

PARTICIPATION OF STRIATAL NEURONS IN LARGE-SCALE OSCILLATORY NETWORKS

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1. INTRODUCTION

In recent years basal ganglia physiologists have become increasingly interested in oscillations and synchrony (Bevan et al., 2002; see also chapters by Boraud et al., and Walters et al., this volume). This reflects a now broad recognition that “box-and-arrow” models of the basal ganglia based upon simple inhibition and excitation are inadequate to explain current data, and too conceptually limited to serve as a basis for further experimentation. In this brief chapter I shall address some recent observations while recording local field potentials (LFPs) and single-units in the striatum of awake, unrestrained rats (Berke et al., 2004). Rather than reiterating previously published points, I shall focus on some *difficulties* encountered in interpretation of field potential recordings, and some (incomplete) solutions to these problems.

The striatum is just one component of the cortex – basal ganglia – thalamus – cortex loops, and may or may not reflect all the dynamic activity seen in subsequent nuclei such as the globus pallidus. However, it will be difficult to understand oscillations in such other structures without considering oscillations in their inputs – so the striatum, together with its relationships to cortex, is a reasonable place to begin.

2. WHAT IS A LOCAL FIELD POTENTIAL, AND WHAT IS IT GOOD FOR?

Extracellular recordings are made by measuring the potential difference between the tip of an electrode in the brain, and some reference location. Fluctuations in this potential difference can be filtered into either higher-frequency (e.g. 300–6000 Hz) or lower-frequency (e.g. 0.1–300 Hz) components. The higher frequency parts of the signal can include brief action potentials (spikes) from individual neurons (“single-units”) or groups of cells (“multi-unit activity”). The lower frequencies are called the local field potential

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(for some examples, see Figs. 1,3), and can be an indication of the collective activity of large populations of neurons and/or synapses. Such population activity may be of interest in itself, especially in a structure such as striatum where individual neurons tend to have highly diverse behavioral correlates (Schultz, 1995). Even more importantly, the relationships between single-units and LFPs may give us useful clues to the phenotypes, connectivity, and temporal coding properties of individual neurons, as discussed below.

3. STRIATAL LFPs ARE TOPOGRAPHICALLY ORGANIZED AND BEHAVIORALLY CONTINGENT

Oscillatory activity is readily apparent in LFPs recorded from rat striatum. The types of oscillation observed reflect both the behavioral state of the animal, and the location within the striatum (Fig. 1). High-voltage spindles (HVSSs, ~ 8 Hz), which engage broad regions of neocortex, are also strongly present in the dorsal/lateral striatum. These oscillations occur in awake but immobile animals, and have increased incidence following striatal dopamine depletion or blockade (Buonamici et al., 1986; Buzsáki et al., 1990). In ventral striatum strong ~ 50 Hz gamma oscillations are frequently observed in alert rats (whether moving or not), and these are sometimes accompanied by beta oscillations (~ 20 Hz; Berke and Kunec, 2004). During maze running ventral striatum also shows the ~ 8 Hz theta rhythm that is widespread in limbic brain regions (Buzsáki, 2002).

4. HOW LOCAL IS THE STRIATAL LOCAL FIELD POTENTIAL?

To understand the significance of such striatal LFP oscillations, we need to know how they arise. Firstly we should establish which components of the striatal LFP signal are actually being generated in striatum, rather than some other structure. While all LFPs rely on conduction through the extracellular medium to some extent, under some conditions electrical field changes generated in one brain area can be detected in another, quite distant, location. This volume conduction is a serious concern when interpreting LFP signals. Secondly, if the LFP is indeed locally generated, we would like to know which neural elements are responsible, and over what spatial scale they contribute to the LFP. Unfortunately, despite a number of useful clues, neither question can currently be answered to our full satisfaction in any brain nuclei, including the basal ganglia.

