Intuition: A Social Cognitive Neuroscience Approach

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This review proposes that implicit learning processes are the cognitive substrate of social intuition. This hypothesis is supported by (a) the conceptual correspondence between implicit learning and social intuition (nonverbal communication) and (b) a review of relevant neuropsychological (Huntington’s and Parkinson’s disease), neuroimaging, neurophysiological, and neuroanatomical data. It is concluded that the caudate and putamen, in the basal ganglia, are central components of both intuition and implicit learning, supporting the proposed relationship. Parallel, but distinct, processes of judgment and action are demonstrated at each of the social, cognitive, and neural levels of analysis. Additionally, explicit attempts to learn a sequence can interfere with implicit learning. The possible relevance of the computations of the basal ganglia to emotional appraisal, automatic evaluation, script processing, and decision making are discussed.

These “feelings” have an efficiency of operation which it is impossible for thought to match. Even our most highly intellectualized operations depend upon them as a “fringe” by which to guide our inferential movements. They give us our sense of rightness and wrongness, of what to select and emphasize and follow up, and what to drop, slur over and ignore among the multitude of inchoate meanings that are presenting themselves . . . These qualities are the stuff of “intuitions.” (Dewey, 1925, p. 244)

In our culture, the legacy of intuition is less than inspiring. Intuition is seen as mysterious and unexplainable at best and as something inaccurate, hokey, or epiphenomenal at worst. Freud quipped that it is “an illusion to expect anything from intuition” (Jones, 1953, p. 327). The veiled processes behind our intuitions do not count as arguments or evidence (Smith & Decoster, 1999), which Goldberg (1983) satirically points out, “[no one every says] give me one good feeling why you think John is wrong” (p. 18).

Our culture is replete with cognitive maxims like “look before you leap” and “think before you act” that suggest that one’s impulses or intuitions tend to be deeply flawed. Our educational institutions give “little attention to the development of intuitive understanding” suggesting that intuition is not highly valued as a product of education (Bruner, 1960, p. 56). Our journals have dedicated many pages to showing that our intuitions systematically ignore important sources of information (Nisbett & Ross, 1980; Tversky & Kahneman, 1974) and that our judgments are improved when situations press us to reason more carefully (Chaiken, Liberman, & Eagly, 1989; Dienes-Raj & Epstein, 1994; Fiske & Neuberg, 1990; GIlbert, 1989; Langer, 1989; but see Bargh, 1997; Wilson & Brekke, 1994).

Although intuitions may often lead to suboptimal decisions, it is still possible that intuitions are sometimes as good or better than judgments derived from deliberation. This quality of intuitions is not necessarily a default circumstance due to deliberative strategies falling short when overused (Nisbett & Wilson, 1977; Schooler, Ohlson, & Brooks, 1993; Wilson & Brekke, 1994), but rather may be the result of the structural properties of intuition once it is considered in its proper information processing context.

It may seem strange to think of intuition in terms of information processing because phenomenologically, intuition seems to lack the logical structure of information processing. When one relies on intuition, one has no sense of alternatives being weighted algebraically or a cost–benefit analysis being undertaken. Oddly, it is just this lack of subjectively experienced reasoning that suggests intuition may have a home in the world of information-processing theory. In recent years, research on implicit learning has suggested that our behavior can be rule-like and adaptive without a concomitant conscious insight into the nature of the rules being used (Knowlton & Squire, 1996; Rieber, 1993). This similarity between intuition and implicit learning suggests that it may be fruitful to consider intuition as the subjective experience associated with the use of knowledge gained through implicit learning. Combining these two theoretical constructs may lend some deserved credibility to the former and some needed generalized applicability to the latter.

Intuitive and Deliberative Decision Making

Consider the prospect of initiating a romantic relationship. If one is deciding whether to start dating a particular person, one can construct a decision tree in which all the factors relevant to the decision are determined, rate the potential relationship on all these factors, and then tally the score (Winterfeldt & Edwards, 1986). Furthermore, one could focus on each factor relevant to the dating decision and begin a new level of the decision tree hierarchy for each of these. Thus, if one of the factors relevant to dating is “Could I conceive of myself ever living with this person on a day
to day basis? one could generate a sublist of factors relevant to
determining how the person in question should be evaluated with
respect to this question. The type of decision making being de-
dscribed here reflects a deliberative or analytic strategy.

Alternatively, an intuitive strategy would involve asking the
question Should I date this person? and waiting for a feeling to
emerge (Gilbert, Pinel, Wilson, Blumberg, & Wheatley, 1998).
Frequently, we engage in mixed strategies in which we enumerate,
and thus activate the cognitive substrate of, factors relevant to the
main decision, but then let intuition work on those factors, yielding
either an overall feeling or at least letting each factor receive its
weighting from our affective reaction rather than from a second
tier of cost–benefit analyses (Damasio, 1994).

Understanding the structural architecture of intuition and deliber-
eration is only of interest to the degree that choice of decision-
aking strategy affects the quality of the decision being made.
Thus, an obvious question is whether it matters which strategy is
used. The very unexciting answer proposed here is, it depends.
This answer is perhaps a bit more intriguing when it is seen in the
context of the general answers from social cognition and decision-
making theory, which give responses closer to, the more deliber-
ative components to decision making, the better. This answer can
be seen as a cultural value, institutionalized in our political, legal,
and school systems, as well as the implied or stated conclusion
from some of the most prominent research in field of decision
making (Arkes & Hammond, 1986; Denes-Raj & Epstein, 1994;
Fiske & Neuberg, 1990; Hammond, 1996; Hastie & Pennington,
1995; Nisbett, Fong, Lehman, & Cheng, 1987; Nisbett & Ross,
1980; Tversky & Kahneman, 1974; but see Bargh, 1997; Wilson &
Brekke, 1994).

Purpose and Structure of Review

The thesis proposed in this review is that intuition is a phenom-
enaological and behavioral correlate of knowledge obtained through
implicit learning. This claim is assessed in two ways. In the first
part of this review, an explication of the conceptual correspond-
ence between implicit learning and intuition, with particular
emphasis on social intuition, is laid out (represented by dotted
arrows in Figure 1). In the second and third sections, evidence
suggesting that the basal ganglia are the neuroanatomical bases of
both implicit learning and intuition is reviewed (represented by
solid gray arrows in Figure 1). Functionalism would suggest that
identical neuroanatomical bases must result in conceptual overlap
between processes of intuition and implicit learning. Using neu-
roanatomical localization to help determine the relationship be-
tween social and cognitive processes could be described as a social
cognitive neuroscience approach.

Logic of Intuition as Implicit Learning

Intuition

What is intuition? Most accounts have limited themselves to its
phenomenology. Bruner (1960) described the intuitive process as
one in which

The thinker arrives at an answer... with little, if any, awareness of
the process by which he reached it. He rarely can provide an adequate
account of how he obtained his answer, and he may be unaware of just
what aspects of the problem situation he was responding to. Usually,
imtuitive thinking rests on familiarity with the domain of knowledge
involved and with its structure... Analytic thinking characteristically
proceeds a step at a time. Steps are explicit and usually can be
adequately reported by the thinker to another individual. Such think-
ing proceeds with relatively full awareness of the information and
operations involved. (p. 57)

This dichotomy is one of the ancestors of dual process models
of cognition and social cognition (Chaiken & Trope, 1999; Fiske
Bruner's remarks allude to both the appeal and the danger of
intuitions. They are fast and take into account nonconsciously
generated information, gathered from experience, about the prob-
abilistic structure of the cues and variables relevant to one's
judgments, decisions, and behavior. These properties of intuition
are demonstrated in domains ranging from speeded chicken sexing
to infant language learning to chess expertise (Aslin, Safran, &
Newport, 1998; Biederman & Shiffar, 1987; DeGroot, 1965; Lunn,
1948; Safran, Aslin, & Newport, 1996). On the other hand, the
lack of conscious awareness of the information contributing to
one's intuitive judgment makes it impossible to delineate in re-
flexion, or for others in discourse, the justification for one's
judgment (Bastick, 1982; Clinchy, 1975; Griffin & Ross, 1991;
Hagafors & Breher, 1983; Smith & DeCoster, 1999).

Before giving a more formal definition of intuition, intuition
must be distinguished from insight or the Eureka! phenomenon
(Schooler & Melcher, 1994; Schooler et al., 1993). Sudden insight
also seems to rely on nonconscious processes, but when awareness
is derived in insight, it is not a judgment, as is usually the case in
intuition. Rather, insight is a process where one suddenly becomes
aware of the logical relations between a problem and the answer.
In the case of intuition, usually there is no insight into the logical relations, but simply an impetus, judgment, hunch, or behavioral response. That said, intuition is the subjective experience of a mostly nonconscious process that is fast, a-logical, and inaccessible to consciousness that, dependent on exposure to the domain or problem space, is capable of accurately extracting probabilistic contingencies.

**Intuitive Social Action and Intuitive Social Cognition**

There is a long, if sparse, history of associating intuition and social judgment (Valentine, 1929). Neisser (1963) suggested that intuition plays a prominent part in interpersonal relations, in our judgments of other people and our behavior towards them. Neither the therapist whose interpretations are appropriate, nor the actor gifted with a keen sense of timing seems to base his actions on rational deliberation. (p. 1)

In everyday experience, we rely on intuitive processes to understand the world around us. Despite our confidence in our intuitions, our intuitions often lead to biased or inaccurate judgments. A variety of low effort mental heuristics have been demonstrated to produce judgmental errors. Most individuals, for instance, will identify the following sequence of coin flips (H = heads, T = tails), HHHHTHTT, as less likely to occur randomly than the sequence HTHHTHTH, in fact when they are equally likely (Kahneman & Tversky, 1972). The latter sequence is taken as more probable because it strikes us as intuitively more representative of a random sequence.

The consequences take on greater importance when the heuristics used in social cognitive processes are considered (Greenwald & Banaji, 1995). Without the motivation or cognitive resources to reconsider and correct our intuitive judgments of others, we tend to mistakenly assume that single instances of behavior are indicative of enduring personality traits (Gilbert & Malone, 1995; Jones, 1979). Stereotypes often automatically guide our impressions of individuals (Banaji & Greenwald, 1994; Fiske, 1998; Kunda & Thagard, 1996) and directly shape our intuitive understanding of behaviors enacted by those individuals (Devine, 1989; Dovidio, Kawakami, Johnson, Johnson, & Howard, 1997; Duncan, 1976; Kunda & Sherman-Williams, 1993). For instance, we automatically experience a shove from a Black person as more aggressive than a shove from a White person (Sagar & Schofield, 1980). More generally, implicit attitudes shape our current intuitive judgments without our awareness of this influence (Bargh, Chaiken, Govender, & Pratto, 1992; Bargh, Chaiken, Raymond, & Hymes, 1996; Fazio, Sanbonmatsu, Powell, & Kardes, 1986; Greenwald, Draine, & Abrams, 1996). Each of these intuitive processes can be thought of in terms of automatic activation of conceptual associations (McClelland, McNaughton, & O‘Reilly, 1995; Smith & DeCoste, 1998, 1999).

There are other social psychological processes that are dependent on sequential or temporal associations. In contrast to the conceptual associations, the sequential associations are more consistently associated with accuracy. In the domain of nonverbal decoding, cue sequences are used to gauge the emotions, personality, intentions, attitudes, or skill level of others. In an extreme example of intuitive social information processing, participants were able to identify facial expressions presented for 5 ms (Rosenthal, Hall, DiMatteo, Rogers, & Archer, 1979). In a recent variation, Edwards (1998) found participants were able to order 14 photographs reflecting the temporal sequence of an emotion unfolding. The 14 photos were taken over a period of less than 1 s, and there is little discernible information in the photos that would suggest the application of deliberative processes. Lewicki (1986) reported that participants were able to detect minute violations of the basic proportions of the human face. The participants reported feeling something was wrong with the faces, but none of the participants were able to specify what was wrong with the faces. In a study with real world import, Ambady and Rosenthal (1993) had undergraduates watch a 6-s clip of an instructor teaching, with the sound turned off. The students were then asked to rate the teaching ability of the teacher. These ratings correlated .71 with the ratings other students gave after having had the same teacher for an entire semester. This correlation did not drop appreciably after covarying out teacher attractiveness. Because participants in these studies often report feeling they have insufficient information on which to base their judgment, researchers often prompt participants to rely on their intuitions.

