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# Social and asocial prefrontal cortex neurons: a new look at social facilitation and the social brain

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## Abstract

A fundamental aspect of behavior in many animal species is 'social facilitation', the positive effect of the mere presence of conspecifics on performance. To date, the neuronal counterpart of this ubiquitous phenomenon is unknown. We recorded the activity of single neurons from two prefrontal cortex regions, the dorsolateral part and the anterior cingulate cortex in monkeys as they performed a visuomotor task, either in the presence of a conspecific (Presence condition) or alone. Monkeys performed better in the presence condition than alone (social facilitation), and analyses of outcome-related activity of 342 prefrontal neurons revealed that most of them (86%) were sensitive to the performance context. Two populations of neurons were discovered: 'social neurons', preferentially active under social presence and 'asocial neurons', preferentially active under social isolation. The activity of these neurons correlated positively with performance only in their preferred context (social neurons under social presence; asocial neurons under social isolation), thereby providing a potential neuronal mechanism of social facilitation. More generally, the fact that identical tasks recruited either social or asocial neurons depending on the presence or absence of a conspecific also brings a new look at the social brain hypothesis.

**Key words:** associative learning; neurophysiology; monkey; social cognition

## Introduction

Recent years have witnessed a tremendous interest in understanding how the brain functions in social context, in particular when people or animals make decisions and behavioral choices (for reviews see Báez-Mendoza and Schultz, 2013; Ruff and Fehr, 2014; Watanabe and Yamamoto, 2015). Of particular interest here are studies demonstrating that prefrontal neurons can be selectively activated in relation with a conspecific's actions, errors and/or rewards (Yoshida *et al.*, 2011, 2012; Azzi *et al.*, 2012; Hosokawa and Watanabe, 2012, 2015), or social status (Fujii *et al.*, 2009; Azzi *et al.*, 2012). In these important studies, however, the conspecific was actively involved in the task, either as a partner or as a competitor. This leaves open the question of how the conspecific's presence, without any overt interaction, contributed to the neuronal modulations. More generally, little attention has been devoted to understanding how the mere presence of others, the most fundamental invariant of behavior in many, if not all, animal species affects neuronal activity.

This is surprising given the evidence accumulated for more than a century in social psychology demonstrating that the mere presence of conspecifics generally improves performance

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on easy or familiar tasks, an effect referred to as social facilitation (for reviews, see Zajonc, 1965; Bond and Titus, 1983; Guerin, 2009). According to Zajonc's (1965) classic view, social presence increases drive or arousal and facilitates dominant responses (considered to be correct on easy tasks thereby resulting in social facilitation). Although Zajonc's physiological assumption received little empirical support (e.g. Cacioppo and Petty, 1986), there is ample evidence with different animal species—from cockroaches to humans—that the mere presence of others increases dominant responses, whether correct or incorrect. When the dominant response is incorrect (difficult or novel tasks), social presence impairs performance, especially when the conspecifics are unfamiliar, unpredictable or threatening. Under these circumstances, the presence of others may also distract a significant portion of attentional resources away from task execution, as indicated by studies with humans and monkeys (Huguet *et al.*, 2014; Belletier *et al.*, 2015; see also Baron, 1986). Conversely, when the presence of others is familiar or reassuring, it may reduce stress, compared with social isolation (Stamm, 1961; Gunnar *et al.*, 1980; Cacioppo *et al.*, 2011). The non-threatening presence of others can also boost attentional control, with a positive effect on tasks requiring the inhibition of incorrect dominant response tendencies (Huguet *et al.*, 1999, 2004; Sharma *et al.*, 2010; Augustinova and Ferrand, 2012).

Despite the high interest devoted to social facilitation at the behavioral level, little is known about its neuronal correlates. As associative learning relies on feedback signals, i.e. rewards and error signals (Sutton and Barto, 1998), one way social presence may affect learning is through direct modulation of neuronal processing of these signals. Our prediction was that both behavioral performance and neuronal activity associated with feedback signals would be modulated by social presence/absence. To test this hypothesis, we recorded single neuron activity from the prefrontal cortex, which constitutes with the basal ganglia the core brain network mediating reinforcement-guided learning (see Passingham and Wise, 2012; Bissonette and Roesch, 2015). We targeted the dorsolateral (PFdl) and medial (anterior cingulate cortex, ACC) prefrontal regions where neuronal activity is known to process feedback signals during learning (Asaad *et al.*, 1998; Holroyd and Coles, 2002, 2008; Nieuwenhuis *et al.*, 2004; Amiez *et al.*, 2006; Kennerley *et al.*, 2006; Kobayashi *et al.*, 2006; Kennerley and Wallis, 2009). Although PFdl and ACC are connected with each other (Procyk *et al.*, 2014), their anatomical and functional properties suggest that they may be differentially modulated by social presence: PFdl is indeed strongly connected with sensory and motor systems, and is part of the attentional network (Corbetta and Shulman, 2002; Noudoost *et al.*, 2010; Miller and Buschman, 2013), whereas ACC is at the convergence of the emotional, cognitive and motor systems (Passingham *et al.*, 2010). As expected, we found that feedback-related neuronal activity in both areas was highly sensitive to social presence.

## Materials and methods

### Subjects

Two adult male Rhesus monkeys (*Macaca mulatta*) were subjects in this study. They were housed together since the age of 3 years, and weighted 8–12 kg at the time of the study. They had established stable and spontaneous social interactions, with monkey A being the dominant as revealed by 'access to water and food' test. Animal care, housing and experimental procedures conformed to the European Directive (2010/63/UE) on the use of nonhuman primates in scientific research. The two monkeys were maintained on a dry diet, and their liquid consumption and weight were carefully monitored.

