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Neurophysiological Studies of Learning and Memory in Pigeons

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The literature on the neural basis of learning and memory is replete with studies using rats and monkey, but hardly any using pigeons. This is odd because so much of what we know about animal behavior comes from studies with pigeons. The unwillingness to use pigeons in neural studies of learning and memory probably stems from two factors, one that the avian brain is seen as radically different from the mammalian brain and as such can contribute little to its understanding, and the other that the behavior of pigeons is not seen as sophisticated as that of mammals, and certainly primates. Studies over the past few decades detailing the remarkable cognitive abilities of pigeons, as well as a newly revised nomenclature for the avian brain, should spark a renewed interest in using pigeons as models to understand the neural basis of learning and memory. Here we review studies on the pigeon's hippocampus and 'prefrontal cortex' and show that they provide information not only on the workings of the avian brain, but also shed light on the operation of the mammalian brain.

Keywords: hippocampus, NCL, avian, memory, single-unit

Background

So much of what we know about the principles of behavior has been learned from studies with pigeons, and there is little doubt that these principles apply to mammalian behavior (Skinner, 1953). Despite this, those conducting neuroscience studies with mammals such as rats and monkeys rarely reference avian neuroscience studies. The purpose of this review is not only to highlight the value of using pigeons in neurophysiological investigations of brain function, but also to illustrate that these studies are relevant for our understanding of not just mammalian brain function, but also primate brain function.

On the basis of cost-effectiveness, ease of use, and more importantly on the basis of the ethical imperative of using the species with the least complicated neural system to achieve our goals, pigeons are an ideal choice of species to use in neurophysiological studies. They can be trained on most, if not all, of the tasks that are used to explore the neural basis of learning and memory in rats and monkeys, and there is little evidence that they solve tasks like discrimination learning, delayed matching-to-sample, and serial-order any differently from them (Colombo, Cottle, & Frost, 2003; Scarf & Colombo, 2010, 2011). In addition, while rats perform spatial tasks with ease and visual tasks with difficulty, and monkeys perform visual tasks with ease and are more difficult to test on spatial tasks, pigeons can easily perform both types of tasks. In the current review we hope to convince researchers, especially those younger scientists embarking on a career studying the neural basis of learning and memory, that pigeons are an excellent species of choice to use in such studies.

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The Avian Brain

Given that pigeons are behaviorally ideal subjects to use, why have so few studies used them to examine the neural basis of learning and memory? It would appear that the biggest obstacle to using pigeons in neurophysiological research seems to be the fact that we are dealing with an avian brain

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rather than a mammalian brain, and therefore the ability to extrapolate from one to the other is perceived as being limited. This issue is interesting because it has not prevented the transfer of behavioral knowledge to the mammalian condition, yet it has prevented the transfer of neurophysiological knowledge.

The unwillingness to accept neural studies in pigeons as relevant to our understanding of the mammalian brain may have been due to an outdated and incorrect avian brain nomenclature that pervaded the literature for many years. In this old nomenclature, most avian brain structures ended with the term 'striatum' (Jarvis et al., 2005; Reiner, 2005; Reiner, Yamamoto, & Karten, 2005; Shimizu, 2009). In mammals, the striatum consists of the caudate nucleus and the putamen, that along with the globus pallidus form a structure known as the basal ganglia. Damage to this region produces a variety of disorders, chief among them are posture and movements disorders (Banich, 1997). The basal ganglia have also been implicated in procedural memory, a low-level type of memory that is used for forming habits such as riding a bike (Packard, Hirsh, & White, 1989; Squire, 1992). Because the avian brain consisted of a number of structures all that ended with the term 'striatum' (e.g., ectostriatum, neostriatum), this fuelled the view that the bird's brain was one complex motor system, and that at best birds could engage in a very low-level reflexive procedural memory. Their value as a model to understand the neural mechanisms of learning and memory, not to mention more complex behavior, at least in the eyes of people conducting research with mammals, was very limited. As stated elegantly by Reiner (2005) "…the outdated terminology has clearly been an impediment to the assimilation of avian brain research findings into the broader body of neuroscience findings" (p. 323).

The fact is, structures such as the avian ectostriatum and neostriatum have nothing whatsoever to do with the striatum in mammals. How then did all structures in the avian brain come to be labelled with the 'striatum' misnomer? The history of this issue was elegantly reviewed by Reiner (2005). Briefly, the telencephalon of mammals is characterized by two main cytoarchitectural regions, an outer six-layered cell region with dendrites arranged in an ascending fashion called the neocortex, and an inner region composed of uniformly distributed cells with dendrites arranged in a radial fashion called the basal ganglia. Inspection of the avian telecephalon, especially with the techniques available at the turn of the 20th century, revealed no layering of cells, but rather one region more similar in appearance to the basal ganglia of mammals. Hence, the avian telencephalon was viewed as one large hypertrophied basal ganglia, a view that fit perfectly with the perception at the time that birds were not terribly smart creatures.

Over the past 20 years, however, our view of the cognitive abilities of birds has changed dramatically. Birds are now seen to posses an amazing repertoire of cognitive abilities such as transitive inference (Paz-y-Miño, Bond, Kamil, & Balda, 2004), tool use (Weir, Chappell, & Kacelnik, 2002), as well as a highest form of memory known as episodic memory (Clayton & Dickinson, 1998). And these amazing abilities are not restricted to members of the corvid family. Pigeons also have been shown to perform as well as monkeys on tasks of concept formation (Colombo et al., 2003; Wright, 1997), and serial-order knowledge (Scarf & Colombo, 2010, 2011; Terrace, 1987; Wright, Santiago, Sands, Kendrick, & Cook, 1985). These behavioral findings, along with modern histochemical techniques that indicate that the avian telencephalon is in fact pallial (i.e., cortical) but organized with a nuclear rather than laminar architecture (Karten & Shimizu, 1989), has prompted a reconsideration of avian brain nomenclature. In July of 2002, the Avian Brain Nomenclature Forum was held at Duke University, and a revised nomenclature for the avian brain was put forth (Jarvis et al., 2005; Reiner et al., 2004). The key notion to emerge from this meeting was that the avian brain consists of a large amount of pallial tissue of which a major fraction is homologous to the cortex of mammals. As a result, most of the 'striatum' terms were discarded and replaced with the more accurate 'pallial' terms.

Armed with a far more accurate nomenclature, it remains for avian neuroscientists now to convince mammalian neuroscientists that the avian brain is an excellent model in which to study the neural basis of learning and memory. More importantly, it is critical for neuroscientists studying the avian brain to not just show that the same mechanisms that are present in the mammalian brain are present in the avian brain, but that avian neuroscience studies can make contributions in their own right that further our understanding of mammalian brain function. The neuroscience research that my colleagues and I have been engaged in over the past 20 years has been one small step in that direction.

Avian Hippocampal Studies

Basic Findings. Does damage to the hippocampus in birds cause the same constellation of deficits as damage to the hippocampus in mammals? It had been known for some time that spatial problems are common following hippocampal lesions in rats (Olton, Becker, & Handelmann, 1979) and humans (Milner, 1965). In some of the early avian studies exploring this issue, Bingman and colleagues had shown that damage to the hippocampus impairs certain aspects of homing in pigeons (Bingman, Ioale, Casini, & Bagnoli, 1990; Bingman & Yates, 1992), and Sherry and Vaccarino (1989) had shown that hippocampal lesions caused disruptions in memory for food caches in black-capped chickadees. Despite this concordance, in the early 90s the idea that the avian hippocampus might have the same function as the mammalian hippocampus seemed unlikely given that the architecture of the avian and mammalian hippocampi are very different. In primates, the hippocampus is a roughly tubular structure that runs along the anterior/posterior extent of the medial temporal lobe (Figure 1A), whereas in rats the hippocampus is a C-shaped structure with both dorsal and ventral components (Figure 1B). In birds, the hippocampus, which consists of the hippocampus and the adjacent area parahippocampalis, is located dorsally and along the midline and posterior extent of the brain (Figure 1C). Even though the avian hippocampus emerges from the same part of the developing telencephalon as the mammalian hippocampus, and has similar input/output connections with sensory regions of the brain (Casini, Bingman, & Bagnoli, 1986; Krayniak & Siegel, 1978), when we began these studies in the early 90s the view was that it lacked an Ammon's horn, dentate gyrus, hilar region, postcommisural fornix, as well as the classic CA subfields that are present in the mammalian hippocampus (Krayniak & Siegel, 1978; Krebs, Erichsen, & Bingman, 1991; but see Atoji & Wild, 2006, for a review of more recent evidence that subareas within the avian hippocampus may indeed resemble the dentate gyrus and Ammon's horn). These significant architectural differences made it unlikely, similarities in spatial impairments notwithstanding, that the avian hippocampus could serve the same function as the mammalian hippocampus.

