay period is close in time to when a response is required. Alternatively, given that the subject knows that soon the comparison period will appear and it will be required to make a selection between two stimuli, the sus-

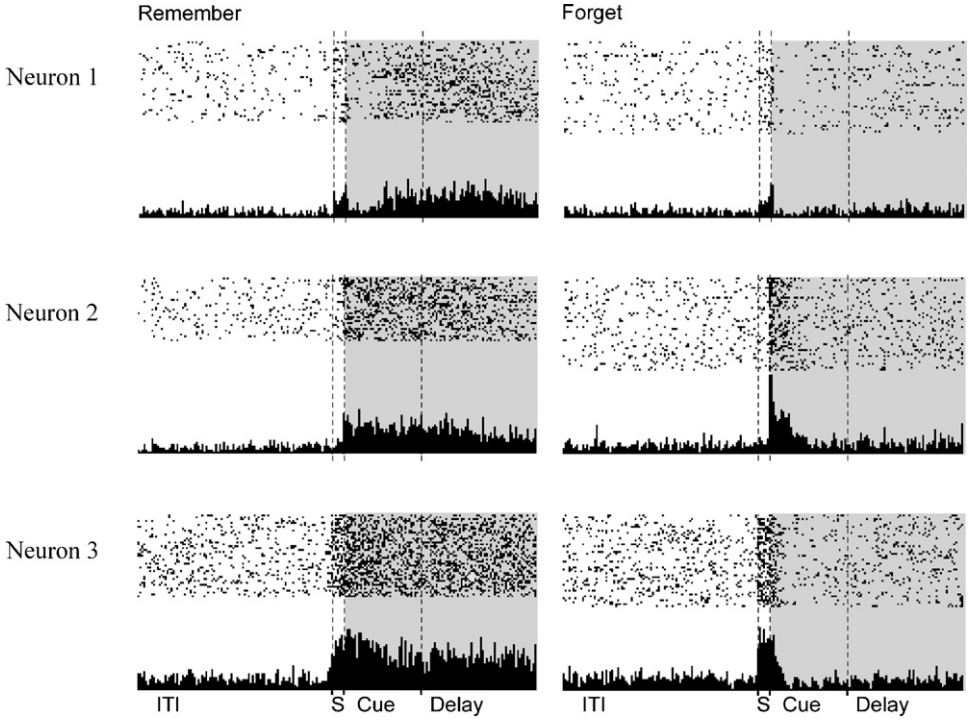

Fig. 2. Three examples of delay neurons. Neural activity was sampled across the middle 5 s of the 15-s ITI. For the sample stimulus, activity was sampled across a 300-ms period from 400 ms prior to a keypeck to 100 ms prior to a keypeck. For the cue and delay periods, activity was sampled across their entire 2-s and 3-s periods, respectively. For further details see [31]. Note the sustained activation seen in the cue and delay periods. The dots represent rasters and the binwidth is 50 ms. To the left is the neural activity on remember trials, whereas to the right is the neural activity on forget trials. Note that on forget trials the sustained activation is abolished. ITI: intertrial interval; S: sample stimulus. Reprinted from [31].

tained activation could simply reflect an increased attentional state directed at the spatial location where the stimuli might be presented [21]. Finally, the sustained activation might indicate the fact that, assuming the subject makes a correct response, a reward is forthcoming. With respect to reward, at the very least the sustained activation in the delay period may signal that the opportunity to obtain a reward is imminent.

In some cases, simpler explanations of the sustained activity in the delay period, such as it reflecting general arousal, or even it being an attentional code or a reward code, can be discounted. For example, there are many situations where neurons show sustained activation in the delay period after only one of the sample stimuli used in the DMS task and not the other [2]. Such findings make it difficult to account for the sustained activation in terms of some arousal code, attentional code, or reward code, because equal arousal and attention ought to be dedicated following both stimuli and, likewise, both stimuli are predictive of the possibility of reward. Yet such selective delay neurons do fit with the idea that the sustained activation represents a sample code, because just as we know that some neurons in the brain code for one color and not another, so too would we expect there to be neurons that are differentially involved in the memory of different colors, especially given that seeing a stimulus activates the same cortical regions as remembering a stimulus [7].

