Activity of striatal neurons reflects social action and own reward

Raymundo Báez-Mendoza[a,1], Christopher J. Harris[b], and Wolfram Schultz[a]

[a]Department of Physiology, Development and Neuroscience, University of Cambridge, Cambridge CB2 3DY, United Kingdom; and [b]Faculty of Economics, University of Cambridge, Cambridge CB2 9DD, United Kingdom

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Social interactions provide agents with the opportunity to earn higher benefits than when acting alone and contribute to evolutionary stable strategies. A basic requirement for engaging in beneficial social interactions is to recognize the actor whose movement results in reward. Despite the recent interest in the neural basis of social interactions, the neurophysiological mechanisms identifying the actor in social reward situations are unknown. A brain structure well suited for exploring this issue is the striatum, which plays a role in movement, reward, and goal-directed behavior. In humans, the striatum is involved in social processes related to reward inequity, donations to charity, and observational learning. We studied the neurophysiology of social action for reward in rhesus monkeys performing a reward-giving task. The behavioral data showed that the animals distinguished between their own and the conspecific's reward and knew which individual acted. Striatal neurons coded primarily own reward but rarely other's reward. Importantly, the activations occurred preferentially, and in approximately similar fractions, when either the own or the conspecific's action was followed by own reward. Other striatal neurons showed social action coding without reward. Some of the social action coding disappeared when the conspecific's role was simulated by a computer, confirming a social rather than observational relationship. These findings demonstrate a role of striatal neurons in identifying the social actor and own reward in a social setting. These processes may provide basic building blocks underlying the brain's function in social interactions.

Social interactions enhance individual fitness by giving agents access to otherwise unobtainable resources (1). To be successful in such interactions, individuals need to recognize the social action resulting in reward. This process allows individuals to assign credit to their partners (2), which is crucial for establishing and maintaining mutually beneficial interactions.

Despite the importance of recognition of social action for obtaining reward, little is known about the underlying neuronal mechanisms. Recent evidence suggests separate coding of social reward and action in the neuronal activity of distinct frontal cortical areas of monkeys. Reward neurons distinguish between own and other's reward (3, 4) and show differential activity during social competition (5) and movement (6). Action neurons respond to the observation of movement of social partners (7), differentiate between own and other's movements (8), and detect other's error commission (9). However, none of these studies investigated the neuronal coding of social action together with reward.

A candidate brain structure for coding social action and reward is the striatum. Its neurons code reward expectation and reception (10, 11), are activated during planning and execution of movements (12), and process influences of reward on movement coding (13, 14). In human imaging, the striatum is activated during social interactions, such as donation giving (15), observational learning (16), and detection of reward inequity (17). A social role of the striatum is further supported by actor specific activations without reward coding in cingulate cortex (2), which projects strongly to striatum (18). Thus, the striatum is engaged in separate reward, action, and social mechanisms, but it is unknown how the same striatal neurons may process social action and reward.

To investigate neuronal mechanisms underlying social action and reward, we recorded neuronal activity from the striatum while two rhesus monkeys interacted via a horizontal computer touchscreen mounted between them (Fig. 1 *A* and *B*). We used imperative trials to avoid choice confounds and dissociated two social dimensions: who was required to act, and who would receive reward. Only one animal acted at a time, and the actor's role switched after every correct trial. Hence, the recorded animal performed as actor or conspecific on alternate trials. There were no behavioral requirements for the animal taking the role as conspecific. The animals experienced four different payoffs: reward to neither, only to the recorded animal, only to the conspecific, or to both (Fig. 1*C*). We used fixed numbers of juice drops on each experimental day, thus testing reward presence vs. reward absence.

Results

Behavior. To investigate the neuronal coding of social variables, it is critical to first establish that the animals reacted to the social components of the task. Four behavioral measures fulfilled this requirement. Reaction times of the recorded animal varied between conspecific's rewarded and unrewarded trials (Fig. 1*D*, conspecific: purple vs. blue, own reward: red vs. blue; $P < 0.05$, post hoc Tukey tests following one-way ANOVA). There were also minor effects of conspecific's reward on error rates (Fig. S1*A*). Eye fixation patterns revealed that the recorded animal closely observed the conspecific's reward-predicting stimuli, face and spout (Fig. 1 *E* and *F*), confirming previous observations during social interaction of nonhuman primates (3, 8, 19). Without being required, the passive, nonacting animal maintained its hand on the resting key (Fig. 1*A*) during the conspecific's turn on a fraction of trials (monkey A: 20%; monkey B: 60%) and released

Significance

A basic requirement for engaging in beneficial social interactions is to recognize the actor whose movement results in reward. We studied the neuronal correlates of social action and reward in the monkey striatum. Behaviorally, the animals distinguished between their own and their conspecific's reward and knew which individual acted. In a subset of striatal neurons, neuronal activity occurred preferentially when either the own or the conspecific's action was followed by own reward. Some of the social action activity disappeared when the conspecific's role was simulated by a computer, confirming a social rather than observational relationship. These findings demonstrate a role of striatal neurons in identifying the social actor and own reward in a social setting.

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[1]To whom correspondence should be addressed. E-mail: raymundobaez@gmail.com.

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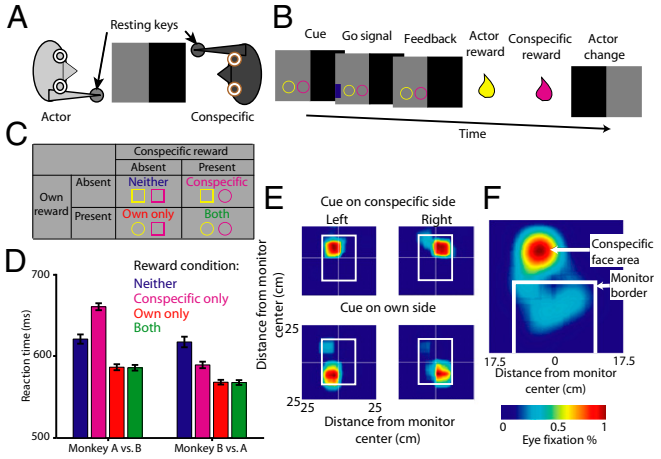

Fig. 1. Reward-giving task and behavioral results. (*A*) Experimental setup. Two monkeys sat opposite each other at a horizontal computer touch screen, each holding a resting key. On each trial, light gray and black backgrounds indicated actor and conspecific roles to the respective animals. (*B*) Task sequence: shape of conditioned cue predicted absence (square) or presence (circle) of reward for each animal (yellow for left, purple for right animal). Appearance of a subsequent blue go signal was followed by key release, stimulus touch and reward for actor, and 1 s later for conspecific. (*C*) The four reward conditions used: reward for neither, own reward only, conspecific's reward only, and reward for both. (*D*) Mean reaction times for the four reward conditions (from go signal to stimulus touch) between the two animals. Error bars show SEM. (*E*) Eye fixation density between onset of conditioned stimuli and go signal. (*F*) Eye fixation density on conspecific's face and spout after reward delivery to conspecific.

it in less than 3% of these trials (monkey A: 3%; monkey B, 2%). These results suggest that the animals distinguished between the active and the passive individual in the task, thus demonstrating crucial components of social behavior in our laboratory situation.

Neuronal Database. We identified 457 statistically significant task-related activations in 192 of 273 tested neurons in the anterior, precommissural caudate, and putamen of two monkeys (A and B; Fig. S2; one-way ANOVA with post hoc Holm test against baseline activity). The activations occurred in one or more of six task epochs: before cues, during cues, during arm movement, after arm movement, before reward, and after reward. Table 1 presents a breakdown of the major types of activations, which failed to vary significantly between monkeys [$\chi^2(20) = 24.97$, $P = 0.2$].

Reward Coding. The next step of our statistical analysis revealed that 177 of the 457 significant activations (38%; 125 neurons) distinguished between rewarded and unrewarded trials for the recorded monkey, as described before (13, 14), or for the conspecific monkey (three-way ANOVA). Specifically, 136 of the 177 significant activations (87 neurons) reflected either the presence ($n = 66$ activations; Fig. 2 *A–D*, red-green vs. blue-purple) or the absence of own reward ($n = 70$; Table 1). By contrast, these activations failed to vary with the conspecific's reward (purple vs. blue). As a confirmation, the activations differed also when both animals received reward compared with the conspecific alone (green vs. purple) or when neither received reward (green vs. blue). Inversely, 16 activations were modulated by the conspecific's, but not own, reward (9% of 177 activations; 15 neurons; Table 1; Fig. S3*A*; *SI Text*). Finally, 25 activations were modulated by both own and conspecific's reward (14% of 177 activations; 22 neurons). Hence, most task-related striatal neurons coded own reward predominantly and separately from conspecific's reward.

Our regression analysis confirmed the predominant coding of own reward compared with conspecific's reward or reward to both (Fig. S4*A*; *SI Text*). The numbers of activations identified by the regressions (123, 32, and 2 activations for own, conspecific's, and both reward, respectively) corresponded to the numbers from the main analysis (three-way ANOVA; 136, 16, and 25 activations; Table 1).

The separate coding of own and conspecific's reward amounted to reward discrimination between the animals (Fig. 2, red vs. purple). To formally assess the discrimination, we subjected activity in these neurons to a receiver operating characteristic (ROC) distinguishing between reward only for the recorded monkey and reward only for the conspecific. A total of 107 of the 152 (70%) activations reflecting either own or conspecific's reward discriminated between reward recipients ($P < 0.05$, permutation test). These results suggest that a population of striatal neurons distinguished between own and conspecific's reward.

Coding of Social Action and Social Reward. For adequate social interactions, it is important to distinguish whether reward coincides with one's own or a conspecific's action (2, 20). Indeed, a subset of striatal activations made this distinction. Ninety-six of the 136 own reward coding activations (70%, 70 neurons) differentiated between the actors (Table 1). Of the 96 activations, 67 (49 neurons) were significantly higher when the recorded animal received reward while acting compared with the conspecific's action (own action; Fig. 2 *C* and *D*, red-green, solid vs. dashed lines). Inversely, 29 of the 96 activations (23 neurons) were higher when the conspecific rather than the recorded animal made the movement and the recorded animal received reward (conspecific's action; Fig. 2 *E* and *F*, dashed vs. solid lines). The remaining 40 of the 136 activations (30%, 32 neurons) were indifferent to whose turn it was (Fig. 2 *A* and *B*). Correspondingly, a fraction of the activations reflecting conspecific's reward also distinguished between the individual performing the action (8 of 16 activations in 8 of 16 neurons; Fig. S3*A*; *SI Text*). Table S1 provides a complete breakdown of activations reflecting social action and reward for the six task epochs.