To address the first question we must rely heavily on mapping out the amplitude and phase of oscillations, within and around the structure of interest. Recording from multiple brain regions and subregions simultaneously can be helpful in establishing the spatial extent of oscillatory activity, and in adding or excluding candidate structures that may be generating rhythmic activity. For example, ~ 50 Hz gamma oscillations appear very similar in ventral striatum and in nearby piriform cortex, but not in medial prefrontal cortex or hippocampal CA1 (Fig. 3A). Recording different signals from many distinct sites, all using the same (distant) reference location, also adds confidence that the signals observed are not actually being detected at the reference site. Similarly, moving an electrode progressively through a structure and observing that a signal disappears at the structure's boundaries can increase confidence that a signal is local. On the other hand, even in large-scale electrophysiological recording one is limited in the number of recording sites, so often it remains possible to argue that another, non-sampled location is actually generating the observed signal.

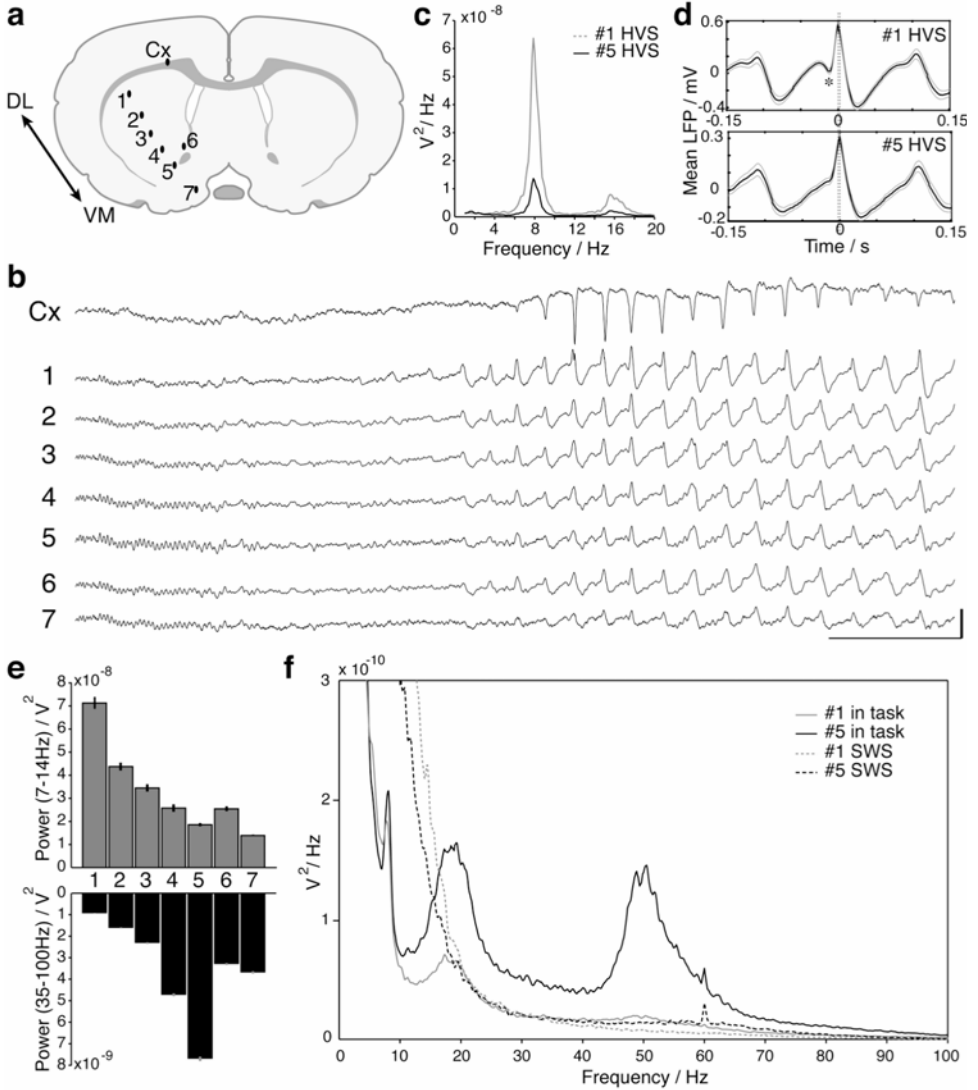


Figure 1. Graded distributions of striatal oscillations. **a)** Example of tetrode recording positions. “1”–“5” were positioned along the dorsal/lateral to ventral/medial axis, “Cx” was on the boundary between deep cortical layers and the white matter of the corpus callosum. **b)** Example of LFPs recorded from the sites shown in a, in an awake but immobile rat. Gamma oscillations (~50 Hz) are visible early in some traces, a high-voltage spindle (~8 Hz) is present on the right hand side. Scale bars: 0.5 s, 1 mV. **c)** Power spectral density for LFPs from tetrodes 1,5 during HVSS. **d)** Averaged shape of HVS cycles from tetrodes 1,5. HVS peaks in ventral-medial striatum lag slightly behind those in dorsal-lateral striatum. In addition to smaller amplitude, they also lack the clear negative deflection (marked by asterisk) slightly before the peak. **e)** HVS (top), and gamma (bottom) power for each striatal location, during detected HVS and gamma epochs respectively. There are opposite gradients of HVS and gamma power between dorsal/lateral and ventral/medial striatum. **f)** Power spectral densities for electrodes 1,5 during well-practiced performance of a radial maze task (solid lines) or slow-wave sleep (dashed lines). Ventral/medial striatum shows pronounced theta (~8 Hz), beta (~20 Hz) and gamma oscillations (~50 Hz). For more details and methods see Berke et al., 2004. Parts of this figure and the next were modified from that work, with permission from Elsevier.

