CURRICULUM VITAE

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PART I: General Information

Name: EDWARD GEORGE MELONI

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Place of Birth: Beverly MA

Education:

1991	B.A. (Biopsychology), Bates College
1994	M.S. (Neuroscience), Yale University
2000	PH.D. (Neuroscience), Yale University

Postdoctoral Training:

01/00-12/01	Postdoctoral Fellow, Psychiatry, Emory University
01/01-12/02	Postdoctoral Fellow, Neurology, Brigham and Women's Hospital

Academic Appointments:

2001-2002	Research Fellow in Neurology, Neurology-Brigham and Women's Hospital, Boston, MA
2002-	Instructor in Psychiatry, Psychiatry-McLean Hospital, Belmont, MA
2006-	Assistant Professor of Psychiatry, McLean Hospital, Belmont, MA

Hospital or Affiliated Institution Appointments:

01/02-	Assistant Psychobiologist, McLean Hospital, Belmont, MA

Other Professional Positions and Major Visiting Appointments:

2006-	Scientific Advisory Board Member, Med Associates, Inc., St. Albans, VT

Major Committee Assignments:

2004-	Institutional Animal Care and Use Committee, Member, McLean Hospital

Professional Societies:

1992-	Society for Neuroscience, Member
2005-	New York Academy of Sciences, Member
2006-	Anxiety Disorders Association of America, Member

Editorial Boards:

2004-	Reviewer, Journal of Neuroscience
2004-	Reviewer, Neuroscience Letters
2004-	Reviewer, Physiology and Behavior
2004-	Reviewer, Biological Psychiatry
2006-	Reviewer, Neuropsychopharmacology

Awards and Honors:

1991-1996	Air Force Office of Scientific Research Fellowship, Yale University
1994-1995	John F. Enders Fellowship, Yale University
2004-2006	Kaneb Fellow in Psychiatry, McLean Hospital
2005-2007	Young Investigator Award, National Alliance for Research on Schizophrenia and Depression (NARSAD)
2005-2005	NYAS Fellowship Award, New York Academy of Sciences
2006	Career Development Travel Award, Anxiety Disorders Association of America (ADAA)
2006	Travel Award, American College of Neuropsychopharmacology (ACNP)
2007	Pope Award for Young Investigators, McLean Hospital

Part II: Research, Teaching, and Clinical Contributions

A. Narrative report of Research, Teaching, and Clinical Contributions

I am interested in the neural systems underlying fear and anxiety with broad relevance to the study of mood and affective disorders. My goal is to identify the various brain substrates (anatomical and molecular) that are involved in the development and expression of psychiatric diseases such as anxiety disorders (e.g. generalized anxiety, post-traumatic stress disorder, and social anxiety disorder), depression and bipolar disorder. With this knowledge, it is my hope that novel therapies can be developed to target these substrates and provide beneficial treatments, or ultimately a cure, for these debilitating diseases. Along these lines, I have currently been studying the role of a particular brain area, the bed nucleus of the stria terminalis (BNST), in the development and manifestation of anxiety-like behaviors. I have recently found that pharmacological inactivation of the BNST enhances dramatically the level of fear and anxiety exhibited by animals in our behavioral paradigm (fear-potentiated startle). These data suggest that the BNST is a critical brain area for controlling the expression of fear and anxiety. Moreover, we have found that the activity of a specific molecule (CREB) in the BNST is directly related to the expression level of anxiety-like behavior (Meloni et. al., 2006a). Together, these findings may have application to the development of new treatments for anxiety disorders. I have recently received a NARSAD Young Investigator Award and an RO3 (N.I.H., 1 RO3 MH076230-01) to further explore the role of CREB in the BNST; using viral-mediated gene transfer we intend to directly increase or decrease the function of CREB in the BNST and assess the effects on anxiety-like behaviors.