1. The prefrontal cortex and executive control of behavior in primates

Damage to the prefrontal cortex (PFC) of primates causes impairments in both working memory and executive control abilities, that is, processes that operate on working memory. In humans, the PFC is activated under conditions that require working memory [24] or executive control [34]. In nonhuman primates, PFC neurons show sustained activation during the delay period of DMS tasks [9]. In addition, evidence that PFC neurons are modulated by attentional demands [28] and encode abstract rules [35] has been taken as evidence that the nonhuman primate PFC is also involved in executive control.

2. The avian NCL: analogue of PFC?

The nidopallium caudolaterale (NCL) [36] is a multimodal telencephalic region situated in the posterior pallium of birds. Divac and coworkers [5,26] were the first to suggest that the avian NCL, what they called the postero-dorsolateral neostriatum, might correspond to the mammalian PFC. Since that time, considerable anatomical and behavioral evidence has led to the current view that although the NCL and PFC are not homologous structures, they are analogous structures [14,15]. Anatomically, both the PFC and NCL are the main integrative areas of the brain, ideally situated to serve the function of executive control by translating sensory information into action. Both have very similar input and output connections. For example, both receive projections from modality-specific secondary visual, auditory, and somatosensory areas, and both project to motor and limbic areas of the brain [18,19]. One of the features of the PFC in primates is that it is densely innervated by midbrain dopaminergic fibers, and the same is true for the NCL [5,15].

This is not to say that there are no anatomical differences between the PFC and the NCL, for that would be unlikely given at least 300 million years of independent evolution. For example, in primates, the mediodorsal (MD) nucleus of the thalamus projects to the PFC [11]. In birds, the main thalamic projection to the NCL is the nucleus dorsolateralis posterior thalami (DLP) [36]. Unfortunately, the afferent and efferent connections of the DLP are not entirely consistent with the afferent and efferent connections of the MD [3]. Nevertheless, recent research suggests that despite the differences in connectional patterns, the DLP may serve the same function as the MD [13].

There is also considerable behavioral/lesion data to suggest that the consequences of damage to the PFC and NCL are similar. For example, damage to the NCL and PFC result in impairments on delayed alternation and pattern-reversal tasks while having little or no effect on simultaneous visual discriminations and basic sensory processes [8,13,16,26]. In addition, blockade of D1 receptors in NCL and PFC both cause impairments on tasks sensitive to PFC and NCL damage [14,15]. In short, while not a homologue of the PFC, the NCL is clearly an analogue of the PFC.

3. Neural correlates of executive control in birds

Perhaps the most elegant example of executive control is the ability to selectively filter information, retaining that which is necessary and discarding that which is not [34]. Rose and Colombo [31] examined whether neurons in the avian NCL also engaged in selective filtering of information. Pigeons were trained on a directed-forgetting version of a DMS task in which following the sample period was a cue period during which either a high-frequency tone (HT) or low-frequency tone (LT) was played (Fig. 3, left and center panels). The cues instructed the animal to either remember or forget the sample stimulus that it had just seen. The HT was the remember cue, and indicated that after the delay period the birds would be shown two comparison stimuli and required to peck the one they had seen previously as the sample stimulus to obtain a reward (remember trial). The LT was the forget cue, and indicated that after the delay period the trial would effectively end (forget trial). Thus, the remember cue instructed the subject that it had better remember the sample stimulus because it was going to be given a memory test, whereas the forget cue instructed the subject that it did not need to remember the sample stimulus because no memory test was forthcoming. Within a session there were an equal number of remember and forget trials.

We recorded from 124 NCL neurons. Of these, 83 were classified as delay neurons in that they showed sustained activation during the delay period on remember trials. By sustained activation we mean that they fired significantly more during the delay period than during the baseline intertrial interval period. Of these 83 memory neurons, the majority (76%) showed sustained activation on remember trials and no sustained activation on forget trials. Examples of three memory neurons whose activity was modulated by the remember and forget cues is shown in Fig. 2.