### Behavioral procedures

The monkeys were trained to associate abstract images with targets on a touchscreen either under social presence or in isolation (Figure 1A and B). Under social presence, the two monkeys were sited in primate chairs facing each other, with their head immobilized (see 'Surgery' section), and alternated the roles of actor and spectator. Only the actor had access to the touchscreen, and thus performed the task and received rewards on correct trials. The spectator was not rewarded, had no incentive to produce any particular behavior, was never tested (as actor) during the same day, and when tested, a new set of stimuli was used (therefore preventing any observational learning to occur). When a monkey was tested in isolation, the other remained in the housing room located at a distance such that the actor was truly alone in the testing box, deprived from any communicative means with the conspecific through visual, auditory or olfactory channels.

During task performance, the actor started trials by touching a white rectangle, which triggered the presentation of a cue at the center of the screen. The monkey was required to indicate among the four white squares (targets), the one associated with this cue. After a variable delay (500–700 ms), the cue went off (go signal) and the monkey had to move the hand and touch the chosen target. If correct, a green circle (positive feedback) informed the monkey that the choice was correct, and a reward (fruit juice) was delivered after 1 s. If the choice was incorrect, a red circle signaled the error (negative feedback), and no reward was delivered.

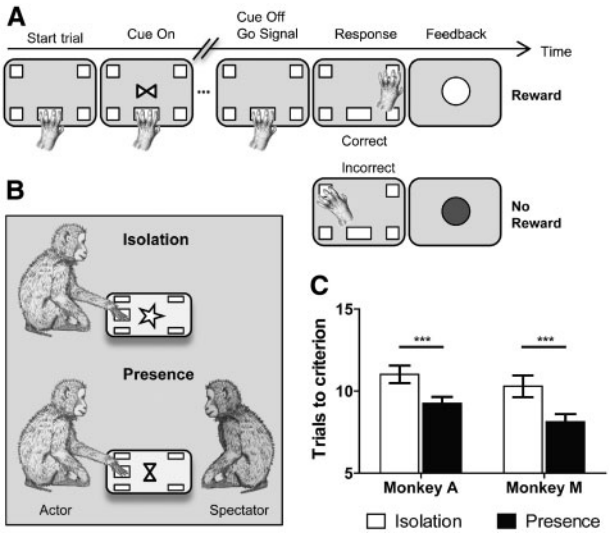


**Fig. 1.** Task design and behavior. (A) The successive grey frames represent the touch screen, as it appeared to the monkey, from the beginning (left) to the end of the trial. The white rectangle represents the lever and the four white squares represent the targets. A cue (white symbol) appeared at the screen center and the monkey was required to touch one of the targets. If correct, a green circle (white circle) appeared followed by a reward. If incorrect a red circle (grey circle) appeared with no reward. (B) Social performance contexts. The actor (bottom left) performed the task in presence of the conspecific (spectator) or in isolation (top) with different sets of cues. (C) Social facilitation of learning speed. Comparison of the number of trials needed to reach the learning criterion across the two performance contexts, for each monkey. The error bars represent one SEM. ***$P < 0.001$.

Typically, the monkeys were tested on a daily basis. A testing session (one per day) started with one monkey learning a set of two associations under isolation, then a second yet equivalent set of two associations under social presence (80 trials for each set). This fixed order was necessary for two reasons. First, the two monkeys were particularly reluctant to perform the task in isolation if this condition came after social presence, perhaps because the presence of a familiar conspecific reduces (or its removal increases) stress in learning contexts (Guerin, 2009). Second, there is evidence that the presence of others may still operate in isolation when this presence comes first (Markus, 1978), making the reversed order (presence then isolation) inappropriate (Guerin, 2009; Huguet *et al.*, 1999). Thus, the most appropriate control condition was a test–retest, where each monkey performed the task twice under isolation.

### Surgery

Surgery was performed under deep anesthesia and aseptic conditions, for implantation of a recording chamber over the hemisphere contralateral to the hand used to perform the task, and a bolt used to immobilize the head. Chamber implantation was guided by magnetic resonance imaging (MRI) scans of each monkey's brain obtained prior to surgery. A single chamber allowed access to the PFdl and ACC. The bone was removed from the area covered by the chamber, and the later was closed with a plastic cap, which was removed during the recording sessions.