The prevailing view throughout the 1980s was that in addition to spatial problems, damage to the hippocampus in monkeys and rats produced visual memory impairments, expressed mainly as steeper retention functions on a delayed nonmatching-to-sample (DNMS) task (Mishkin, 1978; Mumby, Wood, & Pinel, 1992). We found, however, that bilateral damage of the hippocampus in birds had no effect on visual memory, and this was true irrespective of whether the animals were trained preoperatively and then tested postoperatively, or whether they were trained and tested postoperatively (Colombo, Swain, Harper, & Alsop, 1997b; Figure 2C). Furthermore, although both proactive and retroactive interference increases memory loss in rats (Jarrard, 1975), monkeys (Zola-Morgan & Squire, 1985) and humans (Sidman, Stoddard, and Mohr, 1968), birds with hippocampal lesions were no more affected by these types of interference than control animals (Colombo et al., 1997b).

Our initial failure to find any visual memory deficits following hippocampal lesions in birds pointed to a functional difference between the avian and mammalian hippocampi, a finding that was perhaps both interesting from an evolutionary perspective and also in line with the known architectural differences, but one that made the possibility of using pigeons to illuminate mammalian hippocampal function unlikely. At the same time as these studies were being con-

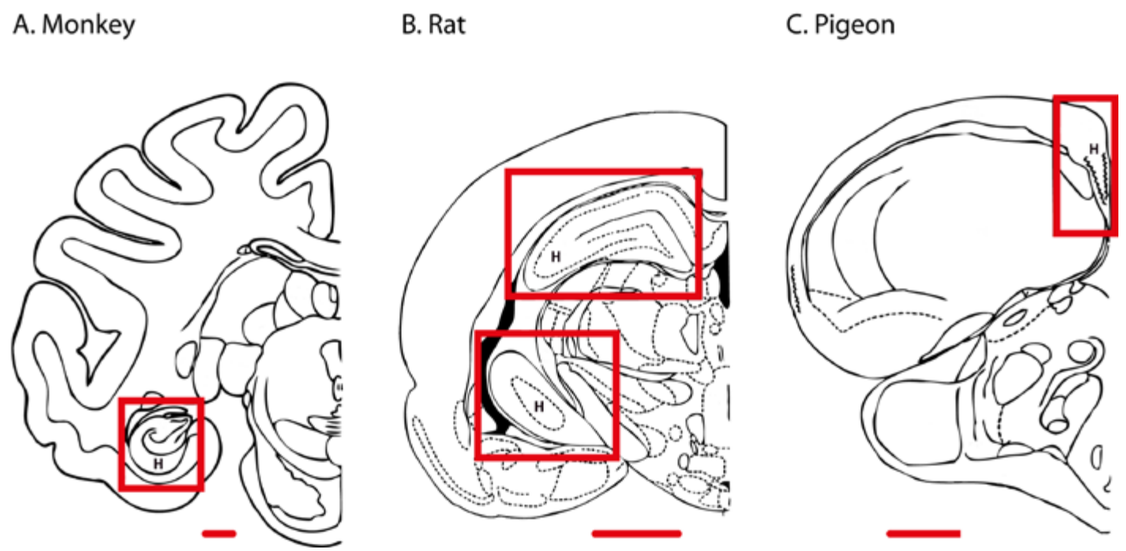

Figure 1. *Coronal sections illustrating the relative position of the hippocampus (H, red box) in the monkey (A), rat (B), and pigeon (C). Adapted from "Is the avian hippocampus a functional homologue of the mammalian hippocampus?," by M. Colombo and N. Broadbent, 2000, Neuroscience and Biobehavioral Reviews, 24, p. 465-484. Copyright 2000 by Elsevier Limited. Adapted with permission*

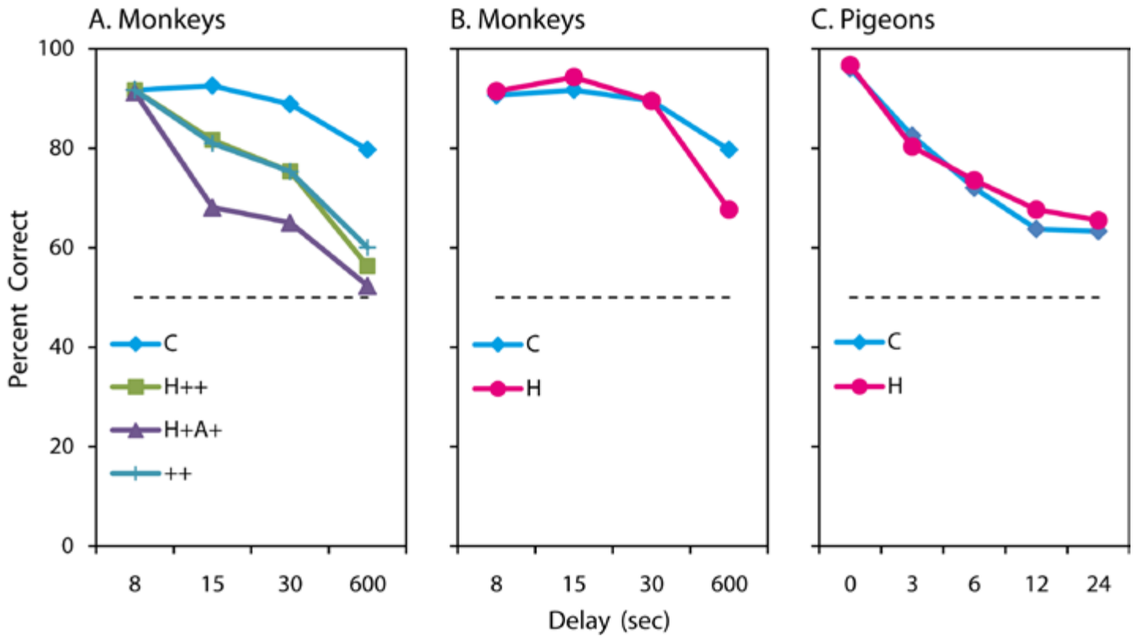

Figure 2. *Visual memory performance following extensive hippocampal lesions in monkeys (A), restricted hippocampal lesions in monkeys (B), and hippocampal lesions in pigeons (C). The dashed line represents chance levels of performance. C: normal unoperated animals; H: hippocampus; H++: hippocampus and tissue adjacent to the hippocampus and amygdala; H+A+: hippocampus, amygdala, and tissue adjacent to both these regions; ++: tissue adjacent to the hippocampus and amygdala. Adapted from "Is the avian hippocampus a functional homologue of the mammalian hippocampus?," by M. Colombo and N. Broadbent, 2000, Neuroscience and Biobehavioral Reviews, 24, p. 465-484. Copyright 2000 by Elsevier Limited. Adapted with permission.*

ducted in pigeons, however, data began to emerge that the visual memory impairments noted after hippocampal damage in monkeys may have been due to damage to the tissue adjacent to the hippocampus rather than the hippocampus itself. When Mishkin (1978) originally developed his animal model of human amnesia, his monkeys received lesions to the hippocampus (H), the tissue adjacent to the hippocampus (+), the amygdala (A), and the tissue adjacent to the amygdala (another +). This lesion, called the H+A+ lesion, was intended to replicate the damage sustained by the classic amnestic patient HM, who received bilateral resection of considerable portions of his medial temporal lobe. Indeed, like HM and other amnestics with similar damage, monkeys with H+A+ lesions show severe visual memory impairments, expressed as steeper retention functions on the DNMS task (H+A+ lesion; Figure 2A). An impairment almost as severe as that following H+A+ lesions, however, can be obtained by damaging the hippocampus, the tissue adjacent to the hippocampus, and the tissue adjacent to the amygdale (H++ lesion; Figure 2A; Zola-Morgan, Squire, Clower, & Rempel, 1993). Indeed, impairments equally as severe as those seen following H+A+ and H++ lesions can be obtained by damaging just the tissue adjacent to the hippocampus and amygdale (++ lesion; Figure 2A; Zola-Morgan, Squire, Amaral, & Suzuki, 1989), a finding that seriously called into question whether the hippocampus was important at all for visual memory.

Although the visual memory impairments following ++ lesions challenged the role of the hippocampus in visual memory, final proof of this came about when a surgical technique was devised that would permit resection of the hippocampus without incurring damage to the adjacent tissue (Alvarez, Zola-Morgan, & Squire, 1995). Under these conditions, monkeys with damage restricted to the hippocampus show almost no impairment in visual memory (Figure 2B), and the small impairment seen at the long delay was later attributed to an artefact of the testing procedure (Nadel, 1992, 1994). In sum, damage to the hippocampus in monkeys does not seem to impair visual memory performance.