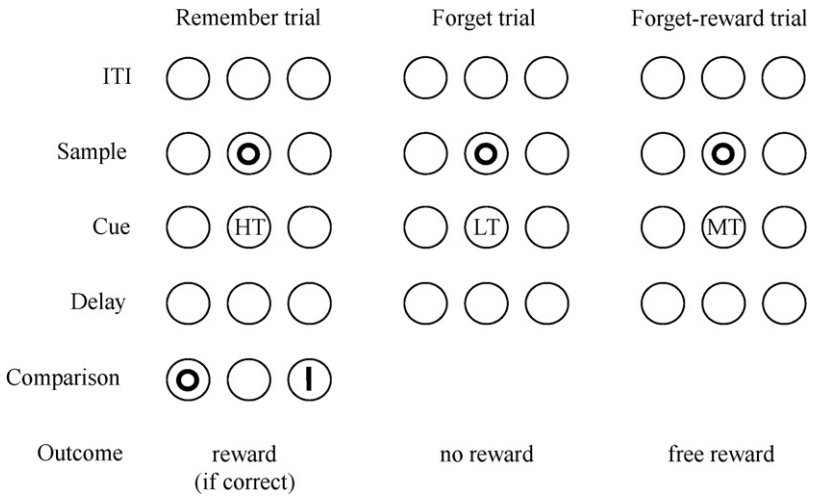

Fig. 3. Sequence of events on remember (left panel), forget (center panel) and forget–reward (right panel) trials. The circle and line are the stimuli used on the DMS task. HT, LT, and MT, refer to a high-frequency (remember), low-frequency (forget), and middle frequency (forget–reward) cue. In [31], only remember and forget trials were used. In the second study, all three trial types were used.

For each of these three neurons the remember cue was followed by an increase in activity that persisted throughout the cue and delay periods. In contrast, the forget cue triggered a drop in activity to baseline (intertrial interval) levels, and this drop persisted throughout the cue and delay periods. In short, following instructions to remember the sample stimulus, the cells exhibited sustained activation throughout the cue and delay periods, whereas following instructions to forget the sample stimulus, the sustained activation was abolished.

The modulation of neural activity by remember and forget cues was not only seen at the level of individual neurons, but also seen across the entire population of delay neurons that we sampled (Fig. 4A). For ease of exposition we only present the data for excitatory neurons, although the effect we describe was also statistically present for inhibitory neurons. (The terms "excitatory" and "inhibitory" generally refer to neuroanatomical classes of neurons, and their effects on another neuron. Thus, an "excitatory" neuron is one that increases the firing rate of another neuron. In the current manuscript, however, "excitatory" and "inhibitory" neurons refer to neurons that show an increase and decrease, respectively, in activity during the delay period relative to the intertrial interval period). On average, the cells increased their firing rate when the subject was presented with the sample stimulus. The cells maintained a high-firing rate while the cue to remember was played (solid line) and continued to fire at a high rate during the delay period when the animal was remembering the sample stimulus. In contrast, these same cells decreased their firing rate after the cue to forget was played (dotted line), and the decreased firing rate was maintained throughout the remainder of the cue period and all of the delay period.

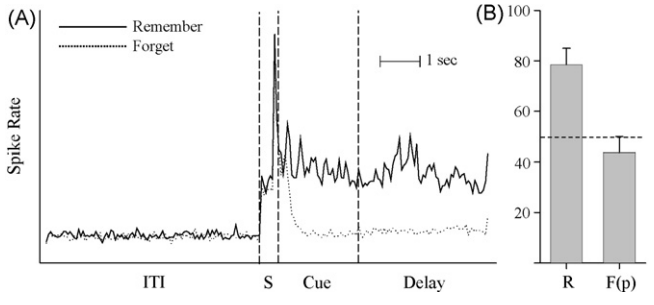

Fig. 4. (A) Performance across the population of 61 excitatory delay cells from the Rose and Colombo [31] study. To account for differences in firing rates across the neurons, each neuron's firing rate was normalized against its baseline activity. The timescales are the same as described in Fig. 2. The dashed vertical lines separate the different periods of the DMS task. (B) Performance on the behavioral probe test sessions. The horizontal line indicates chance levels of performance. ITI: intertrial interval; S: sample stimulus period; R: performance on remember trials during the probe test; F(p): performance on the forget-probe trials during the probe test.