Neurons may simultaneously code two variables in several ways. In the statistical form of interaction, one variable affects the coding of the other variable. This form represents the most interesting part of our results, as the individual who acts affects the coding of the reward for each animal. In the other form, neurons may conjointly code both variables independently from each other. Fifty-four of the 96 activations (46 neurons) reflected the interaction between own reward and social action (28 for reward presence, 26 for reward absence). Of these, 35 activations (30 neurons) reflected the interaction of own action and own

Table 1. Numbers of neuronal activations coding reward and actor

		Reward				
Actor	Animal	Own	Conspecific	Both	None	Subtotal
Own	A	37 (27)	4 (4)	5 (4)	28 (23)	74 (45)
	B	30 (22)	2 (2)	2 (2)	29 (25)	63 (44)
Conspecific	A	20 (16)	1 (1)	9 (8)	16 (13)	46 (30)
	B	9 (7)	1 (1)	0 (0)	7 (6)	17 (13)
None	A	15 (13)	6 (6)	8 (8)	99 (55)	128 (63)
	B	25 (19)	2 (2)	1 (1)	101 (71)	129 (80)
Subtotal	A	72 (47)	11 (10)	22 (19)	143 (79)	248 (93)
	B	64 (41)	5 (5)	3 (3)	137 (83)	209 (99)

Activations were classified into the stated categories only when coding was statistically significant and, if applicable, consistent between main factors and interactions. Some neurons showed activations in multiple periods. Thus, in columns and rows labeled subtotal, the number of activations is the sum across the row or column, but not the sum of neurons as each neuron was counted only once. Three classes of activation were collapsed in the actor none, reward none cell: (*i*) activations that showed incongruence in the significance between the main and interaction factors; (*ii*) activations with significant interactions but insignificant main factors; and (*iii*) neuronal activations without any significant effects. Number of neurons in parentheses.

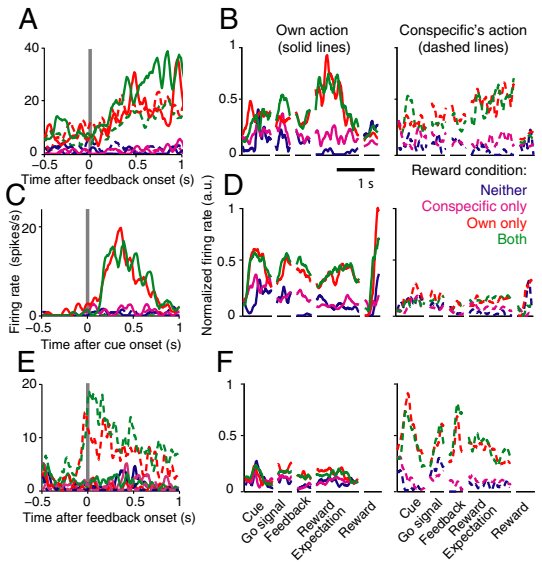

Fig. 2. Coding of reward and actor in striatal neurons. (*A* and *B*) Activations coding own reward irrespective of actor. (*A*) Single neuron (activation after feedback onset). (*B*) Population; $n = 20$ activations showing increased activity with own reward presence (red-green) compared with no own reward (purple-blue). Activity was normalized to maximum firing rate of individual neurons irrespective of trial type and is shown as impulse density. (*C* and *D*) Coding of own action and own reward: higher activations with own compared with conspecific's action in single neuron (*C*; activation after cue onset) and population (*D*; $n = 28$). (*E* and *F*) Coding of conspecific's action for own reward in single neuron (*E*; activations after feedback onset) and population (*F*; $n = 15$). Interrupted axes underneath *B*, *D*, and *F* indicate noncontinuous analysis periods and are labeled in *F*.

reward, and 19 (16 neurons) reflected the interaction of conspecific's action and own reward (Table S2). By contrast, 42 of the 96 activations (39 neurons) reflected own reward conjointly with a specific animal's action (20 for reward presence, 22 for reward absence). Of these, 32 activations (29 neurons) reflected conjointly own action and own reward, and 10 (10 neurons) reflected conjointly conspecific's action and own reward (Table S2).

The regression analysis confirmed the substantial sensitivity to social action and own reward in striatal neurons (Fig. S4*A*; *SI Text*). The numbers of activations identified by the regressions (95 and 7 activations for own and conspecific's reward, respectively) corresponded well to the numbers from the main, three-way ANOVA (96 and 8 activations; Table 1).

Taken together, these data demonstrate that a population of striatal reward neurons distinguished between social actors and between reward receivers.

Social Action Coding Irrespective of Reward. The observed social neuronal coding might be specific for the combination of action and reward. However, earlier studies on human and monkey cingulate cortex reported differential coding of action observation without having tested reward variations (2, 8). Thus, would striatal neurons also code social action irrespective of reward? Indeed, 80 activations of the 457 task-related activations (17%, 65 neurons) discriminated between actors but were not modulated by reward. Of these, 57 activations (48 neurons) were higher when the recorded animal acted rather than the conspecific (Fig. 3 *A*, *B*, and *E*), whereas 23 activations (19 neurons) showed the opposite (Fig. 3 *C*–*E*; Table S1). This result was confirmed by our regression analysis. Of 75 actor sensitive activations, 58 reflected own action and 17 reflected conspecific's action (Fig. S4*A*). In both the ANOVA and regression analysis, social action coding was not restricted to the movement period but could occur during any task period.

Neuronal ROC. We used ROC analysis as a third independent measure to assess the degree of neuronal coding for reward recipient and actor. Activations reflecting the presence or absence of own or conspecific's reward (Fig. 3 *E* and *F*, horizontal axis), as well as activations not modulated by reward, were sensitive to the actor, being higher for either own (Fig. 3 *E* and *F*, below horizontal line) or conspecific's action (above horizontal lines; number of activations on each category is summarized in Table S3). Thus, the ROC analysis confirms the substantial sensitivity to social action for reward as identified by our ANOVA and regression analysis.

Social vs. Observational Nature of Actor Coding. If these activations depended on the social nature of the other agent, they should change in its absence. We investigated this possibility by moving the conspecific out of the recorded monkey's sight while a computer presented identical visual stimuli on the touch table in the same sequence of events used in conspecific's trials (except for reaction time being fixed to 1.2 s). The recorded animal received the same juice amount as the computer, but the computer's juice dropped visibly into an inaccessible bucket. We tested a further 22 actor-related activations from 12 striatal neurons. Of these, nine activations lost the discrimination between own and not

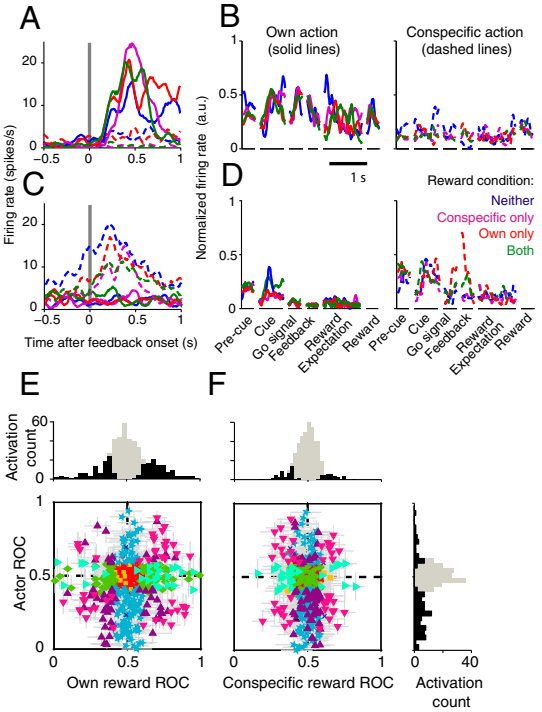

Fig. 3. Sensitivity to actor in striatal neurons not coding reward and neuronal ROC. (*A* and *B*) Coding of own action (solid lines) compared with conspecific's action (dashed lines) in single neuron (*A*) and population (*B*; $n = 57$). Overlapping solid lines suggest lack of reward coding. (*C* and *D*) Coding of conspecific's action (dashed lines) rather than own action (solid lines) in single neuron (*C*) and population (*D*; $n = 23$). Overlapping dashed lines suggest lack of reward coding. (*E*) Neuronal ROC values for own reward vs. actor. Reward ROC varies between 0 and 1 for no reward vs. own reward and actor ROC varies between 0 and 1 for own action vs. conspecific's action. (*F*) Same as *E* but for conspecific's reward vs. actor. Gray bars indicate 95% bootstrap CI. Number of members on each group for *G* and *H* (permutation test, $P < 0.05$): green rhomboids ($n = 73$), own reward and not actor; yellow squares ($n = 11$), conspecific's reward and not actor; turquoise triangles ($n = 23$), own and conspecific's reward and not actor; purple triangles ($n = 61$), own reward and actor; dark blue crosses ($n = 3$), conspecific's reward and actor; blue stars ($n = 118$), not reward but actor; pink triangles ($n = 45$), all categories; red dots ($n = 122$), all insignificant.

own action when the conspecific was out of sight (Fig. 4 *A* and *B*, *Center*; Fig. 4*C*, empty bars). The activations recovered when the conspecific was returned to the task (Fig. 4 *A* and *B*, *Right*). Of the nine activations, seven occurred with own action (Fig. 4*A*) and two with conspecific's action (Fig. 4*B*). The remaining 13 activations differentiated between own and not own action irrespective of the conspecific being present or absent (Fig. 4*C*, filled bars).

Arm movement reaction time was shorter in this control task (monkey A: from 588 ± 5 to 563 ± 6 ms, mean ± SEM, $P > 0.05$, *t* test; monkey B: from 542 ± 6 to 496 ± 13 ms, $P < 0.05$) and correlated only insignificantly with the reductions in actor coding of the nine social sensitive neuronal activations ($r = 0.58$, $P = 0.09$; Pearson's correlation). Thus, the observed loss of actor coding in the computer control task was unlikely to be explained by faster arm movement reactions.

Of 14 agent differential activations recorded together with eye movements, only one showed relationships to ocular positions ($P < 0.05$, ANCOVA). Of the remaining 13 activations, six were social sensitive in being reduced during the computer test, whereas seven were maintained during the computer test. We conclude that a fraction of neurons distinguishing between own and conspecific's action stopped doing so when the social agent was removed, suggesting that these neurons were sensitive to the social context.

Response Inhibition. Neuronal activations classified as being sensitive to social action might instead reflect differences in action vs. no action of the recorded animal. The task did not require specific behavioral action or inhibition by the nonacting animal. In particular, the passive player did not need to contact the resting key, nor release it after the cue, and thus was not required to inhibit a prepotent behavioral response. Nevertheless both animals contacted the key in a proportion of trials (monkey A: 20%; monkey B: 60%) and occasionally released it after the cue (monkey A: 3% of all trials; monkey B, 2%), and thus may not have inhibited a response in these trials. If activations classified as sensitive to own social action might be simply explained by performance of own movement, then neuronal activity during trials without response inhibition should be similar to own action trials as the recorded animal moved in both situations. Similarly, if responses sensitive to conspecific's social action were explained by inhibition of own movement, they should be stronger in trials with response inhibition compared with trials without response inhibition. This analysis included only neurons tested in greater than five uninhibited trials in which the recorded animal released the key during conspecific's trials. Of the 58 activations classified as reflecting own social action (with or without reward sensitivity), 53 remained significantly lower in conspecific's than own trials even when the recorded animal acted (Fig. 4*D*, Response inhibition and No response inhibition vs. Own action; one-way ANOVA followed by post hoc Tukey test; both $P < 0.05$). Similarly, of the 36 activations reflecting conspecific's social action, 29 remained significantly higher in conspecific's than own trials even when the recorded animal moved (Fig. 4*E*). Thus, the reported activations sensitive to social action were unlikely to be explained by own movement or response inhibition.

