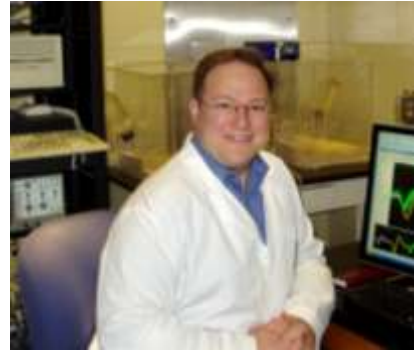


Alberto Eugenio Musto, MD, PhD

Assistant Professor – Research, Neurosurgery and Neuroscience

**Education:**

- 1991 Medicine. School of Medicine, Universidad de Buenos Aires (UBA), Argentina (AR)
- 2002 PhD, Neurobiology, School of Medicine. UBA, AR.
- 2002 Radiology, Board certified, Public Health Ministry. AR.
- 2002 Neurology, Board certified, Universidad de la Plata. AR.
- 2002 Post. Doc. LSUHSC, Neurosciences Ctr. New Orleans, LA. USA.
- 2005 Sr. Post. Doc. LSUHSC, Neurosciences Ctr. New Orleans, LA. USA.

Positions:

- 2007- present: Assistant Professor-Research in Neuroscience and Neurosurgery, Louisiana State University, Health Sciences Center, New Orleans, LA.
- 2002-2006: Postdoctoral Fellow, Louisiana State University, Health Sciences Center, New Orleans, LA USA, Neuroscience Center of Excellence.
- 2001-2002: Senior Chief. Medical Image Department. Sanatorio Guemes. Buenos Aires. AR
- 1999-2000: Chief of MRI and CT Division. Medical Image Department. Sanatorio Guemes. Buenos Aires. AR
- 1998-2002: Assistant Professor. Internal Medicine Department. Neurology Division. Hospital Municipal de San Isidro. AR.
- 1996-2002: Assistant Professor, Lecturer on Human Anatomy, Anatomy Department, School of Medicine. Universidad Austral, Pilar, AR
- 1995-1998: Resident of Neurology. Internal Medicine Department. Neurology Division. Hospital Municipal de San Isidro. AR
- 1994-1998: Research Fellowship: Tesis research conducted by scholarship from CONICET at Instituto de Neurobiologia. AR.
- 1992-1994: Neurophysiology Research Assistant, Neurobiology Institute, Consejo de Investigación Científica y Tecnológica (CONICET), Buenos Aires, AR.
- 1992-2002: Assistant Professor. Anatomy Department, School of Medicine, Universidad de Buenos Aires, AR

Current research:

Dr. Alberto E. Musto's laboratory is focusing in the basic mechanism/s that mediates the genesis of seizures (ictiogenesis) and the occurrence of spontaneous seizures (epileptogenesis). The central hypothesis is that failure of inhibitory neurotransmission mediated by GABA_A receptors lead to the initiation and propagation of seizures. Several repetitive seizures enhance the accumulation of platelet activating factor (PAF) that activates molecular pathways, which triggers inflammatory and degenerative process in the brain (Fig 1). Dr. Musto tests the mentioned hypothesis integrating his background of clinical neurology and neuroradiology into *in vivo* animal models of experimental epilepsy, using behavioral procedures, state of the art of electrophysiology *in vivo*, immunohistology techniques, biochemical protocols and novel chemical compounds (LAU-0901). Dr. Musto's laboratory hypothesizes that modulation of PAF activity through the PAF-antagonist receptor, LAU-0901, the neuroinflammation, neuronal damage and recurrent epileptic seizures will be attenuated. Dr. Musto collaborates with Dr. Nicolas G. Bazan in studying neuroprotective signaling in experimental epilepsy.

