Cell Responses To GSM-1800 MHZ Electromagnetic Fields In The Rat Brain Affected 
By An Acute Inflammation

J. Lameth(1), D. Arnaud-Cormos(2,3), P. Lévêque(2), S. Boillée(1), JM. Edeline(4) and M. Mallat* (1)
(1) Institut du cerveau - Paris Brain Institute -ICM, INSERM, Paris 75013, France  
(2) Univ. Limoges, CNRS, XLIM, UMR 7252, F-87000 Limoges, France  
(3) Institut Universitaire de France (IUF), 75005, Paris, France  
(4) Paris Saclay Institute of Neuroscience, Neuro-PSI, UMR 9197 CNRS, Saclay, France

The extensive use of wireless communications has raised public health concerns, which stimulate investigations 
of how electromagnetic fields (EMF) oscillating in the radiofrequency (RF) range might affect organs at cell and 
molecular levels. Brain cell responses triggered by head or whole-body exposure to RF have been extensively 
studied in healthy subjects. Less is known about the effect of these RF on the brain affected by a pathological 
process. 

Lipopolysaccharide (LPS)-treated rodents model a human acute neuroinflammatory state triggered by benign 
peripheral infections, which affect many people each year, world-wide. We have investigated brain cells responses 
to a 2h single head exposure to GSM-1800 MHz in LPS-treated rats undergoing acute neuroinflammation. The 
analyses were carried out 3 to 72 h after the end of the GSM exposure, in different regions of the cerebral cortex 
in which the mean values of the specific absorption rate (SAR) varied between 1.55 W/kg and 3.22 W/kg. LPS-
treated rats submitted to a sham exposure (null SAR values) served as the negative control. By recording the 
electrophysiological activity in the primary auditory cortex 3 to 6 h after the end of the GSM or sham exposures, 
we found that GSM-EMF triggered functional alterations in neuronal networks, as indicated by significant 
reductions in evoked responses to acoustic stimuli and an increase in response duration. GSM exposure also 
induced changes in the morphology of brain immune cells localized in the auditory cortex such as microglia, 
which consisted in a significant growth of microglial cell processes [1]. A similar growth of microglial cell 
processes could be observed in the dorsal part of the cingulate cortex (mean SAR value: 2.94 W/kg), 24h after the 
end of the GSM-exposure but this effect did not persist 72h post exposure [2]. Reverse transcription-quantitative 
PCR analyses and a genome wide mRNA profiling were carried out 24h post exposure, in a dorsal area of the 
cerebral cortex (mean SAR value: 3.22 W/kg) and showed significant but moderate (fold changes <2) modulations 
of transcripts levels affecting 2.7% of the expressed genes, including genes encoding proinflammatory mediators 
and genes involved in protein ubiquitination or dephosphorylation [3]. GSM-induced protein dephosphorylations 
were directly observed in glutamate ionotropic receptors that are known to be involved in excitatory 
neurotransmission [2]. Importantly, assessments of neuronal activity, microglia cell morphology, gene 
expressions and glutamate receptor phosphorylation showed no significant effect in healthy rats that were 
submitted to a same head-only exposure to GSM-EMF [1, 2, 3]. Together our analyses uncover alterations in brain 
cell functions, morphologies or genes, which are triggered by GSM-1800 MHz under neuroinflammatory 
conditions. We also show that pathological processes affecting the brain can promote brain cell responses to GSM-
EMF. This research was supported by PNREST Anses (grants 2015/2 RF/12 and 2018/2 RF/1).

1. F. Occelli et al., "A Single Exposure to GSM- 1800MHz Signals in the Course of an Acute 
Neuroinflammatory Reaction can Alter Neuronal Responses and Microglial Morphology in the Rat Primary 

2. J. Lameth et al., "Acute Neuroinflammation Promotes Cell Responses to 1800 MHz GSM Electromagnetic 
Fields in the Rat Cerebral Cortex," Neurotox Res, 32, 3, Jun 03 2017, pp. 444-459, doi: 10.1007/s12640-017-
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Gene Responses Under Proinflammatory Conditions," Neurotox Res, 38, 1, Jun 2020, pp. 105- 123, doi: 