Nonverbal decoding skill develops over time with practice. This is an important component of intuition in contrast with instincts and innate abilities. Skill in nonverbal decoding improves from early childhood through early adulthood (Cohen, Prather, Town, & Hynd, 1990; Dimitrovsky, 1964; Hamilton, 1973; Rosenthal et al., 1979).

Nonverbal decoding is a potent example of intuitive processing in social psychology, as it involves drawing inferences with little effort or attention about the internal mental states and dispositions of other individuals on the basis of subtle sequences of nonverbal cues (Ambady, 1999; Lieberman & Rosenthal, 1999). Despite the lack of insight into the nature of our nonverbal inferences, it is more often than not an accurate source of information. As such it is a paradigmatic example of intuitive social cognition for sequential information.

Neisser indicated a second form of intuition in his example of the gifted actor with great timing. This sense of fluidity and timing is the other side of nonverbal communication research: encoding or production. At all times, we are communicating information about our emotional state, attitudes, and evaluations of whatever we are currently confronting (DePaulo, 1992). Encoding refers to the process of translating a mental state into an externally visible signal like a facial expression. Several of the nonverbal cues that reflect our internal state can be controlled consciously to some degree, but this will only occur if one directs one’s conscious attention to the process of nonverbal encoding. Most often we are unaware of our own encoding (Barr & Kleck, 1995). Additionally, there are other cues to one’s internal state (e.g., tone of voice, blinking, posture) that the vast majority of us have little or no control over (Ekman & Friesen, 1969). We produce most of our nonverbal cues intuitively, without phenomenological awareness. Our internal state is translated into nonverbal production without our conscious attention, and the translation process is, for the most part, outside of conscious awareness. As such nonverbal encoding is a compelling example of intuitive social action.

Using nonverbal communication as a reference point, intuitive social action and cognition have relevance for several domains of social psychological investigation. For instance, Schlenker and Wiegold (1992) have argued that much of impression management...
is "the result of habitual patterns of behavior triggered automatic-
ically and unthinkingly by situational cues" (p. 147). Similarly,
DePaulo (1992) suggested that we ordinarily focus on the impres-
tion we want to convey rather than on the nonverbal cues needed
to actually produce the effect:

Instead of trying deliberately to lean forward, smile, and gaze, people
probably instead try to convey an impression of liking, with the
leaning and smiling and gazing following from that, perhaps even out
of their awareness. (p. 225)

Because the skill is fundamentally intuitive rather than con-
ciously analytic, the intentional modification of one's impression
management behavior can often lead to poorer results than not
thinking at all and allowing intuitive social action to hold the
reins (Schlenker, 1987). If this is the case, why do people ever try
to consciously control the behavioral impression they are making?
Bruner (1960) suggested that individuals forgo intuitive processing
for more consciously controlled analytic strategies when the stakes
are high. Consistent with Bruner's hypothesis, consciously con-
trolled impression formation tends to occur more with increases in
the importance of the behavioral performance (Leary & Kowalski,
1990; Schlenker, 1985).

Automatic use of stereotypes was included earlier in the class of
conceptual, rather than temporal, intuitions, but one of the initial
demonstrations of implicit stereotyping was based entirely on
unintentional nonverbal cue production and implicit nonverbal
decoding, which do fall into the class of sequential associations.
Word, Zanna, and Cooper (1974) examined how implicit racial
decoding, which do fall into the class of sequential associations.

Neither applicant nor candidate was aware of the nonverbal sub-
cues and unintentionally encoded the same cues in response.
Black candidates, without awareness, decoded these
viewer that might make the candidate appear less desirable
White interviewers displayed the same coldness cues to Black
interactors, increasingly reciprocal the coldness cues communicated by
the interviewer. Applicants were displaying their racial attitudes
not through their words but through their unintentional nonverbal
production. Black candidates, without awareness, decoded these
cues and unintentionally encoded the same cues in response.
Neither applicant nor candidate was aware of the nonverbal sub-
text of the interview. Similarly, Chen and Bargh (1997) found that
activating an African American stereotype in one interactant re-
sulted in a second interactant behaving in a more hostile fashion.

Thus, Neisser's anecdotal distinction between intuitive social
cognition and intuitive social action appears to be a useful one.
Within the realm of nonverbal communication, one of the basic
elements of social cognition and action, the distinction is a long-
standing one. Data presented in subsequent sections suggest that
the distinction is also borne out at both the cognitive and neural
levels.

Implicit Learning and Intuition

Imagine as a participant in an experiment the task is to deter-
mine as quickly as possible in which of four quadrants a target
stimulus appears. The trials are broken into groups of seven, which
is clear only because every seventh trial is different from the
preceding six. On the first six trials, the target appears by itself on
the screen, but on the seventh trial it is embedded in a screen full
of other distracters. This is the participant's perspective in a matrix
scanning experiment carried out by Lewicki, Czyzewski, and
Hoffman (1987). The participant was unaware of a subtle relation-
ship between the sequence of target locations on the first six trials
and the location of the target on the crucial seventh trial. More
specifically, the sequence of locations on Trials 1, 3, 4, and 6
combined to form a code that predicted the exact location of the
target on Trial 7. There were 24 sequences that could occur across
these four trials, which map on to 24 possible locations of the
target on Trial 7. Note that Trials 2 and 5 were distracters that had
no predictive use. Also, note that the location of the target on any
one trial did not correlate at all with the location on Trial 7. Only
the sequence of the four key predictors together allowed for
accurate prediction of the target on Trial 7.

Participants received 11 hr of practice on the task. During this
time, speed improved, and this would be expected just from greater
familiarity with the task. Lewicki, however, demonstrated that part
of this speed improvement was a result of their having implicitly
learned the relation between the predictor sequences and the target
trials. In the 12th hour, the relation between predictor sequence
and seventh trial target was changed. With this change, partici-
pants showed a significant increase in reaction times. Lewicki et
al. (1987) pointed out that with this change, "intuitive knowledge
about the pattern acquired by the subjects not only was of no help,
but could even slow down their responses" (p. 526). When the
previous predictive sequencing was restored, so were the faster
reaction times. The temporary increase in reaction times was a
reflection of the probabilistic knowledge that had been gained in
the 11 hr that had suddenly become an inaccurate predictor. The
participants had learned the probabilistic contingencies between a
sequence of predictors and target despite the impossibility of
conscious analysis. It is noteworthy that participants were not told
that the seventh trial was a key dependent measure for the exper-
imerter, nor were they told that there was any relation to be
deciphered between any of the trials.

This type of learning has been called implicit learning for more
than three decades (Reber, 1967; for thorough reviews, see Lam-
berts & Shanks, 1997; Seger, 1994; Stadler & Frensch, 1998).
Reber (1993) defined implicit learning as

the acquisition of knowledge that takes place largely independently of
conscious attempts to learn and largely in the absence of explicit
knowledge about what was acquired. One of the core assumptions of
our work has been that implicit learning is a fundamental, "root"
process, one that lies at the very heart of the adaptive behavioral
repertoire of every complex organism. (p. 5)

This definition proposes a central place in human cognition for
implicit learning, but it is only recently that there has been a surge
of more general interest in this learning process. This is the case
for two reasons. First, there are ongoing debates within the re-
search niche regarding what kind of representational structure is
being learned: abstract versus prototype based knowledge (Brooks,
1978), perceptual fluency versus rule learning (Perruchet &
Pacteau, 1990), and hybrid versus hierarchical structures (Curran
& Koele, 1993). The extent to which the learning requires attention
and intention is also not agreed on (Goschke, 1997; Jimenez &
Mendez, 1999; Miller, 1987; Nissen & Bullemer, 1987; Reed &
Johnson, 1994). These are important debates that will shape a more
complete understanding of implicit learning but at the same time may keep the general intellectual journeymen of our field from passing through and mining this research territory.

Second, there is the question of "Neat finding, but what does it do?" Reber initially offered implicit learning as an alternative to Chomsky's (1980) nativist theory of language acquisition, a proposal deserving a second look, in light of Saffran et al.'s (1996; Aslin et al., 1998) finding on infant language acquisition. Although researchers have long considered the broad implications of automaticity in general, until only recently the study of implicit learning has mostly focused on the power and integrity of the process rather than on real world analogues of implicit learning at work. One of the themes of this review is to suggest that implicit learning is the cognitive correlate of some forms of social intuition, with the hope of broadening the domain of applicability of implicit learning.

Lewicki's finding (Lewicki et al., 1987) might be thought of as at least partly a motor learning skill because learning was measured in terms of the speed of a motor response. Other implicit learning studies have focused on classification judgments that have no motor component (Berry & Broadbent, 1984, 1988; Koh & Meyer, 1991). Reber's research program has mainly relied on the artificial grammar task. In these experiments, participants memorize letter strings consisting of consonants (e.g., XTRLRJR). Unbeknownst to the participant, the construction of these letter strings follows a complex set of sequencing rules based on a Markovian grammar (see Figure 2). Participants are then asked to take a new list of words and classify them as grammatical (i.e., rule-based) or not, but they are given no explicit information about the Markovian grammar. Participants consistently perform at higher levels than chance in this discrimination task, though they feel like they are relying on intuitive guesses rather than real information. As with nonverbal communication research, implicit learning research also involves explicit directions for participants to rely on their intuitions (Knowlton, Ramus, & Squire, 1992).

Many variations on the original artificial grammar task have revealed much of what is actually being learned. For instance, participants could be learning the surface structure relations between the specific letters presented in the exposure phase, but Reber (1969; Brooks & Vokey, 1991; Manza & Reber, 1997; but see Dienes & Altman, 1997) found that the deep structure of the artificial grammar transfers to a new set of letters combined according to the previously learned rules. Knowlton and Squire (1996) found that both abstract grammar representations and conscious fluency with letter pairs and triplets are used in the artificial grammar task. Manza and Reber (1997) have also found successful transfer across sensory modalities.

Covariation detection and frequency detection are at the root of implicit learning. Intuitive social cognition and action also appear to be based on implicit covariation and frequency knowledge. There is some debate over the quality of our ability to consciously assess covariation relationships in social phenomena (Alloy & Tabachnik, 1984; Crocker, 1981; Gavanski & Hoffman, 1987; Jennings, Amabile, & Ross, 1980; Malmi, 1986), but there is substantial evidence that implicit learning is a mechanism that could shape accurate covariation detection of social phenomena as a more unintended intuition.

In one attempt to bridge the gap between implicit learning and social cognition, Lewicki (1986) had participants make personality judgments on the basis of a picture and a list of descriptors. The pictures were chosen such that there was a subtle correlation between hair length and a personality trait. In the test phase, participants accurately used this covariation information to rate the personality of new faces. When asked to justify their choices, participants would often point to the look of the eyes but never to hair length. The participants were unaware of the covariations present that were reliably affecting their judgment. This is quite similar to the relationship between subjective experience and the underlying processes in nonverbal decoding and implicit stereotyping, primary examples of intuitive social cognition.

Recently, Seger (1997) suggested a dichotomy of implicit sequence learning processes that parallels the aforementioned split between nonverbal encoding and nonverbal decoding. She proposed that there are judgment implicit learning processes that are qualitatively distinct from motor-response implicit-learning processes. It seems reasonable that to the extent that nonverbal communication does rely on implicit learning processes, nonverbal decoding should rely more on judgment implicit learning, whereas nonverbal encoding should rely more on motor-implicit learning.

So far, an informal case has been presented that, on the basis of the characteristics associated with intuition and implicit learning, they are at least related processes. Although none of the parallels between social intuition (e.g., nonverbal decoding) and implicit learning demand the two be equated, the evidence is suggestive. Nonverbal decoding and implicit learning both (a) involve temporal sequencing and prediction, (b) involve learning that occurs mostly outside of conscious awareness, (c) result in representations and skills that are applied mostly without conscious direction or intent, (d) are most parsimoniously divisible into judgment and action forms, and (e) are dependent on multitrial learning, with skill developing slowly over time. To establish more rigorous empirical links, we turn to a review of the neural bases of implicit learning and social intuition. To the extent that implicit learning and social intuition have the same neural bases, it should follow that they are linked functionally and computationally.