Another line of research that I am currently pursuing involves the neural systems mediating the adaptive and maladaptive response to stress. Specifically, I have been studying the effects of the neuropeptide corticotropin releasing factor (CRF) on behavior. CRF is the principle peptide involved in regulating the stress response and when centrally administered to animals, it produces many of the physiological and behavioral changes seen in both depression and anxiety. In my experiments, I have been using the enhancement of the acoustic startle response by CRF as an animal model of stress and anxiety-like states. The focus of my experiments has been to study how other neurotransmitter systems (e.g. dopamine, norepinephrine, serotonin and anxiolytics/antidepressants that act through these neurotransmitter systems) may interact with CRF systems to affect stress-related behavior. For example, I have recently found that blockade of dopamine D1 receptor-mediated neurotransmission significantly reduces the stress/anxiety-like effects of CRF on startle (Meloni et al., 2006b). This dopamine-CRF interaction may be mediated by a novel dopaminergic pathway I have identified, originating in the periaqueductal gray and terminating in the BNST, where dopamine terminals make heavy synaptic contact with the CRF-containing cells in this brain area. I believe these studies have the potential to identify new targets for pharmacological intervention that could be used in treatment for psychiatric conditions where the brain's CRF system is dysfunctional.

N/AN/A

B. Funding Information

2002-	Investigator, Foundation, Stanley Foundation, Development of animal models of bipolar disorder
2005-2007	P.I., Foundation, NARSAD, Role of CREB function within the bed nucleus of the stria terminalis (BNST) in fear-, anxiety- and depressive-like behaviors
2006-2008	P.I., N.I.H., 1 RO3 MH076230-01, Role of CREB function in the bed nucleus of the stria terminalis (BNST) in fear and anxiety-like behaviors

D. Report of Teaching

1. Local contributions

a. Medical School Courses

2005	Psychiatry Residents Training			
			contact time	*prep time*
	Lecturer	10 Resident	2 hours/year for 1 year(s)	none reported

c. Local Invited Presentations

Seminar

2004 The role of corticotropin-releasing factor (CRF) in affective disorders, Northeastern University
Lecturer: 10 participants, 2 hours contact time per year, no prep time reported

e. Advisory and Supervisory Responsibilities in Clinical or Laboratory Setting

2005- 1 Graduate Students for 50 hrs/year, advising research studies, Harvard University at McLean Hospital

g. Advisees/Trainees

Training Duration	*Name*	*Current Position*
2002-2004	Alexandra Jackson	Graduate Student
2004-2006	Lyle Gerety	Medical Student

2. Regional, national, or international contributions

a. Invited Presentations

Regional

2006 Short Courses in Neuroscience: Animal models of fear conditioning, Med Associates *[Seminar]*

National

2002 Nigro-collicular modulation of motor reflexes and brain circuits involved in fear and anxiety-like behaviors, Winter Conference on Brain Research (WCBR) *[Invited Lecture]*