Eye Movement. Given the known involvement of striatal neurons in eye movements (21), neuronal activations sensitive to social action might reflect oculomotor processes. Particularly important were eye position, which differed between own and conspecific's action (Fig. 1*E*), and eye velocity, which might have reflected differential motivation between own and conspecific's action (general neuronal relationships to saccadic eye movements are described in *SI Text* and Table S4). Of 81 actor-related activations recorded alongside eye movements, 61 (76%) were unrelated to eye position. Furthermore, 71 of the 81 activations (88%) were unrelated to eye movement velocity. The remaining activations could equally reflect actor or eye movements. Thus, differences in eye position or velocity were unlikely to explain the neuronal coding of social action and reward.

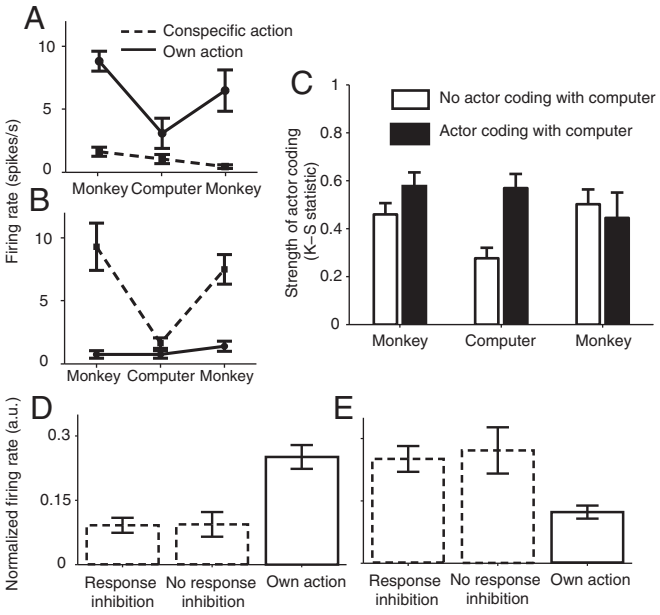

Fig. 4. Decrease of neuronal distinction between own and conspecific's action during computer control test. (*A*) Higher activation with own action compared with conspecific's action (*Left*) decreased when conspecific was replaced by computer (*Center*). The difference recovered with reinstatement of conspecific (*Right*). Data are from a single striatal neuron. (*B*) Same as *A*, but higher activation with conspecific's action. (*C*) Kolmogorov-Smirnov statistics (*D*) for influence of computer opponent on actor specific neuronal responses. Empty bars, decreased difference own vs. conspecific's action with computer (9 activations); filled bars, maintained difference own vs. conspecific's action with computer (13 activations). (*D* and *E*) Simple action relationships fail to explain neuronal sensitivity for social action (*D*, average of 58 activations coding own social action; *E*, 36 activations coding conspecific's social action). Solid bars represent population activity during action of recorded monkey (normalized to maximum firing rate of individual neurons irrespective of trial type). Dashed bars represent activity in recorded monkey during conspecific's action. Response inhibition refers to absence of movements of recorded monkey. In No response inhibition trials, the recorded animal performed movements without being required. In *D*, neuronal activity was high in own trials but low in conspecific's trials irrespective of own inhibition. In *E*, neuronal activity was low in own trials but high in conspecific trials irrespective of own movement. Error bars show SEM.

Reward Timing. The acting monkey received the reward 1 s before the conspecific. Thus, would the activations coding social action and own reward rather reflect temporal discounting of reward value? To address this issue, we included a hyperbolic discounting model in the simultaneous testing procedure using a wide range of discounting parameters typical for rhesus monkeys (22–24) (*SI Text*). In 3 of 95 neuronal activations, we rejected the social action and own reward model in favor of the hyperbolic discounting model. Thus, temporal discounting did not seem to explain the large majority of activations reflecting social action and own reward.

The acting animal received reward 1 s before the passive animal to facilitate distinction between reward recipients. Despite this advantage, the temporal order of reward delivery might explain the actor sensitivity of reward responses. This possibility was investigated in conspecific's trials in which the recorded animal received reward first when the conspecific received no reward, but last when both animals received reward. Only 1 of the 29 neuronal activations reflecting conspecific's action and own reward varied significantly between these two trial types ($P < 0.05$; ROC with 2,000 permutations). A similar comparison was impossible

for actions by the recorded animal, as it always received the reward first. Thus, at least for conspecific's actions, temporal order did not seem to explain actor-sensitive reward processing.

Reward Cost. Movement effort constitutes an economic cost that reduces reward value (25). Thus, would activations coding social action and own reward be better explained by own reward minus cost? To test this possibility, we included an action-cost model in the simultaneous testing procedure with a wide range of cost parameters (*SI Text*). In only 14 of 95 neuronal activations originally reflecting social action and own reward (Fig. S4, *Inset*) was action cost a reasonable model of the data. Thus, movement effort did not seem to explain most activations reflecting social action and own reward.

Discussion

These experiments revealed that a population of striatal neurons coded social action and reward. Other striatal neurons coded social action without coding reward. Almost all reward related striatal neurons processed own rather than conspecific's reward. A fraction of social action-coding neurons required the presence of a social agent, suggesting a biological relationship. This spectrum of neuronal activity demonstrates an active involvement of the striatum in social processes.

During the nonsocial computer control task, the activity of some social action-coding neurons disappeared with the removal of the conspecific. This result implies that the activity was not related to the animal's action per se. Instead, it may have been related to the social context in which the action was embedded. In the subclass of neurons sensitive to conspecific's action, the response reduction could have been due to reduced visual stimulation generated by the conspecific's absence. However, the activity recorded during the computer control task was unrelated to eye position and thus insensitive to the visual stimulation elicited by different eye positions.

Our analysis of potential alternative interpretations included behavioral processes known to engage striatal neurons, including simple motor action, behavioral inhibition, eye position, eye velocity, reward delay, reward order, and effort (economic cost) (13, 21, 22, 26). Remarkably, the combined evidence from these limited control analyses suggests that only a few of the observed striatal activations reflecting social processes could be explained by these behavioral alternatives.

The sensitivity to own vs. conspecific's action could alternatively reflect differential coding of peri-personal space (within reach) during own trials vs. extrapersonal space (out of reach) during conspecific's trials. Movement-related neurons in the frontal cortex are known to code the distance at which task-relevant stimuli are presented (27). In the computer control task, stimulus positions in peri- and extrapersonal space were unchanged; hence, activations reflecting personal space should remain unaltered. However, the social responses were reduced during the control task in a good fraction of neurons. These data suggest sensitivity to social action rather than personal space in these neurons. By contrast, some of the activations not affected by the control task might reflect personal space, requiring additional experimentation for better characterization.

Our data on social action coding in the striatum might challenge the known role of this structure in straightforward movement processing (13, 14). However, previous studies already demonstrated the conditional nature of striatal movement-related activity. For example, groups of putamen neurons are only activated with the first of several consecutive movements (28), many movement-related striatal neurons are only activated with rewarded as opposed to unrewarded movements (14, 15), and some striatal neurons code action values rather than the actions themselves (29). Some of these neurons, and some of the many unmodulated neurons in the anterior striatum in which we recorded here, might have shown social action relationships if the testing involved a conspecific. Thus, rather than challenging the movement role of the striatum, the observed social action coding provides an extension of the conditional nature of movement-related activity of this structure.

Our data might challenge current views that position processing of social information and action primarily in the cerebral cortex and amygdala (30, 31). However, striatal neurons receive afferents from many cortical and subcortical structures involved in social processes (18, 32), which could give rise to the observed activities. As striatal neurons link own action with own reward (33), converging inputs and local interactions in the striatum (34) are also well suited to combine information about other's actions and own reward. Thus, the current data extend social processing to a further subcortical structure.

The current observation of social action-related activity may help to assign credit to a social agent when receiving reward from other individuals (2). The recognition of social agency is an important factor for fostering reciprocal exchanges in primates and maintaining adequate social relationships (1, 2, 20). "Returning a favor" to someone relies on knowing whose action produced the original favor. This type of social interaction would break down without that knowledge. The present data suggest a possible role of the striatum in this form of social behavior.

Materials and Methods

Two adult male *Macaca mulatta* monkeys (monkeys A and B), weighing 9 kg, served in the main study. All experimental procedures were approved by the University of Cambridge Ethics Committee and the Home Office under the Animals (Scientific Procedures) Act 1986.

Monkeys A and B performed an imperative reward giving task under computer control (Fig. 1*A*). The task included two simultaneous cues predicting reward (circle) or no reward (square) separately for each animal (Fig. 1*B*, *Left*), followed by a blue go signal eliciting the actor's arm movement for touching the stimuli (Fig. 1*B*, second from left), and delivery of reward to the actor and 1 s later to the conspecific. We tested presence vs. absence of reward and kept the amount of reward fixed on each day (one to three drops indicated by one to three circles, respectively). We sampled extracellular activity from 273 slowly firing neurons ($n = 115$ and $n = 158$ in monkeys A and B, respectively) with known electrophysiological properties (21) from the anterior striatum of one monkey at a time with conventional electrophysiological techniques during at least 64 trials performed by each individual. We discriminated the activity of single neurons against background noise online with window discriminator hardware and offline with spike sorting software (Plexon).

Analysis of Neuronal Data. We defined the control period as the last 0.5 s of the intertrial interval and defined six trial epochs of interest, namely (*i*) onset of gray background for acting animal (0.5 s before cue onset), (*ii*) cue onset (between 0.25 s after cue onset and go signal onset 0.75 s later), (*iii*) go signal onset (the first 0.5 s after go onset), (*iv*) movement feedback (0.5 s starting at go signal offset), (*v*) before reward (from end of movement feedback period for 1.5 s, which coincided with disappearance of the cue for the acting animal), and (*vi*) reward delivery (from first juice pulse to 0.5 s later).

In the first step, we defined task-related activations as a significant increase in neuronal activity during a task epoch compared with baseline using a one-way ANOVA followed by post hoc Holm test ($P < 0.05$). Subsequently, we explored the relationship of neuronal activity to reward receiver and actor of each task-related activation using a three-way ANOVA ($P < 0.05$). The factors and their levels were as follows: reward for recorded monkey (presence or absence), reward for conspecific (presence or absence), actor (action by recorded monkey or conspecific monkey), and interaction between reward receiver and actor. Because striatal neurons may show either increasing or decreasing activity with increasing reward value (35), this analysis distinguished between activations induced by reward presence (high reward value) and activations induced by reward absence (low reward value). We did not test the interaction between own and conspecific's reward or a triple interaction because they did not capture the variables of interest, namely actor and reward.