Neural Basis of Implicit Learning: The Basal Ganglia

In the following subsections, evidence linking the basal ganglia to implicit learning is reviewed. There are several lines of inquiry surrounding the function of the basal ganglia, and there is often minimal cross-referencing between them. There does, however, seem to be some thematic consensus across these lines. In general, the basal ganglia learn temporal patterns that are predictive of events of significance, regardless of conscious intent to learn the predictor–reward relationship, as long as exposure is repeatedly
instantiated. After the temporal representation is formed, presentation of the early sequence elements will lead to activation of the entire temporal pattern, a process referred to as temporal pattern completion, which is tantamount to prediction of later sequence elements. Additionally, the basal ganglia are capable of notifying different areas of the brain of the presence of the ensuing event, so that individuals may shift their attention or act upon the environment to adjust for the event of significance. Because sequential representations only form if predictor cues really predict rewards, these representations are structurally designed to lead to intuitions that are accurate.

Certainly other parts of the brain, such as the hippocampus and amygdala, constitute memory systems as well that contribute to the recognition of meaningful patterns in the environment (McDonald & White, 1993). Each of these systems is adapted to different components of information in the environment, and each stores the information in a different manner. The hippocampus, for instance, appears adapted for the rapid formation of configural and conceptual associations between cues that occur at the same time and are consciously accessible (McClelland et al., 1995). Information in the basal ganglia develops at a glacial speed, in comparison, and is associated with learning sequences and probabilistic relationships that are not accessible to consciousness (Rauch et al., 1995). These properties suggest the basal ganglia as a prospective neuroanatomical correlate of implicit learning.

Neuroanatomy

The basal ganglia consist of three major components: striatum, substantia nigra (SN; see Appendix for a list of abbreviations), and globus pallidus (GP). The striatum receives inputs from most areas of the cortex and limbic system, and can be subdivided into the caudate, putamen, and nucleus accumbens. Dorsal or upper striatum is made up of the putamen and the body of the caudate, whereas ventral or lower striatum is composed of the head of the caudate and the nucleus accumbens. The substantia nigra can be divided into the substantia nigra pars reticulata (SNr) and substantia nigra pars compacta (SNC), the latter of which is the major dopaminergic (DA) input to the basal ganglia. Five corticostriatal loops or circuits are known to connect the basal ganglia to the rest of the cortex (Alexander, Delong, & Strick, 1986). Each of these circuits starts in multiple related sites in the cortex, converge on the striatum, pass through to GP, to the thalamus, and back to DLPFC, which controls saccades (Akert, 1964; Kunzle & Akert, 1977). These circuits reveal that the basal ganglia are in a privileged position in their potential to affect attention, cognition, emotion, and behavior. The anatomical position and general connectivity of the basal ganglia, however, reveals little of what is done with this potential. To better understand the role of the basal ganglia in cognition, the review turns to the research domains of neuropsychology, neuroimaging, and neurophysiology.

To understand the information-processing role of the basal ganglia, it is necessary to consider the neuroanatomy and neurochemistry of the basal ganglia in finer detail. This detail, some of it still tentative, offers an explanation of how the basal ganglia come to recognize patterns of cortical activation associated with behaviorally significant stimuli and eventually comes to recognize the predictors of these same stimuli. Because of the range of circuit types, the predictor patterns and predicted events might be as disparate as a light predicting the location of a food reward (McDonald & White, 1993) or a retrieval strategy predicting successful recall of an old memory (Gabrieli, 1995).

Neuropsychological Evidence

Evidence for the role of the basal ganglia in implicit learning and other intuitive nonconscious informational processes comes from neuropsychological research on Huntington’s disease (HD) and Parkinson’s disease (PD) patients. HD and PD are both degenerative brain diseases that affect the striatum of the basal ganglia very early in the disease course. Both ultimately ravage the striatum until it is dramatically atrophied and metabolically weakened. If this were the anatomical extent of the damage in these diseases, they would be ideal testing grounds for the functioning of the basal ganglia. Unfortunately, both diseases also lead to atrophy and metabolic changes in other areas of the brain, most notably the prefrontal cortex (PFC). This makes it difficult to localize the brain areas associated with cognitive deficits in HD and PD.

Through neuroimaging and postmortem studies, there is now a relatively clear map of the disease course in the HD and PD affected brains. This affords two other research avenues for studying the function of the striatum using neuropsychological methods. Knowledge of disease course allows us to select HD and PD patients in different stages of neuropathology for which the affected brain areas differ. Second, cognitive and behavioral functions can be correlated with the extent of atrophy and hypometabolism in different areas of the brain as revealed by computed tomography (CT) and positron emission tomography (PET) scans, respectively. Thus, if recall deficits are highly correlated with striatal atrophy and metabolism in HD, but not with frontal atrophy
and metabolism, this would suggest that the recall is likely to be causally linked with striatal constitution. This would not suggest that recall deficits in all diseases (e.g., Alzheimer’s) are rooted in the striatum but rather would provide evidence that the striatum can be one of the contributors to normal recall.

Parkinson’s disease. PD is a neurodegenerative disease affecting 1% of those 65 and older. Patients tend to present symptoms including bradykinesia (slowing of movement), akinesia (absence of spontaneous movement), rigidity, awkward gait and dementia (Gabrielli, 1995; McPherson & Cummings, 1996). Neurochemically, PD causes massive cell death in the SN, the major source of DA for the striatum. DA levels in putamen and caudate are reduced to between 5% and 22% of normal levels (Fahn, Libsch, & Cutler, 1971; Gotham, Brown, & Marsden, 1988), with putamen being affected first and more severely (see Figure 4a; Canavan et al., 1989a; Nahmias, Garnett, Firnau, & Lan, 1985). There is evidence that mesolimbic and mesocortical dopaminergic projections are also reduced because of cell loss in ventral tegmental, but this constitutes a smaller reduction as these DA projections are more resistant to degeneration than the nigrostriatal pathway (Hung & Lee, 1996). Consequently, DA levels of the hippocampus and frontal cortex are reduced, but only to 40%, and the DA levels of the anterior cingulate, amygdala, and hypothalamus are reduced to 50% of normal (Fahn et al., 1971; Gotham et al., 1988; Horykiewicz, 1973; Javoy-Agid & Agid, 1980; McGeer, McGeer, Itagaki, & Mizukawa, 1987; Nahmias et al., 1985; Price, Farley, & Hornykiewicz, 1978; Scatton, Rouquier, Javoy-Agid, & Agid, 1982).

It has been suggested that despite its widespread influence on the brain, PD is the best neuropsychological model of basal ganglia functioning (Marsden, 1982; Saint-Cyr, Taylor, & Lang, 1988). There are at least three reasons for this proposal. First, early damage in PD seems limited to the basal ganglia, and thus studies using PD patients in the early phases of the disease (EPD) should provide data regarding basal ganglia function. Second, reduction in DA levels only affects local neural output if the reduction is 85% or more (Brozoski, Brown, Rosvold, & Goldman, 1979; Goldman & Rosvold, 1970). In PD, the DA reductions in putamen, and potentially caudate, are in this range, but frontal lobe reductions are not. Even though frontal lobe DA levels are reduced, they may not be reduced enough to impact behavior, thus shifting explanatory power to the basal ganglia. Furthermore, even though PD leads to some deficits that are superficially similar to frontal lobe symptoms, the underlying attentional and information processing deficits can be differentiated (Owen et al., 1993).

The third reason is rooted in Marsden’s (1982) suggestion that the basal ganglia are fundamentally motor related, rather than cognitive. Marsden suggests that the functioning of the basal...
Changes over Time

**Early**  
**Late**

**A** Parkinson's Disease

- **Caudate**
- **Putamen**
- **Prefrontal Cortex**

**B** Huntington's Disease

- **Putamen**
- **Caudate**
- **Prefrontal Cortex**

*Figure 4.* (A) Temporal course of degeneration in the Parkinson's disease brain. Initially affects putamen and spreads to caudate and prefrontal cortex (PFC). (B) Temporal course of degeneration in the Huntington's disease brain. Initially affects caudate and spreads to putamen and PFC.

ganglia cannot be cognitive because their output is not accessible to consciousness:

The rather casual use of the term “cognitive” by experimental animal psychologists has led to some confusion. They employ “cognitive” to describe those processes by which animals obtain knowledge of objects or become aware of the environment irrespective of whether the animal is conscious of such awareness. The clinician, however, describes “cognitive functions” as those concerned with recognition, perception, identification, understanding, imagination, learning, thinking and even speech... All these aspects of cognitive state in human patients demand access to conscious thought processes. (1982, p. 517)

Given the general consensus in experimental psychology that most psychological processes are transparent and inaccessible to consciousness, this seems an untenable position, but if one takes this view suggesting the basal ganglia are fundamentally motor areas, then PD, especially in the early stages, becomes a very attractive disease to study because it targets the putamen earlier and more severely than the caudate nuclei (Bernheimer, Birkmayer, Hornykiewicz, Jellinger, & Seitelberger, 1973; Kish, Shannon, & Hornykiewicz, 1988; Martin et al., 1987; Nahmias et al., 1985). Putamen is linked to areas of the cortex that are clearly involved in motor activity, where the caudate nuclei have links to DLPFC, OFC, and AC all considered primary sites of higher cognitive activity (Alexander et al., 1986). If one is interested in the information processing consequences of caudate functioning, the benefits of PD research is less clear since the progression of the disease to the caudate and the frontal cortex may be temporally confounded (see Figure 4a).

The fundamental split in the striatum between the projections of the caudate and putamen suggest that they might be differentially involved in intuitive social action and cognition, hearkening back to Seger’s (1997) finding of a similar split in implicit learning. Putamen ought to be more involved in intuitive social action and caudate more involved in intuitive social cognition. Referring back to nonverbal communication as a prototype of intuitive social action and cognition, this division at the level of the striatum suggests that putamen might be implicated more in nonverbal encoding, whereas caudate would have a larger role in nonverbal decoding.

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**Huntington's disease.** HD is a genetically inherited degenerative brain disease, which causes chorea (random irregular muscle movements), dystarthisia (speech articulation problems), dystonia (involuntary postural movements), bradykinesia, apathy, depression, disinhibition, and dementia as well as an impairment of social judgment (Brant & Butters, 1996; Caine, Hunt, Weingartner, & Ebelt, 1978; Hopkins, 1994; Rothlind, Bysma, Peyser, Folstein, & Brandt, 1993; Shelton & Knopman, 1991; Young et al., 1986). The neuropathology of HD involves severe loss of cells in the caudate nuclei upwards of 80%, with additional, though less substantial, damage to putamen that appears subsequent to initial caudate damage (Albin et al., 1990; Bernheimer et al., 1973; Bruyn, Bots, & Dom, 1979; Dom, Malfroid, & Barn, 1976; Forno & Jose, 1973; Reid et al., 1988; Vonsattel et al., 1985; Willingham, Koroshetz, Treadwell, & Bennett, 1995; Young et al., 1986; 1987). There is considerable disagreement concerning HD's effect on PFC. As with PD, the efficacy of using HD as a model of the cognitive functions of the basal ganglia turns on the whether and the when of cortical damage in HD. Most CT studies have found significant atrophy of the caudate and little or no atrophy of the frontal cortex in HD (Bruyn, 1973; Kuhl et al., 1982; Nemeroff, Youngblood, Manberg, Prange, & Kizer, 1983; Tanahashi et al., 1985; Vonsattel et al., 1985; but see Hedreen, Peyser, Folstein, & Ross, 1991, and Sotrel et al., 1991). There is also little evidence of metabolic change in the frontal cortex, especially in the early and middle stages of the disease, whereas metabolic changes in the caudate are seen from the earliest stages of the disease (see Figure 4b; Berent et al., 1988; Hayden et al., 1986; Hayden et al., 1987; Kuhl et al., 1982; Kuhl, Metter, Riege, & Markham, 1984; Martin et al., 1992; Mazziotta et al., 1985; Mazzuotti et al., 1987; Reid et al., 1988; Weinberger, Berman, Iadarola, Driesen, & Zec, 1988; Young et al., 1986).