4. Behavioral evidence of forgetting

The forget cue directs the subject to forget the sample stimulus because it predicts the absence of a memory test. If we do not test the subject's memory after the forget cue, then how is it possible to know that the subject has indeed forgotten the sample stimulus? We tested this issue by occasionally presenting the subject with forget-probe trials. In a forget-probe trial, the subject is presented with the forget cue, but then, against its prediction, the comparison stimuli appear. Such forget-probe trials must be presented very rarely to the subject, otherwise the ability of the forget cue to predict the absence of a memory test would be jeopardized. The performance on the forget-probe trials and on the remember trials is shown in Fig. 4B. Overall, performance on the forget-probes was 43% correct, a value that did not differ from chance. In contrast, performance on remember trials was 79% correct, a value that was significantly above chance. Thus, the remember and forget cues do seem to be directing the subject to remember and forget the sample stimulus, respectively.

5. What does sustained activation on remember trials represent?

Rose and Colombo [31] showed that when birds are told to remember, there was sustained activation during the delay

period, and when they are told to forget, the sustained activation during the delay period was abolished. To date, this is convincing evidence that the delay period sustained activation is a code for the subject remembering the sample stimulus. We also recognize that the sustained activation may represent a prospective code of the comparison stimulus that needs to be responded to [29]. In our procedure, in which the sample stimulus is repeated as one of the comparison stimuli, whether the subject engaged retrospective processing and remembers the sample stimulus, or prospective processing and remembers the correct comparison stimulus to be responded to, they are effectively remembering the same stimulus. Thus while we recognize that a prospective code is possible, for ease of exposition we only discuss the delay activity in terms of a retrospective code of the sample stimulus.

Unfortunately, our experiment does not distinguish one other possibility for the sustained delay activation on remember trials, that is, that the increased activity may merely represent a neural code for the fact that a reward is possible. Recall that the remember cue tells the subject to remember the sample stimulus, but it also tells the subject that a reward is available if a correct response is made. Likewise, the forget cue tells the subject to forget the sample stimulus, but it also tells the subject that no reward is forthcoming. Thus the increased activity on remember trials and the decreased activity on forget trials could represent the subject remembering and forgetting the sample stimulus, but it could also represent the subject anticipating that a reward is possible and that no reward is possible. We are simply not able to untangle these two possibilities.

In an effort to address the above issues we conducted a second experiment with two new birds. For the most part, the procedure of the second experiment was similar to that of the first experiment. The differences were as follows: in addition to the standard remember and forget cues, the second experiment also used a third, forget–reward cue (Fig. 3, right panel). The forget–reward cue consists of a medium-frequency tone (MT; 2750 Hz) easily distinguishable from the HT (5000 Hz) and LT (500 Hz) not only on the basis of frequency, but also on the basis that, unlike the HT and LT, the MT was pulsed on and off (.5 s on and .5 s off resulting in five on–off periods during its 2-s presentation). Like the LT on forget trials, the MT on forget–reward trials also instructs the subject to forget the sample stimulus, that is, on forget–reward trials there is no memory test after the delay period. However, unlike the LT on forget trials, the MT on forget–reward trials also tells the subject that a free reward will be delivered at the end of the delay period. As in the first experiment, we tested the subjects' memory of the sample stimulus following the forget and forget–reward cues by occasionally delivering probe trials in which the forget and forget–reward cues were followed by a comparison period.