We classified activations as "own reward" or "conspecific's reward" when the respective factors were significant in the three-way ANOVA. Activations reflected own action when the factor actor was significant and mean firing rate during own trials was higher than during conspecific trials. Likewise, an activation reflected conspecific's action when activity was higher during conspecific's than own trials. Activations reflected actor for own reward when the statistical interaction of actor × own reward was significant. These activations revealed the most interesting aspect of the results, namely coding

of reward for the recorded animal only when either that animal or the conspecific performed the action that led to reward for the recorded animal. By contrast, activations reflected conjoint coding of action and own reward when both factors were significant but the interaction of actor × own reward was insignificant. We classified, analogously, the few actor coding activations for conspecific's reward by the significant interaction of actor × conspecific's reward, whereas conjoint coding showed an insignificant interaction.

Eye Movement Analysis. We used an ANCOVA to analyze potential oculomotor relationships in actor-related activations in monkey A. The ANCOVA included the same variables as the main ANOVA and additional horizontal and vertical eye position and, separately, eye velocity. Activations were considered as actor-related when the factors referring to an actor survived addition of the eye movement parameters ($P < 0.05$).

ROC Analysis. To assess differences in neuronal activations in binary comparisons involving reward, actor, or spatial movement positions, we used ROC analysis. A significant ROC indicated discrimination (permutation test with 2,000 iterations; $P < 0.05$). CIs were established from a bootstrap distribution of ROC values (2,000 iterations).

Multiple Linear Regression. We used multiple regressions structured as a simultaneous testing procedure (STP) for refinement and independent confirmation of the ANOVA results. This procedure adequately deals with multiple significant regressors without requiring corrections for multiple tests of significance. In addition, it allowed us to test temporal discounting and action cost. The principal hypothesis is stated in the unrestricted model

$$Y = \beta_0 + \beta_1 A + \beta_2 W + \beta_3 Z + \beta_4 AW + \beta_5 AZ + \varepsilon, \quad [1]$$

where Y is neuronal activity, A is actor (recorded animal acts = 1, conspecific acts = 0), W is reward for recorded animal (reward = 1, no reward = 0), and Z is reward for conspecific animal (reward = 1, no reward = 0). The two models of most interest were

$$Y = \beta_0 + \beta_1 A + \beta_2 W + \beta_4 AW + \varepsilon, \quad [2]$$

which tests neuronal coding of actor for own reward, and

$$Y = \beta_0 + \beta_1 A + \beta_3 Z + \beta_5 AZ + \varepsilon, \quad [3]$$

which tests neuronal coding of actor for conspecific's reward. We also included a model that tests coding of reward only

$$Y = \beta_0 + \beta_2 W + \beta_3 Z + \varepsilon. \quad [4]$$

All models implied by the unrestricted model (Eq. **1**), as well as the temporal discounting and action cost models, are detailed in Eqs. **S5–S16** (for full details of the STP, see *SI Text*).

Computer Control Test. To test the social nature of the observed actor-dependent coding, we moved the conspecific animal 50 cm laterally out of the recorded monkey's sight and away from the spout and delivered its reward visibly into an inaccessible bucket. In these trials, the behavioral control computer timed all stimuli as in the regular trials and mimicked task performance in alternation with the recorded monkey; the go stimulus stayed on for a fixed 1.2 s. We conducted these tests in three consecutive trial blocks, namely with the conspecific present, the computer replacing the conspecific, and the conspecific back again. In each block, we assessed the difference in neuronal activation between the own and the conspecific's action using the Kolmogorov–Smirnov test and the direction of the difference using the ROC. Actor-related social activations were considered as biological if the differences between actors were significant during the initial and the retesting block but not during the computer block.

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Supporting Information

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SI Text

Subjects. Two adult male *Macaca mulatta* monkeys (monkeys A and B), weighing 9 kg, served in the main study. The animals were housed together with other two adult male macaques and showed a constant linear hierarchy throughout the experiments (monkey C > D > A > B). The animals had ad libitum access to water continuously 24 h every 7 d. We ranked dominance as the order in which they accessed a single water spout on these days (1). All experimental procedures were approved by the University of Cambridge Ethics Committee and the Home Office under the Animals (Scientific Procedures) Act 1986.

Behavioral Task. Two monkeys performed in an imperative reward giving and receiving task (Fig. 1 *A* and *B*) in the presence of a white background noise of 60 dB sound pressure level (SPL). Each trial started when the background color on the acting animal's side of the touch screen changed from black to light gray. Subsequently, the animal placed its right hand within 0.5 s on the resting key, followed after 0.5 s by presentation for 1 s of a conditioned cue for the acting animal and, simultaneously, another cue for the conspecific passive animal. The cues appeared either to the left or right of the animal's midline. The cues differed between animals in color and between reward presence or absence in shape (Fig. 1*C*; reward prediction, circle of 2 cm diameter; reward absence: square of 2 cm length). Subsequently a blue rectangle of 5.3 × 2.6 cm appeared as a go signal, which the animal touched within 1.2 s. The blue rectangle disappeared on touch and initiated a 2-s delay for reward delivery to the acting animal. The cue for the acting animal disappeared on reward delivery. The other animal received its reward 1 s after the reward to the acting animal, together with extinction of its cue. The gray background extinguished 0.5 s after reward delivery, and an intertrial interval of 1.5 ± 0.25 s drawn from a uniform distribution began. The roles of active and passive player reversed after every correct trial. Possible errors comprised failure of key touch before the trial, key release before the go signal, or failure of touch of blue rectangle. Errors led to time out (trial duration + 0.5 s) with a black background and then trial repetition. Task performance was interrupted after at least three consecutive errors.

The animals received four different outcomes: reward to neither, reward only to the recorded animal, reward only to the conspecific, or reward to both (Fig. 1*C*). We used one, two, or three drops of 0.2 mL of blackcurrant juice to accommodate different levels of motivation across experimental days. The number of juice drops was kept fixed on each experimental day. We indicated each player's payoff by the number of circles (one to three circles for one to three drops, respectively) or a square (no reward). Rewards consisting of two or three drops were delivered with 0.15-s intervals.

Eye movements of animal A were sampled at 125 Hz via an infrared camera (ISCAN) placed next to the touchscreen (EloTouch 1522L 15"; Tyco). Stimuli and behavior were controlled using custom MATLAB code (The Mathworks) and Psychophysics toolbox (version 3.0.8) (2, 3). The laboratory was interfaced with data acquisition boards (NI 6225; National Instruments) installed on a PC running Windows XP.

Recording Procedures. Conventional techniques of in vivo extracellular recordings served to study the activity of single striatal neurons. A stainless steel tube (0.56 mm outer diameter) guided a single tungsten microelectrode of 0.250 mm diameter and 1- to 5-MΩ impedance (FHC Inc.) through the dura and assured good targeting of subcortical structures. A hydraulic micromanipulator (MO-95; Narishige, Tokyo, Japan) served to advance the microelectrode vertically in the stereotaxic plane. Neuronal signals were amplified, bandpass filtered (300 Hz to 3 kHz), and monitored online with oscilloscopes. Somatodendritic discharges from single, slowly firing, medium spiny striatal neurons were distinguished from background noise and other neurons using a time threshold window discriminator (WD-95; Bak Instruments), which produced a 1.0-ms-long standard transistor-transistor logic (TTL) pulse for each neuronal impulse. We did not study tonically active striatal neurons that differed from medium spiny neurons in spontaneous activity, impulse waveform, and firing rate (4). Behavioral data, digital signals from the impulse window discriminator, and analog eye position data were sampled at 2 kHz on a laboratory computer with custom MATLAB code. We also recorded analog impulse waveforms at 22 kHz and sorted them offline (Offline sorter; Plexon).

Eye Position Analysis. We analyzed ocular fixation patterns to assess whether the monkey inspected the conspecific during and after reward delivery. Because the monkey's head was slightly tilted forward (~10°) for a better view of the touchscreen, we estimated eye position in two steps. First, we assessed eye position in a plane in front of the monkey's eyes, followed by a transformation into the table plane. Once eye position was obtained, we determined whether and when a fixation occurred. We defined a fixation when eye velocity was below 25% of its statistical SD for more than 80 ms (5). To create frequency maps of eye fixations, a histogram matrix (50 × 50 cm) with the possible eye positions was convolved with a Gaussian function ($\sigma = 1.5$).

We investigated eye position and eye velocity in monkey A as confounds for the neuronal coding of our variables of interest, namely actor, own reward, conspecific's reward, actor and own reward interaction, and actor and conspecific's reward interaction. We used an ANCOVA that focuses on the categorical variables that identify grouping or treatment factors, with an adjustment for the confounding effects of at least one continuous variable (6). Our ANCOVA model added horizontal and vertical eye position or eye velocity. We considered that an activation was actor-related when the factors referring to actor remained significant when the eye movement parameters were added to the ANCOVA ($P < 0.05$).

Linear Regression Analysis. We applied multiple linear regressions to the task-related activations identified by the initial one-way ANOVA to get an independent verification of the findings from the three-way ANOVA. We based the analysis on the same three main factors as the three-way ANOVA (own reward, conspecific's reward, actor). We structured the regression analysis as a simultaneous testing procedure (STP), following the general principles set out by Gabriel (7). We shall describe this analysis in detail below, but first we give an overview of the broad ideas underlying an STP.

An STP is a unified procedure for carrying out multiple statistical tests. It is structured in such a way that ad hoc corrections for multiple tests of significance are not required. The STP achieves this by testing all hypotheses, both the main hypotheses of interest and any hypotheses implied by the main hypotheses of interest, directly against a single unrestricted hypothesis, using the optimal test for that comparison. In particular, two sorts of test are avoided: (*i*) sequential tests, in which a later test may depend

on the outcome of an earlier test; and (*ii*) indirect tests, in which a composite hypothesis is evaluated on the basis of its component hypotheses. (For example, the composite hypothesis might be $\beta_1 = \beta_2 = 0$, and the two component hypotheses might be $\beta_1 = 0$ and $\beta_2 = 0$.)

