Beta2 oscillations (23–30 Hz) in the mouse hippocampus during novel object recognition

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Abstract

The oscillatory activity of hippocampal neuronal networks is believed to play a role in memory acquisition and consolidation. Particular focus has been given to characterising theta (4–12 Hz), gamma (40–100 Hz) and ripple (150–250 Hz) oscillations. Beyond these well-described network states, few studies have investigated hippocampal beta2 (23–30 Hz) activity in vivo and its link to behaviour. A previous study showed that the exploration of novel environments may lead to the appearance of beta2 oscillations in the mouse hippocampus. In the present study we characterised hippocampal beta2 oscillations in mice during an object recognition task. We found prominent bursts of beta2 oscillations in the beginning of novel exploration sessions (four new objects), which could be readily observed by spectral analysis and visual inspection of local field potentials. Beta2 modulated hippocampal but not neocortical neurons and its power decreased along the session. We also found increased beta2 power in the beginning of a second exploration session performed 24 h later in a slightly modified environment (two new, two familiar objects), but to a lesser extent than in the first session. However, the increase in beta2 power in the second exploration session became similar to the first session when we pharmacologically impaired object recognition in a new set of experiments performed 1 week later. Our results suggest that hippocampal beta2 activity is associated with a dynamic network state tuned for novelty detection and which may allow new learning to occur.

Introduction

The relation between neuronal oscillations and behaviour has been the focus of many studies over recent decades. Although also detected at the single-neuron level (Kamondi et al., 1998), neuronal oscillations are typically studied at the mesoscopic scale of local field potentials (LFPs; Buzsaki & Draguhn, 2004; Buzsaki, 2006), which represent the activity of an ensemble of nearby neurons (Buzsaki, 2004; Linden et al., 2011; Buzsaki et al., 2012; Reimann et al., 2013). In the hippocampus, many studies have focused on the role of theta (4–12 Hz; Vanderwolf, 1969; Winson, 1978; Buzsaki, 2002), gamma (30–100 Hz; Csicsvari et al., 2003; Montgomery & Buzsaki, 2007; Colgin et al., 2009), high-frequency (110–160 Hz; Scheffer-Teixeira et al., 2012; Tort et al., 2013) oscillations, and sharp-wave associated ripples (150–250 Hz; Buzsaki et al., 1992; Girardeau et al., 2009; Ego-Stengel & Wilson, 2010), in different behaviours and cognitive states. In addition, Berke et al. (2008) have called attention to prominent bursts of 23- to 30-Hz oscillations that appear in CA1 and CA3 when mice explore novel environments. This rhythm, referred to as beta2 oscillations, has been shown to modulate hippocampal neurons and to depend on NMDA transmission (Berke et al., 2008), which is known to be important for rapid learning (Nakazawa et al., 2003). Grossberg (2009) suggested that beta2 oscillations provide a transient plasticity signal able to solve the stability–plasticity dilemma, by which the ability of a neuronal network to learn rapidly must be compatible with stable memory representations without catastrophic forgetting (Grossberg, 1980, 1999, 2009).

The beta2 oscillations reported in Berke et al. (2008) are visible in unfiltered LFPs and, actually, may have amplitude similar to that of theta oscillations. This is by itself a remarkable finding considering that hippocampal oscillations constitute a major focus of research: why have such large-amplitude oscillations not been described before? And, to the best of our knowledge, why has there not been a second report showing similar hippocampal oscillations? Would the putative appearance of beta2 only during specific behaviours account for the fact that these oscillations remained undetected for so long? If so, what kinds of behaviours elicit beta2 oscillations? Are they specific to novel spatial experiences? Can amnesic interventions modulate beta2 appearance in the hippocampus?

In the present study we sought to confirm and extend the original findings of Berke et al. (2008). By recording from freely moving mice, we found that the exploration of novel objects leads to the transient appearance of prominent beta2 oscillations; no such activity occurred when animals were recorded in the home cage before survival.
and after the exploration session. Furthermore, beta2 power depended on the number of novel objects present in the arena, and pharmacologically blocking object memory consolidation was associated with higher beta2 power than when animals showed normal recognition of familiar objects. These results provide support to the proposal that hippocampal beta2 oscillations could play a role in novelty detection and may constitute a signal for new learning to occur (Berke et al., 2008; Grossberg, 2009).

