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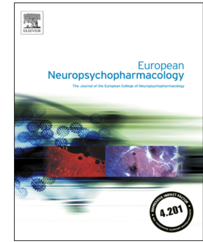

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Characterization of electrically evoked field potentials in the medial prefrontal cortex and orbitofrontal cortex of the rat: Modulation by monoamines

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Abstract
Medial prefrontal cortex (mPFC) and orbitofrontal cortex (OFC) play critical roles in cognition and behavioural control. Glutamatergic, GABAergic, and monoaminergic dysfunction in the prefrontal cortex has been hypothesised to underlie symptoms in neuropsychiatric disorders. Here we characterised electrically-evoked field potentials in the mPFC and OFC. Electrical stimulation evoked field potentials in layer V/VI of the mPFC and layer V of the OFC. The earliest component (approximately 2 ms latency) was insensitive to glutamate receptor blockade and was presumed to be presynaptic. Later components were blocked by 6,7-dinitroquinoxaline-2,3-dione (DNQX (20 μM)) and were assumed to reflect monosynaptic (latency 4–6 ms) and polysynaptic activity (latency 6–40 ms) mediated by glutamate via AMPA/kainate receptor. In the mPFC, but not the OFC, the monosynaptic component was also partly blocked by 2-amino-5-phosphonopentanoic acid (AP-5 (50–100 μM)) indicating the involvement of NMDA receptors. Bicuculline (3–10 μM) enhanced the monosynaptic component suggesting electrically-evoked and/or glutamate induced GABA release inhibits the monosynaptic component via GAB_{AA} receptor activation. There were complex effects of bicuculline on polysynaptic components. In the mPFC both the mono- and polysynaptic components were attenuated by 5-HT (10–100 μM) and NA (30 and 60 μM) and the monosynaptic component was attenuated by DA (100 μM). In the OFC the mono- and polysynaptic components were also attenuated by 5-HT (100 μM), NA (10–100 μM) but DA (10–100 μM) had no effect. We

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propose that these pharmacologically characterised electrically-evoked field potentials in the mPFC and OFC are useful models for the study of prefrontal cortical physiology and pathophysiology.
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1. Introduction

The prefrontal cortex (PFC) is critically involved in executive functions and behavioural control. The rat mPFC mediates attentional set shifting (Birrell and Brown, 2000) and so is regarded as homologous to the dorsolateral prefrontal cortex in primates. In both rodents and primates, the orbitofrontal cortex (OFC), appears to play a pivotal role in reversal learning (McAlonan and Brown, 2003; Rygula et al., 2010) and the motivational control of behaviour and impulsivity (Berlin et al., 2004; Rudebeck et al., 2006; Zeeb et al., 2010).

Both the mPFC and OFC are extensively innervated by 5-HT (serotonin), noradrenaline (NA) and dopamine (DA) (Agster et al., 2013; Descarries et al., 1987; Hoover and Vertes, 2007; Oades and Halliday, 1987; Slopsema et al., 1982). Electrophysiological studies have revealed that monoamines regulate firing activity in neurons of the prefrontal cortex. In the mPFC, inhibition of pyramidal cell firing activity is the predominant response to electrical stimulation of the serotonergic dorsal and median raphe nuclei (DRN and MRN) (Hajos et al., 2003; Mantz et al., 1990), the noradrenergic locus coeruleus (LC) (Mantz et al., 1988) and the dopaminergic ventral tegmental area (VTA) (Godbout et al., 1991; Mantz et al., 1988). Data from the OFC are sparser but DRN stimulation has been shown to alter the electroencephalogram (Dringenberg and Vanderwolf, 1997) and 5-HT agonists have been shown to influence OFC pyramidal cell firing (Rueter et al., 2000). The noradrenaline and DA releasing agent D-amphetamine (Homayoun and Moghaddam, 2008) and the DA reuptake inhibitor cocaine (Guillem et al., 2010) have also been shown to influence pyramidal cell firing in the OFC. Studies employing pharmacological agents that modify monoamine neurotransmission or selective monoamine neurotoxic lesions also point to important roles for the monoamines in the behavioural functions subserved by the mPFC and OFC (McGaughy et al., 2008; Robbins and Arnsten, 2009; Winstanley et al., 2006).