Because all hypotheses are tested, two types of conflict can in principle arise. First, one of the hypotheses (call it hypothesis B) may imply another (call it hypothesis A). (For example, hypothesis B might state that $\beta_1 = \beta_2 = 0$ and hypothesis A might state that $\beta_1 = \beta_2$.) Because both hypotheses are tested, a serious conflict could in principle occur: the less restrictive hypothesis A might be rejected, whereas the more restrictive hypothesis B is accepted. One of the strengths of an STP is that it avoids this type of conflict altogether. Second, two hypotheses (call them hypothesis A and hypothesis B) may imply a third hypothesis (call it hypothesis C). (For example, hypothesis A might state that $\beta_1 = 0$, hypothesis B might state that $\beta_2 = 0$, and hypothesis C might state that $\beta_1 = \beta_2 = 0$.) In that case, hypothesis C would be tested directly, even if it is not of primary interest. This test can create a less serious type of conflict: both A and B might be accepted, whereas their implication, C, is rejected. This result can be interpreted as meaning that, although we know that the restrictive hypothesis C does not hold, the evidence is too ambiguous to allow us to decide which of the two less restrictive hypotheses A and B is to be preferred. Of course, this type of conflict could be swept under the carpet, either by suppressing C altogether or (in the case where C is simply the conjunction of A and B) by accepting or rejecting C on the basis of the separate tests for A and B. Another of the strengths of an STP is that it identifies ambiguous cases like this for what they are.

It may also be helpful to explain what we mean by an optimal test. All of the tests that we use are maximum-likelihood tests. [For a general discussion of the desirability of using maximum-likelihood tests in the context of an STP, see Gabriel (7).] Because most of our hypotheses require that one or more regression coefficients are 0, this means that, in practice, most of our tests are (partial) *F* tests. [If a hypothesis involves only one regression coefficient, then the (partial) *F* test is equivalent to a (two-sided) *t* test.] However, the maximum-likelihood approach is sufficiently flexible to cover those of our hypotheses that involve the positivity or negativity of certain regression coefficients. Indeed, it does so in a particularly simple way.

Finally, we note that a third strength of STPs is that they exert strong control over type I error (i.e., over false positives). Specifically, if the overall significance level of the STP is α, then, under the null hypothesis, the probability that any of the many hypotheses tested is accepted when it is false is α. (It is worth being completely explicit about what is meant here. If there are N hypotheses $H_1, H_2 \ldots H_N$, then the probability that there exists n such that H_n is accepted when it is false is α.) Furthermore, if the null hypothesis does not hold, then the probability that any of the many hypotheses tested is accepted when it is false is less than or equal to α.

Turning now to our own analysis, we note that the first step in setting up an STP is to formulate the principal hypotheses (or models) of interest. All of the models that we considered were special cases of the unrestricted model, namely

$$Y = \beta_0 + \beta_1 A + \beta_2 W + \beta_3 Z + \beta_4 AW + \beta_5 AZ + \varepsilon. \quad \textbf{[S1]}$$

Here, Y is neuronal activity, and the regressors are as follows: A, actor (recorded animal acts = 1, conspecific acts = 0); W, reward for recorded animal (reward = 1, no reward = 0); and Z, reward for conspecific animal (reward = 1, no reward = 0).

Eq. **S1** is analogous to the model tested with the three-way ANOVA, which facilitates comparisons between the two. The two models of most interest were

$$Y = \beta_0 + \beta_1 A + \beta_2 W + \beta_4 AW + \varepsilon, \quad \textbf{[S2]}$$

which can be interpreted as saying that actor and own reward together explain neuronal activity and

$$Y = \beta_0 + \beta_1 A + \beta_3 Z + \beta_5 AZ + \varepsilon, \quad \textbf{[S3]}$$

which can be interpreted as saying that actor and conspecific's reward together explain neuronal activity. We also included the model

$$Y = \beta_0 + \beta_2 W + \beta_3 Z + \varepsilon. \quad \textbf{[S4]}$$

This model can be interpreted as saying that only reward matters. Unlike Eqs. **S1** and **S2**, it does not include an interaction term. Exclusion of the interaction term (WZ in the present case) was justified by the design of our experiment, which investigated modulation of neuronal reward responses by actor, but not the interaction between own and conspecific's reward.

The second step in setting up an STP is to include all models implied by the principal models of interest, which ensures that the model is closed in the sense of Gabriel (7). For example, Eq. **S2** encapsulates the hypothesis that $\beta_3 = \beta_5 = 0$, and Eq. **S3** encapsulates the hypothesis that $\beta_2 = \beta_4 = 0$. Taken together, they imply that $\beta_2 = \beta_3 = \beta_4 = \beta_5 = 0$, i.e., that

$$Y = \beta_0 + \beta_1 A + \varepsilon \quad \textbf{[S5]}$$

holds. Similarly, Eqs. **2** and **4** imply that

$$Y = \beta_0 + \beta_2 W + \varepsilon, \quad \textbf{[S6]}$$

and Eqs. **3** and **4** imply that

$$Y = \beta_0 + \beta_3 Z + \varepsilon. \quad \textbf{[S7]}$$

Finally, Eqs. **2**–**4** together imply that

$$Y = \beta_0 + \varepsilon. \quad \textbf{[S8]}$$

Eq. **S8** is the minimal model in our STP.

The third step in designing an STP is to associate a test statistic with each of the models. This test statistic is used to test whether the model should be accepted or rejected when the alternative is the unrestricted model. Because all of the models described thus far require that a subset of the βs of the unrestricted model be 0, the correct statistic for our purposes is the augmented *F* statistic [cf. example 2.2 on page 226 of Gabriel (7)]. The alternative, for example testing Eq. **S2** using a pair of *t* statistics (for the separate hypotheses $\beta_3 = 0$ and $\beta_5 = 0$) instead of a single *F* statistic (for the joint hypothesis $\beta_3 = \beta_5 = 0$) is not recommended: the two *t* statistics are statistically dependent on one another, and combining them in this way would therefore require a Bonferroni or similar correction.

The fourth step is to choose a critical value ζ for the test statistics. To do this, we begin from the test statistic for the minimal model (Eq. **S8**), which is an augmented *F* statistic with $(p_U - p_M, n - p_U)$ degrees of freedom, where $p_U = 6$ is the number of parameters of the unrestricted model (Eq. **S1**), $p_M = 1$ is the number of parameters of the minimal model (Eq. **S8**), and n is the number of data points. We therefore fix a significance level $\alpha = 0.05$, and we let ζ be the associated critical value of the augmented *F* statistic with $(5, n - 6)$ degrees of freedom, i.e., the point at which the probability that that statistic exceeds ζ is α. Notice that we apply the same critical value to all of the statistics and not the same significance level. Hence, as the models become less restrictive, the significance level at which they are tested gets lower. In other words, the criterion for accepting

a less restrictive model is stricter than the criterion for accepting a more restrictive model. Notice too that an augmented F statistic with $(p_U - p_M,\ n - p_U)$ degrees of freedom is simply $p_U - p_M$ times the corresponding F statistic with $(p_U - p_M,\ n - p_U)$ degrees of freedom.

The fifth step is to test all of the restricted models (i.e., Eqs. **S2**–**S8**) against the unrestricted model (i.e., Eq. **S1**) using the same critical value ζ. For each model, we then either accept or reject the hypothesis that the corresponding parameter restriction holds. For example, for Eq. **S2**, we would either conclude that $\beta_3 = \beta_5 = 0$, or we would conclude that at least one of β_3 and β_5 was nonzero. Two sorts of conflicts might in principle arise at this point. First, the results might be inconsistent. For example, we might accept Eq. **S5**, implying that $\beta_2 = \beta_3 = \beta_4 = \beta_5 = 0$, but reject Eq. **S2**, implying that at least one of β_3 and β_5 is nonzero. In the terminology of Gabriel (7), this would be an example of incoherence. Second, the results might be incomplete. For example, we might accept Eq. **S2**, implying that $\beta_3 = \beta_5 = 0$, accept Eq. **S3**, implying that $\beta_2 = \beta_4 = 0$, but reject Eq. **S5**, implying that at least one of β_2, β_3, β_4, and β_5 is nonzero. In the terminology of Gabriel (7), this would be an example of dissonance.

The results of these tests are most easily understood in terms of a Venn diagram (Fig. S4*A*). In this figure, the set {Unrestricted Model} corresponds to Eq. **S1**, and the sets {Actor for Own Reward}, {Actor for Conspecific's reward}, {Own & Conspecific's reward}, {Actor}, {Own Reward}, {Conspecific's reward}, and {Insignificant} correspond to the Eqs. **S2**, **S3**, **S4**, **S5**, **S6**, **S7**, and **S8**, respectively. We also included two further sets, {Inconsistent} and {Incomplete}, which correspond to the two types of conflict outlined above.

The role of the latter two sets can be further explained by two examples. First, if we were to accept Eq. **S5** and reject Eq. **S2**, then we would be faced with the impossibility of putting the activation both inside the smaller set {Actor} corresponding to Eq. **S5** and outside the larger set {Actor for Own Reward} corresponding to Eq. **S2**. This placement is impossible because {Actor} is contained in {Actor for Own Reward}. The corresponding activation would therefore be placed in the set {Inconsistent}. Second, if we were to accept Eq. **S2**, accept Eq. **S3**, and reject Eq. **S5**, then we would be unable to determine whether the activation should go in the set {Actor for Own Reward}\{Actor} corresponding to Eq. **S2** but not Eq. **S5** or in the set {Actor for Conspecific's reward}\{Actor} corresponding to Eq. **S3** but not Eq. **S5**. The activation would therefore be placed in the set {Incomplete}. Notice that, in theory, our choice of test statistic should ensure that there are no instances of inconsistency. That this is indeed the case is confirmed by the statistical analysis.

All those activations that are situated in the set {Actor for Own Reward}, but not in either of the sets {Actor} or {Own Reward}, involve some form of statistical interaction between A and W. For our purposes, the best way of understanding this interaction is to look at the way in which the own reward signal is modulated by actor. To do this, we can rewrite Eq. **S2** in the form

$$Y = \beta_{11}W + \beta_{12}(1-W) + \beta_{13}AW + \beta_{14}A(1-W) + \varepsilon. \qquad \textbf{[S9]}$$

We can then identify two important special cases. The first of these is the case in which $\beta_{14} = 0$, namely

$$Y = \beta_{11}W + \beta_{12}(1-W) + \beta_{13}AW + \varepsilon. \qquad \textbf{[S10]}$$

Eq. **S10** corresponds to activations that reflect the actor for presence of own reward, with positive values of β_{13} corresponding to own action for presence of own reward and negative values of β_{13} corresponding to conspecific's action for presence of own reward. More explicitly, a neuronal activation for which Eq. **S10** is accepted but Eq. **S6** is rejected provides information about actor in the case in which a reward is received, but no information about actor in the case in which a reward is not received. The second important special case of Eq. **S9** is that in which $\beta_{13} = 0$, namely

$$Y = \beta_{11}W + \beta_{12}(1-W) + \beta_{14}A(1-W) + \varepsilon. \qquad \textbf{[S11]}$$

Eq. **S11** corresponds to activations that reflect the actor for absence of own reward, with positive values of β_{14} corresponding to own action for absence of own reward and negative values of β_{14} corresponding to conspecific's action for absence of own reward. More explicitly, a neuronal activation for which Eq. **S11** is accepted but Eq. **S6** is rejected provides information about actor in the case in which a reward is not received, but no information about actor in the case in which a reward is received.

