

BIOGRAPHICAL SKETCH

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NAME Maria F. M. Braga, D.D.S., Ph.D	POSITION TITLE Assistant Professor		
eRA COMMONS USER NAME			
EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)			
INSTITUTION AND LOCATION	DEGREE (if applicable)	YEAR(s)	FIELD OF STUDY
School of Dentistry, Federal University of Alagoas, Brazil	D.D.S.	1982	Dentistry
School of Medicine, University of Sao Paulo, Brazil	M.S.	1986	Pharmacology
University of Strathclyde, Scotland, UK	Ph.D.	1993	Physiology/Pharmacology

A. Positions and Honors.**Positions and Employment**

- 1986-1988 Assistant Professor of Pharmacology, Department of Physiology and Pharmacology, Federal University of Alagoas, Brazil.
- 1989-1993 Doctoral Student and Teaching Assistant, Department of Physiology and Pharmacology, University of Strathclyde, Scotland.
- 1993-1994 Postdoctoral Fellow, Strathclyde Institute for Drug Research, Glasgow, Scotland.
- 1994-1996 Associate Professor of Biophysics, Federal University of Rio de Janeiro, Brazil.
- 1996-2000 Postdoctoral Fellow, Department of Pharmacology and Experimental Therapeutics. University of Maryland – School of Medicine, Baltimore, MD.
- 2000-2004 Research Assistant Professor, Department of Psychiatry, Uniformed Services University of the Health Sciences, Bethesda, MD.
- 2004- Assistant Professor of Anatomy, Physiology and Genetics, Uniformed Services University of the Health Sciences, Bethesda, MD.
- 2005- Assistant Professor of Neuroscience, Uniformed Services University of the Health Sciences, Bethesda, MD.

Honors and Awards

- 1989-1993 CAPES Student Fellowship by the Brazilian Government for Graduate studies at the University of Strathclyde, Scotland.
- 1993-1994 Research Fellowship, University of Strathclyde, Scotland.

B. Selected peer-reviewed publications (in chronological order).

1. **Braga M.F.M.**, Harvey A.L., Rowan E.G. (1991). Effects of tacrine, velnacrine (HP029), suronacrine (HP128) and 3,4-diaminopyridine on skeletal neuromuscular transmission in vitro. **British Journal of Pharmacology** **102**: 909-915.
2. **Braga M.F.M.**, Anderson A.J., Harvey A.L., Rowan E.G. (1992). Apparent block of K⁺ currents in mouse motor nerve terminal by tetrodotoxin, μ -conotoxin and reduced sodium concentrations. **British Journal of Pharmacology** **106**: 91-94.
3. **Braga M.F.M.**, & Rowan E.G. (1992). Reversal by cysteine of the cadmium-induced block of skeletal neuromuscular transmission in vitro. **British Journal of Pharmacology** **107**: 95-100.
4. **Braga M.F.M.**, Rowan E.G., Harvey A.L., Bowman W.C. (1993). A prejunctional action of neostigmine on mouse neuromuscular preparations. **British Journal of Anaesthesia** **70**: 405-410.
5. **Braga M.F.M.**, Rowan E.G., Harvey A.L., Bowman, W.C. (1994). Interactions between suxamethonium and non-depolarizing neuromuscular blocking drugs. **British Journal of Anaesthesia** **72**: 198-204.
6. **Braga M.F.M.** & Rowan E.G. (1994). The pharmacological effects of cadmium on skeletal neuromuscular transmission. **General Pharmacology** **25** (8): 1729-1739.