Correlational studies reinforce the link between caudate damage and the various cognitive symptoms of HD. Measures of caudate atrophy and metabolism correlate strongly with clinical severity of
cognitive symptoms (Hayden et al., 1987; Kuhl et al., 1982; Starkstein et al., 1988; Young et al., 1986, 1987), components of the Wechsler Adult Intelligence Scale and Wechsler Memory Scale (Berent et al., 1988; Sax et al., 1983), and ability to function in everyday life, as indexed by the Total Functioning Capacity score and the Activities of Daily Living questionnaire scale (Bamford, Caine, Kido, Plassche, & Shoulson, 1989; Rothlind et al., 1993; Shoulson, Bamford, & Caine, 1985; Shoulson, Plassche, & Odoroff, 1982; Sax et al., 1983; Starkstein et al., 1988). Perhaps more importantly, these correlations do not obtain for cortical atrophy or metabolism in HD (Josiassen, Curry, & Mancall, 1983; Kuhl et al., 1982; Neophytides, DiChiro, Barron, & Chase, 1979; Sax et al., 1983; Starkstein et al., 1988; Weinberger et al., 1988; Young et al., 1986; but see Starkstein et al., 1992, and Tanahashi et al., 1985).

Lesion studies examining caudate infarcts and hemorrhages shed additional light on the existence of an independent role for the caudate nuclei in cognition and on the relation of this role to the putamen's role in motor planning. Bhalla and Marsden (1994) examined 43 patients with lesions isolated to the caudate. Of these, 23% presented chorea or dystonia and 77% presented cognitive deficits including disinhibition and abulia (loss of spontaneous thought). Of the 7 patients examined with putaminal lesions all had motor symptoms, but none showed signs of abulia. Caplan et al. (1990) found all 4 caudate infarct patients they examined displayed abulia but only 1 of 5 putamen patients. Given that PD patients often display akinesia, a motor symptom, it is interesting that abulia has also been referred to as psychic akinesia, reinforcing the notion that the caudate operates on cognitive data, similar to the way the putamen operates on motoric data. Mendez, Adams, and Lewandowski (1989) found three different groups of cognitive-affective changes in the patients with caudate infarcts. The first group showed the symptoms of abulia and had damage to dorsolateral caudate which projects to DLPFC. Patients in the second group were socially inappropriate, disinhibited, and impulsive. These patients had damage to ventromedial caudate which projects to orbitofrontal and ventromedial prefrontal cortex. The third group showed affective symptoms including anxiety and had damage to the caudate nuclei that would disrupt projections to the limbic areas.

Considering these different sources of data it appears that the caudate nuclei undeniably play an independent role in cognition. Additionally, there is strong evidence that the neurodegeneration in the HD brain, particularly in the early stages of the disease, affords the opportunity to study the functions of the caudate without the competing hypothesis of cortical causality.

One caveat must be made. Given the circuit-like connections between caudate and the cognitive cortex (Alexander et al., 1986), it can be argued that a damaged caudate is sending noisy data that disrupts the processing in the cortex, still maintaining that all the important information processing features of higher cognition are located in the cortex. If one is to make this argument though, it must be conceded that the argument can be reversed suggesting that the caudate is the seat of higher cognition and its processes are disrupted in frontal lobe patients whose cortex sends noisy signals to the caudate. We might fairly speculate that the cortex and the caudate process similar data, given their circuitry, but extract from and contribute to the data flow in different ways. This chicken and egg conundrum is not resolvable at this time given that HD and frontal lobe damage cause many similar symptoms. It is interesting to note, though, that Knowlton and her colleagues have reported data that reflect a functional dissociation between caudate and cortex (Knowlton, Mangels, & Squire, 1996; Knowlton et al., 1996; also Owen et al., 1993). Knowlton found frontal patients performed normally on a probability classification task, whereas HDs were impaired. If damage to the caudate is relevant to cognitive processes only in the distracting noise delivered to the cortex, frontal lobe damage should include all caudate related cognitive impairments within its own catalogue of cognitive impairments.

Characterizations of basal ganglia functions. If we accept that HD and PD are good models of basal ganglia function, it is appropriate to examine the deficits associated with these neurological syndromes and the conclusions of various researchers regarding the general function of the basal ganglia. The range of suggested functions is wide, spanning the continuum from low-level motor functions to high-level executive functions.

At the lowest level of the information-processing hierarchy, there is a long-standing view that the basal ganglia are involved in motor coordination. This is undoubtedly a component of basal ganglia functioning given the plethora of motor symptoms associated with the early stages of both PD and HD. Hallett and Koshin (1980) have suggested that the basal ganglia help select specific muscles to be used in movements and foster the inhibition of those muscles irrelevant to the movements. Others have pointed to a higher level motor function for the basal ganglia, suggesting they are involved in the automatic execution of learned motor plans (Marsden, 1982; Saint-Cyr & Taylor, 1992; Shelton & Knopman, 1991).

More recently, several proposals have surfaced linking basal ganglia function and frontal lobe function, given the anatomical connections between the two areas (Alexander et al., 1986). Patients with PD, and to a lesser degree HD, present cognitive symptoms that bear a resemblance to frontal lobe syndrome. These patients have difficulty shifting mental set or strategy, initiating planning, and monitoring goal-directed behaviors (McPherson & Cummings, 1996). Additionally, both HD and PD patients show marked deficits in recall but not recognition (Brandt, 1985; Butters, Wolfe, Martone, Granholm, & Cermak, 1985; Faglioni, Botti, Scarpa, Ferrari, & Saeiti, 1995; Faglioni, Scarpa, Botti, & Ferrari, 1997; Gabrieli, 1995; Heindel, Butters, & Salmon, 1988; Kramer et al., 1988; Kramer, Levin, Brandt, & Delis, 1989; Vriezen & Moscovitch, 1990; for opposite findings see Caine, Ebert & Weingartner, 1977). Out of this area of research comes a variety of similar proposals that the basal ganglia are involved in spontaneous generation of task specific planning and sequencing (Bloxham, Mindel, & Frith, 1984; Brown & Marsden, 1991; Caine et al., 1978; Harrington & Haaland, 1991; Lange et al., 1992; Saint-Cyr & Taylor, 1992).

Although these proposals account for a good deal of extant data, there are two limitations on this line of thought. First, these proposals are heavily steeped in PD research with few replications with HD patients. Given the disease course of PD, the causal properties of the frontal cortex and caudate may be confounded (see Figure 4a). This point is crucial because the caudate is the only part of the striatum that projects to nonmotor areas of the cortex. It is unclear whether frontal-like deficits in PD are a result of DA depletion in caudate or in the frontal cortex itself. A second
issue is that the frontal function proposals do not differentiate the information processing done by the basal ganglia and the sites of its cortical projections. The circuitry linking the basal ganglia and cortex suggests that the different brain areas are operating on the same data but transform the data in different ways just as different stations on an assembly line contribute different features to a single finished product. For instance, whereas both PD patients and frontal lobe patients appear to perseverate on a particular way of responding, Owen et al. (1993) found that frontal lobe patients have difficulty disengaging from a particular way of responding, whereas PD patients have difficulty re-engaging attention to a new pattern of responses that was earlier learned to be irrelevant.

Another set of hypotheses of striatal functioning addresses the division of labor between the cortical and subcortical areas in question. On the basis of animal research, Mishkin and colleagues have suggested that the basal ganglia are the primary site of habit learning (Mishkin, Malamut, & Bachevalier, 1984; Mishkin & Petri, 1984). This proposal is broad enough in scope to encompass both the motor and cognitive findings in neuropsychological research. There are certainly motor habits, tendencies, and biases, but there is also a strain of cognition that is habitual, namely, implicit learning (Anderson, 1982; Reber, 1993).

Additionally, the habit-learning proposal suggests a delineation of cortical versus striatal contributions to cognition. Frontal lobe theories focus on the ability of the cortical areas to flexibly incorporate new data, to shift data in and out of working memory, and to shift attention on the basis of internal goals (Baddeley, 1986; Norman & Shallice, 1986; O’Reilly, Braver, & Cohen, 1997). If anything, the frontal lobes are in the business of inhibiting prepotent or habitual processes, not generating them.

Many neuropsychologists extending the habit theory include skill learning among the functions of the basal ganglia (Gabrieli, 1995; Knowlton & Nissen, 1991; Knowlton & Squire, 1996; Knowlton et al., 1996; Mendez et al., 1989; Prado-Alcala, 1985; Taylor & Saint-Cyr, 1995; Taylor, Saint-Cyr, & Lang, 1986), though there is no consensus as to whether the basal ganglia are necessary for the expression of a well-learned skill (Gabrieli, 1995; Shelton & Knopman, 1991). Two elements of skill learning that should be considered separately are nonconscious processing and predictive sequencing.

Nonconscious processing, with respect to basal ganglia functions, refers to the process of the skills being learned slowly switching from being under conscious supervision in the PFC and hippocampal region (McClelland et al., 1995) to nonconscious operation in the striatum (Ashby, Alfonso-Reese, Turken, & Waldron, 1998; Marsden, 1982; Taylor et al., 1986). For instance, when learning a new domain of knowledge, one might rely heavily on consciously invoked mnemonics to facilitate accurate retrieval of the new knowledge. Over time and with practice, the knowledge comes to mind readily without the use of an intervening cue. The skill of recall and use of the new knowledge domain becomes increasingly automatized. This does not imply that automatized functions are completely independent of consciousness. Conscious intentions are certainly involved in the mobilization of automatic processes (Cohen, Dunbar & McClelland, 1990; Wegner & Bargh, 1998).

Caine et al. (1978) provided anecdotal evidence of striatal involvement in predictive sequencing from an HD patient that was still practicing dentistry, "I would be standing there with a tool in my hand and with the [patient’s] mouth open, and I wouldn’t remember what to do next. I had done these things for years" (p. 379). Another of Caine’s patients noted that she could no longer prepare Thanksgiving dinner because she could not remember the sequence of steps involved. She could remember each element, just not the sequence. If the computational notion of constraint satisfaction is applied, some light is shed on the relationship between sequencing errors and prediction errors. When part of a well-learned representation is activated in a neural network, pattern completion occurs such that the rest of the pattern also becomes active to result in a coherent overall interpretation of partial data. Thus, a partly occluded object can be recognized (Rumelhart & McClelland, 1986). Similarly, if part of a well-learned temporal sequence is activated, particularly an early temporal element, then the representations of the later elements in the sequence would also become active. Thus, sequenced cognition and reward prediction can be construed as the same neural representations being used in different ways. In the one case, the temporal associations guide one through a set of sequential operations, whereas in the other case, the presence of early cues activates later cues that bring them to mind as intuitions. The fact that the temporal pattern completion is automatic will leave individuals unable to recognize explicitly that the early cues are guiding their intuitions (Bargh, 1989).

Consider playing defense in a game of basketball. Often times the offensive player being guarded will try to fake out the defense by making a set of gestures and movements suggestive of going in one direction while actually going in the opposite direction. The speed with which the defensive player can detect the fake out and defend the direction the offensive player wants to travel is one essential determinant of the player’s level of defensive skill. The fake-out sequence is fast and subtle, too fast to be interpreted and responded to efficiently by serially processed frontal lobe representations, but slowly over time the basal ganglia may form a representation of the sequence and eventually temporal pattern completion processes would activate the later elements of the sequence (e.g., fake out) to the defensive player as the earlier elements of the sequence unfold. This would prompt the defensive player to make the behaviorally relevant response (e.g., getting back on defense) earlier and earlier in the sequence.

Procedural learning, implicit learning, and predictive sequencing. Automatic predictive sequencing appears to be a plausible function of the striatum that would account for much of the data driving cognitive and motor theories of the basal ganglia without sacrificing function specificity. In this section, the neuropsychological evidence on implicit learning and related automatic predictive sequencing phenomena bearing on this hypothesis is reviewed.

Neuropsychological investigations of nonconscious predictive skill learning began with investigations of procedural learning, a functional equivalent of implicit learning that tends to be more motor oriented. Cohen and Squire (1980) found amnesics became faster at reading and recognizing mirror-reversed words over several repetitions, despite the continued inability to explicitly recognize the words. Cohen and Squire suggested that this preserved form of memory for procedures, or procedural memory, must be subserved by a different memory system than declarative memory for which amnesics are deeply impaired.

At the same time, HD and PD researchers were reporting sequencing difficulties as part of the cognitive profile of these
diseases (Caine et al., 1977; Caine et al., 1978). Martone and colleagues (Martone, Butters, Payne, Becker, & Sax, 1984) joined these ideas and found a double dissociation between amnesics and HD patients. Martone replicated the Cohen and Squire finding of spared mirror reading and impaired recognition in amnesics but found the performance of HD patients was reversed with normal recognition but impaired mirror reading. Since 1984, there has been a steady stream of neuropsychological studies of procedural learning, predictive sequencing and more recently implicit learning. Like characterizations of the functional domain of the basal ganglia, these studies span the motor–cognitive continuum.