Although Eqs. **S9–S11** cannot be obtained from the unrestricted model (Eq. **S1**) by simply setting a suitably chosen subset of the coefficients of the latter model to 0, they are all (linear) restrictions of the unrestricted model. Like the earlier models (Eqs. **S2–S8**), they can therefore all be tested against the alternative of the unrestricted model with an F test. In the expanded view in Fig. S4*A*, those activations from the main diagram for which Eq. **S2** is accepted but Eqs. **S5** and **S6** are rejected are further classified according to which of the two models (Eq. **S10** or Eq. **S11**) is accepted. In that view, the sets {Own Action for Presence of Own Reward}, {Conspecific's Action for Presence of Own Reward}, {Own Action for Absence of Own Reward}, and {Conspecific's Action for Absence of Own Reward} correspond, respectively, to Eq. **S10** with $\beta_{13} > 0$, Eq. **S10** with $\beta_{13} < 0$, Eq. **S11** with $\beta_{14} > 0$, and Eq. **S11** with $\beta_{14} < 0$.

Temporal Discounting. The value of a given reward is greater when it is delivered earlier rather than later. This drop in reward value with delay is well modeled in rhesus monkeys by hyperbolic temporal discounting. To test whether our results could be explained by hyperbolic temporal discounting, we incorporated a hyperbolic temporal discounting model into our STP. First, we noted that delays only occurred when the conspecific acted. Temporal discounting can therefore be modeled by the equation

$$Y = \beta_0 + \beta_1\{W/[1 + k(1-A)]\} + \varepsilon, \qquad \textbf{[S12]}$$

where k is a discount parameter. Second, based on the typical temporal discounting parameters for rhesus monkeys reported in refs. 8–10, it is reasonable to require that k is drawn from the interval [0.04, 0.31]. Third, because A takes only the values 0 and 1, Eq. **S12** can be rewritten in the form

$$Y = \gamma_0 + \gamma_1 W + k\gamma_1 AW + \varepsilon. \qquad \textbf{[S13]}$$

Fourth, Eq. **S13** is the special case of the unrestricted model (Eq. **S1**), in which $\beta_1 = \beta_3 = \beta_5 = 0$, $\beta_0 = \gamma_0$, $\beta_2 = \gamma_1$, and $\beta_4 = k\gamma_1$. The discounting hypothesis can therefore be stated as follows:

$$Y = \beta_0 + \beta_2 W + \beta_4 AW + \varepsilon, \qquad \textbf{[S14]}$$

with $0.04 \le \beta_4/\beta_2 \le 0.31$ (or with $\beta_4 = 0$ if $\beta_2 = 0$).

Notice that Eq. **S14** is a nonlinear hypothesis. It is weaker than Eq. **S8**, which is the special case of Eq. **S14** in which $\beta_2 = \beta_4 = 0$, and stronger than Eq. **S2**, which is the model that is obtained when the requirement that β_4/β_2 lies in the interval [0.04, 0.31] is dropped and the extra regressor A is added. It can be incorporated into our STP by calculating its maximum-likelihood statistic. This statistic is directly comparable with the augmented F statistics used to test the linear hypotheses (Eqs. **S2–S11**), and with the maximum-likelihood statistics used to test the positivity/

negativity of β_{13} and β_{14} in Eqs. **S10** and **S11**, respectively. It is favorable to the discounting hypothesis in that it allows the discounting parameter k to be chosen optimally within the interval [0.04, 0.31] for each neuronal activation. It is also favorable to the discounting hypothesis in that, in cases where the discounting parameter k lies at one of the endpoints of the interval [0.04, 0.31], it takes into account the fact that the discounting model is operating with two parameters fewer than Eq. **S2**. Overall, it provides a systematic test of the discounting hypothesis.

Finally, although we obtained the maximum-likelihood statistic for this model (and all models) in all neuronal activations, we only report the results of this analysis on neuronal activations in which we rejected Eqs. **S5** and **S6** but accepted Eq. **S2**, i.e., neuronal activations that lie in the set {Actor for Own Reward}.

Reward Cost. Effort involves an economic cost that could reduce the value obtained from own reward. To test whether our results could be explained in terms of an economic cost incurred by the acting monkey, we incorporated an action-cost model into our STP. First, we noted that a cost would only be incurred when the recorded animal acted. Action cost can therefore be modeled by the equation

$$Y = \delta_0 + \delta_1(W - cA) + \varepsilon, \qquad \textbf{[S15]}$$

where c is the cost of acting. Second, in the absence of a specific cost assessment, we considered a very wide interval of values for c, namely [0.2, 0.8]. Third, Eq. **S15** is the special case of the unrestricted model, in which $\beta_3 = \beta_4 = \beta_5 = 0$, $\beta_0 = \delta_0$, $\beta_1 = -c\delta_1$, and $\beta_2 = \delta_1$. The action-cost hypothesis can therefore be stated as follows:

$$Y = \beta_0 + \beta_1 A + \beta_2 W + \varepsilon, \qquad \textbf{[S16]}$$

with $0.2 \leq -\beta_1/\beta_2 \leq 0.8$ (or with $\beta_1 = 0$ if $\beta_2 = 0$).

Like Eq. **S14**, Eq. **S16** is a nonlinear hypothesis. It is weaker than Eq. **S8**, which is the special case of Eq. **S16** in which $\beta_1 = \beta_2 = 0$, and stronger than Eq. **S2**, which is the model that is obtained when the requirement that $-\beta_1/\beta_2$ lies in the interval [0.2, 0.8] is dropped and the extra regressor AW is added. It can be incorporated into our STP by calculating its maximum-likelihood statistic. It is favorable to the action-cost hypothesis in that it allows the cost parameter c to be chosen optimally within the interval [0.2, 0.8] for each neuronal activation. It is also favorable to the action-cost hypothesis in that, in cases where the cost parameter c lies at one of the endpoints of the interval [0.2, 0.8], it takes into account the fact that the action-cost model is operating with two parameters fewer than Eq. **S2**. Overall, it provides a systematic test of the action-cost hypothesis.

Finally, although we obtained the maximum-likelihood statistic for this model in all neuronal activations, we only report the results of this analysis on neuronal activations in which we rejected Eqs. **S5** and **S6** but accepted Eq. **S2**, i.e., neuronal activations that lie in the set {Actor for Own Reward}.

Reaction Time Decomposition. The reaction time (RT) in our task (from go signal to stimulus touch) can be decomposed into two measurable behaviors: key release latency after go signal (RL) and elapsed time between key release and stimulus touch [also called movement time (MT)]. We performed the same statistical analysis for these two measures as for the overall reaction time, namely one-way ANOVA followed by post hoc Tukey test. The four reward conditions had an effect on RL and MT in both monkeys: monkey A, RL: $F(3,5975) = 31.08$, $P = 0$; MT: $F(3,5975) = 5.52$, $P = 0.0008$; monkey B, RL: $F(3,5977) = 25.77$, $P = 1E\text{-}16$; MT: $F(3,5977) = 39.21$, $P = 0$ (Fig. S1*A*). Reaction times decreased with increasing own reward magnitude by 25 and 15 ms per extra juice drop of 0.2 mL in monkeys A and B, respectively.

The differences in RT between the different reward conditions were due to differences in RL and MT, but at different degrees in the two animals. Monkey A distinguished between own and no reward in RL (Fig. S1*A*, red and green vs. purple and blue) and between conspecific's reward and no reward in MT (purple vs. blue). Monkey B distinguished between own and no reward in MT (red and green vs. purple and blue) and between conspecific's reward and no reward in RL (purple vs. blue). Both RL and MT were shorter in this animal when giving reward to the conspecific compared with no reward for conspecific (RL: purple vs. blue and red; MT: purple vs. blue). Both RL and MT correlated well with RT in the two animals ($r = 0.76$, $P = 0$ and $r = 0.58$, $P = 0$, respectively) but poorly with each other ($r = -0.07$, $P = 0.000004$).

The differences in RT and MT for giving reward to conspecific vs. no reward (purple vs. blue) varied inconsistently between monkeys A and B. The two monkeys had different hierarchical positions in their social housing group. Although we tested each animal with two different opponents, the number of tested dyads is too low to make firm conclusions relative to hierarchy; therefore, we did not investigate the issue further. We have considered other possibilities for explaining RT differences, such as age, training duration, task experience, and breeding group, which were all similar, and found only a difference in the arrival date at the holding facility that might have affected their hierarchical ranking.

Errors. To further support the animals' distinctions of task components suggested by the RTs, we assessed the percentage of behavioral errors during task performance. We measured errors in nonoverlapping five trial bins to account for different trial numbers per session and different session numbers. Reward condition had a main effect on errors [monkey A: $F(3,1425) = 21.397$, $P = 0$; monkey B: $F(3,1718) = 192.64$, $P = 0$; Fig. S1*B*]. Specifically, monkey A made significantly less errors whenever it received reward compared with not receiving reward (red and green vs. blue and purple; $P < 0.05$, post hoc Tukey test). However, in contrast to its RT differences, monkey A made no more errors when giving reward to the conspecific compared with no one (blue vs. purple). Monkey B's error pattern mirrored its RT differences: it made the most errors when no one received reward (blue), followed by errors when only the conspecific received reward (purple); it showed low error rates whenever expecting own reward (red and green). These results suggest that monkey A's error rate was influenced by its own reward but indifferent to the conspecific's reward, whereas monkey B's error rate was sensitive to both own and conspecific's reward. Thus, although the variations in error rate varied slightly from the differences in RT, they support the notion that the animals distinguished between the individuals that received reward.

Coding of Conspecific's Reward. The three-way ANOVA analysis revealed that most reward-related activations reflected the reward for the recorded animal, whereas only few activations reflected the reward for the conspecific (16 of the 177 reward activations in 15 striatal neurons; Table 1). Of these 16 activations, 7 were significantly higher and 9 were significantly lower with conspecific's reward delivery compared with conspecific's reward absence. Fig. S3*A* shows higher neuronal activity when the conspecific received reward compared with no reward (purple and green vs. red and blue). In detail, activations were high when only the conspecific animal (purple) or both animals received reward (green) and low when only the recorded animal or nobody received reward (red and blue). These activations showed the mirror image to those reflecting own reward shown in Fig. 2*A*.

Coding of Social Action and Conspecific's Reward. Similar to the modulations with own reward, some activations reflecting conspecific's reward were sensitive to the animal that acted. Of the 16 activations reflecting the conspecific's reward, 6 were modulated when the recorded animal acted (six neurons), and 2 were modulated when the conspecific's acted (two neurons; three-way ANOVA). The remaining eight conspecific's reward-related activations were not modulated by actor (eight neurons). Thus, one half of the few activations reflecting the conspecific's reward were modulated also with the individual that acted, in analogy to the much larger group of activations modulated by own reward.