### Neuronal recordings and data analysis

Extracellular neuronal activity was recorded using a multichannel system (Alphalab from Alpha Omega). Single tungsten electrodes (FHC Instrument, 0.8–1.2 MΩ impedance) were inserted in the brain, and were moved down to the target cortical areas identified on the basis of MRI scans. Up to four electrodes were used simultaneously, two in the PFdl and two in ACC, whose location within each area varied from session to session in order to cover the area as much as possible. The signals recorded by each electrode were high- (6 kHz) and low-pass (250 Hz) filtered, and amplified using Alphalab software. The neuronal signals were synchronized with task events (visual stimuli onset and offset, behavioral events, reward delivery timestamp) derived from the Cortex software (NIH, Bethesda, MD USA), and stored as analog signals for off-line analyses. A home-made Matlab toolbox was used off-line to process the data for spike sorting under MClust Spike sorting toolbox (A. David Redish, http://redishlab.neuroscience.umn.edu/MClust/MClust.html). Specifically, this step aimed to distinguish action potentials emitted by the same neurons and separate the activity of different neurons from one another and from noise. The isolated spike clusters were then processed using Neuroexplorer, and neurons were classified based on their response to the various task events. Here, we focused on neuronal activity extracted for epochs surrounding the onset of feedback (see Supplementary Figure S1), and normalized for statistical analyses. A Z score was computed for each bin (a 10 ms time window) using the following formula: $Z(\text{bin})_{i,j} = (\text{bin count})_{i,j} - \text{mean}(\text{bin count baseline})_i / \text{std}(\text{bin count baseline})_i$; where 'i' is the number of trials, 'j' the number of bins, '$(\text{bin count})_{i,j}$' the number of spikes in bin $j$ of trial $i$ during a given time window, 'mean (bin count baseline)' is the average baseline bin count, 'std(bin count baseline)' the standard deviation of the mean spike count relative to the baseline. This formula normalizes the spike counts in each bin to the baseline on a trial-by-trial basis, and produces normalized rasters in the form of Z scores, which then produces peri-stimulus time histograms (PSTHs). PSTHs were then used to examine the activity profile and identify neuronal categories. Three main parameters of the neuronal discharge were examined (latency, amplitude and duration), to determine the changes of activity in relation with the feedback signal. A neuron was considered to be active in relation with a particular event (e.g. feedback signal) if the Z score of the PSTH was higher than 1.96 (CI of 0.95) in at least three consecutive bins. The latency of the response was defined as the first occurrence of this value ($Z > 1.96$). Conversely, the end of the response was indicated by the first of three consecutive bins with a Z value $< 1.96$. Statistical analyses compared the neuronal activity in the two epochs across conditions (see Supplementary Figure S1 for the definition of epochs). A Mann-Whitney U-test ($P < 0.05$) was used to compare the activity of each neuron across conditions (isolation, presence), for the same time epoch, and thus determine whether neurons were preferentially active in one or the other condition.

## Results

### Behavior

Behavioral data were collected over a total of 258 sessions (monkey A, $n = 181$; monkey M, $n = 77$), and analyzed using the session as unit of analysis to determine whether social presence affected learning speed, i.e. the number of trials required to reach the learning criterion (defined as the third of five consecutive correct trials). Repeated measure ANOVAs revealed a main effect of presence on learning speed in both monkeys (Figure 1C). Social presence reduced the number of trials to criterion, compared with isolation (from 11.02 to 9.26 across sessions in monkey A, $F(1, 180) = 10.84$; $P < 0.001$, $\eta^2_p = 0.06$; from 10.29 to 8.12 across sessions in monkey M, $F(1, 76) = 10.97$, $P < 0.001$, $\eta^2_p = 0.13$), indicating a social facilitation effect in both monkeys (skewness values inferior to 1.96 in both conditions for both monkeys). This effect was also confirmed by a non-parametric test (Mann-Whitney U-test; $Ps < 0.05$).

Analyses of the test–retest (control) data showed that improved performance under social presence cannot be accounted for by task repetition, as the number of trials to criterion at retest did not decrease (quite the contrary for monkey A) relative to test (Figure 2). Furthermore, although both monkeys were reluctant to perform the task in the opposite order

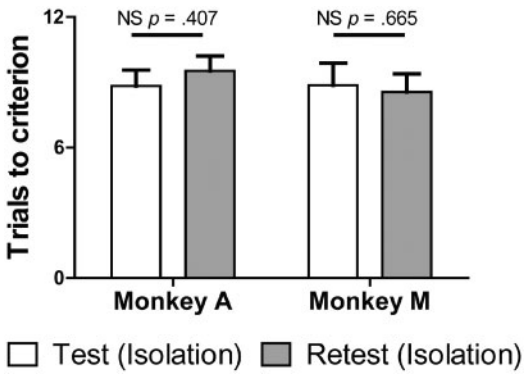


**Fig. 2.** The null effect of task repetition. Comparison of the number of trials needed to reach the learning criterion in test–retest isolation sessions for each monkey. The error bars represent one SEM. NS, non-significant.

(see 'Methods' section), we successfully collected additional control data over 11 sessions in monkey M in presence then in isolation. Analysis of the data showed that even in this opposite order, social presence had a significant positive effect on learning speed ($M$ presence = 5.82, s.d. = 0.58; $M$ isolation = 12.41, s.d. = 1.39; $F(1, 10) = 18.42$, $P < 0.002$, $\eta^2_p = 0.65$).

## Neural data

We recorded the activity of 592 single neurons in the PFdl ($n$ = 376) and ACC ($n$ = 216) of the two monkeys during task performance, and found that more than half of them were related to the processing of outcome signals (60% in PFdl, 53% in ACC). In each area, a given neuron increased its firing rate in at least one of two epochs surrounding the feedback signal (see Supplementary Figure S1 and 'Methods' section). Activations were analyzed in each area, and separately for positive and negative feedback-related signals (see Supplementary Table S1 for proportions). Because of similar profiles and proportions of outcome-related activations in PFdl and ACC, the data were pooled together in a single neuronal sample, hereafter referred to as prefrontal cortex neurons. Thus, the present analysis was based on a total of 502 feedback-related activations.

Social presence modulated outcome-related activity of a vast majority of prefrontal neurons, whether encoding errors (85%) or rewards (82%). We actually discovered two main categories of neurons depending on the modulation of their firing rate amplitude (categorization relying on Mann-Whitney U-tests based on the PSTHs). In one category, 'social neurons' (Figure 3A N1 and N2), the firing rate increased under social presence, compared with isolation. These neurons represented 41% and 38% of negative and positive feedback-related activations, respectively. In a second category, 'asocial neurons', the pattern was opposite, as these neurons were preferentially active during isolation (Figure 3A N3 and N4), and represented 41% and 47% of negative and positive feedback-related activations, respectively. Figure 3B shows the distribution of these activations for the entire neuronal sample in each performance context (presence, isolation). We tested further the validity of our neuronal categorization process into social and asocial categories by using a hierarchical, unsupervised clustering analysis. This analysis (see Supplementary Figure S2) confirmed the existence of two main categories of neurons (with finer grouping into slightly different sub-categories) corresponding, with very minor inter-classes overlap (<3%), to social and asocial categories reported based on the Mann-Whitney U-tests. Finally, social and asocial neurons were found in similar proportions in both prefrontal areas (PFdl and ACC), and histological reconstruction revealed no spatial segregation within individual areas (Figure 4).