At the lowest level of predictive motor sequencing, researchers have compared performance on the simple and choice reaction-time tasks. In choice reaction-time tasks, participants must make one of two responses as fast as possible once a cue is given. The cue to respond and the cue designating the correct response for the trial are given simultaneously. In simple reaction-time tasks, the cue for the correct response is given prior to the cue to respond, thus allowing a motor plan to be selected and ready when the go cue is given. Comparing performance in these two tasks indicates impairments in predictive sequencing or motor planning. A number of studies have found that PD patients show performance deficits on the simple reaction-time task relative to normals but are unimpaired on the choice reaction-time task (see Table 1; Bloxham et al., 1984; Evarts, Teravainen, & Calne, 1981; Goodrich, Henderson, & Kennard, 1989; Pullman, Watts, Juncos, Chase, & Sanes, 1988; Sheridan, Flowers, & Hurrell, 1987; but see Willingham et al., 1995).

In a more naturalistic study, patients with PD were slower to make a series of hand and arm gestures in sequence (Benecke, Rothwell, Dick, Day, & Marsden, 1987). In part, the speed impairment was due to general motor difficulties associated with making each gesture, but performed in sequence, performance times were even longer. Another lab, however, found that PD patients early in the disease course were unimpaired at sequencing hand gestures, whereas patients with frontal lobe damage were impaired at the same sequencing task (Canavan et al., 1989b). This result suggests that the basal ganglia may not be heavily involved in explicit or intentional learning of novel sequences, and Benecke’s finding may reflect the eventual spread of PD to the cortex.

Another set of investigations has explored skill learning in the motor domain using the pursuit rotor task. In this task, a device like a record turntable spins at a set speed while the participants attempt to keep a stylus in contact with a small patch of material attached to the turntable. A number of studies have found rotary

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<td>Choice vs. simple RT</td>
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<td>Bloxham et al. (1984)</td>
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Note. HD = Huntington’s disease; PD = Parkinson’s disease; RT = reaction time. (+) spared, (−) impaired.
A few studies have followed up on Martone's study of procedural learning in the realm of perception. In three studies, PD and HD patients demonstrated less adaptation to the visual displacement that results from looking through prisms (Canavan et al., 1990; Paulsen, Butters, Salmon, Heindel, & Swenson, 1993; Stern, Mayeux, Hermann, & Rosen, 1988). Interestingly, three studies attempting to replicate Martone et al. (1984) have failed to find a mirror reading or tracing deficit in HD or PD patients (Daum et al., 1995; Gabrieli, 1995; Harrington et al., 1990). Gabrieli has suggested that mirror reading performance may be more dependent on learning new perceptual–motor mappings than procedural learning.

Tower of London (TOL), a puzzle task, has been used in a number of studies to assess the cognitive end of procedural learning. In this puzzle a set of five discs are stacked by size on the first of three pegs, smallest disc on top. The object of the puzzle is to move all the discs to another peg such that they are again stacked in the same order from largest to smallest. There are two rules to the game. First, only the top disc from any peg can be moved at any time, and second, a disc can never be placed on top of a smaller disc. There is a sequential principle of movement that must be repeated several times to most efficiently solve the puzzle. Even as the algorithm is used increasingly with practice, most participants do not have explicit knowledge of the steps in the algorithm.

More than a half dozen studies have found speed and accuracy deficits in PD and HD patients' TOL performance (Butters et al., 1985; Daum et al., 1995; Lange et al., 1992; Lawrence et al., 1996; Morris et al., 1988; Owen et al., 1992; Owen et al., 1995; Saint-Cyr et al., 1988). In both PD and HD, it is not yet clear whether TOL performance is impaired early in the disease course. Lawrence et al. (1996) found HD patients early in the disease course showed deficits on TOL, but Saint-Cyr et al. (1988) found that only the most amnesic-like HD's were at impaired at TOL early in the disease, and Owen et al. (1992) found EPD patients to be unimpaired. Two studies, however, have found performance deficits in EPD, one of which altered the TOL by removing the need to physically move the discs so that the task was purely cognitive (Daum et al., 1995; Owen et al., 1995). It is also unclear whether amnestic patients are impaired at the task (Butters et al., 1985; Saint-Cyr et al., 1988). It is, however, clear that frontal lobe syndrome patients do have difficulty with the task (Owen et al., 1995).

A limitation of the TOL's capacity to illuminate the cognitive functions of the basal ganglia is that it is not a process pure task. Although automatic predictive sequencing that improves with practice might play a part in the task, it is just one part in a cast of characters. PFC must be involved in the future-oriented counterfactual thought processes (e.g., "what if I move . . . ?"). and the anterior cingulate is probably involved in detection of strategies gone awry. Implicit learning tasks have also been used in neuropsychological investigations and are better suited to test the cognitive functions of the basal ganglia. The implicit learning task used most frequently is the serial reaction time task (SRT), in which participants indicate as quickly as possible, on each trial, which of the four quadrants on a screen contains the target. In most studies using the SRT, the movement of the target from trial to trial is determined by an 8- to 10-trial sequence that repeats. If participants are gaining implicit facility with the sequence, they should become faster with practice, as they are better able to nonconsciously predict the location of subsequent targets. During the key block of test trials, the repeated sequence is disrupted so that the predictive structure of the sequence is altered. Comparing performance on test trials against the most recent practice trials gives a measure of implicit learning.

Studies of both HD and PD indicate that these participants are impaired on the SRT (Ferraro, Balota, & Connor, 1993; Jackson, Jackson, Harrison, Henderson, & Kennard, 1995; Knopman & Nissim, 1991; Willingham & Korschoetz, 1993). One of these studies used only early PD patients and found the same impairment (Jackson et al., 1995). In contrast, amnesics are unimpaired on the SRT (Knopman & Nissim, 1987; Nissim & Bullemer, 1987). Dominey and colleagues looked at SRT performance when the sequence was explicitly accessible and found that PD patients were not impaired relative to normals (Dominey, Ventre-Dominey, Brousseloe, & Jeannerod, 1996). This collection of studies provides clear evidence for the localization of a higher cognitive function, nonconscious predictive sequencing, within the basal ganglia.

Although the SRT appears to require a predominantly cognitive form of implicit learning, it also relies on the speed of motor output (e.g., pressing one of four buttons). To remedy this potential concern, Knowlton and her colleagues have used implicit tasks that are entirely nonmotor in nature: a probabilistic-classification task and an artificial-grammar task (Knowton & Squire, 1996; Knowton et al., 1996). In the probabilistic-classification task, participants learn to predict the weather. Each of four predictor cues is either present or absent, and each cue is probabilistically related to the outcome judgment that participants make.

Knowlton and colleagues found PD and HD patients were impaired on the probabilistic-classification task, whereas amnesics and frontal patients demonstrated as much implicit learning as normals. These compelling data suggest that the basal ganglia have a unique role in automatic predictive sequencing of cognitive as well as motor phenomena. Somewhat surprisingly, Knowlton found HD patients performed normally on the artificial-grammar task. She concluded that artificial-grammar task performance largely depends on perceptual fluency with the bigrams and trigrams from the practice set (Knowlton & Squire, 1996). HDs show intact perceptual priming and should perform normally on a fluency-based task.

The neuropsychological data are relatively consistent, supporting the conclusion that the basal ganglia are centrally involved in nonconscious predictive sequencing in both the motor and cognitive domains. Although the neuroanatomical connectivity of the striatum suggests that putamen should be more involved with the former and caudate with the latter, this suggestion is not clear from the HD and PD data given that both groups show the same pattern of impairment of predictive sequencing. This result may be partly a result of the relative paucity of studies in this domain, especially toward the cognitive end of the continuum. It is probably also the case that there are inherent limitations in our ability to use neuropsychological methods to differentiate caudate and putamen contributions to predictive sequencing.
Neuroimaging Evidence

The evidence from functional imaging studies is clear in assigning a role to the basal ganglia in predictive sequencing. Unfortunately, most of the existing studies have focused on the motor domain. More than half a dozen studies have found significant activation of the basal ganglia during the learning and/or execution of well-learned procedural skills, including the pursuit rotor task (Grafton et al., 1992; Grafton, Woods, & Tyszka, 1994), sequencing of finger movements or keypresses (Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Schlag, Knorr, & Seitz, 1994), SRT (Berns, Cohen, & Mintun, 1997; Grafton, Hazeltine, & Ivry, 1995; Hazeltine, Grafton, & Ivry, 1997; Rauch et al., 1995; Rauch et al., 1997), TOL (Baker et al., 1996), and learning to manipulate a robot arm in a complex virtual environment (Krebs et al., 1998). In general, the tasks that emphasize motor performance result in contralateral putamen activation along with the cortical components of the motor circuit, the primary motor cortex, and supplementary motor area. In two of the studies, increased striatal activation was not present during the initial learning but only after the procedural skill became overlearned (Grafton et al., 1992; Grafton et al., 1994).

Learning in the SRT shows a neuroanatomical shift during the course of learning from caudate and ventral striatum (Berns et al., 1997; Rauch et al., 1995; Rauch et al., 1997) to putamen (Grafton et al., 1995; Hazeltine et al., 1997). Consistent with the neuropsychological evidence (Dominey et al., 1996), it appears the basal ganglia may be uninolved or less involved in explicit sequence learning (Rauch et al., 1995). In an fMRI study of the probabilistic-classification task, Foldrack, Prabakaran, Seger, and Gabrieli (in press) found caudate activation coupled with medial temporal lobe deactivations. The findings of Rauch et al. (1995) and of Foldrack et al. (in press) are consistent with animal lesion studies showing that tasks for which learning is impaired by basal ganglia lesions are actually learned more quickly after hippocampal lesions (McDonald & White, 1993; Packard, Hirsh, & White, 1989). This raises the intriguing prospect that the process of explicit sequence learning interferes with the implicit learning mechanism in the basal ganglia. Given that explicitly learned sequences are only applied with the explicit intention to do so, the lack of parallel learning in the basal ganglia could leave individuals unprepared to spontaneously recognize stimuli predictive of reward when there is no explicit goal to do so.

Additionally, the neuroimaging findings are consistent with the hypothesis that motor and judgment forms of implicit learning are dependent on separate components of the striatum. The purely motor learning tasks indicate putamen, but not caudate, activations. The SRT, which is both motor and cognitive, tends to show both caudate and putamen activations, though at different points in the learning process. Finally, in the probabilistic-classification task, which involves cognitive judgment but not motor learning component, Foldrack et al. (in press) found caudate, but not putamen, activation.

Neurophysiology

Recognizing behaviorally relevant stimuli. Although there are several studies implicating the basal ganglia in procedural or implicit learning, the neurophysiology and neurochemistry of the basal ganglia are the essential conceptual components that allow us to understand why the basal ganglia should function the way that it does. First, the dense connectivity from nearly the entire cortex suggests a pattern recognition function. Each spiny neuron in the striatum receives over 10,000 inputs, which is second in connectivity density only to Purkinje cells in the cerebellum, and its cellular architecture, with this high resolution input, resembles that used in connectionist network models of pattern recognition (Houk, Adams, & Barto, 1995). Additionally, all the elements exist local to the striatum necessary for long-term potentiation, which enables a cell assembly to encode a pattern into changes in the synaptic efficiency of corticostriatal afferents (Wickens & Kotter, 1995). This change in synaptic efficiency is what the change in connection strengths in connectionist architectures is intended to model (Bechtel & Abrahamsen, 1991). The three elements necessary for long-term potentiation are presynaptic activity, postsynaptic activity, and neurochemical reinforcement. The convergence of the cortical inputs on the striatum involves the presynaptic release of the neurotransmitter glutamate. Additionally, neurochemical reinforcement arrives in the form of DA release from SNC, which projects to the striatum. DA release is, at least in early stages of striatal pattern learning, due to limbic and hypothalamic inputs to SNC, which innervate SNC in response to primary rewards and valenced events (Schultz et al., 1995). Thus, the timed release of DA with enormous convergence on each SP neuron allows the striatum to code for complex, but subtle, patterns or contexts associated with reward.

DA is thought to have a modulatory or gating role in helping the striatal neurons distinguish useful patterns from less useful ones. DA functions by increasing the activation of striatal neurons that are already active and suppressing the activation of less active striatal neurons (Robbins & Brown, 1990; Taylor & Saint-Cyr, 1995; Wickens & Kotter, 1995). In effect, this creates greater contrast between the cells that were already firing in conjunction with the behaviorally significant event and those that were not.