Signals that are the result of volume conduction should propagate at the speed of light (in brain), and hence have no detected phase lag. This is not the case for striatal high-voltage spindles (Fig. 1d), which show a small but measurable phase-lag between dorsal-lateral and ventral-medial sites, as well as a pronounced change in shape (more on this below). This is fairly good evidence that the high-voltage spindles in striatum actually do reflect local processes.

Usually, some of the strongest evidence for local generation of LFPs is the observation of complete phase reversals within the structure. Note that there are two quite distinct meanings of phase reversal (Duffy et al., 1989). “Instrumental” phase reversal can be observed when a series of electrode contacts are placed in a line, with each contact serving as the reference point for the next. A single focus of current can produce voltage deflections in opposite directions for two adjacent channels in this chain of bipolar recordings, simply as a result of the referencing arrangement. Such useful evidence for focal oscillatory activity located between two contacts has been obtained in macroelectrode investigations of the human subthalamic nucleus (e.g. Brown et al., 2001). However, such electrode and recording configurations are not well suited for mapping out oscillations. For example if two adjacent contacts are both located within similar parts of the same structure, or in two structures oscillating in phase, the bipolar arrangement can make it appear as if there is no oscillation present at all. In addition these human recordings have so far suffered from the limitations of low spatial resolution and imprecise electrode localization without post-mortem data.

“True” phase reversal occurs between recording sites whose local potential changes are truly out of phase – because of opposite directions of current flow at the two locations. One of the best-studied examples is the hippocampal CA1 region (see Fig. 3A). Here the phase of theta oscillations changes systematically with depth (Brankack et al., 1993; Buzsáki, 2002), as electrodes pass through successive layers of synapses from distinct afferents, and cell bodies. The organized separation of these current “sources” and “sinks” in coherently aligned neurons allows large electrical fields to be generated. High-density recording methods, together with current-source-density analysis, have now allowed systematic investigation of current sources and sinks within such laminar structures (e.g. Kandel and Buzsáki, 1997; Csicsvari et al., 2003).