Materials and Methods

Animals

Five C57BL/6 mice were used in this study. Animals were housed individually, with a 12-h cycle of light and dark (lights on at 06.00 h), and no food or water restriction. All procedures followed guidelines of the National Institutes of Health and were approved by the Edmond and Lily Safra International Institute of Neuroscience of Natal Ethics Committee (protocol number 08/2010).

Task design

Animals were submitted to a novel object recognition task. Each experiment (saline or haloperidol protocol; see below) consisted of two sessions 24 h apart with three periods each: pre-exploration (home cage), object exploration (open field), and post-exploration (home cage; Fig. 1A).

In the first session, four objects (A, B, C and D) were presented for 10 min during the exploration period in a circular arena (50 cm diameter and 30 cm high). Immediately after, animals were subjected to intraperitoneal (i.p.) injection of saline (saline protocol) before returning to the home cage. Twenty-four hours later (i.e., in the second session), animals explored two familiar objects (A and B from the first session) and two novel objects (E and F).

One week after the first experiment, four of the animals were subjected to a similar behavioural task but with different objects and with injection of haloperidol (0.3 mg/kg) immediately after the first exploration session (haloperidol protocol), which impairs object recognition in the second session (Lobão-Souares et al., 2009). As in experiment 1, objects are also referred to as A, B, C, D, E and F for computing the novelty index (see below). Animals were video-recorded throughout the experiments.

Surgery

Animals were implanted with multielectrode arrays (dimensions 0.9 × 2.1 mm) composed of 50-µm-diameter tungsten wires, 1.5 mm in length; four electrodes targeted the primary motor cortex (M1), four electrodes targeted the somatosensory cortex (S1) and five electrodes targeted the CA1 region of the dorsal hippocampus. The arrays were...
implanted through a rectangular opening in the skull (coordinates from begma, 0.55 and 1.65 mm mediolateral and 0.0 and −2.2 mm antero-posterior). Electrode placement was confirmed by inspecting histological brain sections stained with Cresyl Violet (Fig. 1B).

Recordings
Electrophysiological recordings were made using a multichannel acquisition processor (Plexon Inc., Dallas, TX, USA). LFPs were preamplified (1000×), filtered (1–500 Hz) and sampled at 1000 Hz. A high-impedance homemade headstage and a PBX preamplifier (Plexon Inc., Dallas) model were used. Spikes from multiunit activity were obtained by amplifying (1000×), filtering at 500–8000 Hz, and sampling at 40 kHz.

Data analysis
Data analyses were performed using built-in and custom-written routines in Matlab (MathWorks, Natick, MA, USA). The raw signal was first visually inspected and electrodes with prominent noise were discarded from further analysis.

Filter settings
Filtering was achieved with the eegfilt.m routine from the EEGLAB Matlab toolbox (http://sccn.ucsd.edu/eeGLAB/). This routine uses a linear finite response filter and applies the filter forward and then backwards to eliminate phase distortions. The instantaneous phase and amplitude of a filtered signal were obtained from the analytical representation of the signal using the hilbert.m routine from the Signal Processing Toolbox. Theta and beta2 oscillations were obtained by bandpass filtering at 4–12 Hz and 23–30 Hz, respectively.

Spectral analyses
The power spectrum density was computed by the pwelch.m routine from the Signal Processing Toolbox (50% overlapping Hamming window of 4 s). Band power was defined as the mean over power values in the analysed frequency range. The time–frequency representation shown in Fig. 3A was obtained by the spectrogram.m routine from the Signal Processing Toolbox. The latency to peak beta2 activity was estimated from the mean amplitude of beta2-filtered signals in 10-s sliding windows with 50% overlap. To detect beta2 bursts, for each electrode we first computed the mean amplitude of the beta2-filtered signal. A burst event was defined as occurring when the instantaneous amplitude was > 2 SD from the mean. Burst duration was defined as the period > 1 SD from the mean. For the burst analysis shown in Fig. 7, each animal contributed with a single LFP, selected as the hippocampal electrode with the highest number of beta2 bursts (electrodes in any individual animal had similar numbers of beta2 bursts; not shown).

Behavioural analysis
Object exploration time was considered to be the time animals spent with the whiskers or both front paws in contact with objects. Mice have a natural tendency to explore novel objects (Hughes, 1997, 2007; Dere et al., 2007; Heyser & Chemero, 2012), and spend roughly the same amount of time exploring each of the four objects if they are all novel. On the other hand, if only two objects are novel, animals spend less time exploring the familiar objects, giving rise to unequal exploration time between novel and familiar objects (Dere et al., 2007). Therefore, we defined the novelty index as the ratio of the time spent exploring the two objects that were the same in sessions 1 and 2 (objects A and B) divided by the time exploring the other two objects (objects C and D in session 1 and E and F in session 2). Notice that, under normal conditions, the novelty index should be lower in session 2 than 1 because objects A and B become familiar and are less explored.