Spatial Preferences. Neurons in the striatum may show spatial preferences during stimulus presentation or action preparation and execution (11). To assess the possible coding of spatial preferences, we estimated neuronal discriminations between left and right stimuli and targets from an egocentric perspective using receiver operating characteristic (ROC) analysis with permutation statistics ($P < 0.05$). In the 457 task-related activations, spatial preferences were rarely significant (Fig. S3*B*, black). The few significant preferences were evenly distributed between left (n = 27, 6%) and right stimulus or target positions (n = 26, 6%). These low incidences suggest that spatial preferences did not explain the observed overall actor coding.

Saccadic Eye Movement and Neuronal Activity. We analyzed whether neurons with or without saccadic eye movement–related activity differed in reward or actor coding, We searched for all saccades during one session, binned them into eight cardinal directions, and measured the average firing rate between 250 ms before and 50 ms after each saccade onset on each direction. To estimate saccade direction selectivity, we measured the mean resultant vector length of the vector of mean firing rates for each saccade direction using the CircStats toolbox for MATLAB (11). Mean resultant length estimates range from 0, indicating no direction selectivity, to 1 indicating perfect saccade direction selectivity. We used a permutation test with 2,000 iterations to test for statistical significance.

We found that only 5 of 80 (6.2%) neurons recorded with eye position monitoring showed significant saccade direction selectivity, whereas the remaining 75 neurons showed no such saccadic relationships. The five neurons showed 15 task activations, of which 7 were modulated by social reward or actor (46%; Table S4). Although this fraction is lower than the overall incidence of social reward or actor coding in the whole population (57%), the number of five neurons was too low to draw any firm conclusions as to the incidence of social reward or actor coding in neurons with or without saccadic relationships.

Linear Regression Analysis. Our null hypothesis stated that none of the three variables of interest (actor A, own reward W, and conspecific's reward Z) would explain the observed changes in neuronal activity. This hypothesis was accepted for 155 activations (155/457, 34%), which were accordingly classified as {Insignificant} in the main Venn diagram in Fig. S4*A*. By contrast, the null hypothesis was rejected for 302 activations (302/457, 66%), suggesting that at least one of the three variables of interest explained the observed variance in neuronal activity. The 66% rejection rate compares favorably with the 5% rejection rate that would be expected under the null hypothesis.

The 302 activations for which the explanatory variables were significant need to be assigned to one of the neuronal categories that we identified. For 70 activations (70/457, 15%), this could not be done unambiguously. These activations were therefore classified as {Incomplete}. Of the remaining 232 activations, the overwhelming majority fell into one of three leading categories: those explained by actor alone (75/457, 16%, classified as {Actor}); those explained by own reward alone (28/457, 6%, classified as {Own Reward}); and those explained by the interaction of actor and own reward (95/457, 21%, classified as {Actor for Own Reward}). As expected on the basis of the theoretical properties of our procedure, none of the activations was classified as {Inconsistent}.

Each of the three leading categories deserves further discussion. For the first, namely those activations explained by actor alone, one would go on to ask: "Was activity higher when the recorded animal was the agent or when the conspecific was the agent?" To answer this question, we used the sign of the coefficient β_1 of the actor term in Eq. **5**. If positive, it indicated that the neuronal activation was higher when the recorded animal was the agent; if negative, it indicated that the neuronal activation was higher when the conspecific was the agent. In this sense, 58 activations (58/75, 77%) reflected own actor and 17 activations (17/75, 23%) reflected conspecific actor.

Similarly, for the second of the leading categories, namely those activations explained by own reward alone, one could go on to ask: "Was activity higher for presence or absence of own reward?" To answer this question, we used the sign of the coefficient β_2 of the own reward term in Eq. **S6**. If positive, it indicated that an activation occurred in trials in which own reward was presented; if negative, it indicated that that an activation occurred when own reward was absent. In this sense, 13 activations (13/28, 46%) reflected the presence of own reward and 15 activations (15/28, 54%) reflected the absence of own reward.

The most interesting of the three leading categories was, however, that consisting of activations explained by interaction of actor and own reward. In a social setting, it is crucial that an animal that receives reward would be able to distinguish whether this reward derives from its own action or from the action of a conspecific. Similarly, an animal that does not receive a reward should be able to distinguish whether this lack of reward is the result of its own action or the result of the action of a conspecific. All activations shown in the expanded view of Fig. S4*A*, namely those activations belonging to {Actor for Own Reward} but not {Actor} or {Own Reward}, reflected one or other or both of these pieces of information. Specifically, of the 95 activations in the expanded view of Fig. S4*A*, 44 reflected the actor for presence of own reward (but not the actor for absence of own reward), 30 reflected the actor for absence of own reward (but not the actor for presence of own reward), and 21 reflected both.

The 44 activations that reflected the interaction of actor and presence of own reward were further subdivided into those that occurred when the recorded animal was the agent and those that occurred when the conspecific was the agent. If the coefficient β_{13} of the regressor AW in Eq. **S13** was positive, this would indicate that the activation reflected the interaction of own action and presence of own reward. If it was negative, then the activation would reflect the interaction of conspecific's action and presence of own reward. In this sense, 22 activations reflected the interaction of own action and presence of own reward, and 22 activations reflected the interaction of conspecific's action and presence of own reward (Fig. S4*A*, *Inset*). It is striking that such a large number of activations reflected the interaction of conspecific's action and presence of own reward.

Similarly, the 30 activations that reflected the interaction of action and absence of own reward were further subdivided into those that occurred when the recorded animal was the agent and those that occurred when the conspecific was the agent. A positive coefficient β14 of the regressor $A(1 - W)$ in Eq. **S14** indicates the interaction of own action and absence of own reward. A negative coefficient β14 indicates the interaction of conspecific's action and absence of own reward. In total, 25 activations reflected the interaction of own action and absence of own reward and 5 activations reflected the interaction of conspecific's action and absence of own reward.

Fig. S4 *B–D* shows averaged histograms of the populations of striatal neurons reflecting the interaction of own action and presence of own reward (*B*), the interaction of conspecific's action and presence of own reward (*C*), and the interaction of own action and absence of own reward (*D*).

A comprehensive overview of the nature of the interaction between actor and own reward coding can be obtained by using a scatter plot with the coefficient β13 of actor for presence of own reward on the horizontal axis and the coefficient β14 of actor for absence of own reward on the vertical axis (Fig. S4*C*). On both axes, a positive direction indicates own action and a negative direction indicates conspecific's action.

Finally, moving outside the set {Actor for Own Reward}, a total of 34 of the 457 activations (6%) reflected conspecific's reward. Of these, 3 reflected conspecific's reward alone, 7 reflected the interaction of conspecific's reward and actor, 2 reflected the interaction of conspecific's reward and own reward, and the remaining 22 reflected all three explanatory variables.

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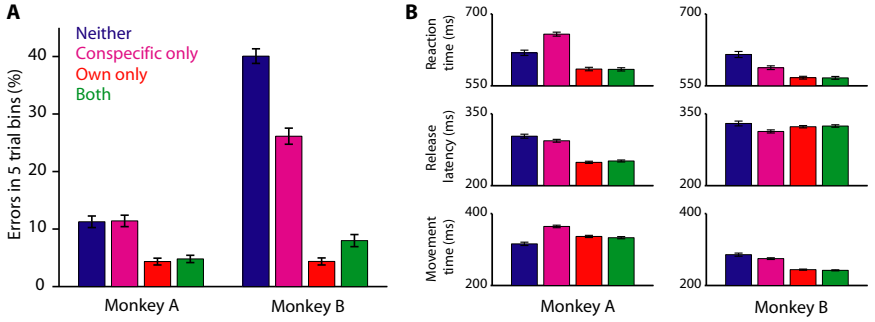

Fig. S1. Supplementary analysis of behavior. (*A*) Error rates per five trials. (*B*) Reaction time (RT) decomposed in release latency (RL) and movement time (MT) for monkeys A and B performing the task with each other. Monkey A had a higher social rank than monkey B. All bars are means ± SEM.

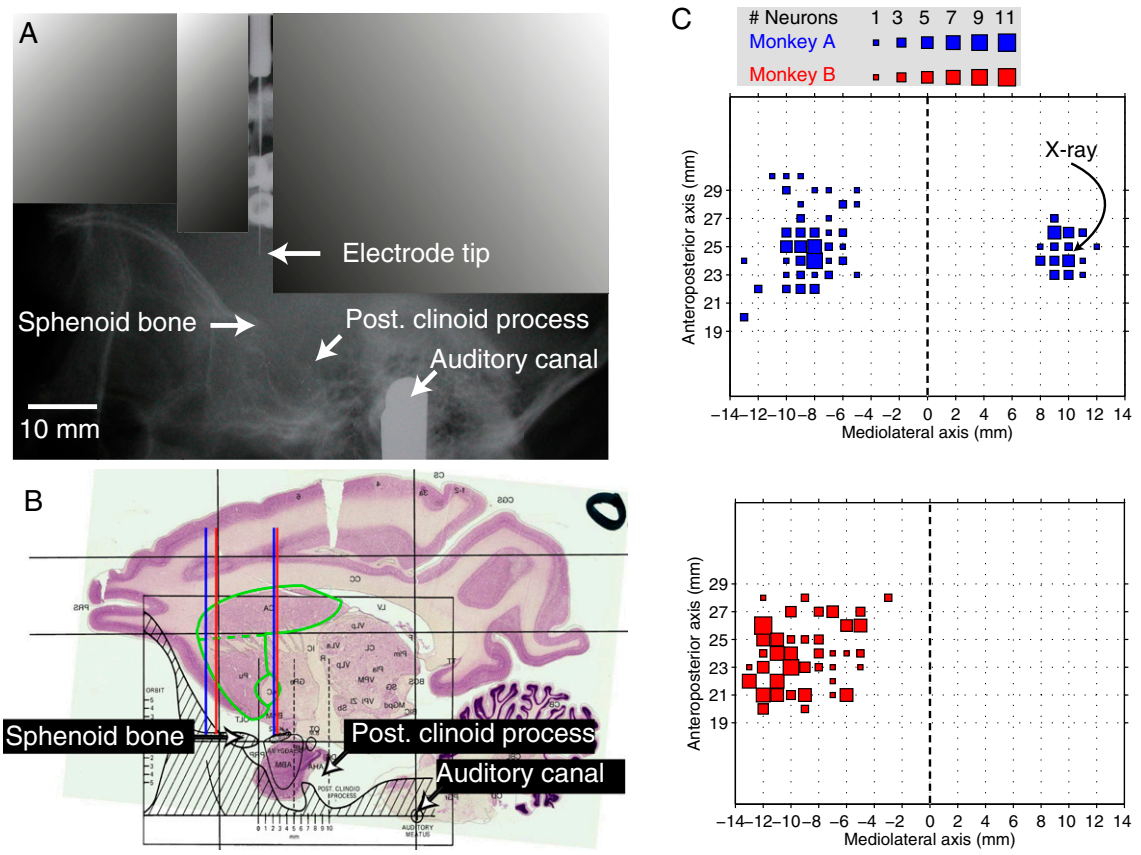

Fig. S2. Localization of recording sites in rhesus monkeys. (*A*) X-ray of lateral view of monkey A's brain with an electrode directed to the anterior striatum. (*B*) Composite figure of recording area in striatum. Colored vertical lines indicate anterior and posterior boundaries of recording area (monkey A, blue; monkey B, red). These lines overlaid on a Nissl-stained standard sagittal histological section from a rhesus monkey at +6 were obtained from brainmaps.org (12). Schematic black and white drawing of the base of the rhesus monkey skull was obtained from ref. 13, reproduced with permission. Our recordings in the striatum (green outline) were situated above the sphenoid bone and dorso-anterior to the amygdala (which is located between the sphenoid and posterior clinoid). All figure components are displayed at the same scale as the X-ray shown in *A*. (*C*) Surface view of recording tracks in monkey A (blue symbols) and monkey B (red symbols) in mediolateral and anteroposterior axes. Symbol sizes indicate numbers of neurons recorded in each track (left and right hemispheres of monkey A, left hemisphere of monkey B).