7. **Braga M.F.M.**, Rowan E.G., Harvey A.L. (1995). Modification of ionic currents underlying action potentials in mouse nerve terminals by the thiol-oxidizing agent diamide. *Neuropharmacology* **34** (11): 1529-33.
8. Henning R.H., Rowan E.G., **Braga M.F.M.**, Nelemans A., Harvey A.L. (1996). The prejunctional inhibitory effect of suramin on neuromuscular transmission in vitro. *European Journal of Pharmacology* **301** (1-3): 91-97.
9. Camara A.L., **Braga, M.F.M.**, Rocha E.S., Santos, M.D., Cortes W.S., Cintra W.M., Aracava Y., Maelicke A., Albuquerque E.X. (1997). Methamidophos: an anticholinesterase without significant effects on postsynaptic receptors or transmitter release. *Neurotoxicology* **18** (2): 589-602.
10. Edson X. Albuquerque, Edna F.R. Pereira, **Maria F.M. Braga**, Manickavasagom Alkondon. (1998). Contribution of nicotinic receptors to the function of synapses in the central nervous system: The action of choline as a selective agonist of $\alpha 7$ nicotinic receptors. *Journal of Physiology* (Paris) **92**: 309-316.
11. Edson X. Albuquerque, Edna F.R. Pereira, **Maria F.M. Braga**, Hiroaki Matsubayashi, Manickavasagom Alkondon. (1998). Neuronal nicotinic receptors modulate synaptic function in the hippocampus and are sensitive to blockade by the convulsant strychnine and by the anti-Parkinson drug amantadine. *Toxicology Letters* **102-103**: 211-218.
12. **Maria F.M. Braga**, Edna F.R. Pereira, Edson X. Albuquerque. (1999). Nanomolar concentrations of lead inhibit glutamatergic and GABAergic transmission in hippocampal neurons. *Brain Research* **826**: 22-34.
13. **Maria F.M. Braga**, Edna F.R. Pereira, Murilo Marchioro, Edson X. Albuquerque. (1999). Lead increases tetrodotoxin-insensitive spontaneous release of glutamate and GABA from hippocampal neurons. *Brain Research* **826**: 10-21.
14. Manickavasagom Alkondon, **Maria F.M. Braga**, Edna F.R. Pereira, Alfred Maelicke, Edson X. Albuquerque. (2000). $\alpha 7$ Nicotinic acetylcholine receptors and modulation of gabaergic synaptic transmission in the hippocampus. *European Journal of Pharmacology* **393**: 59-67.
15. Emerson P. Pecanha, Carlos A.M.Fraga, Eliezer J. Barreiro, **Maria F.M. Braga**, Edna F.R. Pereira and Edson X. Albuquerque. (2001). Synthesis and Pharmacological Evaluation of a New 2-Azabicyclo[3.3.0]octane Derivative. *Journal of the Brazilian Chemical Society*, **12**: 408-412.
16. **Maria F. M. Braga**, Vassiliki Aroniadou-Anderjaska, Robert M. Post and He Li. (2002). Lamotrigine reduces spontaneous and evoked GABA_A receptor-mediated synaptic transmission in the basolateral amygdala: Implications for its effects in seizure- and affective disorders. *Neuropharmacology*, **42**: 522-529.
17. **Maria F. M. Braga**, Vassiliki Aroniadou-Anderjaska, Jianwu Xie, and He Li. (2003). Bidirectional Modulation of GABA Release by Presynaptic Glutamate Receptor 5 Kainate Receptors in the Basolateral Amygdala. *Journal of Neuroscience*, **23** (2): 442-452.
18. **Maria F. M. Braga**, Vassiliki Aroniadou-Anderjaska and He Li. (2004). The physiological Role of Kainate Receptors in the Amygdala. *Molecular Neurobiology*, **30**(2):127-42.
19. **Maria F.M. Braga**, Edna F.R. Pereira, Arpad Mike, Edson X. Albuquerque. (2004). Pb²⁺ via protein kinase C inhibits nicotinic cholinergic modulation of synaptic transmission in the hippocampus. *Journal of Pharmacology and Experimental Therapeutics*, **311**(2): 700-710.
20. **Maria F. M. Braga**, Christopher J. Hough, Sean T. Manion, Vassiliki Aroniadou-Anderjaska, and He Li. (2004). Stress impairs α_{1A} adrenoceptor-mediated noradrenergic facilitation of GABAergic transmission in the basolateral amygdala. *Neuropsychopharmacology*, **29**(1): 45-58.
21. Aroniadou-Anderjaska V, Qashu F, **Braga MF**. (2007). Mechanisms regulating GABAergic inhibitory transmission in the basolateral amygdala: implications for epilepsy and anxiety disorders. *Amino Acids*; **32**(3):305-15.
22. Vassiliki Aroniadou-Anderjaska, Brita Fritsch, Felicia Qashu and **Maria F.M. Braga**. (2007). Pathology and Pathophysiology of the Amygdala in Epileptogenesis and Epilepsy. *Epilepsy Research*, *in press*.