The correlation between beta2 and the time spent in locomotion or exploring objects was obtained by first computing the mean (over electrodes) normalised beta2 power per animal, and then by pooling
The hippocampus exhibits transient beta2 oscillations during the exploration of novel objects. (A) Top panel, time–frequency decomposition of a representative hippocampal LFP signal during the first object exploration session (four novel objects). The bottom panel highlights the transient increase in beta2 power. (B) Normalised hippocampal beta2 power along the first object exploration session of experiment 1 in non-overlapping 1-min windows (mean ± SEM). Power values were normalised to the last window (*P < 0.01, t-test against 1, Bonferroni-corrected for ten comparisons). Inset shows mean latency (±SEM) to peak beta2 activity (see Materials and Methods).

Values among animals. We used 1-min non-overlapping windows; beta2 power in each window was normalised to the power value in the last window.

Results

Transient hippocampal beta2 oscillations appear during the exploration of novel objects

Each experiment consisted of two sessions separated by 24 h, in which animals were allowed to explore four objects in an open field for 10 min (Fig. 1A). The four objects were novel in session 1, while only two objects were novel in session 2 (the other two objects were the same as in session 1). Behavioural analysis showed that animals explored the objects in sessions 1 and 2 of experiment 1 (saline injection after session 1; see Materials and Methods). In session 1, animals spent roughly the same amount of time exploring the four novel objects, while in session 2 animals spent more time exploring the only two novel objects (paired t-test, \( t_4 = 3.83, P = 0.0186; \) Fig. 2), which shows that animals recognised the two familiar objects.

We started the electrophysiological analyses by first characterising the oscillatory content of hippocampal LFPs during the exploration of the four novel objects in session 1 of experiment 1. As expected, spectral analyses revealed robust theta oscillations throughout the 10-min exploration period (see Figs 3A and 4B). Interestingly, and consistent with Berke et al. (2008), we found prominent beta2 activity mostly during the beginning of session 1 (Fig. 3B). The mean latency to peak beta2 activity among animals was 55 ± 9 s (range 35–80 s; Fig. 3B inset). Beta2 power in the first 100 s of object exploration was 2.30 ± 0.24 times higher than in the last 100 s (paired t-test compared to 1, \( t_4 = 5.41, P < 0.0001; \) Fig. 4A). The power of other frequency bands such as theta and gamma was also higher in the beginning of the exploration session (Fig. 4), but to a much lower extent than that observed for beta2 [maximum power ratio of 1.31 ± 0.07 (first 100 s/last 100 s) for theta oscillations]; indeed, the increase in power seen at the beginning of session 1 was statistically significantly larger for beta2 than for four other analysed frequency bands (\( F_{4,80} = 15.44, P < 0.0001, \) one-way ANOVA; Fig. 4). As shown in Fig. 5, the decrease in beta2 power along the session correlated with the decrease in locomotion (\( r = 0.49, P < 0.01 \)), but not with the time animals spent exploring objects (\( P = 0.35 \); compare Figs 2A and 3B).

We next confirmed the results above by visual inspection of raw LFPs. To that end, we first filtered the LFP into the beta2 band and localised the periods of high beta2 amplitude (which, consistent with the power analysis, occurred mostly at the beginning of the session; Figs 6A and 7A). We then examined the unfiltered LFP at these periods and found that both sustained theta oscillations and bursts of beta2 activity could be directly observed; the latter was characterised by high-amplitude, sharp LFP deflections (Figs 6B and 7B inset). The mean duration of beta2 bursts was 167 ± 42 ms (Fig. 7B). Therefore, beta2 oscillations are a genuine LFP activity and not harmonics or artifacts of the spectral analysis. In all, these results show that beta2 has a unique dynamics along the first object exploration session, with most beta2 bursts occurring at the beginning of the session.