Our hypothesis was as follows: if the increased activity of delay neurons represents a code of the sample stimulus, then we should see increased activity in the delay period on remember trials, and a decrease in activity on forget and forget–reward trials. On the other hand, if the delay activity represents a code of the possibility of obtaining a reward, then we should see an increase in activity in the delay period on remember and forget–reward trials, and a decrease in activity on forget trials.

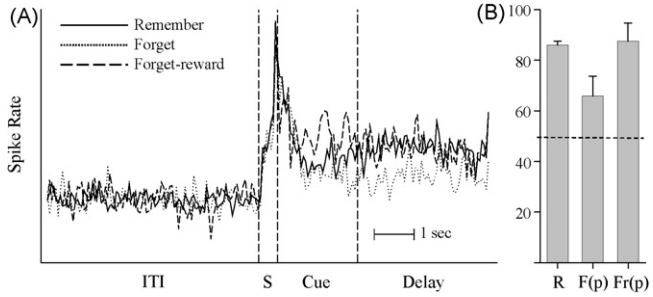

Fig. 5. (A) Performance across the population of 16 excitatory delay cells in the second study. To account for differences in firing rates across the neurons, each neuron's firing rate was normalized against its baseline activity. The timescales are the same as described in Fig. 2. The dashed vertical lines separate the different periods of the DMS task. (B) Performance on the behavioral probe test sessions. The horizontal line indicates chance levels of performance. ITI: intertrial interval; S: sample stimulus; R: performance on remember trials during the probe test; F(p): performance on the forget-probe trials during the probe test; Fr(p): performance on forget–reward probe trials during the probe test.

We recorded from 126 NCL neurons. Of these, 81 (64.3%) were classified as delay neurons, that is, they showed an increase in activity during the delay period on remember trials. The results from this second study are shown in Fig. 5A. Although the data are more variable than that reported in Fig. 4A, a fact likely due to a smaller number of excitatory delay neurons encountered, the pattern across the two birds was consistent. Again, for ease of exposition we only present the data from excitatory neurons (neurons that show an increase in activity during the delay period), although the effect we describe is also statistically present for inhibitory neurons (neurons that show a decrease in activity during the delay period). We conducted a two-way analyses of variance with condition (3: remember, forget, and forget–reward) and bins (100 and 60 in the intertrial interval and delay periods, respectively) as factors, with repeated measures over both condition and bins. The main effect of condition was significant for the delay period, $F(2, 34) = 5.50$, $p < .05$ period, but not for the ITI period ($p = .80$). For the delay activity, paired t-tests based on the average values of the delay activity revealed a significant difference in activity between the remember and forget conditions, $t(17) = 3.53$, $p < .01$, and between the forget–reward and forget conditions, $t(17) = 2.13$, $p < .05$, but not between the remember and forget–reward conditions ($p = .87$). In other words, activity on the forget–reward trials mirrored that on the remember trials.

The sustained delay activity on the forget–reward trials clearly maps onto the sustained delay activity on the remember trials. This would suggest that the sustained activity is a code of the reward rather than a code of the sample stimulus. But for this to be the case one other condition has to be met, that is, the animals should be forgetting on forget–reward trials. If the animals are forgetting on forget–reward trials and the forget–reward activity maps onto the remember activity, then the sustained delay activity must clearly be a code of the upcoming reward. The reason is that given that the neural activity of forget–reward and remember trials is very similar, we must look for a dimension on which these trials are also similar. They are not similar on the basis of memory for the sample stimulus,

because one tells the subject to remember and the other tells the subject to forget. But they are similar on the basis of the reward because both remember and forget–reward trials instruct the subject that a reward is at least possible. Once again, the key is how they perform on the behavioral probe tests.

The data for the behavioral probe tests is shown in Fig. 5B. Once again, performance on the forget-probe trials was not different from chance. More importantly, the performance on the forget–reward probe trials mirrored performance on remember trials. In other words, contrary to the fact that the forget–reward cue was a cue to forget the sample stimulus, the animals were not forgetting the sample stimulus. As such, it is not possible to conclude whether the sustained activation is a code of the sample or a code of the reward. The only way we could have come to this conclusion is if the birds had shown evidence of forgetting on forget–reward trials, and they did not.