The fact that the visual memory impairments seen in monkeys can be attributed to extrahippocampal damage rather than hippocampal damage appears to be true also for humans (Zola-Morgan, Squire, & Amaral, 1986) and rats

(Mumby & Pinel, 1994). Thus, what was initially perceived as evidence that the avian hippocampus had a different function to the mammalian hippocampus rapidly became an example of how the avian and mammalian hippocampi shared a similar function: damage to the hippocampus of both species impaired performance on spatial tasks but caused no impairments on visual memory tasks. Over the subsequent years, this similarity of function was extended to a variety of tasks, both those impaired by damage to the hippocampus as well as those not impaired by hippocampal damage. With respect to tasks not impaired, damage to the hippocampus in mammals and birds has little or no effect on acquisition or retention of procedural or habit tasks (Packard et al., 1989) such as simple visual discriminations, complex visual discriminations, or concurrent discriminations (Alvarez et al., 1995; Colombo, Broadbent, Taylor, & Frost, 2001; Colombo, Cawley, & Broadbent, 1997a). With respect to tasks that are impaired, damage to the hippocampus in mammals and birds significantly impairs performance on a variety of navigation tasks (Maguire, Frackowiak, & Frith, 1997; Pearce, Roberts, & Good, 1998; Bingman & Yates, 1992), as well as tasks such as the radial-arm maze and water maze tasks used with rats, and analogues of these tasks used with pigeons (Colombo et al., 1997a; Fremouw, Jackson-Smith, & Kesner, 1997; Morris, 1984; Olton & Samuelson, 1976).

The similarity in the consequences of hippocampal lesions in mammals and birds extends to more subtle effects as well. For example, within the spatial domain, it is interesting that although rats with hippocampal lesions are impaired on the radial-arm maze task in that they take longer to learn the task than controls, they do eventually learn the task. It appears, however, that their method of solution is different to that of control animals: while control animals adopt a nonstereotypic response strategy of entering arms in a random fashion, hippocampal rats solve the maze by adopting stereotypic response strategies, such as exiting from an arm and entering the next arm to the right, a strategy that achieves success without requiring spatial memory of which arms have been visited. It appears hippocampal pigeons also adopt stereotypic response strategies in solving mazes. Colombo et al. (2001) trained pigeons on a radial-arm maze analogue task and showed that, like in rats, although hippocampal pigeons were significantly slower to learn the maze task than controls, they were eventually able to learn the task. Colombo et al. (2001) then analyzed the paths the control and hippocampal animals performed over the two criterial acquisition days and found that compared to control pigeons, hippocampal pigeons had a greater tendency to follow the same paths from one criterial day to the next (Figure 3). Thus, like rats with hippocampal damage, hippocampal pigeons adopted stereotypic response strategies in the solution of a maze task.

Outside of the spatial domain, we have also noticed that

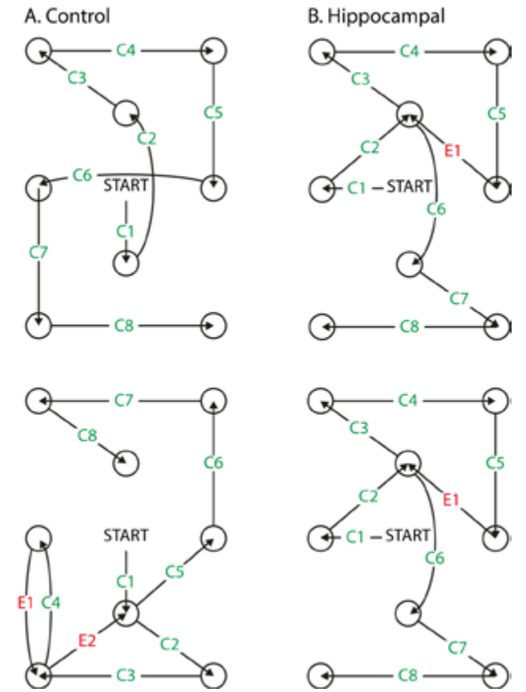

Figure 3. *Paths taken by control (A) and hippocampal (B) animals on the first (top) and second (bottom) criterial days on acquisition of a radial-arm maze analogue task. The task was conducted on an elevated table. The circles represent cups on a table that hid food, and the arrows represent the paths taken by one representative control and one representative hippocampal animal. The animals were trained until they satisfied a criterion of two consecutive days with no more than three errors. An error was defined as revisiting a cup that had already been visited. Notice that for the control animal the paths taken on the first and second criterial days are very different, whereas for the hippocampal animal there is perfect overlap. C, a correct path that leads to a cup not yet visited; E, an incorrect path (error) in which the subject revisits a cup already visited. Adapted from "The role of the avian hippocampus in orientation in space and time," by M. Colombo, N. J. Broadbent, C. S. R. Taylor, & N. Frost, 2001, Brain Research, 919, p. 292-301. Copyright 2001 by Elsevier Limited. Adapted with permission.*

hippocampal birds show less fear and/or are less distractible than control birds (Broadbent & Colombo, 2000), a finding in line with what has been shown in rats (Kaplan, 1968; Raphelson, Isaacson, & Douglas, 1965; Wickelgren & Isaacson, 1963). In fact, despite nearly two decades of research by ourselves and others (Good & Macphail, 1994; Hampton & Shettleworth, 1996), we have encountered only

two instances in which damage to the hippocampus in pigeons produces an impairment different to that produced by damage to the hippocampus in mammals. In both these cases, however, there are either inconsistencies within the mammalian literature itself concerning whether hippocampal lesions cause impairments (Bingman, Strasser, Baker, & Riters, 1998; see Colombo & Broadbent, 2000, for a review of the inconsistencies in the mammalian studies), or the exact task used with pigeons (Strasser, Ehrlinger, & Bingman, 2004) has never been examined in hippocampal mammals, so it is unknown whether they would be impaired. In fact, the similarity in findings between the avian and mammalian hippocampus prompted Colombo and Broadbent (2000) to suggest that the avian hippocampus is a functional homologue (i.e., an analogue) of the mammalian hippocampus, and further proclaim that "despite 300 million years of independent evolution, there are no degrees of freedom in the evolution of hippocampal function" (p. 480).

Future Directions. The fact that the avian hippocampus is an analogue of the mammalian hippocampus makes it possible to now use the avian hippocampus as a model to understand mammalian hippocampal function. Several theories have been advanced concerning the role of the hippocampus, ranging from the initial view that the hippocampus was important for memory in general (Mishkin, 1982), followed by refinements that it was important only for declarative memories (Squire, 1992), then configural processing of information (Rudy & Sutherland, 1989, 1995), to the more recent view that the hippocampus is important for representational flexibility (Eichenbaum, Otto, & Cohen, 1994). All these theories share the same underlying theme that the hippocampus fulfils an important role in 'cognitive' behavior. For example, Eichenbaum and colleagues believe that the hippocampus is important for the ability to make transitive (logical) judgements (Bunsey & Eichenbaum, 1996).

In contrast to 'cognitive' theories of hippocampal function, other theories view the hippocampus as having a much simpler role in behavior. According to these theories, damage to the hippocampus causes impairments in response inhibition and perseveration (Douglas, 1967; Gray, 1982; Gray & McNaughton, 1983; Kimble, 1968), and it is these 'simpler' impairments that underlie the 'cognitive' deficits seen after hippocampal lesions. We have also noted failures in hippocampal birds that don't fall into the traditional memory-based or cognitive-based theories of hippocampal function. One such impairment is the autoshaping deficit. A number of investigators have noticed that hippocampal pigeons are slower to acquire an auto-shaped response (Reilly & Good, 1989; Good & Macphail, 1994; Richmond & Colombo, 2002). Operationally, birds with hippocampal lesions peck

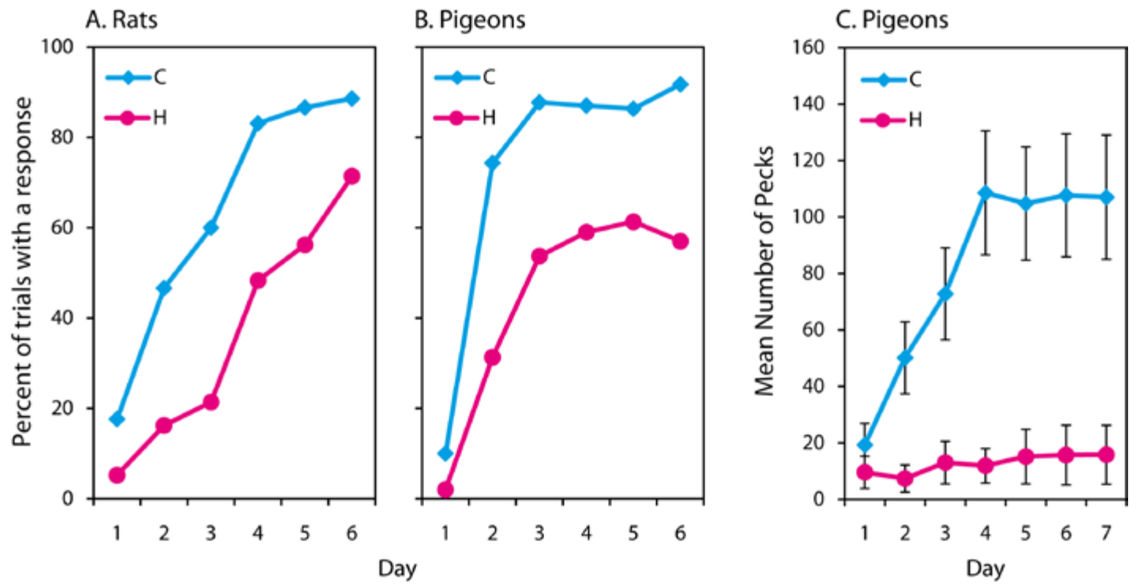

Figure 4. *Autoshaping performance by rats (A) and pigeons (B and C). Panels A and B show the autoshaping impairment expressed as a percentage of trials with a response to the CS, whereas panel C shows the impairment expressed in terms of overall responses to the CS. Adapted from "Is the avian hippocampus a functional homologue of the mammalian hippocampus?," by M. Colombo and N. Broadbent, 2000, Neuroscience and Biobehavioral Reviews, 24, p. 465-484. Copyright 2000 by Elsevier Limited. Adapted with permission.*