Beta2 power is lower when animals explore two familiar and two novel objects

We next examined LFP power content in the second exploration session of experiment 1, as well as performed spectral analyses during the pre- and post-exploration periods in the home cage. As shown in Fig. 8A, we found that the transient increase in beta2 power occurred only during the object exploration period in both the first and second sessions (repeated-measures ANOVA, \( F_{2,462} = 25.19, P < 0.0001 \) and \( F_{2,393} = 24.26, P < 0.0001 \), for the first and second sessions, respectively), but not when animals were recorded in their home cage. Interestingly, when power values were normalised by dividing by the mean power in the last minute of exploration, we found that the transient increase in normalised beta2 power was larger in the first than in the second object exploration session (repeated-measures ANOVA, \( F_{1,283} = 5325.16, P < 0.0001 \); Fig. 8B). These results suggest that a greater level of novelty in the session compared to the second (four vs two novel objects, respectively) is associated with higher beta2 activity.
Hippocampal beta2 oscillations

**Beta2 oscillations modulate hippocampal but not S1 and M1 neurons**

We next analysed LFP signals simultaneously recorded from S1 and M1. In contrast to hippocampal signals, S1 and M1 LFPs exhibited no power peak in the beta2 range (Fig. 9A). We then investigated whether spiking activity was coupled to beta2. Fig. 9B shows an example of multiunit activity in CA1, which was highly modulated by both theta and beta2 oscillations (see also Berke *et al.*, 2008). At the group level, while hippocampal beta2 modulated CA1 neurons (Rayleigh test, $P < 0.0001$), population activity recorded at neocortical regions was not coupled to beta2 phase (Fig. 9C). Thus, novelty-related beta2 oscillations seem to occur and modulate spiking activity in the hippocampus but not in S1 or M1 (see Discussion).

**Impairing object recognition is associated with resurgence of prominent beta2 activity**

One week after the first set of recordings (experiment 1) we subjected animals to a similar protocol, but using different objects (experiment 2). In addition, in experiment 2 animals were treated with haloperidol (0.3 mg/kg i.p.) instead of saline immediately after the first object exploration session; haloperidol is amnesic in this paradigm (Lobão-Soares *et al.*, 2009; A.S.C. França, B. Lobão-Soares, L. Muratori, G.C. do Nascimento, J. Winne, C.M. Pereira, S.M.B. Jeronimo, S. Ribeiro, unpublished observations). While animals treated with saline after the first session of experiment 1 showed lower preference for the two familiar objects (A and B) in the second session (paired *t*-test, $t_{4} = 4.759$, $P = 0.0089$; Fig. 10A left panel), haloperidol injected after the first session of experiment 2 led animals to display no object preference in the second session (Fig. 10B left panel). Thus, in the saline protocol the novelty index in the second exploration session was lower than in the first session (paired *t*-test, $t_{4} = 4.262$, $P = 0.0130$; Fig. 10C, left bars), while in the haloperidol protocol the novelty index was not different between sessions 1 and 2 (Fig. 10C, right bars), suggesting that amnesic animals perceived the four objects as equally novel in the second session.

Interestingly, while animals displayed a lower increase in beta2 power in the beginning of the second exploration session of experiment 1 (c.f. Fig. 8), animals treated with haloperidol exhibited...
prominent beta2 activity in both the first and second sessions of experiment 2 (see Fig. 10A and B, right panels, for representative examples). Thus, the beta2 power ratio (first 100 s/last 100 s) decreased from 2.3 in the first session of experiment 1 to ~1.2 in the second session ($t$-test, $t_{31} = 4.011, P < 0.001$; Fig. 10D, left bars), while it was close to 1.8 in both the first and second exploration sessions of experiment 2 (Fig. 10D, right bars). In all, these results suggest that changes in beta2 power within sessions parallel changes in behaviour (compare Fig. 10C and D), with greater beta2 activity in sessions associated with a greater number of novel objects as putatively perceived by the animals.

Discussion

We have characterised hippocampal beta2 oscillations through extracellular recordings, pharmacological intervention and behavioural...
analysis in a novel-object recognition task. We found prominent bursts of beta2 oscillations when mice were allowed to explore four novel objects in an open field. The occurrence of beta2 bursts was transient and concentrated at the beginning of the exploration session. Compared to basal levels, beta2 activity was also higher in the beginning of a second session in which animals explored two novel and two familiar objects, but to a lower extent than in the first exploration session with four novel objects. Interestingly, however, the transient increase in beta2 activity was higher in the second session of experiment 2, when animals putatively perceived familiar objects as novel due to the injection of an amnesic drug immediately after the first exploration session. Taken together, our results suggest that the appearance of beta2 activity in the hippocampus is associated with novel experience.