Our understanding of LFPs in structures such as striatum, in which cells have radial morphology and lack organized orientation, is far more limited. Current theories that treat neurons as electric dipoles (balanced sources and sinks) would predict that the electric fields generated by a set of randomly arranged dipoles should cancel one another out, unless the LFP signal reflected activity from just one or a small number of cells (this would be a “very local” field potential). This is not the case, since striatum LFPs can remain nearly unchanged during electrode movements of a millimeter or more, while extracellular spikes from individual units typically disappear with electrode movements of 100 μm or less. Thus, in so far as the LFPs within striatum are indeed being striatally-generated, they are reflecting synchronized population activity of many neural elements. In cortex it has long been believed that the longer duration of synaptic currents (especially EPSCs) compared to spikes results in synaptic activity dominating the LFP (Mitzdorf, 1985). More recent work suggests that other cellular processes that affect membrane potentials may also make a substantial contribution (Nadasdy et al., 1998; Logothetis, 2003). It would be useful to have more theoretical analyses to tell us what processes may contribute to the striatal LFP, and over what spatial scales.

If striatal LFPs are generated largely by EPSCs, one could conceivably observe substantial LFP phase changes in striatum if there were sharp boundaries between the terminal

fields of distinct afferents, and these afferents oscillated with different phases. For the most part, the divergent nature of corticostriatal innervation would seem to discourage this – corticostriatal axons tend to innervate rather wide areas within striatum, and make only a small number of synapses on each striatal neuron they pass (e.g. Cowan and Wilson, 1994; Zheng and Wilson, 2002). Although focal zones within striatum can receive rather different innervation (e.g. Gerfen, 1989; Ragsdale and Graybiel, 1990), we have not yet observed any sharp transitions in the striatal LFP during electrode movements that might correspond to distinct LFPs in these zones.

To summarize so far, while we have observed phase changes within striatum that are suggestive of a local origin, we have not observed the sort of LFP phase reversals that are used to make inferences about LFP generators in laminar structures. Nor, given the micro-anatomical organization of the striatum, would one necessarily expect to. On the other hand, one at least has the consolation of never being in the “wrong” layer within striatum – allowing useful analyses of LFP–single unit phase relationships with only moderately precise anatomical reconstruction.

5. RELATIONSHIP BETWEEN INDIVIDUAL STRIATAL NEURONS AND LFPs

In the absence of extracellular phase reversals, some of the best evidence for local generation of striatal LFPs comes from examination of the timing relationships between LFPs and the activity of individual cells. Some useful demonstrations that the local field potential in the striatum can closely mirror local individual neuronal potentials have come from combined intracellular and extracellular recordings in ventral striatum (Leung and Yim, 1993; Goto and O'Donnell, 2001). In these anesthetized rats, prominent slow-wave/delta (~1 Hz) oscillations were seen in both types of recording, but inverted with respect to each other. This may reflect current flow between the intracellular and extracellular compartments. Such slow membrane changes occur simultaneously across populations of medium-spiny striatal neurons, showing a greater degree of synchrony than individual action potentials (Stern et al., 1998). Hence the striatal LFP may be a good indicator of striatal population changes in intracellular membrane potential.

However, as such slow-wave oscillations are synchronously present across wide regions of the brain during sleep and anesthesia, the causal relationship between intracellular and extracellular potentials could in principle be indirect (Tseng et al., 2001). Further, intracellular recording is currently limited to anesthetized and/or head-restrained preparations. As the rhythms observed in the basal ganglia are highly dependent on behavioral state, it is hard to gain intracellular data on the faster rhythms associated with active behaviors – a particular problem when investigating the functional roles of the basal ganglia in action selection and learning.

We have therefore focused on the relationship between extracellularly-recorded single-units and LFPs (Fig. 2) To briefly address one methodological point first: Magill and colleagues have argued that “. . . the utility of analyses of the correlation or coherence between units and LFPs recorded through the same electrode is limited by overlaps in the frequency content of the two signals, which may lead to the spurious detection of temporal coupling” (Magill et al., 2004). There is indeed a potential pitfall, but this can be readily avoided. One common method of relating spikes to LFP oscillations is to find the peaks (or troughs) of the rhythm, and for each single-unit spike that falls between two peaks calculate the corresponding phase. This produces a phase histogram (e.g. Fig. 2e) and one can use standard circular statistics to calculate the significance of any entrainment. A problem can