the CS on fewer trials than control birds (Figure 4B) and/or exhibit fewer overall pecks to the CS across a session (Figure 4C). It is interesting to note that, opposite to the general trend of first being discovered in mammals, the autoshaping deficit was first noted in pigeons and only later confirmed in mammals (Good & Honey, 1991; Figure 4A).

We have recently turned our attention to understanding the nature of the autoshaping deficit in the hope that this will shed light on the functions of the mammalian hippocampus. Our view is that understanding the source of these simpler deficits is more likely to explain why hippocampal animals are impaired on more complex tasks such as transitivity. We are currently examining whether the failure of the hippocampal animals on the autoshaping task lies in a failure of detecting contingencies, reward processing, or navigation to points in space. The point is that, armed with the knowledge that the avian hippocampus has the same function as the mammalian hippocampus, we can explore the role of the avian hippocampus with confidence that our results will be relevant for understanding mammalian hippocampal function.

Avian NCL Studies

The Avian NCL: Analogue of the Mammalian PFC? The nidopallium caudolaterale (NCL) is a multimodal telencephalic region situated in the posterior pallium of birds (Waldmann & Güntürkün, 1993; Figure 5). Divac and colleagues were the first to suggest that the NCL may correspond to the mammalian prefrontal cortex (Divac, Mogensen, & Björklund, 1985; Mogensen & Divac, 1982), an area involved in the executive control of behavior in primates (Miller & Cohen, 2001), and an area that is the focus of much current mammalian research. Anatomically, there is considerable correspondence between the NCL and the PFC. Both are considered the main integrative areas of the brain, receiving sensory information and translating that information into action. For example, both the NCL and PFC receive projections from visual, auditory, and somatosensory areas, and both project to motor and limbic areas of the brain (Jones & Powell, 1970; Kröner & Güntürkün, 1999). In addition, both the NCL and the PFC are densely innervated by midbrain dopaminergic fibers (Divac et al., 1985; Divac, Björklund, Lindvall, & Passinghman, 1978; Güntürkün, 2005b). Naturally, given 300+ million years of independent evolution, there are going to be some anatomical differences. For example, in primates, the mediodorsal (MD) nucleus of the thalamus projects to the PFC (Giguere & Goldman-Rakic, 1988), whereas in birds the thalamic projection to the NCL, the nucleus dorsolateralis posterior thalami (DLP), does not share the same afferent and efferent connectional patterns as the MD (Csillag & Montagnese, 2005). Despite the different connectional patterns, the DLP does seem to have the same functions as the MD (Güntürkün, 1997a).

The behavioral consequences of damage to the NCL are also similar to the effects of damage to the PFC. For example, damage to the NCL and PFC result in impairments on delayed alternation and pattern-reversal tasks while sparing performance on simultaneous visual discriminations and basic sensory processes (Fuster, 1997; Güntürkün, 1997a; Hartmann & Güntürkün, 1998; Mogensen & Divac, 1982, 1993). In addition, blockade of D1 receptors in the NCL and the PFC cause impairments on tasks sensitive to NCL and PFC damage (Güntürkün, 2005a, 2005b). In short, the evi-

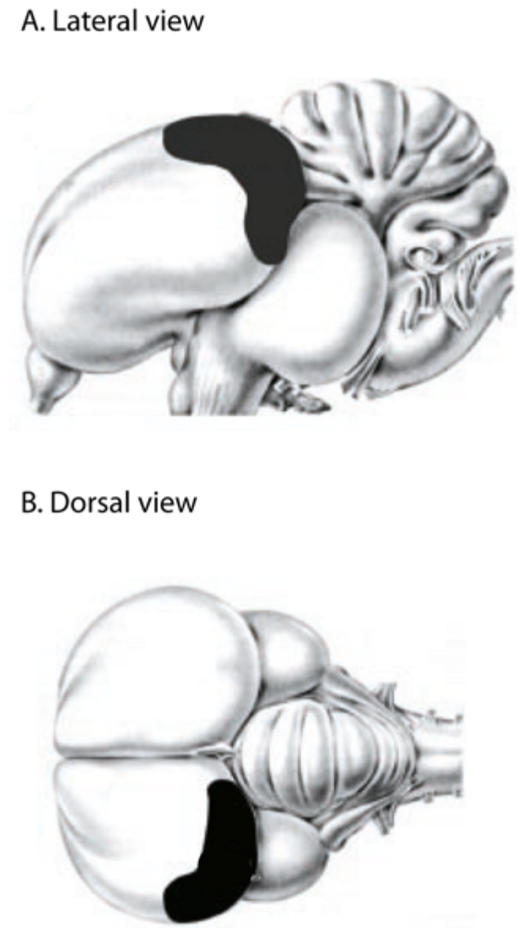

Figure 5. *Lateral (A) and dorsal (B) view of the pigeon brain. The dark area marks the location of the NCL. Adapted from "Delay activity in avian prefrontal cortex – sample code or reward code?," by R. Browning, J. B. Overmier, and M. Colombo, 2011, European Journal of Neuroscience, 33, p. 726-735. Copyright 2011 by John Wiley and Sons. Adapted with permission.*

dence suggests that the NCL is clearly an analogue of the PFC.

Memory Neurons. Fuster and Alexander (1971) reported that some cells in the monkey's prefrontal cortex fired not when the animals were looking at a stimulus, but rather during the delay period when the animal presumably was remembering the stimulus. Such 'memory' cells have now been found in a number of brain regions including the visual cortex, auditory cortex, and hippocampus, as well as in a number of species such as monkeys, rats, and humans (for a review see Colombo & Gross, 1994; Sakurai, 1990). The prevailing view is that the enhanced activity during the delay period represents active maintenance of the stimulus, that is, a neural correlate of the to-be-remembered stimulus (Colombo & Gross, 1994; Fuster & Jervey, 1982; Miyashita & Chang, 1988). With respect to pigeons, memory cells have been found in the entopallium, an area functionally similar to higher-order visual cortex in mammals (Colombo, Frost, & Steedman, 2001), as well as in the NCL (Diekamp, Kalt, & Güntürkün, 2002).

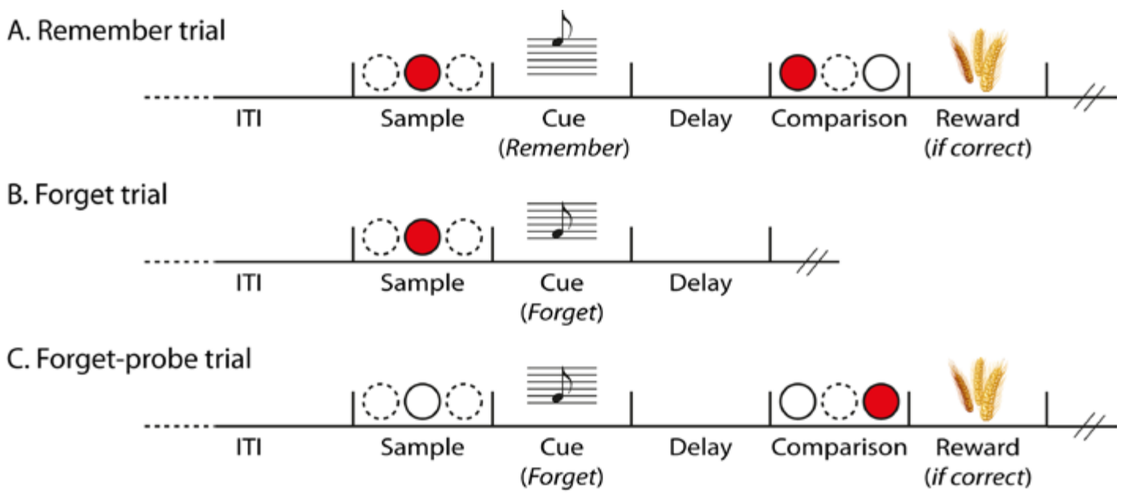

Figure 6. *Sequence of events on remember trials (A), forget trials (B), and forget-probe trials (C). The three horizontally arranged circles represent the projectors on which the stimuli were displayed. During the cue and delay periods, the projectors were turned off. ITI, intertrial interval. The reward consisted of grains of wheat. Adapted from "Neural correlates of executive control in the avian brain?," by J. Rose and M. Colombo, 2005, PLoS Biology, 3, p. 1139-1146. Copyright 2005 by Public Library of Science. Adapted with permission*