The hippocampus plays a key role in memory formation (Squire, 1992; Eichenbaum, 2004), spatial navigation (O’Keefe & Dostrovsky, 1971; O’Keefe & Nadel, 1978), context discrimination (Frankland et al., 1998; Mizumori et al., 2007; Tort et al., 2011), ‘match–mismatch’ operations and novelty detection (Knight, 1996; Lisman & Ot�akho, 2001; Kumaran & Maguire, 2007; Duncan et al., 2012). Growing evidence indicates that network oscillations are important for the hippocampus to execute its functions (Montgomery & Buzsaki, 2007; Tort et al., 2009; Jutras et al., 2013). Given the large amplitude of the beta2 oscillations observed here, which allow their direct observation by visual inspection of raw field potentials, it is quite surprising that (to the best of our knowledge) these hippocampal oscillations have only been reported in vivo once (Berke et al., 2008). This could be due to a selective appearance of beta2 in specific behavioural states of mice. In contrast, theta and gamma oscillations always accompany exploratory activity, while ripple oscillations are common during consummatory behaviour such as drinking and grooming (Buzsaki et al., 2003).

Our results corroborate several of the beta2 characteristics first reported in Berke et al. (2008), such as their large amplitude, transient appearance in the beginning of a novel exploration session, and modulation of hippocampal units. Interestingly, in both studies peak beta2 activity was not at the onset of the novel experience but occurred after a latency period. This latency period may reflect the time animals take to perceive the experience as novel (or to generate a mismatch from previous expectations; Grossberg, 2009), and/or for stable place field representations to emerge. It should be noted that, while we found a positive correlation between beta2 power and locomotion activity (as both decreased along the session), we believe there is no causal relationship between them: peak locomotion tended to occur at the very onset of the exploration session and did not coincide with peak beta2 activity. Moreover, Berke et al. (2008) reported the disappearance of beta2 activity after a couple laps in a novel rectangular arena, despite the fact that animals continued to run. The decrease in beta2 activity...
with time, along with no changes in spatial context or animals’ behaviour, reported in Berke et al. (2008) is consistent with our findings showing no correlation between beta2 and the time animals spent exploring the objects.

It should be mentioned that Berke et al. (2008) and the present study obtained results from different mouse strains. Specifically, Berke et al. (2008) observed beta2 bursts in a genetically modified mouse strain (tNR1 mouse; Tsien et al., 1996) and pointed to the importance of examining whether their findings would hold true for wild-type animals. Here we show that this is indeed the case. However, whether similar hippocampal beta2 activity exists in other species such as rats remains to be demonstrated. In addition to corroborating previous findings, here we went on to demonstrate that blocking familiar object recognition is associated with the reappearance of prominent beta2 oscillations as in the first exploration session. This result goes well with previous theoretical accounts (Grossberg, 2009) and further supports a role for hippocampal beta2 oscillations in novel experience.

We found that hippocampal but not S1 or M1 recordings exhibited beta2 oscillations, and, moreover, beta2 phase-modulated only CA1 but neither M1 nor S1 neurons. These results suggest a certain specificity of novelty-related beta2 activity to the hippocampus. Nevertheless, beta2 oscillations were also recently reported in the basal forebrain of rats during an associative learning task (Quinn et al., 2010). In this study, beta2 power was higher in the first day of learning in which the object–reward pairs were novel than in subsequent days when pairings became familiar (Quinn et al., 2010). Moreover, within the first day of learning, beta2 power was lowest in the first trials but increased with later encounters with the objects (Quinn et al., 2010), akin to the latency period for maximal beta2 power observed here. In contrast, however, Quinn et al. (2010) did not observe a disappearance of beta2 activity after objects became familiar. The basal forebrain possesses cholinergic, glutamatergic and GABAergic neurons that project to widespread regions of the cortex, including the hippocampus (McKinney et al., 2000; Maier et al., 2003). Basal forebrain projections modulate synaptic plasticity in the cortex (Kilgard & Merzenich, 1998; Conner et al., 2005) and are involved in attentional processes at the behavioural level (Muir et al., 1993; Voytko et al., 1994; Chiba et al., 1995). Beta2 oscillations could be thus involved in modulating the saliency of novel stimuli in downstream areas; nevertheless, whether the beta2 oscillations described in Quinn et al. (2010) are related to the beta2 in the hippocampus remains to be determined.