## Correlations

As social presence modulated both behavior and neuronal activity, we tested whether they co-varied across sessions by computing Pearson's correlation coefficients. The finding that stands out most prominently is the significant correlations between the firing rate associated with negative—but not positive—feedback and behavior (Figure 5A). For social neurons, activity and trials to criterion correlated negatively under social presence ($r = -0.24$, $P < 0.02$): the higher the firing rate, the less trials needed (faster learning), suggesting that social facilitation may reflect largely the activity of social neurons. For asocial neurons, activity and trials to criterion also correlated

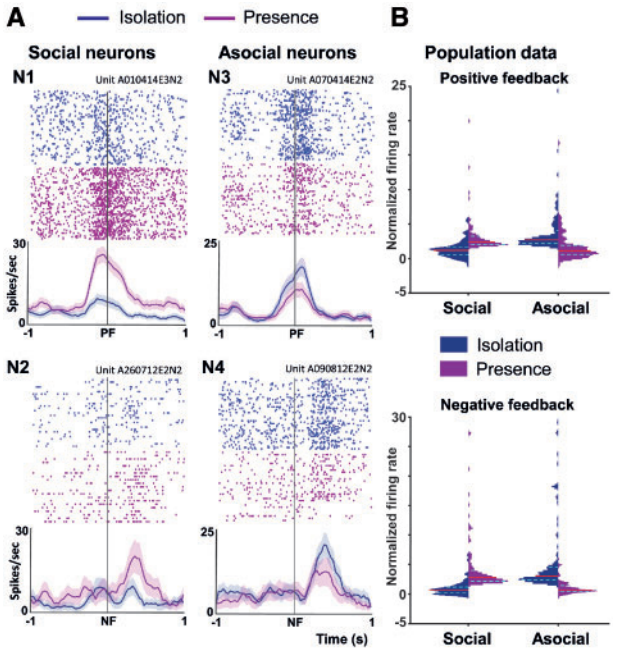


**Fig. 3.** Illustration of neuronal data. (A) N1–N4 are examples of social and asocial neurons recorded from the prefrontal cortex of monkey A. For each neuron, the activity is represented by two raster displays in different colors, blue for isolation, purple for social presence. Each raster is constructed following the usual standards: dots represent action potentials, and each line of dots represents the firing of the neuron over a trial. Trials of the same condition are aligned (the vertical line) on the onset of the feedback signal: PF for positive feedback, NF for negative feedback. Below the raster displays, the mean firing rate (spikes/sec) is represented in the form of PSTHs; the color code is the same as for the raster displays, and the shading represents the variability of the firing rate (mean standard error). (B) Violin plots. The plots show the distribution of neuronal outcome-related activations (firing rate) for positive (top) and negative feedback (bottom) in the two performance contexts (presence *vs* isolation), using the same color code as in A. The distribution of the activations is illustrated as probability density curves calculated using a kernel density estimation. The percentiles (lower and upper quartiles in light blue dotted lines) and the median (red lines) of the data are also illustrated for each performance context.

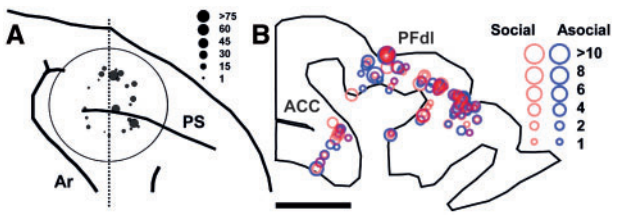


**Fig. 4.** Recording sites. (A) Location of the recording chamber (circle) and recording sites shown on the lateral view of the right frontal portion of the brain. (B) Recording sites of social (pink) and asocial (blue) neurons shown on a coronal section taken at the center of the chamber (dashed vertical line in A). Intermediate colors are due to overlap of the initial colors. In both A and B the diameter of circles represents the number of activations analyzed. Abbreviations: ACC, anterior cingulate cortex; Ar, Arcuate sulcus; PFdl, dorsolateral prefrontal cortex; PS, principal sulcus. The bar scale represents 10 mm for A, 5 mm for B.

negatively but in isolation ($r = -0.22$, $P < 0.02$). Perhaps the most striking result is that these correlations were systematically and significantly reversed for both categories of neurons in the two other conditions: activity of social neurons now correlated positively with trials to criterion in isolation ($r = 0.46$, $P < 0.001$): the higher the firing rate, the *more* trials needed (slower learning); whereas activity of asocial neurons tended to