Many psychiatric disorders, including bipolar disorder, schizophrenia, obsessive compulsive disorder and substance abuse disorders, are characterised by deficits in prefrontally-mediated cognitive functions and/or behavioural control (Cunha et al., 2010; de Geus et al., 2007; McKirdy et al., 2009). Given the efficacy of drugs which target monoamine neurotransmission in the treatment of these disorders, it is reasonable to hypothesise that alterations in monoamine modulation of prefrontal function may underlie the functional deficits and/or the therapeutic effects of drugs. Models to assess prefrontal cortical function and the influence of monoaminergic neurotransmission in experimental animals would be of potential utility in investigating these hypotheses.

In specific brain regions with coherent laminar structures such as the cerebral cortex and hippocampus, synaptic transmission can be measured using electrically evoked field potentials (Empson and Heinemann, 1995; Langdon and Sur, 1990; Lomo, 1971). Here we characterised intra-laminar field potentials stimulated and recorded in layer V/VI of the infralimbic region of the mPFC and in layer V of the ventral/lateral portions of the OFC. The roles of glutamate and GABA and the potential modulation of the field potentials by monoamine neurotransmitters were examined.

2. Experimental procedures

2.1. Animals and tissue

All animal procedures were approved by the Newcastle University Ethical Review Committee and licenced by the UK Home Office. All efforts were made to minimise the number of animals used and the suffering of individual animals.

Adult male Lister hooded rats (250–330 g) ($n=45$) (Charles River, UK) were group housed in controlled conditions of light and humidity and were allowed free access to food (SDS diet) and water. Rats were killed by decapitation and the brain was rapidly removed and placed in ice-cold sucrose slush (composition: 176 mM Sucrose, 9.9 mM HEPES, 25 mM $NaHCO_3$, 10 mM D-Glucose, 1.2 mM NaH_2PO_4, 0.5 mM $CaCl_2$, 2.5 mM KCl, 7 mM $MgSO_4$) oxygenated with 95% O_2:5% CO_2 (carbogen). Coronal slices (350 or 400 μm) of the PFC (containing both mPFC and OFC) were prepared using a Vibratome™. A single slice was placed on lens tissue (Whatman 105) in an interface perfusion chamber and was perfused with oxygenated artificial cerebrospinal fluid (aCSF) (0.5 ml/min). The slice and aCSF were warmed to 37 °C with a water chamber and a stream of warm humidified carbogen passed continuously over the slice. Spare slices were maintained in oxygenated aCSF at room temperature for later use.

2.2. Electrophysiological recording

Stimulating electrodes, comprising two ultra-fine (0.111 mm) Teflon insulated silver wires, (Advent Research Materials, England) twisted together and cut square at the end to reveal two contacts separated by approximately 100 μm. Recording electrodes were glass microelectrodes (1.5 mm OD; Clarke Electromedical, Reading, UK) pulled on a vertical pipette puller (Narashige, Japan) and filled with 2.5 M NaCl coloured with 2% Pontamine Sky Blue. For the mPFC, the stimulating electrode was placed in layer V/VI of the ventral infralimbic cortex immediately medial to the white matter of the claustrum and the recording electrode was placed 500–1000 μm dorsal to the stimulating electrode in layer V/VI (Figure 1). For the OFC, the stimulating electrode was placed in layer V of ventral or lateral OFC immediately ventral to the claustrum and the recording electrode was placed 500–1000 μm lateral to the stimulating electrode also in layer V (Figure 1). It was not possible to visualise the cell layers during recordings and placement in layer V/VI (mPFC) and layer V (OFC) is presumed. Extracellular potentials were amplified (1000 ×) and filtered (band pass:0.2 Hz–15 kHz) and were fed via an interface (micro1401, CED, Cambridge, UK) to a PC and recorded at a sampling rate of 10 or 50 kHz using Spike2 software (version 4, CED, Cambridge, UK). Data sampled at 50 kHz

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