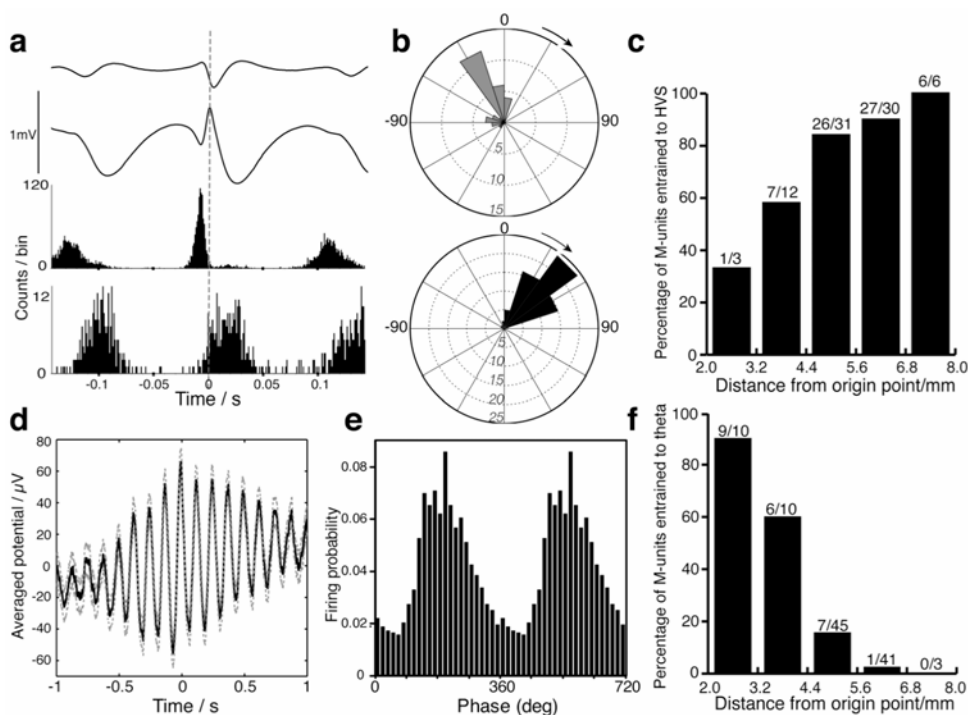


Figure 2. Single-unit entrainment to HVS and theta oscillations. **a**) Upper traces: EEG from frontal cortex (top) and dorsal/lateral striatal LFP (bottom), averaged around the peak of the striatal HVS cycles. Bottom: Perievent spike histograms for two single-units, triggered by the same HVS peaks. Top unit is a presumed fast-spiking interneuron ("F-unit"), bottom is a presumed medium-spiny cell ("M-unit"). **b**) Firing phase for all entrained F-units (top) and M-units (bottom). F-units as a population fire with distinct phase to the M-unit population. **c**) Distribution of HVS-entrained M-units, relative to an arbitrary origin point on the ventral midline. Increasing distance from this point indicates more dorsal, lateral, and/or posterior locations. **d**) Spike-triggered average between a medial striatal M-unit and LFP recorded in dorsal hippocampus. Dashed lines indicate $\pm 2 \times \text{S.E.M.}$. The strong oscillation centered around zero indicates that this unit tends to fire at a specific phase of the hippocampal theta rhythm. **e**) Phase histogram for the same unit and hippocampal LFP (theta troughs are at $0^\circ/360^\circ/720^\circ$). This cell tends to fire at or shortly after hippocampal theta peaks. Entrainment was significant at $p < 10^{-57}$. **f**) Distribution of theta-entrained M-units, relative to same origin as in c. Note opposite gradients of entrainment to HVS and hippocampal theta.

arise when, depending on the signal filtering and the peak detection algorithm, the action-potentials of a single-unit contribute transients to the LFP that are falsely detected as peaks of the LFP oscillation under investigation. The result can be the incorrect assignment of these spikes as having a zero-phase relationship to the LFP oscillation. Therefore any phase histogram showing just a narrow peak of high entrainment precisely at zero phase should be viewed with considerable scepticism. In a similar way, spike-triggered averages of LFPs can show the averaged action potential contribution to the LFP – though only at time zero, so oscillations on either side of this are not affected. One can, of course, escape any such problems altogether by correlating units recorded on one electrode to the LFP recorded on another. As field potentials change only slowly with distance in the striatum (as discussed above), clear oscillatory entrainment of single-units is easily detected even when using LFPs from a considerable distance away (e.g. Figs. 3c,d).