Part III: Bibliography

Original Articles

1. Lee Y, López DE, Meloni EG, Davis M. A primary acoustic startle pathway: obligatory role of cochlear root neurons and the nucleus reticularis pontis caudalis. J Neurosci. 1996;16(11):3775-89.
2. Medina R, Grishina G, Meloni EG, Muth TR, Berlot CH. Localization of the effector-specifying regions of Gi2alpha and Gqalpha. J Biol Chem. 1996;271(40):24720-7.
3. Meloni EG, Davis M. The dorsal cochlear nucleus contributes to a high intensity component of the acoustic startle reflex in rats. Hear Res. 1998;119(1-2):69-80.
4. Meloni EG, Davis M. Enhancement of the acoustic startle response in rats by the dopamine D1 receptor agonist SKF 82958. Psychopharmacology (Berl). 1999;144(4):373-80.
5. Meloni EG, Davis M. Muscimol in the deep layers of the superior colliculus/mesencephalic reticular formation blocks expression but not acquisition of fear-potentiated startle in rats. Behav Neurosci. 1999;113(6):1152-60.
6. Meloni EG, Davis M. GABA in the deep layers of the superior Colliculus/Mesencephalic reticular formation mediates the enhancement of startle by the dopamine D1 receptor agonist SKF 82958 in rats. J Neurosci. 2000;20(14):5374-81.
7. Meloni EG, Davis M. Synergistic enhancement of the acoustic startle reflex by dopamine D1 and 5-HT1A agonists and corresponding changes in c-Fos expression in the dorsal raphe of rats. Psychopharmacology (Berl). 2000;151(4):359-67.
8. Meloni EG, Davis M. Enhancement of the acoustic startle response by dopamine agonists after 6-hydroxydopamine lesions of the substantia nigra pars compacta: corresponding changes in c-Fos expression in the caudate-putamen. Brain Res. 2000;879(1-2):93-104.
9. Goldberg MS, Fleming SM, Palacino JJ, Cepeda C, Lam HA, Bhatnagar A, Meloni EG, Wu N, Ackerson LC, Klapstein GJ, Gajendiran M, Roth BL, Chesselet MF, Maidment NT, Levine MS, Shen J. Parkin-deficient mice exhibit nigrostriatal deficits but not loss of dopaminergic neurons. J Biol Chem. 2003;278(44):43628-35.
10. Meloni EG, Davis M. The substantia nigra pars reticulata mediates the enhancement of startle by the dopamine D1 receptor agonist SKF 82958 in rats. Psychopharmacology (Berl). 2004;174 (2):228-36.
11. Carlezon WA, Rohan ML, Mague SD, Meloni EG, Parsegian A, Cayetano K, Tomasiewicz HC, Rouse ED, Cohen BM, Renshaw PF. Antidepressant-like effects of cranial stimulation within a low-energy magnetic field in rats. Biol Psychiatry. 2005;57(6):571-6.
12. Meloni EG, Jackson AV, Cohen BM, Carlezon WA. Corticotropin-releasing factor from the rat brain measured by protein immunoblot. Peptides. 2005;26(11):2252-6.
13. Goussakov I, Chartoff EH, Tsvetkov E, Gerety LP, Meloni EG, Carlezon WA, Bolshakov VY. LTP in the lateral amygdala during cocaine withdrawal. Eur J Neurosci. 2006;23(1):239-50.
14. Meloni EG, Gerety, LP, Knoll, AT, Cohen BM, Carlezon WA. Behavioral and anatomical interactions between dopamine and corticotropin-releasing factor (CRF) in the rat. Journal of Neuroscience. 2006;26(14):3855-3863.
15. Youngs RM, Chu MS, Meloni EG, Naydenov A, Carlezon WA, Konradi C. Lithium administration to preadolescent rats causes long-lasting increases in anxiety-like behavior and has molecular consequences. Journal of Neuroscience. 2006;26(22):6031-6039.

Proceedings of Meetings

1. Meloni EG, Jackson AV, Cohen BM, Carlezon WA. Role of the bed nucleus of the stria terminalis (BSNT) in the expression of conditioned fear. In: Annals of the New York Academy of Sciences;

September 11-13, 2005; Mount Sinai School of Medicine. New York (NY): New York Acadamy of Sciences;2006. p. 538-541.

Thesis

1. Meloni, EG. Dopaminergic modulation of reflex behavior: studies with the acoustic startle reflex in rats. New Haven (CT): Yale University;2000.

Abstracts

1. Knoll AT, Gerety LP, Cohen BM, Carlezon WA, Meloni EG. Behavioral effects of 6-hydroxydopamine lesions of the bed nucleus of the stria terminalis (BNST) measured with the fear-potentiated startle paradigm in rats. Society for Neuroscience Abstracts. 2005;35.
2. Meloni EG, Gerety LP, Knoll AT, Cohen BM, Carlezon WA. Behavioral and anatomical interactions between dopamine and corticotropin-releasing factor (CRF). ACNP Abstracts. 2005;44.
3. Meloni EG, Gerety L, Cohen B, Carlezon WA. Anxiety-like effects of corticotropin-releasing factor (CRF) are reduced by 6-hydroxydopamine lesions of the bed nucleus of the stria terminalis (BNST). ADAA Abstracts. 2006;26.