In primates, the PFC is believed to be involved in the executive control of behavior, and one manifestation of executive control is the ability to filter information, in other words, retain that which is important and discard that which is not (Smith & Jonides, 1999). We wondered whether delay activity in pigeons could be turned on and off by instructing the animals to remember and forget the sample stimulus, something that had not been investigated in any species at that time. Rose and Colombo (2005) trained pigeons on a directed forgetting (Maki, 1981) version of a delayed matching-to-sample task (Figure 6). Following pecks to the sample stimulus, the animals heard either a 2-sec high-frequency remember cue or a 2-sec low-frequency forget cue. The cue period was followed by a 3-sec delay period. On remember trials (Figure 6A), after the delay period there was a memory test in which the subjects were shown two comparison stimuli and had to respond to the comparison stimulus that had appeared as the sample stimulus. A correct response resulted in a reward followed by the intertrial interval (ITI) and the next trial. On forget trials (Figure 6B), at the end of the delay period there was no test, and following the ITI the next trial began. Effectively, the remember cue instructed the birds to remember the sample stimulus, whereas the forget cue instructed the birds to forget the sample stimulus. A session consisted of an intermixed number of remember and forget trials.

Figure 7 shows examples of three delay cells recorded from the NCL of three different birds. Shown in the figure is the activity of a neuron on both remember (left) and forget (right) trials. The pattern is roughly the same across the three neurons. When the animals were instructed to remember the sample stimulus, there was a sustained level of activity in the cue period that persisted into and throughout the

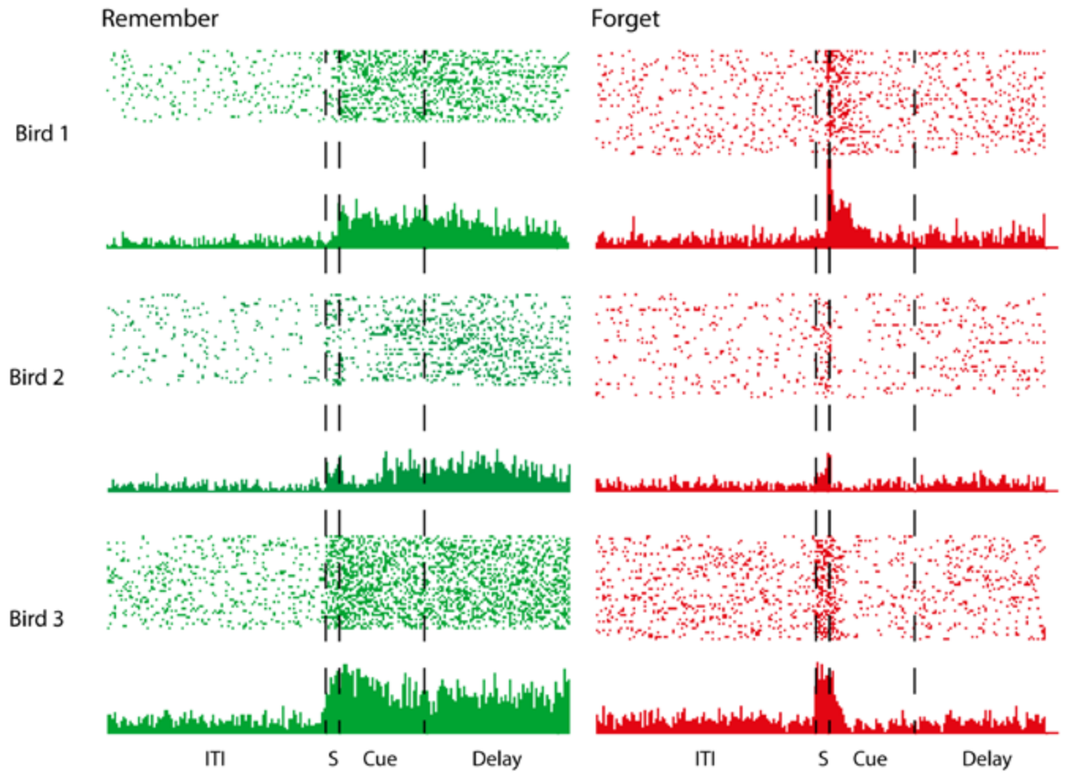

Figure 7. *Examples of three NCL neurons from three different birds. The left panel shows performance across remember trials, whereas the right panel shows the performance of the same neurons on forget trials. On remember trials, there is sustained activation in the cue and delay periods. On forget trials, the sustained activation is abolished. The binwidth is 50 ms. The vertical dashed lines separate the different periods of the task. ITI, intertrial interval; S, sample period. Adapted from "Neural correlates of executive control in the avian brain?," by J. Rose and M. Colombo, 2005, PLoS Biology, 3, p. 1139-1146. Copyright 2005 by Public Library of Science. Adapted with permission.*

delay period. In contrast, when the animals were instructed to forget the sample stimulus, the neural activity in the cue and delay periods returned to baseline (ITI) levels.

Figure 8A shows the activity on remember and forget trials averaged across all the memory neurons encountered in the study. Clearly, across all the memory neurons in the NCL, the remember cue resulted in an increase in activity in the cue period that persisted undiminished in the delay period, whereas the forget cue resulted in activity rapidly returning to baseline levels. We concluded that the increased delay activity represented a neural code of the animal remembering the sample stimulus, and the decrease in activity to baseline levels on forget trials indicated that the pigeons were forgetting the sample stimulus. However, just because we told the animals to 'forget' the sample stimulus, and just because there was only baseline-level activity in the delay following the forget cue, it doesn't necessarily follow that the birds were forgetting the sample stimulus. In order to test whether the animals were forgetting the sample stimulus, on occasion we delivered forget-probe trials in which we presented the forget cue but then presented the animals with the memory test (Figure 6C). As is shown in Figure 8B, performance on forget-probe trials was no different from chance (50%), a finding that supports the idea that the forget cue was indeed instructing the animals to forget the sample stimulus.

The notion that the enhanced delay activity and the abolished delay activity represented remembering and forgetting the sample stimulus, respectively, is not the only possible explanation for the different levels of delay activity on remember and forget trials. Rose and Colombo (2005) also raised the possibility that the differential delay activity might be related to reward. On remember trials, not only is

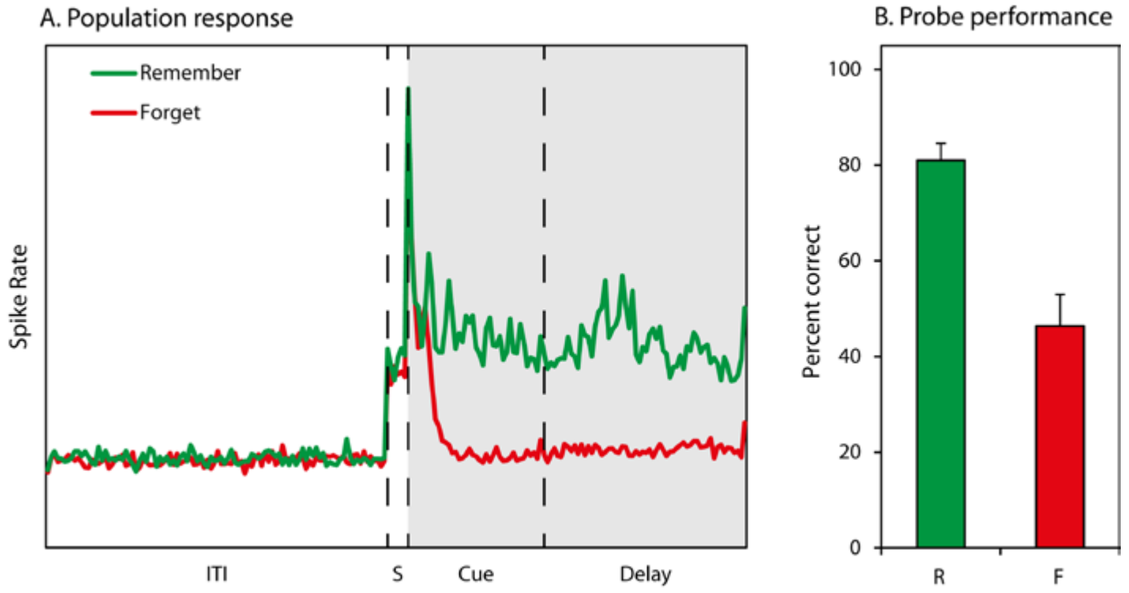

Figure 8. *The population response profile of NCL memory cells on the remember and forget trials (A), and performance on the remember and forget-probe trials averaged across all the birds in the study (B). The vertical lines in panel A separate the different periods of the task. ITI, intertrial interval; S, sample period; R, remember trials; F, forget-probe trials. Adapted from "Neural correlates of executive control in the avian brain?," by J. Rose and M. Colombo, 2005, PLoS Biology, 3, p. 1139-1146. Copyright 2005 by Public Library of Science. Adapted with permission.*

the animal being told to remember, but the remember cue is also instructing the animal that the opportunity for a reward is upcoming (Figure 6). The forget cue, on the other hand, clearly instructs the animal that no reward will be available. Thus, although the heightened and abolished delay activity may represent a neural code of remembering and forgetting the sample stimulus, respectively, it could also represent a neural code of the opportunity to received a reward (remember trials) and no opportunity to receive a reward (forget trials).

