

ELENA BATTAGLIOLI, CV

EDUCATION AND TRAINING:

- 2004-now Assistant Professor in the Dept. of Molecular Biotechnology and Translational Medicine
- 2002-2003 Research Scientist, The Research Foundation SUNY in Stony Brook, Dept. of Neurobiology and Behavior, SUNY in Stony Brook, NY
- 1998-2002 Post doc, Howard Hughes Medical Institute, Department of Neurobiology and Behavior, SUNY in Stony Brook, NY.
- 1997 PhD in Cellular and Molecular Biology, Dept. of Pharmacology and Medical Toxicology, University of Milan.
- 1993 Graduate School, University of Milan, Dept. of Genetics and Microbiology. 110/110 cum laudae.
- 1987 BS, Maturità Scientifica

RESEARCH INTEREST

Principal aim of my research interests is to unravel how alternative splicing enhances LSD1 epigenetic tuneability in the mammalian nervous system. Splicing is an important source of proteome diversity. Indeed, as a result of a neuro-specific “exon-gain” event, the active-site of LSD1 is expected to be partly altered, suggesting that splicing may contribute to a fine regulation of enzyme activity in the brain.

The long-range research goal of my research group is to understand the precise control of gene expression that determines the acquisition of a cell phenotype during development, as well as its maintenance throughout cell life span. I am interested in the study of the flavin-dependent histone demethylase activity of LSD1 (Lysine specific demethylase 1), a key enzymatic component of the CoREST co-repressor machinery focusing in particular on the neuronal isoform of this enzyme recently identified in our laboratory. The primary aim is now to study the relationships between the unique neurospecific epigenetic function of LSD1 and genes known to be involved in mental retardation, testing the hypothesis that an altered activity of LSD1 in the brain may affect the acquisition of specialized and complex neuronal functions. Indeed, while LSD1 can be found from *S. pombe* till humans, the neurospecific isoforms of LSD1 are a mammal prerogative. To test this hypothesis, we want to determine the neurological consequences of loosing expression of the neurospecific isoforms of LSD1. Mutant animals will help to unravel the specific neuronal functions in which LSD1 takes part and the nature the target genes directly regulated by its epigenetic activity. We also plan to assess whether specific loss of neuronal LSD1 affects the social and cognitive behaviors of these mice.

PUBBLICATIONS

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Zuccotti P, Cartelli D, Stropi M, Pandini V, Venturin M, Aliverti A, **Battaglioli E**, Cappelletti G, Riva P. Centaurin interacts with tubulin and stabilizes microtubules. *PLoS One*. 2012;7(12):e52867. doi: 10.1371/journal.pone.0052867.

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B. Bodega, G.D. Ramirez, F. Grasser, S. Cheli, S. Brunelli, M. Mora, R. Meneveri, A. Marozzi, S. Mueller, **E. Battaglioli**, E. Ginelli. "Remodeling of the chromatin structure of the facioscapulohumeral muscular dystrophy (FSHD) locus and upregulation of FSHD-related gene 1 (FRG1) expression during human myogenic differentiation". *BMC Biol.* 7:41, 2009.

F. Forneris, **E. Battaglioli**, A. Mattevi, C. Binda. New roles of flavoproteins in molecular cell biology: histone demethylase LSD1 and chromatin. *FEBS J.* 276:4304-4312. Review, 2009.

A. Karytinis, F. Forneris, A. Profumo, G. Ciossani, **E. Battaglioli**, C. Binda and A. Mattevi "A novel mammalian flavin-dependent histone demethylase". *J Biol Chem.* 2009

F. Forneris, C. Binda, **E. Battaglioli** and A. Mattevi. "Lsd1: Oxidative chemistry for multifaceted functions in chromatin regulation". *Trends Biochem Sci.* 33:181-9, 2008.

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F. Forneris, C. Binda, M.A. Vanoni, A. Mattevi and **E. Battaglioli**. "Demethylation pathways for histone methyllysine residues". 24th Volume of "The Enzymes"(Elsevier/ Academic Press) 229-242, 2006.

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N. Ballas#, **E. Battaglioli**#, F. Atouf, M.E. Andres, J. Chenoweth, M. Anderson, C. Burger, M. Moniwa, J. Davie, W.J. Bowers, H.J. Federoff, D.W. Rose, M.G. Rosenfeld, P. Brehm and G. Mandel. "Regulation of Neuronal Traits by a Novel Transcriptional Complex", *Neuron*, 3. 353-365 (2001). # Equal contribution

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