C. Research Support

Ongoing Research Support

R070SG (Braga - PI)	06/30/2005 - 09/30/2008	1.2 calendar
DOD/USUHS		\$300,000 (direct cost)
GABAergic Transmission in the Amygdala and Anxiety Disorders		

Principal Investigator/Program Director (Last, First, Middle): Braga, Maria F.M.

The major goal of this project is to investigate alterations in GABAergic synaptic transmission in the basolateral amygdala of fear-conditioned mice.

F170TM-C4 (Braga - PI) 05/01/2006 – 04/31/2008 1.2 calendar
DOD/DBSCIP \$208,650 (direct cost)
Epileptogenesis in the Amygdala and the Role of GluR5 Kainate Receptors

The major goal of this project is to determine the role of GluR5 kainate receptors in epileptogenesis, which is the process whereby progressive, pathophysiological alterations in neuronal networks after an acute brain insult, such as traumatic brain injury, lead to the development of epilepsy.

1.E0021-07-US-C (Braga - PI) 10/01/2006 – 09/30/2009 3.6 calendar
Defense Threat Reduction Agency \$615,000 (direct cost)
Efficacy of GluR5 Kainate Receptor Antagonists and Caramiphen against Nerve Agent-Induced Brain Seizures and Neuronal Damage

The major goals of this project is 1) to determine the involvement of the GABAergic and glutamatergic neurotransmission systems in the mechanisms by which organophosphates such as the nerve agent SARIN induces brain seizures, and 2) to determine whether a novel and an FDA-approved, mixed GluR5 kainate receptor antagonist, alone or in combination with the anticholinergic agent caramiphen, can prevent the initiation or block the expression of SARIN-induced seizures.

1 U01 NS058162-01(Braga - PI) 10/01/06 – 09/30/11 3.6 calendar
National Institutes of Health – NINDS \$1,246,898 (direct cost)
Efficacy of GluR5 Kainate Receptor Antagonists against Soman- Induced Seizures and Neuropathology

The major goal of this project is to evaluate the effectiveness of the GluR5 kainate receptor antagonists UBP302 and LY293558, a new class of anticonvulsant drugs, against chemical warfare nerve agents. We are testing the effectiveness of these two compounds by using *in vivo* exposure of rats to soman, as well as *in vitro* exposure of rat brain slices to soman. UBP302 and LY293558 are administered as a prophylactic treatment before exposure to soman or as a therapy at different time points post-exposure. The efficacy of these antagonists against seizures will be correlated with their efficacy in preventing brain damage, as well as pathophysiological alterations in the amygdala and hippocampus brain regions. There is no overlap between the research proposed in grant # 1.E0021-07-US-C (DTRA) and the research proposed in this NIH grant. The GluR5 antagonist LY293558 will be used in both research projects. However, it will be used to answer different questions.

Completed Research Support

USO0488HQ He Li (PI) 03/01/2004 - 02/28/2006
Department of Defense
Protection of Traumatic Stress-Induced Adrenergic Impairment of the Amygdala

This study investigated the effectiveness of α_{1A} adrenoceptor antagonists in preventing the stress-induced alterations in the excitability of the amygdala.
Role: Co-Investigator