We next pursued the issue of whether the delay activity represented a sample code or reward code by combining a differential-outcomes procedure with a directed-forgetting procedure (Browning, Overmier, & Colombo, 2011). The structure of the task is shown in Figure 9. As in the previous studies, the high-frequency cue indicated that the sample should be remembered, and the low-frequency cue indicated that the sample could be forgotten. In contrast to the previous procedure, however, in the differential-outcomes procedure a correct response on red remember trials resulted in a reward (Figure 9A), whereas a correct response on white remember trials was followed by no reward (Figure 9B). The key was that on both red and white remember trials the animal had to remember the sample, yet only one stimulus, red, was associated with the possibility of an upcoming reward.

The reasoning behind the experiment was as follows. If delay activity occurred following both red and white remember trials, then the delay activity must be related to the sample and not the reward, because both red and white trials share the same feature of requiring memory of the sample stimulus. On the other hand, if delay activity occurred following red remember trials and not white remember trials, then the delay activity must represent a reward code, because only the red trials are followed by the opportunity to gain a reward. Browning et al. (2011) found that delay activity occurred on red remember trials but not on white remember trials, thus supporting the idea that delay activity was a reward code (Figure 10A). Probe tests confirmed that the animals were forgetting on forget trials (Figure 10B).

While the data to date suggest that the delay activity we observed in the NCL represents a reward code rather than a sample code, several points must be kept in mind. First, we are not arguing that all delay activity in the NCL (or PFC in mammals) represents a reward code, and indeed there is evi-

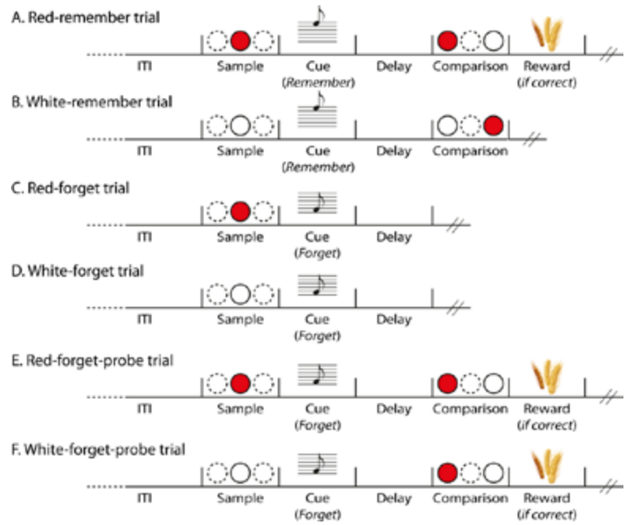

Figure 9. *Sequence of events on red-remember trials (A), white-remember trials (B), red-forget trials (C), white-forget trials (D), red-forget-probe trials (E), and white-forget-probe trials (F). The three horizontally arranged circles represent the projectors on which the stimuli were displayed. During the cue and delay periods, the projectors were turned off. ITI, intertrial interval. The reward consisted of grains of wheat. Adapted from "Delay activity in avian prefrontal cortex – sample code or reward code?," by R. Browning, J. B. Overmier, and M. Colombo, 2011, European Journal of Neuroscience, 33, p. 726-735. Copyright 2011 by John Wiley and Sons. Adapted with permission.*

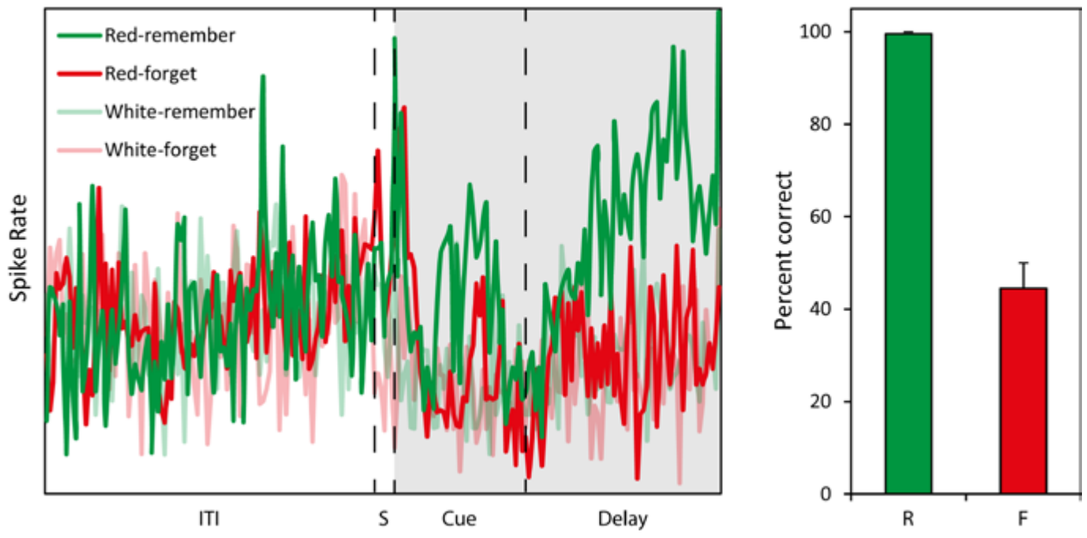

Figure 10. *The population response profile of NCL memory cells on red-remember, white-remember, red-forget, and white-forget trials (A), and performance on the remember and forget-probe trials averaged across all the birds in the study (B). The vertical lines in panel A separate the different periods of the task. ITI, intertrial interval; S, sample period; R, remember trials; F, forget-probe trials. Adapted from "Delay activity in avian prefrontal cortex – sample code or reward code?," by R. Browning, J. B. Overmier, and M. Colombo, 2011, European Journal of Neuroscience, 33, p. 726-735. Copyright 2011 by John Wiley and Sons. Adapted with permission.*

dence that some neurons in the PFC (and likely the NCL as well) do indeed code for the retention of a sample stimulus (Rainer, Asaad, & Miller, 1998). Rather, what our studies have shown is that the long-held belief that delay activity represents a neural correlate of the animal remembering the sample stimulus is too simplistic, and that delay activity can represent codes of different things across different brain regions. In NCL (or PFC in mammals), delay activity could represent either a sample code or a reward code depending on the nature of the task. Indeed the NCL and PFC are areas that are important for both memory (Miller & Cohen, 2001; Güntürkün, 2005b) as well as processing information related to reward (Watanabe, 1996; Güntürkün, 2005b). On the other hand, our prediction is that in entopallium, an area that should be involved in the memory of the sample and not reward processing, delay activity should be apparent on both red and white remember trials. These studies are currently underway, but again, the main point that we wish to convey is that these studies are easily conducted in pigeons, and the results are relevant for understanding how information may be processed in the mammalian brain.

Impulsive-Choice Neurons. Recently, avian neuroscientists have explored the topical area of the neural basis of impulsive choice behavior. Impulsive choice refers to the preference for a small immediate reward over a large delayed reward. Such behavior underlies a variety of pathologies in humans such as gambling, drug addiction, ADHD, as well as general damage to the frontal lobes of the brain (Kalenscher, Windmann, Diekamp, Rose, Güntürkün, & Colombo, 2005).

In pigeons, the preference for a small immediate reward over a large delayed reward is a function of a bird's subjective reward value, defined by Mazur (1984) as:

$$V=A/(1+yD)$$

where 'V' refers to the subjective reward value, 'A' a fixed reward amount, 'D' the delay to the reward, and 'y' a discount factor that determines the slope of the decay function. A number of studies in mammals have shown that cells in the frontal regions of the brain code for 'D' (Brody, Hernandez, Zainos, & Romo, 2003), 'A' (Leon & Shadlen, 1999; Wallis & Miller, 2003) and 'V' (Tremblay & Schultz, 1999). We wondered whether cells in the NCL also coded subjective reward value. Specifically, if a cell is sensitive to the subjective reward value the following criteria should be met:

1. For constant reward amounts but increasing delays the subjective reward value and neural activity should decrease.

2. For constant delays and reward amounts the subjective reward value and neural activity should remain constant.

3. For constant delays but different reward amounts the value of the larger reward, and hence the neural activity to it, should be greater than the value of the smaller reward and the neural activity to it.