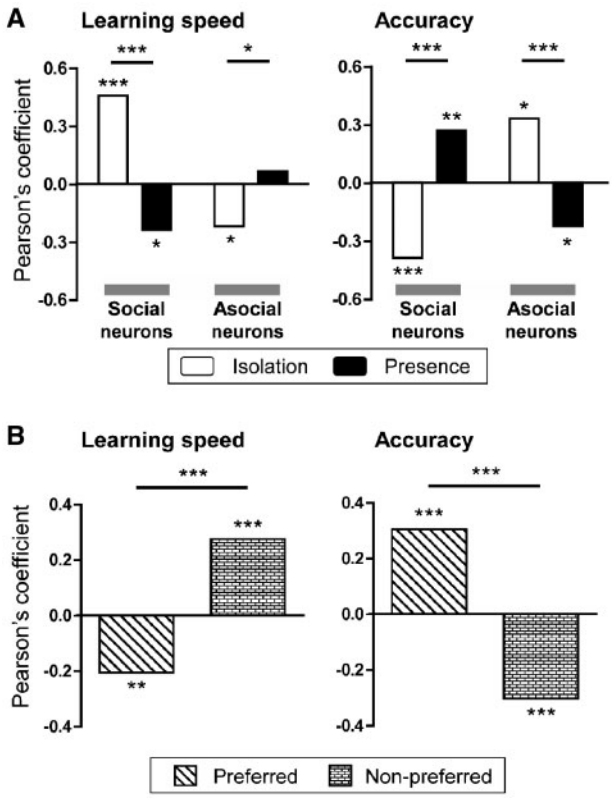


**Fig. 5.** Correlation between neuronal activity and behavior. Pearson's correlation coefficient (*r*) is shown for the analysis of the relationships between the amplitude of firing rate related to negative feedback and the number of trials to criterion (learning speed) on one hand, and the percentage of correct responses (accuracy) on the other hand. (A) For each category of neurons (social *vs* asocial), each graph shows the correlation coefficient for isolation (open bars) and social presence (dark bars) condition. (B) Illustration of the pattern of correlations shown in Figure 5A, between neuronal activity of social and asocial populations of neurons pooled together by preferred social context, and learning speed and accuracy. Note that, under the preferred context (dashed symbol) the higher the firing rate, the less trials needed to reach the learning criterion ($r = -.21$, $P < 0.002$), and the better the accuracy ($r = 0.31$, $P < 0.001$). Conversely, in the non-preferred context (dotted symbol), the higher the firing rate, the more trials needed to reach the learning criterion ($r = 0.28$, $P < 0.001$) and the worse the accuracy ($r = -0.30$, $P < 0.001$). Horizontal lines above graphs depict comparisons between *r* values across conditions.*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

correlate positively with trials to criterion under social presence ($r = 0.07$, *ns*). Consistent with this pattern on learning speed, neuronal activity and performance accuracy correlated positively (the higher firing rate, the better accuracy) for social neurons under social presence ($r = 0.27$, $P < 0.005$) and asocial neurons under isolation ($r = 0.33$, $P < 0.001$). Again, these correlations reversed systematically and significantly for both categories of neurons in the two other conditions: neuronal activity now correlated negatively with performance accuracy (the higher firing rate, the worse accuracy) for social neurons under isolation ($r = -0.39$, $P < 0.001$) and asocial neurons under social presence ($r = -0.22$, $P < 0.02$). In other words, error-related activity (but not success-related activity, see Supplementary Table S2) of both social and asocial neurons correlated with learning speed and accuracy, but in opposite directions depending on the social context of performance (social presence *vs* social isolation). Figure 5B summarizes this pattern of correlations depending on the neurons' preferred context (social neurons under social presence, asocial neurons under social isolation) and non-preferred context (social neurons under social isolation, asocial neurons under social presence).

All the present findings were robust to changes in parameters such as learning criterion (from five to nine consecutive correct trials), or thresholds (three to five temporal bins) used to consider a neuron to be active in relation with feedback signals. For the few neutral neurons found with the discriminant analysis, the relationships between firing rate and learning speed or performance accuracy were not significant (neither under social isolation nor under social presence), however the limited number of these neurons did not allow conclusive statistical analyses.

## Discussion

This study reveals that the mere presence of a familiar conspecific modulates the activity of a vast majority of learning-related prefrontal neurons, whether encoding errors or rewards. For exactly the same task, some outcome-related neurons operate more actively under social presence (social neurons), whereas others are more active in isolation (asocial neurons). These neuronal properties complement earlier findings demonstrating that prefrontal neurons can be selectively activated in relation with the conspecific's actions, errors and/or rewards (Yoshida *et al.*, 2011, 2012; Azzi *et al.*, 2012; Hosokawa and Watanabe, 2012, 2015), or social status (Fujii *et al.*, 2009; Azzi *et al.*, 2012). The question remained, however, whether the 'mere' presence of others, a fundamental aspect of social life, also modulates neuronal activity. In this study, one may argue that the conspecific was not completely passive and may have emitted positive or negative cues through vocalization, gestures, mimicry or eye contact. This is unlikely to account for the strong effects observed, given the conditions of our experiment, which contrasts clearly with the above studies where the partners either interacted overtly or behaved in an interdependent way (e.g. rewards for the self *vs* others), and were generally present in all conditions, preventing the study of mere presence effects. Conversely, we have not addressed the issue of how modulations by the mere presence of others would change if the conspecific had an active role in task performance, nor do we have any evidence that a non-familiar spectator would lead to the same or different results as those reported here. However, taken together, our findings provide the first evidence that a large proportion of outcome-related prefrontal neurons are highly sensitive to the mere presence or absence of familiar conspecifics.