We observed that at least some properties of the striatal local field potential do closely reflect striatal spike activity (Fig. 2). High-voltage spindles (HVS) are strong in dorsal-lateral striatum, and almost all single-units recorded there are strongly entrained to this rhythm. The proportion of entrained units drops off medially and ventrally, along with the HVS power in the LFP. By contrast, ventral-medial striatum contains a high proportion of units that are entrained to hippocampal theta rhythm, and the LFP there shows a theta peak with clear coherence to hippocampus. Thus there are opposite gradients of single-unit entrainment between sensorimotor and cognitive/limbic striatum, which match the properties of the LFP.

Further, the shape, as well as power, of striatal HVS oscillations reflects local unit properties. The striatum shows a graded distribution of tonically-active, brief-waveform cells that are probably parvalbumin-staining, fast-spiking GABAergic interneurons (see Fig. 1 of Berke et al., 2004). Given that fast-spiking interneurons have been shown to strongly influence the spike timing of wide populations of spiny neurons *in vitro* (Koos and Tepper, 1999), it is quite likely that they play an important role in entraining the wider population of spiny neurons into the HVS oscillation *in vivo* – though this remains to be directly demonstrated. These cells fire just before the peak of the HVS oscillation, while the main population of single-units (almost certainly medium-spiny cells) fire after the peak. The activity of the two populations corresponds to two negative deflections in the LFP; in ventral-medial striatum, where there are relatively few fast-spiking units, there is a corresponding relative loss of power of the first deflection (Fig. 1d). As this dip has a similar width to the HVS peak itself, and thus contributes similar frequency components to the LFP, this waveform change is unlikely to be the result of some passive filtering property of the striatal neuropil.

Such close correlations between such HVS variations in the striatal LFP and the variation in the single-unit properties strongly support the view that at least this oscillation is locally generated in striatum. It may be possible to gain further confirmatory evidence by, for example, a selective lesion of striatal fast-spiking interneurons – though there is always the possibility that this would alter the incidence or nature of the oscillation too much to be useful. The very high proportion of theta-entrained units in ventral striatum, the fact that these cells as a population fire with coherent phase (Berke et al., 2004), and the spatially graded decrease of unit entrainment, LFP theta power, and LFP theta coherence with hippocampus argues for local generation of striatal LFP theta as well.

However, there are also some dissociations between spike activity and LFP, both in the striatum and in other structures (Logothetis, 2003). Consider the rather complex case of striatal beta and gamma oscillations (Fig. 3). These rhythms show interesting power changes over the course of habit learning, as well as frequency modulations in response to both dopaminergic drug manipulations and behavioral task features such as reward receipt (Berke et al., 2003; Berke and Kunec, 2004). However, the evidence for these LFP oscillations reflecting local striatal activity is decidedly mixed. It is possible to find single-units in striatum that are strongly entrained to gamma and beta oscillations (e.g. Figs. 3c, d), so these rhythms undoubtedly have some impact on striatal activity. On the other hand, the proportion of clearly entrained units is very small compared to (for example) theta-entrained units, even though ~50 Hz gamma oscillations in particular are very prominent in the LFP. In addition, extremely similar beta and gamma oscillations are observed in the adjacent piriform cortex (e.g. Fig. 3a), as well as related structures such as endopiriform cortex (unpublished observations). Given the potential for large electric field generation by the laminar piriform cortex, it seems quite likely that a large component of the LFP gamma