Kalenscher et al. (2005) trained birds on a delayed-reward choice paradigm in which they had a choice between two response keys, one that delivered a small reward (2 sec access to food) after 1.5 sec, and the other that delivered a large reward (4 sec access to food). Initially the delay-to-reward for the large reward was also set at 1.5 sec, and naturally under these circumstances the bird pecked the key that delivered the large reward after 1.5 sec than the key that delivered a small reward after 1.5 sec. Throughout the session, however, the delay-to-reward for the large reward increased, and at some point the pigeons showed a preference shift to the small immediate reward over the large delayed reward.

An example of a cell that integrated reward amount and delay-to-reward, that is, was modulated by subjective reward value, is shown in Figure 11. The left panel shows the neural activity across the last three blocks of trials prior to the subject's preference shift, in other words, the three blocks in which the animal selected the large immediate reward over the small immediate reward. The right panels shows the neural activity for the first three blocks of trials after the preference shift, that is, when the delay was increased to a point where the animal now chose the small immediate reward over the large delayed reward. The figures show neural activity during the first 1500 msec of the delay period, and for our analysis we examined a window of 500 msec from 1000 to 1500 msec after the start of the delay (the area shaded).

The neuron shown in Figure 11 satisfies the previously mentioned criteria to be classified as encoding subjective reward value. With respect to the first criterion, in the three blocks before a preference shift the subject was always choosing the large reward, but as the delay increased from block -3 to -2 to -1 and the subjective reward value therefore decreased, the neural activity also decreased (Figure 11A, shaded area). With respect to the second criterion, in the three blocks following the preference shift when the animal was always selecting the small immediate reward, the subjective value remains the same, and so does the neural activity across blocks 1, 2, and 3 (Figure 11B, shaded area).

The fact that the neuron is modulated by delay-to-reward amount (first criterion) and not modulated when the delay is constant (second criterion) satisfies 2 of the 3 criteria to be classified as a cell that codes subjective reward value. Evi-

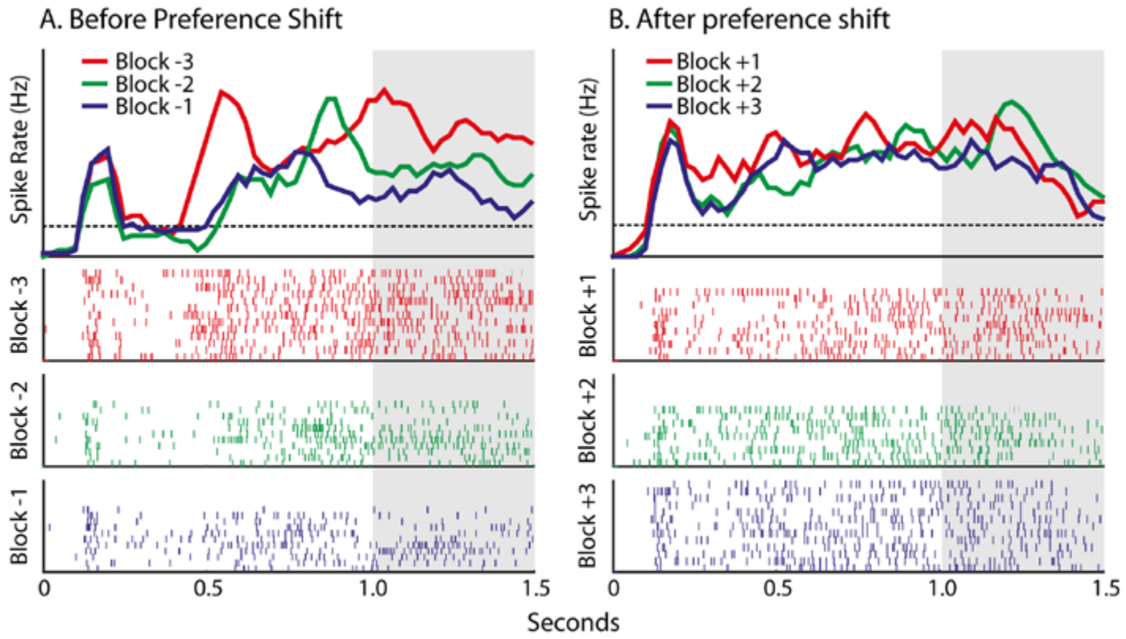

Figure 11. *An example of a neuron coding subjective reward value. The neuron's activity is shown for the three blocks preceding the preference shift in which the animal was choosing the large reward (A), and in the three blocks following the preference shift in which the animal was choosing the small reward (B). The horizontal dotted line marks the averaged baseline spike rate. The grey area delineates the window used for statistical analysis. Adapted from "Single units in the pigeon brain integrate reward amount and time-to-reward in an impulsive choice task," by T. Kalenscher, S. Windmann, B. Diekamp, J. Rose, O. Güntürkün, and M. Colombo 2005, Current Biology, 15, p. 594-602. Copyright 2005 by Elsevier Limited. Adapted with permission.*

dence for the third criterion can be seen in the neural activity from block -3 to block +1. In these two blocks, the delay-to-reward is the same. In block -3, the subject is choosing the large reward with a 1.5 sec delay, and in block +1, the subject is now choosing the small reward, also with a 1.5 sec delay. Yet the neural activity in block -3 is greater than the neural activity in block +1, as is predicted by the third criterion that states that for constant delays but different reward amounts the neural activity should be greater to the large (block -3) rather than the small (block +1) reward.

In summary, the NCL neurons integrated reward amount and time-to-reward and, as predicted by impulsive choice theory, their relative activation level correlated with the pigeon's reward preference. These findings not only shed light on impulsive decision making, but might also help in the understanding of human conditions that are characterized by a decreased ability to wait for a large reward, such as drug addiction, gambling, frontal lobe syndrome, and attention deficit disorders.

Gambling Neurons. Another recent area of research for which pigeons have proven to be an excellent model concerns the neural basis of gambling (Zentall & Stagner, 2011). Although gambling can be a harmless leisure activity, there is evidence that prolonged involvement in leisure gambling can lead to problem gambling and pathological gambling, along with which also comes higher rates of suicide, depression, and substance abuse (Ramirez, McCormick, Russo, & Taber, 1983). Of all the forms of gambling, the one most associated with problem and pathological gambling are slot machines (Fisher & Griffiths, 1995), most likely because they incorporate well-established learning principles (established with pigeons) that promote gambling behavior, such as high rates of reinforcement and rapid event frequency (Griffiths, 1999; Skinner, 1953). So effective are these learning parameters that the addiction to slot machines has been likened to the addiction to crack-cocaine (Breen & Zimmerman, 2002).

Although a large network of structures contribute to gambling behavior in humans, a critical region appears to be the frontal lobes. Damage to the frontal lobes can lead to impaired decision-making abilities on gambling tasks (Fellows & Farah, 2005), and fMRI studies have revealed that

decision-making under uncertainty, high reward, and high-risk situations, all aspects of gambling behavior, result in activation of various regions of the frontal lobe (Fukui, Murai, Fukuyama, Hayashi, & Hanakawa, 2005; Gehring & Willoughby, 2002). Similarly, single neurons in the prefrontal cortex of monkeys code for the type or magnitude of an expected reward (Watanabe, 1996), and the economic value of a stimulus (Padoa-Schioppa & Assad, 2006), conditions also required for gambling behavior.

Despite the recent surge in interest in studies examining aspects of reward and risk-taking behavior, no study had yet examined the responses of single neurons in experimental animals actually playing a slot machine task. We decided to examine how neurons in the pigeon's brain behave while they played a slot machine task similar in many ways to a slot machine at any casino (Scarf et al., 2011). Our slot machine had an upwards pointing "arm" that when pecked assumed a downwards position and activated four tumblers. The pigeons pecked at each tumbler to stop its motion, and if the four tumblers displayed four identical stimuli the bird received a wheat reward.

We recorded from NCL neurons and were able to identify three neural correlates of slot machine gambling. Reward-Proximity neurons showed a significant linear increase or decrease in activity as each successive tumbler displayed an identical stimulus and the opportunity for a reward drew near (Figure 12). These same neurons showed no linear trend when the tumblers displayed a nonrewarded outcome.