From this evidence, it appears that the pattern detected is the pattern of the rewarding or valenced event itself, rather than the predictor of the event. The DA neurons from SNCs train the striatum and, consequently, groups of striatal neurons have the capacity to duplicate limbic and hypothalamic output. Schultz et al. (1995) found that once an experimental task has been mastered, however, multiple populations of striatal neurons in primates respond selectively to different steps in the sequence leading up to a reward and that striatal neurons stop firing at the time of expected reward. This training process yields a much more exciting view of pattern recognition in the striatum, as it appears that through extensive exposure the basal ganglia shifts from responding to behaviorally significant events themselves to instead responding to the predictors of behaviorally significant events.

This predictive ability allows for important adaptations in behavior especially because it can be utilized automatically and outside of awareness (Ashby et al., 1998; Grafton et al., 1995; Marsden, 1982). Processes varying from from walking and typing to speaking and recalling a name involve complex movements, either in space or in cognition, that require significant prediction-based coordination to be accomplished accurately. These are mundane examples that differ little, qualitatively, from events that we would more willingly call intuitive thinking or intuitive behavior. It does not seem unreasonable to suggest that the same process
described here could be extended to explain an experienced farmer's, not to mention a cow's, accurate intuition that the weather is about to change.

Will the real predictor please stand up? The neural events involved in the process of learning to predict are only partially understood. Houk et al. (1995) offered a plausible and compelling, if tentative, model of how the different populations of striatal neurons might come to predict reward and even predict the predictors of reward. The problem to be resolved here is how the striatum can learn that a predictor event (P) predicts reward (R) when the DA release occurs in temporal conjunction with R not P. How is a system without a homuncular agent able to go backward in time and decide which events previously processed should receive the benefit of the current DA reinforcement, especially when P was not consciously attended to? An ideal, but implausible, solution would be for the striatal correlates of P, but only P, to remain active while waiting for reinforcement from the eventual DA release. This solution begs the question of how the system would ever know which events are plausible candidates to be P. The correlates of the external events in the basal ganglia will show some residual activation after the event has passed, but most neural events take place on a time scale far too small (e.g., 100 ms) to allow the breadth of explanatory power we are looking for (Newell, 1990). We want to be able to explain the learning of a P that occurs more than 100 ms before R. The extremely high concentration of calcium-calmodulin-dependent kinase II (CaM PK II; Newman-Gage & Graybiel, 1988) present in the striatum provides a plausible solution. CaM PK II, which potentiates glutamate receptors receiving cortical inputs and thus is central to long-term potentiation, has an extremely slow autophosphorylation rate extending its activation up to hundreds of seconds (Meyer, Hanson, Stryer, & Schulman, 1992).

To illustrate how this extended activation would account for P learning, consider an example. Imagine that a person is listening to a seemingly random sequence of tones but that exactly 3 s after a tone of 440 Hz is played (event P), a reward (R) will always be presented that can be obtained if responded to quickly. Learning the P → R relation would allow for faster reaction times. Each time R is obtained, DA will be released into the striatum, thus strengthening the striatal representation of R. Because of the long activation of CaM PK II, the neural correlates of P will still be active and able to enhance its synaptic efficiency via long-term potentiation. The problem immediately arises that P is not the only neurally represented event active in the striatum to receive the benefit of CaM PK II. Many other events (E₁, E₂, etc.) that occurred in the seconds prior to the reward will also be strengthened. Here it becomes apparent why the basal ganglia, unlike the hippocampus, cannot engage in single trial learning. Although many potential predictor events (P, E₁, E₂, etc.) will be reinforced on any given trial, only the valid predictor P will receive repeated reinforcement over several trials. Moreover, DA functions to depress the activation of events that are not currently occurring and will consequently weaken accidental Eᵢ → R associations. That is, DA will function to weaken the activation of Eᵢ on the second trial if Eᵢ is not still active during the second instance of DA release.

Essentially, the extended autophosphorylation increases the length of time that all current activity transmitted from the cortex remains active in the basal ganglia. If R occurs, limbic or hypothalamic sites will innervate the basal ganglia with DA leading to the reinforcement of striatal representations of the rewarding event, the predicting event and all other events that inadvertently occur in the seconds prior to the rewarding event. Only valid Ps will reliably precede R and thus should form stronger representations than the other extraneous cues that are not real Ps. Furthermore, DA functions to extinguish the representations of unreliable Ps.

Learning to predict the predictors. The striatum is reciprocally connected to SNc, and thus each site is capable of activating the other. The striatum projects to SNc directly and also by an indirect route via the subthalamic nuclei. The indirect route is the faster of the two and is excitatory in nature, whereas the slower direct route is inhibitory. Houk et al. (1995) have pointed out that this configuration allows the striatum to learn progressively earlier and earlier units in a predictive sequence. As just described, initially SNc releases DA during R, and this process occurs in conjunction with the continued activation of the neural correlates of P, allowing P to be learned slowly over time. Once P is learned, the presence of P will lead the striatum to signal cognitive and motor areas of an impending event of significance, R, but it will also send a feedback signal to SNc along the two routes. The slower direct inhibitory route counteracts the excitatory limbic input to SNc, which always results from the presence of the R, thus preventing SNc DA release in conjunction with R. In other words, once P is learned, the striatum signals the SNc to no longer transmit the limbic activation associated with R to the striatum. This fits with Schultz and Romo's (1992) finding that over time, the striatum ceases to respond in conjunction with R. Additionally, the indirect but faster excitatory connection to SNc initiates earlier DA release in temporal conjunction with the predictor event itself. Once P is learned, the striatum sends a signal to SNc in temporal conjunction with P that mimics the normal R-based limbic input to SNc. Because of this excitatory input, SNc responds to P as if it were an R consequently releasing DA into the striatum. Once the DA release is synchronized with the P, rather than R, the same process that allowed for learning the value of P can be used to train the striatum to learn events that predict P.

In summary, the interconnections between the limbic system, the substantia nigra, and the striatum in conjunction with the long lasting activation of CaM PK II in the striatum suggest a neurally plausible resolution to the problem of assigning predictive value in the brain to an event that has already passed and was never consciously attended to. The second function of basal ganglia functioning, notifying cognitive and motor areas of these predictive events is far more straightforward given the known circuitry (Alexander et al., 1986) linking the basal ganglia to the cognitive and motor areas of the cortex, by way of the thalamus. The combination of being able to learn complex and potentially long and subtle sequences of predictive patterns along with the ability to notify and modify frontal cortical activity in both cognitive and motor domains makes the basal ganglia a rather attractive prospect for being a large component of the neuroanatomical substrate of implicit learning and procedural knowledge, which has been argued is the cognitive substrate of intuition and of intuitive social action and cognition. This is made all the more plausible when striatal processes are thought to be nonconscious or at least very low on the continuum of conscious accessibility (Brothie, Iaione, & Horne, 1991; Marsden, 1982; O'Reilly et al., 1997).
Neural Basis of Intuition: The Basal Ganglia

The preceding sections call for our recognition of the unique and vital link between implicit learning and the nonconscious predictive sequencing performed by the basal ganglia. It is the purpose of this article to extend this link to intuition as well. In this section, a variety of social, emotional, and linguistic findings that more directly link the basal ganglia to intuition are reviewed. If intuition and implicit learning are both largely dependent on the basal ganglia, then this would constitute strong evidence that intuition and implicit learning are related overlapping processes.

Nonverbal Communication

Both in terms of comprehension and production, nonverbal communication is a central domain of intuitive social action and cognition. Think of the temporal and spatial sequences of cues that compose facial, vocal, and gestural cues as probabilisticaly associated with internal states of emotion and attitude. The dance of nonverbal communication between two individuals often goes unnoticed by either participant (Word et al., 1974). It is, however, noticeable when it is absent or out of sync (Bernieri & Rosenthal, 1991). The dance occurs intuitively, and when we get a sense of the other's state of mind as a result of the nonverbal cues the other has emitted, we often have nothing other than our intuition to justify our inferences.

There are a number of studies suggesting a role for the basal ganglia in both production and comprehension of nonverbal communication (Cancellier & Kertesz, 1990; Cohen, Riccio, & Flannery, 1994; Hornak, Rolls, & Wade, 1996; Morris et al., 1996; Philips et al., 1997; Ross, 1981; Ross & Mesulam, 1979; Van Lancker & Pachana, 1995; Wedell, 1994). Seven of these studies examined the nonverbal skills of patients with basal ganglia lesions, along with minimal or no cortical damage in most cases (see Table 2). All seven found impairments in the production of emotional prosody, the nonlinguistic vocal cues or melody line of speech (Monad-Krohn, 1947), and/or the production of spontaneous facial expression. Of the four studies also measuring nonverbal comprehension abilities, three of the four studies found comprehension deficits. Two neuroimaging studies also implicated the basal ganglia in the decoding of nonverbal expressions. A PET study found greater activation in the basal ganglia when participants were viewing happy as compared with fearful facial expressions (Morris et al., 1996). Additionally, the participant's task was gender discrimination, not expression discrimination, so presumably this activation reflects unintentional or automatic nonverbal decoding. Morris, Ohman, and Dolan (1999) found basal ganglia activation even when the faces were presented such that participants did not even consciously detect the presence of the face stimuli. Additionally, an fMRI study found more basal ganglia activation when participants were viewing faces of disgust compared with neutral faces (Philips et al., 1997).

It has been suggested in this review that the components of the striatum, the caudate, and putamen might serve different roles in intuitive social action and cognition given the neural circuits of which they are each a part. Accordingly, the caudate should have a greater role in the decoding of nonverbal cues, whereas the putamen should be invoked in the production or encoding of nonverbal cues. None of the above lesion studies report differential damage to caudate and putamen and consequently cannot be used to assess this hypothesis. The different courses of neuronal loss in HD and PD do allow us to examine the separate contributions of the caudate and putamen (see Figures 4a and 4b), though perhaps not with the greatest of precision.

Table 2

<table>
<thead>
<tr>
<th>Affected area</th>
<th>Study</th>
<th>NV comprehension</th>
<th>NV production</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undifferentiated lesion in basal ganglia</td>
<td>Cohen et al. (1994)</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Cancellier &amp; Kertesz (1990)</td>
<td>-</td>
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<td></td>
<td>Hornak et al. (1996)</td>
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<td></td>
<td>Wedell (1994)</td>
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<td></td>
<td>Van Lancker &amp; Pachana (1995)</td>
<td>-</td>
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<td></td>
<td>Ross &amp; Mesulam (1979)</td>
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<td>-</td>
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<tr>
<td></td>
<td>Ross (1981)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HD</td>
<td>Speedie et al. (1990)</td>
<td>-</td>
<td>-</td>
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<tr>
<td></td>
<td>Sprengelmayer et al. (1996)</td>
<td>-</td>
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<tr>
<td></td>
<td>Gray et al. (1997)</td>
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<tr>
<td></td>
<td>Jacobs et al. (1996)</td>
<td>-</td>
<td>-</td>
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<tr>
<td>PD</td>
<td>Benke et al. (1998)</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Blonder et al. (1989)</td>
<td>+</td>
<td>-</td>
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<tr>
<td></td>
<td>Caektekebeke et al. (1991)</td>
<td>+</td>
<td>-</td>
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<tr>
<td></td>
<td>Scott et al. (1984)</td>
<td>+</td>
<td>-</td>
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<tr>
<td></td>
<td>Buck &amp; Duffy (1980)</td>
<td>-</td>
<td>-</td>
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<tr>
<td></td>
<td>Pitta et al. (1990)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Pell (1996)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lesion to putamen (more than caudate)</td>
<td>Speedie et al. (1993)</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. (+) spared, (-) impaired. HD = Huntington's disease; PD = Parkinson's disease; NV = nonverbal.
All six studies examining nonverbal cue production in PD, which initially affects putamen more than caudate, have found impairments in the production of vocal prosody and facial expressions (Benke, Busch, & Andreu, 1998; Blonder, Gur, & Gur, 1989; Buck & Duffy, 1989; Caekebeke et al., 1991; Pell, 1996; Piteira, Clemie, Gray, & Pentland, 1990; Scott, Caird, & Williams, 1984). In contrast, four of the five studies of PD comprehension of nonverbal cues found no impairment relative to non-PD patients (Benke et al., 1998; Blonder et al., 1989; Caekebeke et al., 1991; Pell, 1997; Scott et al., 1984). A lesion patient with more putamen than caudate damage also showed this pattern of impaired production but spared comprehension (Speedie, Wertman, Ta’ir, & Heilman, 1993). Pell was the only investigator to find impaired comprehension in PD patients, but disease duration was longer in Pell’s study (avg. 8.5 years) than the others, indicating the possibility of more severe caudate damage.