6. Future directions

At present we are left with only indirect means of assessing whether the sustained activation reflects either a sample code or a reward code. For example, there is considerable evidence that the neural activity of PFC neurons is modulated by the magnitude, preference, or certainty of a reward [22,27,37]. Likewise, neural correlates of magnitude and preference of rewards have also been noted in the avian brain [17,38]. With respect to the issue of certainty, the certainty of reward following forget–reward trials (100%) is surely greater than following a remember trial, where performance averaged around 85–90% correct. Yet the neural data were such that the level of neural activity following forget–reward trials was identical to that following remember trials. This would not be predicted if the certainty of a reward has an effect on neural activity. However, if both the remember and forget–reward trials were invoking rehearsal mechanisms, then you would predict similar levels of neural activity. Thus one could interpret the similar levels of neural activity as evidence, albeit indirect, that the sustained activation represents a code of the sample stimulus. Unfortunately, these indirect means are interesting to pursue, but they do not provide us with definitive answers. The key for future studies is to find ways to present free rewards yet at the same time ensure that the forget cue functions in its intended role [12].

Earlier we argued that selective delay activity, that is, delay activity that occurs after one sample stimulus and not the other, rules out simple nonmemorial explanations of delay activity. It is interesting to wonder whether selective neurons might be coding for the sample stimulus, whereas the nonselective neurons might be coding for the reward. If this is the case, we might expect that for the selective neurons, which hypothetically code for the sample stimulus, the forget–reward activity would map onto the forget activity, whereas for the nonselective neurons, which hypothetically code for the reward, the forget–reward activity would map onto the remember trials. Unfortunately we found no evidence for a differential pattern of mapping between selective and nonselective neurons; in both cases the forget–reward activity mapped onto the remember activity.

Just as one can categorize delay units on the basis of whether they are selective or nonselective, units can also be categorized on the basis of the pattern of delay activity they exhibit. Delay units have been shown to exhibit, across the delay period, either slow increasing (ramping) activity, slow decreasing (decaying) activity, or consistent (stable) activity [33]. In both the Rose and Colombo [31] study and the second study we noticed all three of these types of activity in our NCL neurons, although in contrast to Shafi et al. [33], who found that ramping activity was more common in PFC, we found that stable activity was more common in NCL. It must be kept in mind that Shafi et al.'s [33] findings are based on two PFC studies, both conducted with monkeys and in the same laboratory. The extent to which our findings of a greater number of stable delay cells in NCL reflect an avian peculiarity, or subtle procedural differences between the two laboratories, is still unclear. That said, Shafi et al. [33] have highlighted an important issue that there may be different types of delay cells and these different delay cells may each be involved in coding different aspects of the task, such as memory for the sample and memory for upcoming reward.

Another issue concerns the fact that there is a difference in the burden on memory between directed-forgetting tasks used in humans and those used with nonhuman subjects [39]. In the case of humans tested on a directed-forgetting task [1], a list of stimuli are presented, each followed by either a remember or a forget cue. Because the list is typically long, there is a premium on memory allocation, and as a result the remember and forget cues are used to allow access into memory that information which is relevant, and to prevent access into memory that information which is not. In other words, the remember and forget cues can actually be used to relieve the load on memory, and it appears that this is exactly what humans do. In the case of the DMS tasks used in most studies of directed forgetting in pigeons, however, there is only one cue to remember (or forget). Increasing the list of items to remember may place a sufficient burden on memory to encourage the animals to utilize the remember and forget cues for their intended purpose [30]. Such studies are currently underway.