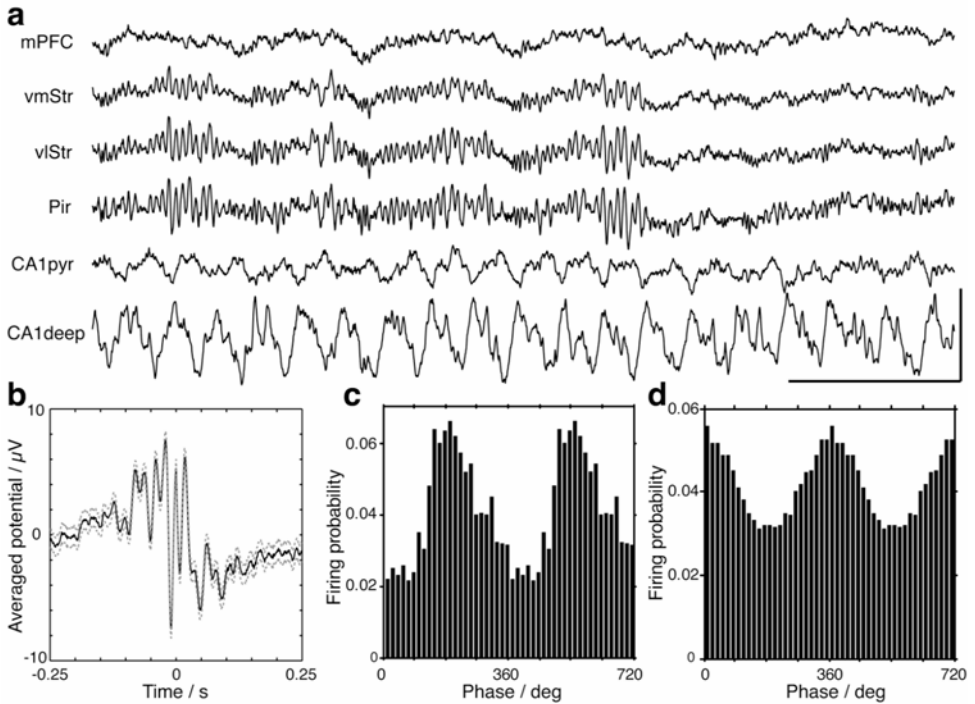


Figure 3. Gamma oscillations in striatum and piriform cortex. **a)** Simultaneously recorded LFPs from medial prefrontal cortex (“mPFC”), ventral-medial striatum (“vmStr”), ventral-lateral striatum (“vlStr”), piriform cortex (“Pir”), cell body layer of dorsal hippocampal CA1 (“CA1pyr”), and deep dorsal hippocampal CA1 (“CA1deep”). All signals were referenced to the same skull screw at a distant, posterior midline location. Scale bars, 0.5 s, 1 mV. Note phase reversal of hippocampal theta between the two hippocampal recording sites, and that the large gamma oscillations are very similar in both ventral striatum and piriform cortex. **b)** Spike-triggered average between spikes from an F-unit recorded at one location in ventral striatum and the LFP recorded at another ventral striatal site. Dashed lines indicate $\pm 2 \times$ S.E.M. The complex-appearing averaged signal reflects entrainment to multiple oscillations. **c)** Phase histogram showing entrainment of the same F-unit to beta oscillations (LFP filtered at 12–25 Hz before peak detection). Entrainment is significant at $p < 10^{-62}$. **d)** Phase histogram showing entrainment of the same F-unit to ~ 50 Hz gamma oscillations (LFP filtered at 42–58 Hz before peak detection). Entrainment is significant at $p < 10^{-147}$.

oscillations recorded in striatum is not produced there. We will need to complete phase mapping and possibly lesion studies before drawing any firm conclusions; initial analyses suggest that piriform cortex and striatum may have zero phase difference most of the time, but not under all behavioral conditions. In any case, it appears that at least a large part of rat striatal beta and gamma oscillations are a manifestation of olfactory information processing (e.g. Kay and Freeman, 1998) (Vanderwolf, 2000; Neville and Haberly, 2003; Ravel et al., 2003) rather than some particular striatal mechanism.