Our findings also suggest that the same task can be learned using different neuronal populations depending on the social *vs* asocial nature of the performance context. Consistent with this suggestion, and possibly the key finding of this work, it appears that the relationship between behavior and error-related neuronal activity strongly depends on whether the neuronal populations at hand are compatible or incompatible with the presence/absence of a familiar conspecific. As a matter of fact, this relationship was systematically reversed when the neurons discharged in their non-preferred performance context, suggesting a functional antagonism: the higher firing rate in the non-preferred context, the worse the behavior. This leads to the tentative conclusion that optimal performance requires the recruitment of appropriate neurons not only based on task features, but also on the social context (social neurons under social presence, asocial neurons under social isolation), at least on learning tasks where the probability of producing errors is high. According to this conclusion, this 'neuron-context

compatibility' relationship should be less relevant for well-learned tasks, but this aspect remains to be investigated in future research.

We still lack direct evidence on causal relationships between neuronal activity and behavior, on how social and asocial neurons interact during learning, how social presence might modulate these interactions, and how attention might have contributed to the present effects. There is ample evidence that the mere presence of others can boost attentional processes with beneficial effects on task performance (for reviews see Baron, 1986; Huguet *et al.*, 2000), as also suggested by a recent PET study in the monkey (Monfardini *et al.*, 2015) where the presence of a familiar conspecific led to a greater activation of the attentional network. Although we did not control for attention, the fact that social presence facilitated learning is consistent with increased attentional resources dedicated to the focal task. Socially facilitated learning in our study is also consistent with past research demonstrating the beneficial effects of familiar conspecifics that generally reduce stress in subjects facing new tasks, contrasting with the deleterious effects of non-familiar conspecifics (Guerin, 2009). More generally, it is possible that social *vs* asocial neurons are actually best described by a factor only incidentally associated with the presence or absence of a familiar conspecific. For example, could the differences in neuronal activity of social and asocial neurons simply reflect different levels of stress (higher stress under isolation *vs* lower stress under social presence of a familiar conspecific)? Although stress may have contributed to the differences in firing rates of social and asocial neurons, we believe that this factor alone cannot explain the present results. If social neurons simply reflected 'lower level of stress', their proportion should have increased (and the proportion of asocial neurons decreased) over time throughout the experiment. However, the proportions of social and asocial neurons were relatively stable over time (see Supplementary Figure S3). Likewise, one may wonder whether the two populations reflect different error strategies that the animals' may adopt under social presence *vs* isolation. There is indeed evidence, e.g. that monkeys (like humans) voluntarily regulate their performance given that errors may be especially costly to make in front of a conspecific (Drea and Wallen, 1999). In this case, errors led to reward suppression and so were costly in both conditions (alone *vs* presence), but possibly more costly under social presence than in isolation. Nevertheless, whatever the exact psychological processes involved, it remains that the findings reported here shed light on the existence of neurons in the prefrontal cortex whose activity encodes outcomes in a manner that is dependent on social presence.

The fact that the activity of the two neuronal populations correlates with behavior but in opposite directions in their non-preferred contexts suggests a dynamic interplay determining learning under social presence/isolation: social neurons under social presence may somehow inhibit (directly or indirectly) asocial neurons, thereby producing social facilitation. Conversely, asocial neurons under social isolation may inhibit social neurons, with a beneficial effect on learning. This reciprocal inhibition, if any, is only partial however as neurons may also discharge in their non-preferred context, although to a much lesser extent. In the non-preferred context, the neuronal firing seems to interfere with behavior as suggested by the reversed relationships found between neuronal activity and learning. These dynamic interactions between social and asocial neurons may take place not only locally within each of the two prefrontal areas investigated here, but also across the two areas through cortico-cortical connections (Barbas and Pandya, 1989; Paus, 2001; Constantinidis and Procyk, 2004; Medalla and Barbas, 2012), and most likely across a larger network that encompasses the so-called social brain (Blakemore, 2008; Rushworth *et al.*, 2013), including the amygdala, the orbitofrontal cortex, the striatum (Báez-Mendoza *et al.*, 2013), the superior temporal sulcus, and the insula (Perrett *et al.*, 1984, 1985a, b; Báez-Mendoza and Hoffman, 2009).

Finally, and again regardless of the exact underlying mechanisms, these findings suggest a new conception of the social brain. A recurrent debate about the social brain is whether identical, different, or partly overlapping neuronal representations support the processing of social and non-social information (see for a review Ruff and Fehr, 2014). However, this debate neglects the possibility that the processing of both types of information, and the control of behavior, be it social or not, may rely on relatively distinct neuronal populations within the same brain area depending on basic features of the social context of performance (for evidence that the social context indeed impacts the processing of both social and non social information, see Monteil and Huguet, 1999). As indicated by our findings, even non-social tasks can recruit social or asocial neurons depending on whether conspecifics are present or absent during task performance. Social and asocial neurons such as those reported here could be ubiquitous in the brain, but cannot be detected by neuroimaging techniques typically used in social neurosciences in humans (Decety and Cacioppo, 2011; Ruff and Fehr, 2014) and nonhuman primates (e.g. Monfardini *et al.*, 2015). Although these techniques, particularly fMRI, proved useful to identify brain networks engaged in various social cognitive functions, they do not allow a detailed breakthrough such as the one provided here using single unit recordings. It is possible that the proportions of social and asocial neurons vary from one brain region to another, and that the regions identified by neuroimaging techniques as supporting the social brain (Adolphs, 2009) actually reflect higher proportions of social neurons. These neurons may yet be present in many regions outside the social brain and operate efficiently under social circumstances, such that a variety of perceptual, cognitive, emotional and motor tasks would be modulated by the social context.

## Supplementary data

Supplementary data are available at *SCAN* online.

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## References

Adolphs, R. (2009). The social brain: neural basis of social knowledge. *Annual Review of Psychology*, **60**, 693–716.