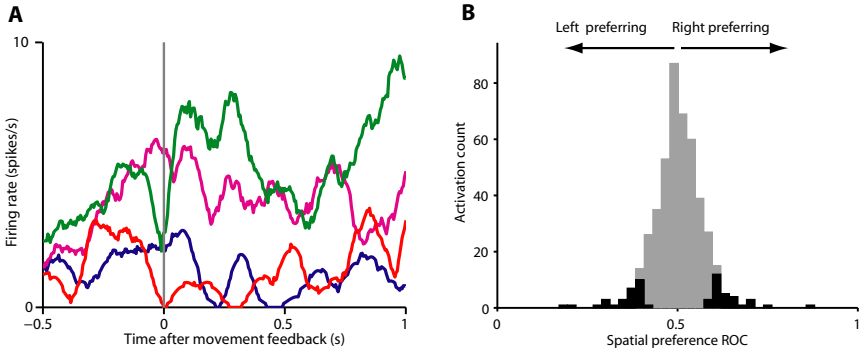

Fig. S3. Sensitivity of striatal neurons to conspecific's reward and spatial preferences. (*A*) Higher activations with conspecific's reward (purple-green) compared with own reward (red) and no reward (blue) in a single striatal neuron. Activations were high when only the conspecific animal received reward (purple) and when both animals received reward (green), and low when only the recorded monkey or nobody received reward (red and blue). This activation was the mirror image to that for own reward shown in Fig. 2*A*. (*B*) Black bars, activations showing significant spatial discrimination between left and right stimulus positions ($n = 56$; permutation test). Gray bars, insignificant activations ($n = 399$).

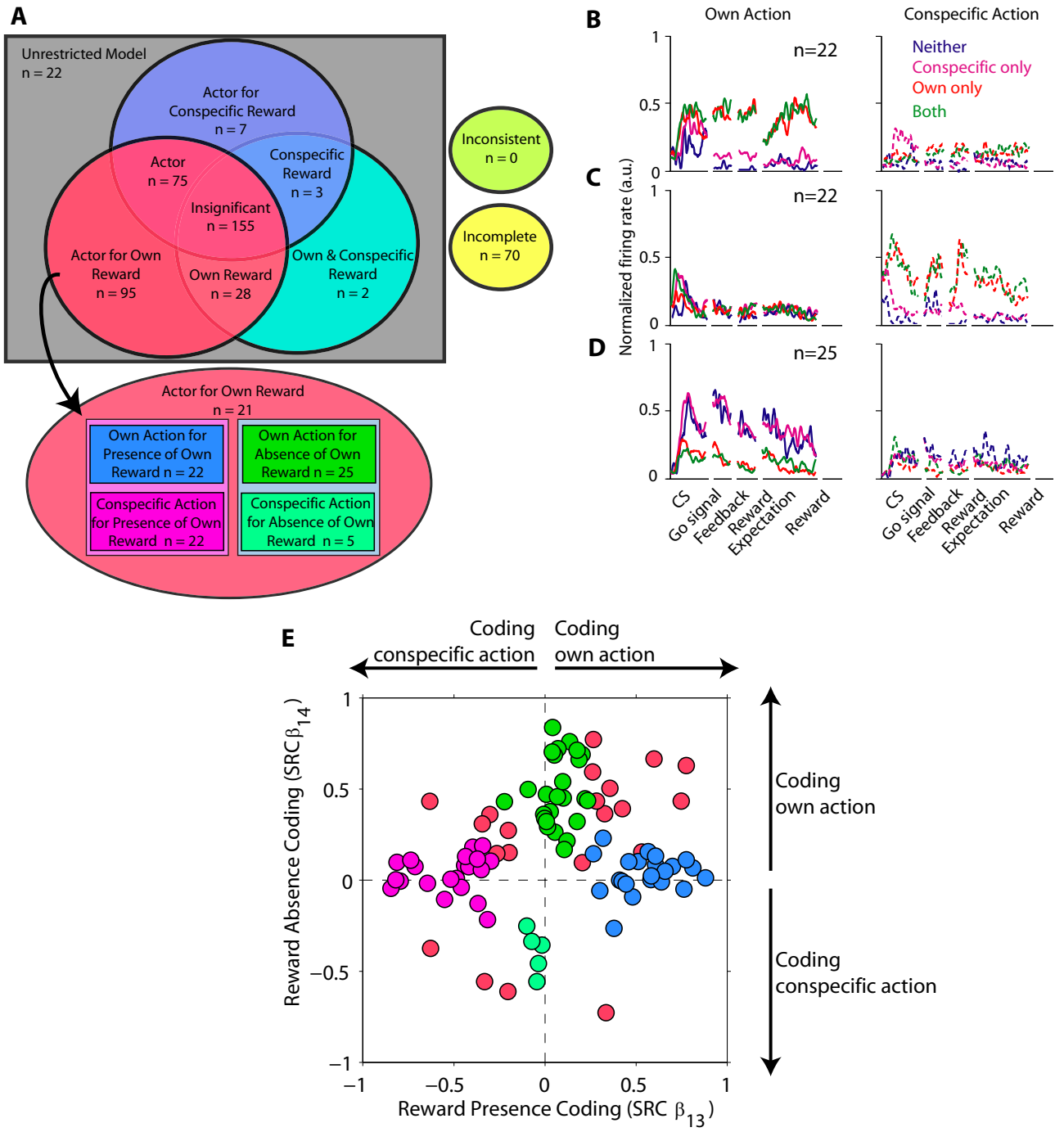

Fig. S4. Results from simultaneous testing procedure. (*A*) Venn diagram of the sets of interest and their intersections as defined by the simultaneous testing procedure. The arrow points to the diagram illustrating the dissection of the set containing neuronal activations reflecting the actor for own reward (red; $n = 95$ activations). The latter contained activations reflecting the interaction of actor and presence of own reward (*Left*, blue and purple; $n = 22 + 22$) and activations reflecting the interaction of actor and absence of own reward (*Right*, light and dark green; $n = 25 + 5$). Both sets were further split into own action coding (*Upper*, blue dark green; $n = 22 + 25$) and conspecific's action coding (*Lower*, purple and light green; $n = 22 + 5$). The remaining activations could not be unambiguously allocated to any of these four categories (*Upper*; $n = 21$). (*B–D*) Spike density function plots of the population of neuronal activations reflecting the actor for own reward. (*B*) Average activations reflecting the own action for presence of own reward. Notice that the neuronal activity was highest when the recorded monkey (solid lines vs. dashed lines) was the agent of its own reward (red and green vs. blue and purple). (*C*) Same as in *B* but for neuronal activations reflecting the conspecific's action (dashed lines vs. solid lines) for presence of own reward. (*D*) Same as in *B* but for neuronal activations reflecting the own action for absence of own reward. Notice that the activity was highest when the recorded monkey was the agent but did not receive a reward (blue and purple) compared with receiving a reward (red and green). (*E*) Standardized regression coefficients for activations reflecting the actor for own reward. Coefficients [β_{13}] of the regressor for actor for presence of own reward [*AW*] plotted against coefficients [β_{14}] of the regressor for actor for absence of own reward [$A(1 - W)$]. Coefficients with a positive sign indicate own action coding and coefficients with a negative sign indicate conspecific's action coding. Green, activations reflecting own action for presence of own reward ($n = 22$); light blue, activations reflecting conspecific's action for presence of own reward ($n = 22$); dark blue, activations reflecting own action for absence of own reward ($n = 25$); pink, activations reflecting conspecific's action for absence of own reward ($n = 5$); red, activations reflecting the actor for own reward ($n = 21$).

Table S1. Distribution of all activations divided by epoch and classification based on three-way ANOVA

Activation class		Epoch						
Reward	Actor	Before cue	During cue	Movement	Feedback	Reward expectation	Reward	Subtotal
Own	Own	0	20	20	11	15	1	67
Own	Conspecific	0	10	6	4	9	0	29
Own	None	0	7	10	10	4	9	40
Conspecific	Own	0	0	0	4	1	1	6
Conspecific	Conspecific	0	0	0	1	0	1	2
Conspecific	None	0	0	1	1	0	6	8
Both	Own	0	2	2	1	2	0	7
Both	Conspecific	0	3	2	4	0	0	9
Both	None	0	2	2	2	0	3	9
None	Own	12	6	18	10	4	7	57
None	Conspecific	12	4	3	2	1	1	23
Not significant		17	26	9	8	9	24	93
Incongruent		8	25	26	20	18	10	107

Table S2. Number of neuronal activations coding the statistical interaction of actor and reward or their conjunction based on three-way ANOVA

		Reward			
Actor	Type of coding	Own	Conspecific	Both	Subtotal
Own	Interaction	35 (30)	1 (1)	4 (4)	40 (34)
	Conjoint	32 (29)	5 (5)	3 (2)	40 (35)
Conspecific	Interaction	19 (16)	1 (1)	7 (6)	27 (21)
	Conjoint	10 (10)	1 (1)	2 (2)	13 (12)
Subtotal		96 (70)	8 (8)	16 (14)	120 (85)

Note that some neurons showed activations in multiple periods. Thus, in the column and row labeled 'subtotal' the number of activations is the sum across the row or column, but each neuron was only counted once. Number of neurons in parentheses.

Table S3. Numbers of neuronal activations coding reward and actor based on ROC analysis

	Reward				
Actor	Own	Conspecific	Both	None	Subtotal
Own	47 (10)	1 (<1)	23 (5)	87 (19)	158 (35)
Conspecific	14 (3)	2 (<1)	23 (5)	31 (7)	70 (15)
None	73 (15)	11 (2)	23 (5)	122 (27)	229 (50)
Subtotal	134 (29)	14 (3)	69 (15)	240 (52)	457 (100)

Percentage on each category in parentheses.

Table S4. Distribution of activations in neurons with selective saccade direction activity

	Reward				
Actor	Own	Conspecific	Both	None	Subtotal
Own	2	1	0	2	5
Conspecific	1	0	0	0	1
None	0	1	0	8	9
Subtotal	3	2	0	10	15