Another unresolved observation is that the few gamma-entrained striatal units recorded so far have all been presumed fast-spiking interneurons (Berke and Kunec, 2004). These cells may receive a more convergent pattern of afferents from cortical structures (Ramanathan et al., 2002) that contributes to higher sensitivity (Parthasarathy and Graybiel, 1997). It is also easier to detect oscillatory entrainment in tonically active units simply due to a larger number of spikes available for analysis. Nonetheless, given the tight control

these interneurons can exert over the spike timing of nearby medium spiny cells, it would be interesting to know what membrane properties or other factors prevent the clear entrainment of projection neuron spikes. Reduced synaptic efficacy at gamma frequencies might also contribute (Thomson and West 2003).

6. CONCLUSIONS

Given our limited understanding of LFP generation (and not just in the striatum), one might ask why it is worth recording the LFP at all, rather than just oscillations in spike trains. One major reason is that the LFP serves as a form of temporal reference. By comparing spike times to the LFP, one can indirectly compare the timing properties of individual neurons recorded from different sessions or even different rats. This has allowed us to confirm that groups of units with different waveforms and firing patterns actually do represent functionally distinct neuronal subpopulations. The relationship between individual neurons and particular rhythms may also give clues as to the sorts of information they are receiving. For example, both theta-entrained and -unentrained neurons can be recorded simultaneously from the same electrode. It would be interesting to know whether neurons that are more entrained to hippocampal theta have behavioral correlates that are more closely related to hippocampal representations than those that are not.

Recording of striatal LFPs may also be useful when attempting to make connections to the human neuroimaging literature. Recent studies have found that (at least in cortex) the fMRI BOLD signal is much more closely related to the LFP than to neuronal spiking (Logothetis, 2003). Both signals appear to more closely reflect synaptic input and local processing than the output of a brain structure, which is encoded in the activity of projection neurons. Because of this, the LFP may contribute useful information about population-wide subthreshold changes in membrane potential – if we can learn more about how it is generated.

The oscillatory entrainment of striatal units shows that oscillations are indeed a feature of rat striatal activity. Do such oscillations play an important role in the functional properties of the basal ganglia? The significance of neural oscillations has been the subject of much debate, and the possible advantages of organizing neural information processing using oscillations have been recently reviewed (Engel et al., 2001; Buzsaki and Draguhn, 2004). While mechanisms for oscillatory pacemaking may exist within striatum (Wilson, 2005), our current evidence suggests that most oscillatory activity found within the normal rat striatum is instead the result of oscillations in striatal afferents. Hence, theta oscillations in striatum are found in striatal areas that receive afferents from theta-entrained brain regions, including hippocampus and medial prefrontal cortex. While there have been few studies of olfactory innervation of striatal regions (e.g. Haberly and Price, 1978; Luskin and Price, 1983), it appears that striatal ~50 Hz gamma oscillations are another manifestation of the widespread olfaction-related gamma. And sensorimotor striatum shows the high-voltage spindles that are most reliably initiated in sensorimotor cortex (Meeren et al., 2002). Intra-striatal circuit properties may, however, influence the spread, extent, and occurrence of oscillations both locally and in other brain structures. For example, high-voltage spindles are far more synchronized in striatum than between cortical EEGs, which likely reflects both divergence of corticostriatal pathways and the coupled activity of striatal fast-spiking interneurons (Berke et al., 2004). The fact that loss of striatal dopamine alters the nature and incidence of oscillations in the basal ganglia and cortex (e.g. Buonamici et al.,

1986; Magill et al., 2001; Goldberg et al., 2002; Belluscio et al., 2003) demonstrates the influence of basal ganglia mechanisms over the dynamic activity of cortex – basal ganglia – thalamus – cortex loops. We have a long way to go, however, before we are able to prove or disprove the intriguing hypothesis of Brown and Marsden (1998) that basal ganglia control of oscillatory activity is central to their functional role in action selection and initiation.

7. ACKNOWLEDGEMENTS

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