Amiez, C., Joseph, J.P., Procyk, E. (2006). Reward encoding in the monkey anterior cingulate cortex. *Cerebral Cortex*, **16**(7), 1040–55. DOI: 10.1093/cercor/bhj046.

Asaad, W.F., Rainer, G., Miller, E.K. (1998). Neural activity in the primate prefrontal cortex during associative learning. *Neuron*, **21**/(6),1399–407.

Augustinova, M., Ferrand, L. (2012). The influence of mere social presence on Stroop interference: New evidence from the semantically-based Stroop task. *Journal of Experimental Social Psychology*, **48**, 1213–6.

Azzi, J.C.B., Sirigu, A., Duhamel, J.-R. (2012). Modulation of value representation by social context in the primate orbitofrontal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, **109**(6),2126–31.

Báez-Mendoza, R., Harris, C.J., Schultz, W. (2013). Activity of striatal neurons reflects social action and own reward. *Proceedings of the National Academy of Sciences of the United States of America*, **110**(41): 16634–9.

Báez-Mendoza, R., Hoffman, K.L. (2009). Object ontology in temporal lobe ensembles. In: Jenkin, M., Harris, L., editors. *Cortical Mechanisms of Vision*, 1st edn, pp. 237–53. Cambridge, UK: Cambridge University Press.

Báez-Mendoza, R., Schultz, W. (2013). The role of the striatum in social behavior. *Frontiers in Neuroscience*, **7**(7), 1–14.

Barbas, H., Pandya, D.N. (1989). Architecture and intrinsic connections of the prefrontal cortex in the rhesus monkey. *The Journal of Comparative Neurology*, **286**(3), 353–75.

Baron, R.S. (1986). Distraction-conflict theory: progress and problems. *Advances in Experimental Social Psychology*, **19**, 1–39.

Belletier, C., Davranche, K., Tellier, I.S., *et al*. (2015). Choking under monitoring pressure: being watched by the experimenter reduces executive attention. *Psychonomic Bulletin and Review*, **22**, 1410–6. DOI: 10.3758/s13423-015-0804-9.

Bissonette, G.B., Roesch, M.R. (2015). Neurophysiology of reward-guided behavior: correlates related to predictions, value, motivation, errors, attention, and action. *Current Topics in Behavioral Neurosciences*, **27**, 199–230.

Blakemore, S.-J. (2008). The social brain in adolescence. *Nature Reviews. Neuroscience*, **9**(4), 267–77.

Bond, C.F., Titus, L.J. (1983). Social facilitation: a meta-analysis of 241 studies. *Psychological Bulletin*, **94**(2), 265–92.

Cacioppo, J.T., Hawkley, L.C., Norman, G.J., Berntson, G.G. (2011). Social isolation. *Annals of the New York Academy of Sciences*, **1231**(1), 17–22.

Cacioppo, J.T., Petty, R.E. (1986). Social processes. In: Coles, M.G.H., Donchin, E., Porges, S., editors. *Psychophysiology: Systems, Processes, and Applications*, pp. 646–79. New York: Guilford Press.

Constantinidis, C., Procyk, E. (2004). The primate working memory networks. *Cognitive, Affective, and Behavioral Neuroscience*, **4**(4), 444–65.

Corbetta, M., Shulman, G.L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews. Neuroscience* **3**, 201–15.

Decety, J., Cacioppo, J.T. (2011). *The Oxford Handbook of Social Neuroscience*. In: Cacioppo, J.T., Decety, J., editors. New York: Oxford University Press.

Drea, C.M., Wallen, K. (1999). Low-status monkeys 'play dumb' when learning in mixed social groups. *Proceedings of the National Academy of Sciences of the United States of America*, **96**(22), 12965–9.

Fujii, N., Hihara, S., Nagasaka, Y., Iriki, A. (2009). Social state representation in prefrontal cortex. *Social Neuroscience*, **4**(1), 73–84.

Guerin, B. (2009). *Social Facilitation*. Cambridge, UK: Cambridge University Press.

Gunnar, M.R., Gonzalez, C.A., Levine, S. (1980). The role of peers in modifying behavioral distress and pituitary-adrenal response to a novel environment in year-old rhesus monkeys. *Physiology and Behavior*, **25**(5), 795–8.

Holroyd, C.B., Coles, M.G.H. (2002). The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, **109**(4), 679–709.

Holroyd, C.B., Coles, M.G.H. (2008). Dorsal anterior cingulate cortex integrates reinforcement history to guide voluntary behavior. *Cortex*, **44**(5), 548–59. DOI: 10.1016/j.cortex.2007.08.013.

Hosokawa, T., Watanabe, M. (2012). Prefrontal neurons represent winning and losing during competitive video shooting games between monkeys. *Journal of Neuroscience*, **32**(22), 7662–71.

Hosokawa, T., Watanabe, M. (2015). Egalitarian reward contingency in competitive games and primate prefrontal neuronal activity. *Frontiers in Neuroscience*, **9**, 1–10.

Huguet, P., Barbet, I., Belletier, C., Monteil, J., Fagot, J. (2014). Cognitive control under social influence in baboons. *Journal of Experimental Psychology: General* **143**(6), 2067–73.

Huguet, P., Dumas, F., Monteil, J.-M. (2004). Competing for a desired reward in the Stroop task: when attentional control is unconscious but effective versus conscious but ineffective. *Canadian Journal of Experimental Psychology*, **58**(3), 153–67.

Huguet, P., Galvaing, M.P., Dumas, F., Monteil, J.M. (2000). The social influence of automatic responding: controlling the uncontrollable. In: Forgas, J.P., Williams, K.D., Wheeler, I., editors. *The Social Mind: cognitive and Motivational Aspects of Interpersonal Behavior*, pp. 371–88. Cambridge, UK: Cambridge University Press.