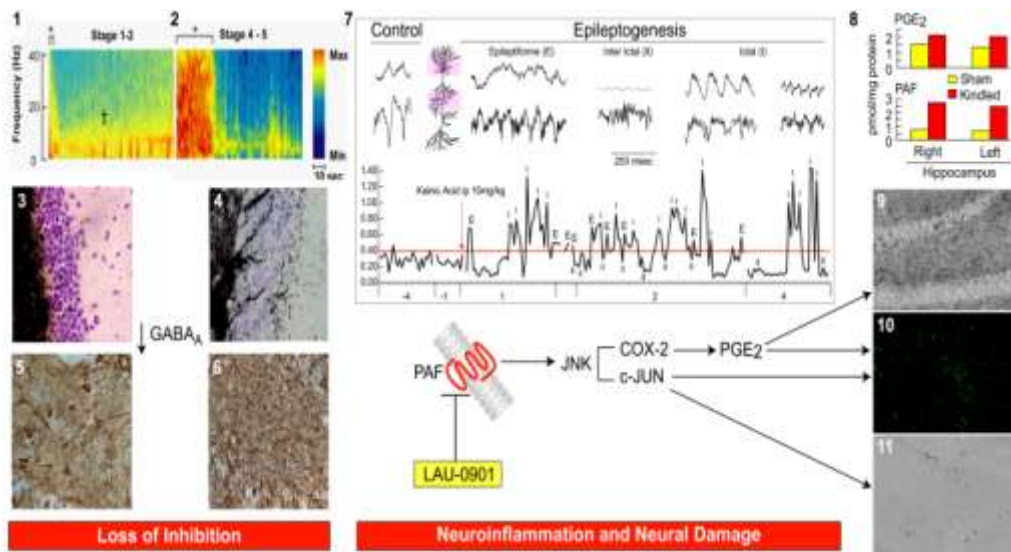


Figure 1: Proposed mechanism of the basic process involved in ictogenesis and epileptogenesis. Spectral profile from hippocampal EEG of induced partial seizure (1) and generalized seizures (2) associated with morphological changes in hippocampus using Tim's staining (3,4); Somatostatin IR for interneuronal cells (5,6). Generalized seizure is mediated by loss of GABA_A agonism and is associated with aberrant mossy fiber sprouting (4) and loss of SOM(6). 7: Local field potential (LFP) from dorsal hippocampus through silicon probe with multi-array electrode at different days before and after status epilepticus. LFP changes from stratum pyramidal (upper traces) and molecular layer (lower traces) of CA1. Epileptiform, interictal and ictal activity associated with disruption of amplitude ratio of these layers are observed during epileptogenesis. These electrophysiological changes are associated with PAF accumulation (8) that induces JNK and in turn increases PGE₂ (8) through COX-2 activation and c-jun gene expression. Hippocampal consequences of these activated molecular pathways: microgliosis (9), degenerative neurons (10) and loss of SOM interneurons (11).

55
56
57
58
59
60
61
62
63
64
65
66
67
68
69
70
71
72
73
74
75
76
77
78
79
80
81
82
83
84
85
86
87
88
89
90
91
92
93
94
95
96
97
98
99
100

Research Interests and Goals:

The long-term goal is to understand the molecular and cellular mechanisms of how the normal brain develops an impaired neuronal network and degenerative process during epileptogenesis. Using a combination of experimental approaches, I can identify the critical factors involve in the genesis of epileptic seizures. These results will be translated into alternative therapeutics pathways for treatment of epilepsy.

Selected Papers:

- Musto, AE**, MS Sammi, JF Hayes. Different phases of afterdischarge during rapid kindling procedure in mice (Submitted to *Epilepsy Research*, 2009.)
- Musto, M**, Hardy, F.R. Jackson, M.S. Samii, B. B. Chiappinelli, H. Thompson, V.L. Marcheselli Attenuation of epileptogenesis by hippocampal neuroprotectin D1 synthesis. Bazan, N.G., **A.E.** (In revision, 2008)
- NG. Bazan and **AE. Musto**. Inositol lipid signaling in synaptic activity, neuronal plasticity and epileptogenesis. *Encyclopedia of Basic Research in Epilepsy*, Elsevier, 2009 (in press).
- Cole-Edwards KK, **Musto AE**, Bazan NG. c-Jun N-terminal kinase activation responses induced by hippocampal kindling are mediated by reactive astrocytes.. *J Neurosci*. 2006 Aug 9; 26:8295-304.
- Musto AE**, Bazan NG. Diacylglycerol kinase epsilon modulates rapid kindling epileptogenesis. *Epilepsia*. 2006 Feb; 47 (2): 267-76.
- Lukiw WJ, Cui JG, **Musto AE**, Musto BC, Bazan NG. Epileptogenesis in diacylglycerol kinase epsilon deficiency up-regulates COX-2 and tyrosine hydroxylase in hippocampus.. *Biochem Biophys Res Commun*. 2005 Dec 9; 338:77-81.
- Musto AE**, Hardy M and Bazan NG Arachidonoyl-Inositol Lipid Signaling is required for Hippocampal Excitability in Kindling Epileptogenesis.. *Neurobiology of Lipids*, Vol. 3, 4, 2004
- Mc Dermott CM, LaHoste GJ, Chen C, **Musto A**, Bazan NG, Magee JC. Sleep deprivation causes behavioral, synaptic and membrane excitability alterations in hippocampal neurons. *J Neurosci*. 2003 Oct 22;23: 9687-95
- Marcheselli VL, Hong S, Lukiw WJ, Tian XH, Gronert K, **Musto A**, Hardy M, Gimenez JM, Chiang N, Serhan CN, Bazan NG. Novel docosanoids inhibit brain ischemia-reperfusion-mediated leukocyte infiltration and pro-inflammatory gene expression. *J Biol Chem*. 2003 Oct 31; 278 (44): 43807-17. Epub 2003 Aug 15. Erratum in: *J Biol Chem*. 2003 Dec 19; 278:51974.

Present Funding:

2007-2012: PJI- CoBRE (Center of Biomedical Research Excellence) NIH. Title: Mentoring Neuroscience in LA: A biochemical program to enhance neuroscience. Grant # P20RR016816. (N.Bazan, PI).