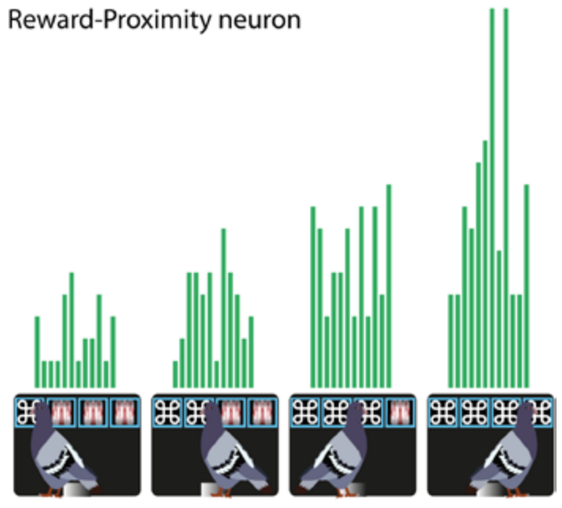

Figure 12. *An example of a Reward-Proximity neuron. The neuron shows a steady increase in firing on rewarded trials as the opportunity of a reward draws near. Adapted from "Brain cells in the avian 'prefrontal cortex' code for features of slot-machine-like gambling," by D. Scarf, K. Miles, A. Sloan, N. Goulter, M. Hegan, A. Seid-Fatemi, D. Harper, and M. Colombo, 2011, PLoS ONE, 6, p. 1-7. Copyright 2011 by Public Library of Science. Adapted with permission.*

We also noted what we called *I-Won* and *I-Lost* neurons. *I-Won* neurons showed activation only to a stimulus on the fourth and final tumbler, and only when the four tumblers displayed a winning combinations (i.e., four of a kind). An example of an *I-Won* neuron is shown in Figure 13. These neurons did not fire to any stimuli displayed on the first three tumblers, nor did they fire to a fourth stimulus on nonrewarded combinations.

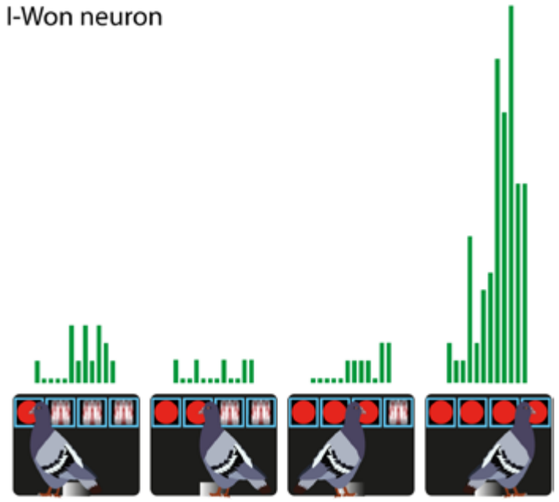

Figure 13. *An example of an I-Won neuron. The activity on the first three tumblers is no different to baseline activity, but activity to the fourth identical stimulus results in an increase in activity. Adapted from "Brain cells in the avian 'prefrontal cortex' code for features of slot-machine-like gambling," by D. Scarf, K. Miles, A. Sloan, N. Goulter, M. Hegan, A. Seid-Fatemi, D. Harper, and M. Colombo, 2011, PLoS ONE, 6, p. 1-7. Copyright 2011 by Public Library of Science. Adapted with permission.*

In contrast to *I-Won* neurons, *I-Lost* neurons either significantly increased or decreased their activity the moment it became apparent that the opportunity to obtain a reward was no longer available. An example of an *I-Lost* neurons is shown in Figure 14. This neuron fired strongly when the opportunity of a reward was still possible, but reduced its firing rate at the presentation of the first nonidentical stimulus, that is, when there was no longer an opportunity to gain reward.

What is the evidence that a pigeon playing a slot machine is a behavioral analogue of a human playing a slot machine?

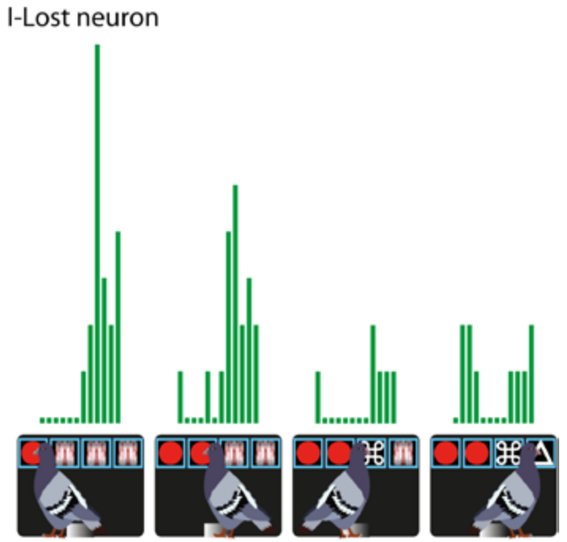

Figure 14. *An example of an I-Lost neuron. The activity drops the moment it becomes apparent that no reward will be delivered. Adapted from "Brain cells in the avian 'prefrontal cortex' code for features of slot-machine-like gambling," by D. Scarf, K. Miles, A. Sloan, N. Goulter, M. Hegan, A. Seid-Fatemi, D. Harper, and M. Colombo, 2011, PLoS ONE, 6, p. 1-7. Copyright 2011 by Public Library of Science. Adapted with permission.*

We would argue that playing a slot machine is not a cognitively demanding task, and that the ability to appreciate that four identical stimuli result in a reward, and that the first nonidentical stimulus results in no reward, is well within the cognitive repertoire of all vertebrates. The only parametric data that would allow us to compare human and pigeon slot-machine behavior is that humans show an increase in latency to initiate the next trial following a rewarded (compared to nonrewarded) outcome (Schreiber & Dixon, 2001). We noted the exact same latency increase in our pigeons: latency to initiate the next trial was significantly longer after rewarded than nonrewarded outcomes. Outside the domain of slot machines, it is interesting that recent studies in birds have shown that they engage in gambling-like behavior (Zentall & Stagner, 2011).

Previous electrophysiological studies with animals have shown that neurons in the frontal lobe are sensitive to elements that underlie gambling behavior, such as risky decisions, reward prediction and expectancy, as well as impending reward size and type. No study, however, had examined neural activity while subjects are actually engaged in a gambling task. We showed that neurons in the NCL show correlates of aspects of slot machine behavior, such as the impending approach of a reward, as well as whether the subject won or lost. Such a pigeon model of slot machine gambling may pave the way for studying disorders of impulse control and their effective treatments in humans.

Conclusions

We have reviewed a number of studies on the neural basis of learning and memory in pigeons. These studies, of course, represent far less than the proverbial tip of the iceberg of avian neuroscience research. Studies on the avian brain have explored such diverse topics as neurogenesis (Barnea & Nottebohm, 1994), synaptic plasticity (Wieraszko & Ball, 1993), the role of immediate early genes (Brito, Britto, & Ferrari, 2006), neural synchronicity (Kirsch & Güntürkün, 2005), motion detection (Wang & Frost, 1992), as well as the neural basis of choice behavior (Kalenscher, Diekamp, & Güntürkün, 2003), lateralization (Güntürkün, 1997b), and birdsong (Konishi, 1994; Nottebohm, 1991), to name but a few areas of research. Yet despite the wide range of interesting studies, few have managed to incorporate themselves into the mammalian literature. The reason for this lies most likely with what was an outdated nomenclature that viewed the avian brain as consisting of non-cortical structures, and therefore unlikely to support 'cognitive' behaviors and contribute to our understanding of the richness of mammalian brain function. Over the past twenty years our understanding of avian cognition has changed dramatically, and birds, even pigeons, are now seen to posses an amazing repertoire of abilities, on par with those of mammals. In line with this new understanding of avian cognition comes a revised nomenclature for the avian brain that highlights its similarities to the mammalian brain. As a result of the behavioral and anatomical advances, avian neuroscience has never had a better platform from which to advance our understanding of the complexities of the mammalian brain.

Finally, it is important to comment on an issue central to those of us conducting comparative cognition work. Macphail (1985) argued not too long ago that we have to accept the null hypothesis and conclude that there are no differences in intelligence, either qualitative or quantitative, across vertebrates. While this debate still rages on, it is now clear that abilities that we thought were the exclusive domain of primates are no longer. The fact that pigeons can engage in mirror recognition (Epstein, Lanza, & Skinner, 1981), executive control (Rose & Colombo, 2005), impulsive decision making (Kalenscher et al., 2005), and gambling (Scarf et al., 2011), and that we can study the neural basis of these behaviors in pigeons, should lead to a deeper understanding of the neural mechanisms that underlie such behavior in primates. Similarly, when you show that the pigeon's hippocampus is in every respect an analogue of the

mammalian hippocampus (Colombo & Broadbent, 2000), while at the same time structurally totally different (Szekely, 1999), we must take this into consideration when we devise computational models of primate hippocampal function. In short, neurophysiological studies in pigeons have as much to contribute as studies in primates to our understanding of the workings of the human mind.

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