Only four studies have examined nonverbal decoding in HD, in which the caudate is selectively targeted early in the disease. In contrast with the PD studies, all four studies of HD patients found them to be impaired in the comprehension of nonverbal cues (Gray, Young, Barker, Curtis, & Gibson, 1997; Jacobs, Shuren, & Heilman, 1996; Speedie, Brake, Folstein, Bowers, & Heilman, 1990; Sprengelmayer et al., 1996). Speedie found HD patients early in the disease course to be impaired, and Gray et al. (1997) found HD gene carriers who were presymptomatic were also impaired. Sprengelmayer carried out the most comprehensive analysis examining nonverbal decoding ability separately for each of the six basic emotional expressions (Ekman & Friesen, 1971). Using Ekman and Friesen’s face pictures, HDs were impaired at recognizing surprise, fear, sadness, disgust, and anger, but not happiness. With vocal cues, IIDs were impaired at recognizing all the basic emotions except sadness.

Although all the impairments were sizable, the deficit in recognizing disgust was extreme almost to the point of having no capacity to recognize the related emotion cues at all. In the facial-expression test, for instance, non-HD patients accurately identified 8.1 of 10 disgust faces, whereas the HD patients accurately identified 1.9 of 10.

Unfortunately, there have been no tests of HD nonverbal production, so the fourth cell of the hypothetical nonverbal-mode-versus-disease matrix is currently empty. Nevertheless, there is strong evidence that the caudate is selectively involved in nonverbal decoding, whereas the putamen is not. Instead, the evidence suggests that the putamen is involved in the production of nonverbal cues.

This is a key set of findings for linking the social, cognitive, and neural levels of analysis considered in this article. First, it establishes the direct relationship between the basal ganglia and social intuition. Second, both nonverbal communication and implicit learning depend on intact basal ganglia. Additionally, the dichotomizing of caudate and putamen function into nonverbal comprehension and production fits parsimoniously with Seger’s (1997) suggestion that there are two domains of implicit learning: judgment and motor output (see Figure 5).

Nonverbal communication research has been less influential in social psychological research, as compared with its actual role in social psychological processes, in part because it has been largely atheoretical without clear cognitive or neural bases (but see Patterson, 1995). A clear understanding of these underpinnings should contribute to the integration of nonverbal research into both social cognition and cognitive psychology. On the other side of the coin, implicit learning tends to be a finding without a phenomenon. There is hesitation in the literature to say just what implicit learning does for us in everyday life. It is consequently an important step to demonstrate that implicit learning provides much to the cognitive underpinnings of nonverbal communication.

**Experience of Emotion**

Intuitive thinking often has been linked to emotional thinking and all the baggage that comes with emotional thinking (e.g., irrationality). This has changed of late as emotion-as-information theories are gaining popularity in models of decision making (Damasio, 1994; Schwarz & Clore, 1988). A number of recent neuroimaging studies show activation in the basal ganglia of participants exposed to emotional stimuli.

Elliot and Dolan (1998) found caudate activation while participants made preference judgments, but not during memory judgments, in a mere exposure paradigm (Zajonc, 1968). In this paradigm, participants show increased liking for stimuli that they were previously exposed to subliminally. LaBar, Gatenby, Gore, LeDoux, and Phelps (1998) found striatal activations in both fear conditioning and extinction.

Four studies (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998; Lane, Reiman, Ahem, et al., 1997; Lane, Reiman, Bradley, et al., 1997; Philips et al., 1997) have found caudate activation during the presentation of positive, but not negative, emotional stimuli. These results fit nicely with Morris et al.’s (1996) finding that automatic nonverbal decoding of positive facial expressions activated the basal ganglia, but not the amygdala, whereas the reverse activations obtain for negative facial expressions (see also Anderson & Phelps, 1998). Additionally, Hamann, Ely, Grafton, and Kilts (1999) found ventral striatum to be the single best correlate of the memory advantage for positive stimuli over neutral stimuli. Ventral striatum activity was not, however, associated with the memory advantage for negative stimuli over neutral stimuli. Beauregard et al. (1997) found caudate activation during the presentation of emotion words but did not report differences across word valence.

London et al. (1990) scanned participants while experiencing the euphoria of a morphine injection and found activation in both

**Intuitive Social Cognition**

![Intuitive Social Cognition Diagram](https://via.placeholder.com/150)

**Intuitive Social Action**

![Intuitive Social Action Diagram](https://via.placeholder.com/150)

*Figure 5. Intuitive cognition and intuitive action at the social, cognitive, and neural levels.*
caudate and putamen. Breiter et al. (1997) examined the initial rush and later craving associated with cocaine infusion into cocaine-dependent individuals. Of the 10 participants, 8 showed significant activation in the caudate associated with the rush, and 9 showed significant activation in ventral striatum associated with the craving. The results from Breiter et al. suggest the human basal ganglia are involved in both reward registration and motivation to seek future reward.

In one of Lane’s studies (Lane, Reiman, Ahem, et al., 1997) participants were also scanned while generating and focusing on their own positive and negative memories. Interestingly, during this internally generated emotional experience caudate and putamen were activated for sad but not happy emotions. This finding replicates George et al.’s (1995) finding of caudate activation in women thinking of sad but not happy memories.

Taken as a whole, these findings suggest a role for the basal ganglia in evaluating positive affective stimuli and in positive emotional experience. Many emotion researchers agree that the first stage of emotional appraisal is an automatic distinction drawn between positive and negative stimuli (Lazarus, 1991; Ochsner, in press; Ochsner & Schacter, in press). This process is generally attributed to the amygdala, but the vast majority of findings linking affective appraisal to the amygdala have focused on fear appraisals (LeDoux, 1996; Morris et al., 1999). Other studies have suggested that the involvement of the amygdala is not necessary for positive affective appraisal (Breiter et al., 1997; Holland & Gallagher, 1999; Morris et al., 1996; Tranel & Damasio, 1993). It might be the case that the basal ganglia and amygdala, being subcortical neighbors, perform the automatic appraisal of only positive or negative affective stimuli, respectively. Although the majority of the findings fit this pattern, the Lane, Reiham, Ahem, et al. (1997) and George et al. (1995) findings do stand apart, not so much for the basal ganglia activation found during a negative affective state but because of the absence of activation during a positive state. Without explaining away these anomalies, it seems relevant to note that these are the only two studies that involve reprocessing of stimuli that were previously encoded as positive or negative.

Consistent with the hypothesis that the basal ganglia plays an important role in positive emotional appraisal and experience, most neuropsychological conditions that affect the basal ganglia are associated with nonreactive forms of depression. Approximately 40% of PD patients also show depressive symptoms (McPherson & Cummings, 1996). Depression has also been found in a large minority of patients with HD (Hopkins, 1994). Two other neuropsychological disorders involving basal ganglia dysfunction, Tourette’s syndrome and obsessive–compulsive disorder, are also associated with depression (den Boer, 1997; Robertson & Baron-Cohen, 1996). Although depression frequently accompanies brain trauma, regardless of location, a number of studies of bipolar and unipolar depression have found reduced metabolism in the basal ganglia (Baxter et al., 1985; Buschbaum, 1986; Buschbaum, Wu, Delisi, & Holcomb, 1986; Mayberg, Lewis, Regenold, & Wagner, 1994). Additionally, in a study of depression resulting from stroke-related lesions (Morris, Robinson, Raphael, & Hopwood, 1996), 75% of patients with lesions to the left basal ganglia or left PFC were depressed, whereas only 21% of patients with lesions anywhere else in the brain were depressed. It seems reasonable to conclude that the basal ganglia damage might contribute to depression by impairing the ability to identify and appraise positive stimuli.

**Linguistic Intuition**

Speedie et al. (1993) reported a fascinating case of a 75-year-old Jewish man with basal ganglia damage who could no longer use automatic speech composed of the linguistic sequences that are “ready made, more holistically, without effortful formulation and are composed of invariant word sequences” (Code, 1989, p. 155). His use of nonautomatic speech was unimpaired. Automatic speech includes greetings, cliches, swear words, songs, and prayers among its kin. Speedie’s patient was unable to produce any of these spontaneously. Perhaps most surprising was his inability to recite a Jewish prayer that he had recited before each and every meal since the age of 5. Speedie et al. (1993) concludes,

> Like the consecutive movements of tying one’s shoes, each triggering the next step, every phrase of a blessing is the trigger for the next phrase. Both the manual task and the blessings are performed without conscious planning of the component segments of the act. They are used without overt awareness. Reciting overlearned texts may be another example of procedural knowledge. (p. 1772)

Speedie’s equating of automatic speech, procedural knowledge, and basal ganglia damage is a rare example of the three levels being considered jointly. It suggests that the basal ganglia may have a role in linguistic automatic in general. A few others have followed up this lead by investigating bilinguals for whom the native tongue (L1) was learned before the second language (L2). Agliotti, Beltramello, Girardi, and Fabbro (1996) reported a case of a bilingual patient, EM, with selective damage to the left basal ganglia. EM was raised speaking Venetian (L1) and learned standard Italian (L2) later on in school. After the basal ganglia lesion, EM lost most of her ability to speak L1. She could still comprehend L1, but tended to respond in L2 and had severe deficits in producing L1 when forced. When asked to translate from L2 to L1 she often repeated the same words in L2. One interpretation of these data is that the basal ganglia aids in the automatic retrieval of words from the lexicon and L2 words are not well rehearsed enough to rely on automatic retrieval. The L1 lexicon still exists and can be accessed as demonstrated by the ability to respond meaningfully in L2 when spoken to in L1. Intact linguistic comprehension in L1 might be akin to cued recall in which the search strategy is more externally driven than in free recall, which might be more analogous to linguistic production.

Two PET studies by Klein and colleagues (Klein, Milner, Zatorre, Meyer, & Evans, 1995; Klein, Zatorre, Milner, Meyer, & Evans, 1994) found the only activation differences across L1 and L2 in bilinguals occurred in the left putamen during the production of L2, either in the form of translating L1 into L2 or in the repetition of a word in L2. Translation into L1 did not activate putamen, however. It seems plausible that this activation would reflect the ongoing process of learning and striatal automation of L2. The results of these studies along with EM’s deficits are mirror images. In both cases, production of either L1 or L2 seems to depend on the integrity of the basal ganglia, but at the same time the data conflict with each other regarding the influence of the basal ganglia on languages automatized to differing degrees. Perhaps the putamen eventually operates so efficiently that blood flow...
changes are undetectable by PET. With lower efficiency in L2 putaminal representations, activations would be present.

One recent study examined the extent to which production differences within a single language are correlated with basal ganglia function (Ullman et al., 1997). Ullman et al. reasoned that the use of regular and irregular verbs rely on procedural and declarative knowledge structures, respectively. The generation of the past tense of regular verbs follows a simple rule, add -ed. Neologisms are always regular and follow the same rule (e.g., email--emailed). There is also a finite set of irregular verbs that have idiosyncratic tense changes (e.g., run--ran). It follows then that regular verbs might be inflected based on a rule stored procedurally in the basal ganglia, whereas inflecting irregular verbs might depend on a list of examples stored in declarative knowledge structures.

Participants in their study were required to inflect regular (e.g., "Today I cook food. Yesterday I _____ food."), irregular (e.g., "Today I run a mile. Yesterday I _____ a mile."), and novel verbs (e.g., "Today I garp my favorite story. Yesterday I _____ my favorite story."). PD patients had difficulty inflecting the regular and novel verbs that follow a rule, but no deficit in inflecting the irregular verbs. Alzheimer’s patients, having declarative rather than procedural memory deficits, showed the opposite pattern in which the rule-based inflection of regular and novel verbs was intact, but irregular verb inflection was impaired.