A final issue concerns the role of dopamine in working memory, and our observation that it was not possible to determine whether the sustained activation in the delay period represents a sample code or a reward code. According to Durstewitz et al. [6], dopamine functions to maintain the stability of the sustained activation in the delay period. Given that reward-predictive stimuli activate midbrain dopaminergic neurons and cause a release of dopamine in the PFC [32], and given that the remember and the forget–reward cues act as predictors of a reward, it is very likely that they cause dopamine release in the NCL. In contrast, the forget cue, which predicts the absence of a reward, should not cause a release of dopamine. If dopamine contributes to the stability of delay activity, the mere fact that the forget–reward cue predicts a reward might cause the maintenance of sample related activity and in turn stabilize delay activity and working memory for the sample. In other words, the mechanism underlying executive control of what to maintain in working memory might be very closely tied to the expectation of a reward. This close tie between reward prediction and delay activity might

explain the experimental difficulties in untangling whether the delay period sustained activation is a code of the sample or a code of the reward.

Acknowledgements

This research was supported by a Neurological Foundation of New Zealand Grant 0528-PG to M. Colombo. We thank Arii Watanabe for assistance with data collection.

References

[1] R.A. Bjork, Theoretical implications of directed forgetting, in: A.W. Melton, E. Martin (Eds.), Coding Processes in Human Memory, Winston, Washington, DC, 1972, pp. 217–235.
[2] M. Colombo, C.G. Gross, Responses of inferior temporal cortex and hippocampal neurons during delayed matching-to-sample in monkeys (*Macaca fascicularis*), Behav. Neurosci. 108 (1994) 443–455.
[3] Csillag, C.M. Montagnese, Thalamotelencephalic organization in birds, Brain Res. Bull. 66 (2005) 303–310.
[4] R. Desimone, The physiology of memory: recordings of things past, Science 258 (1992) 245–246.
[5] I. Divac, J. Mogensen, A. Bjorklund, The prefrontal 'cortex' in the pigeon. Biochemical evidence, Brain Res. 332 (1985) 365–368.
[6] D. Durstewitz, M. Kelc, O. Güntürkün, A neurocomputational theory of the dopaminergic modulation of working memory functions, J. Neurosci. 19 (1999) 2807–2822.
[7] M.J. Farah, The neural basis of mental imagery, Trends Neurosci. 12 (1989) 395–399.
[8] J.M. Fuster, The Prefrontal Cortex: Anatomy, Physiology and Neuropsychology of the Frontal Lobe, 3rd ed., Lippincott–Raven, Philadelphia, 1997.
[9] J.M. Fuster, G.E. Alexander, Neuron activity related to short-term memory, Science 173 (1971) 652–654.
[10] J.M. Fuster, J.P. Jervey, Neuronal firing in the inferotemporal cortex of the monkey in a visual memory task, J. Neurosci. 2 (1982) 361–375.
[11] M. Giguere, P.S. Goldman-Rakic, Mediodorsal nucleus: areal, laminar, and tangential distribution of afferents and efferents in the frontal lobe of rhesus monkeys, J. Comp. Neurol. 277 (1988) 195–213.
[12] D.S. Grant, A.S. Soldat, A postsample cue to forget does initiate an active forgetting process in pigeons, J. Exp. Psychol. Anim. Behav. 21 (1995) 218–228.
[13] O. Güntürkün, Cognitive impairments after lesions of the neostriatum caudolaterale and its thalamic afferent in pigeons: functional similarities to the mammalian prefrontal system? J. Hirn. 38 (1997) 133–143.
[14] O. Güntürkün, Avian and mammalian prefrontal cortices: zero degrees of freedom in the evolution of the cellular mechanisms of goal-state maintenance, Brain Res. Bull. 66 (2005) 311–316.
[15] O. Güntürkün, The avian 'prefrontal cortex' and cognition, Curr. Opin. Neurobiol. 15 (2005) 686–693.