Several types of oscillatory activity can be obtained in the hippocampus in vitro, and many believe that these would correspond to their in vivo counterparts (Traub et al., 1996; Whittington & Traub, 2003). Previous work has shown that the internal circuits of the hippocampus are able to produce theta, gamma and ripple frequency oscillations (Whittington et al., 2000; Maier et al., 2003; Whitting-

![Fig. 9. Beta2 modulates hippocampal but not S1 and M1 neurons. (A) Mean power spectra for CA1 (black), S1 (red) and M1 (blue) LFPs during the first 100 s of object exploration. Shaded area represents ±SEM. Notice beta2 activity only in CA1. (B) Spike-phase histograms for an example CA1 unit coupled to theta and beta2 oscillations. (C) Firing probability as a function of hippocampal beta2 phase for multiunit activity recorded from CA1, M1 and S1 (group results). Only CA1 spikes were significantly modulated by beta2 phase (P < 0.05, Rayleigh test).](https://example.com/fig9.png)
Fig. 10. Blocking object recognition in the second exploration session leads to a resurgence of beta2 activity. (A) Left, percentage of time exploring objects in the first and second sessions of experiment 1. Objects A and B were present in both sessions. Animals received saline injection after the first object exploration session (see Fig. 1A). Notice that animals explored less the familiar objects (A and B) in session 2. Right, power spectra in the beta2 range for the first (light gray) and last (dark gray) 100 s of object exploration in a representative animal. Notice prominent difference in beta2 power levels only for session 1. (B) As above, but for experiment 2, in which animals were treated with haloperidol after session 1. Notice that animals did not display preference for the new objects (E and F) in session 2 and that there was a prominent power difference in the beta2 range between the beginning and end of both session 1 and session 2. (C) Novelty index, defined in session 1 (white) as the exploration time of objects A and B divided by the exploration time of objects C and D, and in session 2 (gray) as (A and B)/(E and F). Notice that saline injection after session 1 allowed animals to recognise objects A and B in session 2, which led to a lower novelty index; on the other hand, animals treated with haloperidol after session 1 did not display object preference in session 2, and had equal levels of novelty index in sessions 1 and 2. (D) Beta2 power ratio (first 100 s/last 100 s) for sessions 1 (white) and 2 (gray) of the two experimental protocols. Results are expressed as mean ± SEM; *P < 0.01, t-test; n.s., nonsignificant.
received less attention than gamma due to the lack of its in vivo correspondent. Further work combining in vitro and in vivo techniques should test whether ‘gamma’ in hippocampal slice preparations might in reality correspond to the novelty-related beta2 activity reported here.

An influential model proposes that the hippocampus and the ventral tegmental area (VTA) form a loop that controls the formation of long-term memory (Lisman & Grace, 2005). Accordingly, the hippocampus would be responsible for detecting new information and sending novelty signals to the VTA, which would integrate this information with others (such as saliency) and in turn release dopamine in the hippocampus, enhancing LTP and learning. Such a model is consistent with electrophysiological, molecular and behavioural findings (Gasbarri et al., 1996; Otmakhova & Lisman, 1996; Lemon & Manahan-Vaughan, 2006; Morice et al., 2007; Terry et al., 2007; Rossato et al., 2009). However, there is not much literature about the influence of D2 antagonists on the novel object recognition task used here. We have recently found that haloperidol blocks object memory consolidation (A.S.C. França, B. Lobão-Soares, L. Muratori, G.C. do Nascimento, J. Winne, C.M. Pereira, S.M.B. Jeronimo, S. Ribeiro, unpublished observations), as observed in the present study. These findings are consistent with a role of dopaminergic projections to the hippocampus in controlling the formation of long-term memory (Lisman & Grace, 2005; Rossato et al., 2009). Whether hippocampal beta2 oscillations would take part in the communication between the hippocampus and VTA remains to be established.

In summary, our results demonstrate the appearance of transient beta2 oscillations in the hippocampus during exploration of novel objects. As hypothesised by Grossberg (1999), these findings suggest that beta2 may be involved in signaling specific time periods for new plasticity to occur. However, much yet should be done to characterise their biophysical mechanisms of generation, regions of occurrence, as well as cognitive roles. In particular, it would be interesting to know whether disruption of beta2 activity during memory acquisition affects behaviour and LFP spectral content when testing memory retrieval. Most importantly, the field would benefit from independent labs reporting a similar pattern of organised electrical activity; we concur with many others (Button et al., 2013) in the belief that replication is key for constructing solid knowledge.

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Abbreviations

LFP, local field potential; M1, primary motor cortex; S1, primary somatosensory cortex.

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