**Women’s Intuition**

A review on intuition would be incomplete without reference to women's intuition, the colloquial notion that women have a sixth sense or a more able intuition faculty than men. Like intuition itself, women's intuition is often shrugged off as an urban myth. No strong antedote is offered here, but there are some interesting leads that fit within the scope of this article. There is strong and consistent evidence that women are better encoders and decoders of nonverbal communication (Hall, 1984), and this evidence has frequently been cited as possible evidence of women's intuition (Graham & Ickes, 1997). Additionally, the hormone estrogen, present in greater quantities in women than men, directly affects the amount of DA released into the striatum (Becker, 1999; McDermott, Liu, & Dluzen, 1994; Mermelstein & Becker, 1995; Van Hartesveldt & Joyce, 1986). Greater DA release into the striatal in conjunction with reward should lead to the development of stronger representations of of P → R relationships that form more quickly, thus resulting in women's intuition. Along these lines, Jennings, Janowsky, and Orwoll (1998) found that estrogen levels in women correlated with performance speed on a sequential learning task.

**Summary**

There are definite empirical links between striatal function and intuitive phenomena including nonverbal communication skills, emotional experience, and language use. Although these phenomena do not cover the whole range of intuition or even social intuition, the results regarding this subset of intuitive phenomena are consistent in indicating a striatal basis to their functioning.

Of particular interest is the finding that nonverbal comprehension is impaired in HD, but not PD, suggesting caudate involvement. Alternatively, PD is associated with nonverbal production deficits suggesting putamen involvement. This distinction has been demonstrated separately at each of the social, cognitive and neural levels of analysis. When the social and neural levels are examined simultaneously, by testing the nonverbal skills of HD and PD patients, the distinction is present again. Parsimony strongly points toward the equating these different phenomena.

**Discussion**

**General Summary**

The claim of this review is that intuition is a phenomenological and behavioral correlate of implicit learning, which has been established both through an inspection of the similarities between the two phenomena as well as through a review of neuropsychological, neuroimaging, neurophysiological, and neuroanatomical data suggesting that both intuition and implicit learning rely critically on the integrity of the basal ganglia. There is substantial evidence that there is a major division in social, cognitive and neural components of intuition between judgment and action. Additionally, there is mounting evidence that the basal ganglia are not involved in explicit or intentional sequence learning and that explicit attempts to learn the relevant sequence can interfere with striatal learning. Finally, it appears that (a) different components of the basal ganglia perform the same kinds of computations on different sorts of information (affective, cognitive, motor) and (b) the basal ganglia and the cortex are linked together in circuits such that they process the same information, in any given circuit, but contribute different functions to the informational product.

**Intuition Versus Loss of Explicit Knowledge**

Implicit learning suggests a mechanism, whereby sequential associations can be learned without the learner ever being aware of the learning process or its ultimate product. There are other cases however, when the representations formed by the basal ganglia are the result of intentional efforts to learn some set of associations. In these cases, the associations were initially processed effortfully, presumably by PFC. Over many repetitions, the explicit knowledge is thought to recede from conscious access while the associations become compiled into more efficient representations in the basal ganglia (Anderson, 1982). One unresolved dilemma with this account is the finding that explicit sequence learning has been found to preempt the formation of an implicit representation in the basal ganglia (Rauch et al., 1995).

**Striatal Contributions to Automatic Social Inference**

This review has provided an empirical case for the role of the basal ganglia in nonverbal communication and potentially emotional experience, but the computations performed by the basal ganglia suggest a broader role in intuitive social cognition. Three processing features of the basal ganglia suggest the scope of its influence within the domain of social cognition. Representations formed in the basal ganglia (a) reflect temporal, not conceptual or configurural associations, (b) predict rewards, and (c) form incrementally over trials without conscious awareness to the temporal associations learned and can function automatically. These features suggest the basal ganglia could play specific roles in script-
based inferences and behavior, distributed decision making, and automatic evaluations.

Scripts. Many of our basic sequences of social behavior and our inferences about social behavior of others is facilitated by the use of scripts (Read, 1987; Schank & Abelson, 1977). As the name suggests, scripts are narratives that indicate the culturally agreed on order of events in social episodes. The application of a script allows us to infer elements of behavior and cognition that were not explicitly seen or described but are ordinarily present in the scripted sequence of events. PD patients have been shown to have script-deficits with simple real-world tasks such as going out for ice cream (Zaila et al., 1998), and there is little reason to expect patients with impaired striatal functioning to perform any better with scripts that are more social in nature. The results of the current review would suggest that PD patients might be differentially impaired at following social scripts, whereas HD patients should be differentially impaired at making inferences about the behavior of others on the basis of social scripts.

Decision making. Although it is likely that many forms of behavioral decision making will function independent of the basal ganglia, at least one type of decision making appears to rely on the computational advantages supplied by the basal ganglia. In distributed choice decision making (Heinstein & Prelec, 1991), (a) decisions that are made repeatedly over time (e.g., who should cater each lunchtime colloquium event) and (b) each decision not only affects the current utility obtained, but affects the utility of future choice options (e.g., choosing mexican food repeatedly makes it less appealing in future weeks). Conscious processing seems unlikely to successfully estimate the change in utility as a function of prior choice. This limitation combined with the repetitive and temporal nature of distributed choice problems suggests that the basal ganglia may be well suited to aid in the necessary computations.

Automatic evaluations. Recent evidence suggests that in addition to automatic social categorization (Uleman, Newman, & Moskowitz, 1996) and automatic characterization (Gilbert, Pelham, & Krull, 1988) of social entities, a process of automatic evaluation also occurs in the presence of an attitude object (Bargh et al., 1995). Smith and DeCoster (1998) have suggested that this evaluation may be an element in a network of conceptual associations that have been developed slowly by way of an autoassociative computational process in the same way that other implicit semantic associations form.

Certainly it is the case that we have evaluations for phenomena that are represented by conceptual representations, but it is an open question as to whether the affective evaluation itself is actually part of this representation. For some time now, memory researchers have indicated that multiple distinct representations compose our knowledge of any object in the world. For instance, our knowledge of our computer keyboard is simultaneously represented in episodic memory, semantic memory, procedural memory, and implicit perceptual memory (McClelland et al., 1995). It may be that in at least some cases, the automatic evaluation of an object is represented separately and some evidence suggests that these evaluations may be constructed in the basal ganglia and the amygdala.

Bargh, Raymond, and Chaiken (as cited in Bargh, 1997) recently tested Osgood’s (1953; Osgood, Suci, & Tannenbaum, 1957) hypothesis that the evaluative component of an object’s representation is qualitatively distinct from other semantic components. They found that priming could be used to automatically activate the evaluative component but not the other semantic components. This suggests two possibilities. First, the evaluative component may be part of the same network of associations that represent other semantic associations of a given attitude object, but tagged so as to be more accessible. A second possibility is that the representation of affective evaluation for any given object is stored separately from the conceptual representation.

One point in favor of the latter position is that classical conditioning of associations between an primary affective stimuli and neutral cues only occurs if the neutral cue is presented earlier than the unconditioned stimulus (McAllister, 1953). Smith and DeCoster’s (1998) autoassociative model is more amenable to learning associations between conceptual features that occur at the same time. Additionally, there is an automatic connection between positive and negative evaluations and approach versus avoidance behavioral responses (Chen & Bargh, 1999; James, 1890; Solarz, 1960), which is not an obvious consequence of conceptual associative representations but does fit well with the finding that the basal ganglia is necessary for learning stimulus-response contingencies (McDonald & White, 1993). Furthermore, neither the amygdala or hippocampus are necessary for stimulus–response associations to form (McDonald & White, 1993).

Social Cognitive Neuroscience Approach

The approach used in this review is a social cognitive neuroscience approach as it requires the insights from each of the social, cognitive, and neuroscience levels of analysis to reach the stated conclusions. Each of these contributing areas asks different questions and uses different techniques to address their domain of inquiry (Klein, Loftus, & Kihlstrom, 1996; Ochsner & Kosslyn, in press). Each of these three areas would benefit from an understanding of and ultimately an integration with the other areas.

The goal of this review was to provide evidence that certain forms social intuition, those most likely to yield accuracy, rely in large part, on the cognitive process of implicit learning. It is impossible to directly associate social intuition with implicit learning, and thus it is necessary to examine the neuroanatomical bases of each. Listing the similarities between the two is mere correlational handwaving, not proof. However, by demonstrating that both social intuition and implicit learning rely on the integrity of the basal ganglia, we can then draw the inference that social intuition operates by way of implicit learning. Thus, neuroscience allows us, in this case, to go further than social psychology and cognitive psychology alone. Furthermore, once the association between social intuition and the basal ganglia is made, then all the knowledge regarding the computational properties of the basal ganglia can be brought to bear on theories of social intuition. These properties provide new insights into the type of training environments likely to yield accurate intuitions. They also suggest which forms of social inference processes are likely to have a shared underlying intuitive component (e.g., scripts and automatic evaluation effects) on the basis of the match between the computational properties of the basal ganglia and the processing needs inherent in the inference category.

The social cognitive neuroscience approach can also be used to assess the necessity of various processing mechanisms in a given
social psychological process. For instance, both cognitive dissonance theory and self-perception theory implicitly rely on the untested assumption that explicit memory for one’s counterattitudinal behavior is essential for the occurrence of behavior-induced attitude change. Festinger’s (1957) cognitive dissonance theory proposes that counterattitudinal behavior leads to dissonance arousal. When the arousal becomes psychologically disturbing, the individual is motivated to alter the initial attitude in the direction of the counterattitudinal behavior, thus restoring consonance between behavior and action. Explicit memory is implicated at the point of conscious detection of the dissonance arousal because the disturbance must be attributed to a cause, presumably to the conflict between behavior and action. If there were no memory for the counterattitudinal behavior, it would be impossible to make this attribution and do the subsequent mental work to bring one’s attitude into alignment with the behavior. Bem’s (1965) self-perception theory suggests that attitudes are often constructed on the basis of our observation and memory for our recent behavior. Accordingly, counterattitudinal behavior should not influence a newly constructed attitude unless the behavior can be explicitly recalled. Although both of these influential social psychological theories rely on explicit memory in their explanation of behavior-induced attitude change, this assumption has never been tested. Social and cognitive psychology have no methodologies that allow researchers to assess whether explicit memory is a causal necessity in behavior-induced attitude change, but neuropsychology does. Anterograde amnesia severely impairs the ability to form new memories that can be explicitly retrieved at a later point. Lieberman, Ochsner, Gilbert, and Schacter (1999) recently tested a group of anterograde amnesic on a behavior-induced attitude change paradigm (Brehm, 1956). Although amnesia evidenced no memory for the counterattitudinal behavior, they actually showed more attitude change than healthy controls. This surprising finding is one that could only be obtained by going down a level to neuropsychology.

The current research and the study of attitude change in amnestic subjects are just two of a growing number of research programs that are taking advantage of the social cognitive neuroscience approach to answer enduring questions. Damasio (1994) used the methods of social cognition in order to understand the processing impairment associated with damage to ventromedial prefrontal cortex. Klein et al. (1996) tested a patient with temporary amnesia to show that self-knowledge does not depend on episodic information. Caicoppi, Crites, and Gardner (1996) demonstrated that evaluative self-knowledge does not depend on episodic information. Crites, Cacioppo, & Gardner (1996) tested a patient with temporary amnesia to show that self-knowledge does not depend on episodic information. Cacioppo, Crites, & Gardner (1996) showed that social psychologists to form the hypotheses formed by neuroscientists regarding the processing mechanisms in the brain, as well as allow the growing knowledge of the computational properties of neuroanatomical regions and their interactions to refine and clarify our understanding of social psychological phenomena.

References
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performance in tests sensitive to frontal lobe dysfunction. Psychopharmacology, 107, 394–404.


Appendix

Abbreviations

AC = anterior cingulate
APA = arcuate premotor area
CaM PK II = calcium-calmodulin-dependent kinase II
CT = computed tomography
DA = dopamine
dLPFC = dorsolateral prefrontal cortex
FEF = frontal eye fields
GP = globus pallidus
HCMP = hippocampus
HD = Huntington’s disease
OFC = orbitofrontal cortex
PD = Parkinson’s disease
PET = positron emission tomography
PFC = prefrontal cortex
PPC = posterior parietal cortex
SMA = supplementary motor area
SN = substantia nigra
SNC = substantia nigra pars compacta
SNR = substantia nigra pars reticulata
SP = spiny neuron
SRT = serial reaction time
TOL = Tower of London
VMPFC = ventromedial prefrontal cortex


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