[16] B. Hartmann, O. Güntürkün, Selective deficits in reversal learning after neostriatum caudolaterale lesions in pigeons: possible behavioral equivalencies to the mammalian prefrontal system, Behav. Brain Res. 96 (1998) 125–133.
[17] E. Izawa, N. Aoki, T. Matsushima, Neural correlates of the proximity and quantity of anticipated food rewards in the ventral striatum of domestic chicks, Eur. J. Neurosci. 22 (2005) 1502–1512.
[18] E.G. Jones, T.P.S. Powell, An anatomical study of converging sensory pathways within the cerebral cortex of the monkey, Brain 93 (1970) 793–820.
[19] S. Kröner, O. Güntürkün, Afferent and efferent connections of the caudolateral neostriatum in the pigeon (*Columba livia*): a retro- and anterograde pathway tracing study, J. Comp. Neurol. 407 (1999) 228–260.
[20] K. Kubota, H. Niki, Prefrontal cortical unit activity and delayed alternation performance in monkeys, J. Neurophysiol. 34 (1971) 337–347.
[21] M.A. Lebedev, A. Messinger, J.D. Kralik, S.P. Wise, Representation of attended versus remembered locations in prefrontal cortex, PLoS Biol. 2 (2004) 1919–1935.
[22] M.I. Leon, M.N. Shadlen, Effect of expected reward magnitude on the response of neurons in the dorsolateral prefrontal cortex of the macaque, Neuron 24 (1999) 415–425.
[23] A.J. Lock, M. Colombo, Cognitive abilities in a comparative perspective, in: A. Lock, C.R. Peters (Eds.), Handbook of Human Symbolic Evolution, Clarendon Press, Oxford, 1996, pp. 596–643.
[24] E.K. Miller, J.D. Cohen, An integrative theory of prefrontal cortex function, Annu. Rev. Neurosci. 24 (2001) 167–202.
[25] Y. Miyashita, H.S. Chang, Neuronal correlate of pictorial short-term memory in the primate temporal cortex, Nature 331 (1988) 68–70.
[26] J. Mogensen, I. Divac, The prefrontal 'cortex' in the pigeon. Behavioral evidence, Brain Behav. Evol. 21 (1982) 60–66.
[27] J. Quintana, J.M. Fuster, Mnemonic and predictive functions of cortical neurons in a memory task, Neuroreport 3 (1992) 721–724.
[28] G. Rainer, W.F. Asaad, E.K. Miller, Selective representation of relevant information by neurons in the primate prefrontal cortex, Nature 393 (1998) 577–579.
[29] G. Rainer, S.C. Rao, E.K. Miller, Prospective coding for objects in primate prefrontal cortex, J. Neurosci. 19 (1999) 5493–5505.
[30] K.L. Roper, D.H. Kaiser, T.R. Zentall, True directed forgetting in pigeons may occur only when alternative working memory is required on forget-cue trials, Anim. Learn. Behav. 23 (1995) 280–285.
[31] J. Rose, M. Colombo, Neural correlates of executive control in the avian brain, PLoS Biol. 3 (2005) 1139–1146.
[32] W. Schultz, Getting formal with dopamine and reward, Neuron 36 (2002) 241–263.
[33] M. Shafi, Y. Zhou, J. Quintana, C. Chow, J. Fuster, M. Bodner, Variability in neuronal activity in primate cortex during working memory tasks, Neuroscience 146 (2007) 1082–1108.
[34] E.E. Smith, J. Jonides, Storage and executive processes in the frontal lobes, Science 283 (1999) 1657–1661.
[35] J.D. Wallis, K.C. Anderson, E.K. Miller, Single neurons in prefrontal cortex encode abstract rules, Nature 411 (2001) 953–956.
[36] C. Waldmann, O. Güntürkün, The dopaminergic innervation of the pigeon caudolateral forebrain: immunocytochemical evidence for a 'prefrontal cortex' in birds? Brain Res. 600 (1993) 225–234.
[37] M. Watanabe, Reward expectancy in primate prefrontal neurons, Nature 382 (1996) 629–632.
[38] S. Yanagihara, E. Izawa, K. Koga, T. Matsushima, Reward-related neuronal activities in basal ganglia of domestic chicks, Neuroreport 12 (2001) 1431–1435.
[39] T.R. Zentall, K.L. Roper, L.M. Sherburne, Most directed forgetting in pigeons can be attributed to the absence of reinforcement on forget trials during training or to other procedural artefacts, J. Exp. Anal. Behav. 63 (1995) 127–137.