Huguet, P., Galvaing, M.P., Monteil, J.M., Dumas, F. (1999). Social presence effects in the Stroop task: further evidence for an attentional view of social facilitation. *Journal of Personality and Social Psychology*, **77**(5), 1011–25.

Kennerley, S.W., Wallis, J.D. (2009). Evaluating choices by single neurons in the frontal lobe: outcome value encoded across multiple decision variables. *European Journal of Neuroscience*, **29**(10), 2061–73.

Kennerley, S.W., Walton, M.E., Behrens, T.E.J., Buckley, M.J., Rushworth, M.F.S. (2006). Optimal decision making and the anterior cingulate cortex. *Nature Neuroscience*, **9**(7), 940–7.

Kobayashi, S., Nomoto, K., Watanabe, M., Hikosaka, O., Schultz, W., Sakagami, M. (2006). Influences of rewarding and aversive outcomes on activity in macaque lateral prefrontal cortex. *Neuron*, **51**(6), 861–70.

Markus, H. (1978). The effect of mere presence on social facilitation: an unobtrusive test. *Journal of Experimental Social Psychology*, **14**(4), 389–97.

Medalla, M., Barbas, H. (2012). The anterior cingulate cortex may enhance inhibition of lateral prefrontal cortex via m2 cholinergic receptors at dual synaptic sites. *Journal of Neuroscience*, **32**(44), 15611–25.

Miller, E.K., Buschman, T.J. (2013). Corticol circuits for the control of attention. *Current Opinion in Neurobiology* **23**(2), 216–22.

Monfardini, E., Redoute, J., Hadj-Bouziane, F., *et al.* (2015). Others' sheer presence boosts brain activity in the attention (but not the motivation) network. *Cerebral Cortex*, **26**(6), 2427–39. DOI: 10.1093/cercor/bhv067.

Monteil, J.M., Huguet, P. (1999). *Social Context and Cognitive Performance: Towards a Social Psychology of Cognition*. Hove, East Sussex: Psychology Press.

Nieuwenhuis, S., Holroyd, C.B., Mol, N., Coles, M.G.H. (2004). Reinforcement-related brain potentials from medial frontal cortex: origins and functional significance. *Neuroscience and Biobehavioral Reviews*, **28**(4), 441–8.

Noudoost, B., Chang, M.H., Steinmetz, N.A., Moore, T. (2010). Top-down control of visual attention. *Current Opinion in Neurobiology*, **20**, 183–90.

Passingham, R.E., Bengtsson, S.L., Lau, H.C. (2010). Medial frontal cortex: from self-generated action to reflection on one's own performance. *Trends in Cognitive Sciences*, **14**(1), 16–21.

Passingham, R.E., Wise, S.P. (2012). *The Neurobiology of the Prefrontal Cortex*. Oxford: Oxford University Press.

Paus, T. (2001). Primate anterior cingulate cortex: where motor control, drive and cognition interface. *Nature Reviews. Neuroscience*, **2**(6), 417–24.

Perrett, D.I., Smith, P.A.J., Mistlin, A.J., *et al.* (1985a). Visual analysis of body movements by neurones in the temporal cortex of the macaque monkey: a preliminary report. *Behavioral Brain Research*, **16**, 153–70.

Perrett, D.I., Smith, P.A.J., Potter, D.D., Mistlin, A.J., Head, A.S., Milner, A.D. (1985b). Visual cells in the temporal cortex sensitive to face view and gaze direction. *Proceedings of the Royal Society*, **223**(1232), 293–317.

Perrett, D.I., Smith, P.A., Potter, D.D., *et al.* (1984). Neurones responsive to faces in the temporal cortex: studies of functional organization, sensitivity to identity and relation to perception. *Human Neurobiology*, **3**(4), 197–208.

Procyk, E., Wilson, C.R.E., Stoll, F.M., Faraut, M.C.M., Petrides, M., Amiez, C. (2014). Midcingulate motor map and feedback detection: converging data from humans and monkeys. *Cerebral Cortex*, **26**(2), 467–76. DOI: 10.1093/cercor/bhu213.

Ruff, C.C., Fehr, E. (2014). The neurobiology of rewards and values in social decision making. *Nature Reviews. Neuroscience*, **15**(8), 549–62.

Rushworth, M.F.S., Mars, R.B., Sallet, J. (2013). Are there specialized circuits for social cognition and are they unique to humans?. *Current Opinion in Neurobiology*, **23**(3), 436–42.

Sharma, D., Booth, R., Brown, R., Huguet, P. (2010). Exploring the temporal dynamics of social facilitation in the Stroop task. *Psychonomic Bulletin and Review*, **17**(1), 52–8.

Stamm, J.S. (1961). Social facilitation in monkeys. *Psychological Reports*, **8**(3), 479–84.

Sutton, R., Barto, A. (1998). *Reinforcement Learning: An Introduction*. Cambridge: MIT Press.

Watanabe, N., Yamamoto, M. (2015). Neural mechanisms of social dominance. *Frontiers in Neuroscience* **9**, 154.

Yoshida, K., Saito, N., Iriki, A. (2011). Representation of others ' action by neurons in monkey medial frontal cortex. *Current Biology*, **21**(3),249–53.

Yoshida, K., Saito, N., Iriki, A., Isoda, M. (2012). Social error monitoring in macaque frontal cortex. *Nature Neuroscience*, **15**(9), 1307–12.

Zajonc, R.B. (1965). Social facilitation. *Science*, **149**(3681), 269–74.