

DATOS PERSONALES		Fecha del CVA	30/01/2024
Nombre y apellidos	Lucía Tabares Domínguez		
Identificadores de investigación	Researcher ID	K-8768-2015	
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Situación profesional actual

Institución	Universidad de Sevilla		
Dpt./Centro	Fisiología Médica y Biofísica / Facultad de Medicina		
Dirección	Avda. Sánchez Pizjuán 4 – 41009 Sevilla (España)		
Teléfonos	954556574	email	ltabares@us.es
Categoría profesional	Catedrática	Fecha de inicio	03/07/2009
Código UNESCO	249000- Neurosciences		
Palabras clave	Motor neurons, spinal muscular atrophy, synaptic transmission		

Formación académica (Títulos, instituciones, fechas)

Licenciatura/Master/Doctorado	Universidad	Año
Licenciada en Medicina y Cirugía	Sevilla	1981
Doctora en Medicina y Cirugía	Sevilla	1986

Historial y cualificación profesional

2009-	Catedrática.Universidad de Sevilla Dept. Fisiología Médica y Biofísica
2006	Visiting Professor, Univ. Colorado Dept. of Medical Physiology and Biophysics
2001-2002	Visiting Professor, Rinat Neuroscience, Palo Alto (CA)
1989-2009	Profesor Titular. Universidad de Sevilla Dept. Fisiología Médica y Biofísica
1989-1990	Research Fellow, Mayo Clinic (MN)
1988-1989	Research Associate, Marine Biological Laboratory (MA)
1987-1988	Research Associate, University of Pennsylvania (PA)
1982-1986	Profesor no Numerario Univ. de Sevilla Dept. Fisiología Médica y Biofísica

Resumen personal

Mi formación es en Electrofisiología e Imagen. Durante mi doctorado, descubrí que las células suprarrenales corticales son eléctricamente excitables y caractericé sus conductancias iónicas. En el postdoctorado trabajé en los mecanismos de permeación de los canales de calcio con Clay Armstrong en la Univ. de Pensilvania y el MBL en Woods Hole (EE.UU.). Durante un segundo postdoctorado, en el laboratorio de David Clapham, Clínica Mayo (Rochester, MN), caractericé dos canales de cloruro en la membrana del núcleo celular. Cuando regresé a España conseguí una plaza de profesor titular y monté mi laboratorio donde estudié la señalización del calcio intracelular y exocitosis en células neuroendocrinas. Tomé un año sabático en 2001 (15 meses) en Rinat Neuroscience Corp. (empresa biotecnológica, Palo Alto; CA, EE. UU.) donde estudié modelos de ratón con ELA con técnicas de electrofisiología. En 2002 regresé a mi laboratorio y comencé mi trabajo en Atrofia Muscular Espinal (AME) con el objetivo de investigar los defectos de la neurotransmisión y las alteraciones morfológicas sinápticas en esta enfermedad y en otros modelos murinos de sinaptopatías. Paralelamente, estudié cómo se relaciona la organización de las zonas activas con la regulación de la transmisión sináptica, mediante imagen dinámica de la exo-/endocitosis en las terminales nerviosas motoras en ratones transgénicos sinaptopHluorin. Saqué la Catedra en 2009.

Financiación pasada y actual

Financiación ininterrumpida desde 1991 por parte del Ministerio de Ciencia e Innovación. Otras: Integrated Project (VI Framework Program of the EU), Muscular Dystrophy Association (USA), GENOME, Marató Foundation, SMA Europe. Actual: MCIN (PID2019-110272RB-I00). (IP).

Participación en R&D y contratos con empresas:

“Effect of some antibodies on one or more mouse models of neurodegenerative diseases”

Rinat Neuroscience (Palo Alto, CA, US). 2003.

Patentes

Tabares, L., Lin, J., Rosenthal, A. Title: Methods for Treating Lower Motor Neuron Diseases. PCT/US2006/016046; USA application # 60/675393; Priority date: 26 April 2005. Actual owner: Rinat Neuroscience Corporation (Pfizer).

Premios y otras actividades profesionales

1986 Premio Extraordinario Tesis Doctoral Universidad de Sevilla

1987-89 Fogarty International Fellowship (Fullbright) (NIH)

Servicios como consultor científico y pertenencia a sociedades científicas

Associate editor for Frontiers (since 2022)// Associate editor for Biomolecules (since 2022)
Scientific Advisory Board European Neuroscience Institute, Göttingen (Germany), 2010-16
Member Research panel of multicenter grants, German Research Foundation (DFG), Ulm Univ. (2016) & Univ. Leipzig/Würzburg (2017) (Germany), and Project evaluator since 2018
Evaluator Muscular Dystrophy UK Project (2019)//Reviewer for Full Professorship (2016), Royal Holloway Univ. London (UK)//Member Parkinson's UK Projects evaluator (2016)
Member of evaluating committees for FPU fellowships (2014 y 2015)//Member of evaluating committee for 'Científicos Titulares del CSIC' (2011)//Council Member Spanish Biophysical Society (SBE) (2010-13)//Program Committee Member Spanish Neuroscience Society (2009-13)//Member Society for Neuroscience (USA) (2007-12)//Projects evaluator Spanish Agencia Nacional de Evaluación y Prospectiva (ANEP)//Projects evaluator Spanish Instituto de Salud Carlos III//Member of evaluating committees for 'Titulares y Catedráticos de Universidad'//Member of evaluating committees for Ramón and Cajal y Juan de la Cierva programs//Member Spanish Neuroscience Society, since 2005//Member American Biophysical Society (2001-09)//Corporate MBL (Woods Hole, USA (1991-1995)//Member Mayo Alumni Association (1990-1995) //Miembro SECF desde 1991//Member European Science Foundation (1984-89)// Peer-reviewer for more than 20 journals.

Labor docente

Profesora de Fisiología (Medicina) desde 1986. Directora de Tesis doctorales desde 1991. Coordinadora de programas de doctorado en Fisiología y Biología Celular.

PUBLICACIONES CIENTÍFICAS RELEVANTES

Kim JK et al. A spinal muscular atrophy modifier implicates the SMN protein in SNARE complex assembly at neuromuscular synapses. **Neuron**. 2023 S0896-6273(23)00082-X.

Franco-Espin J et al. SMN Is Physiologically Downregulated at Wild-Type Motor Nerve Terminals but Aggregates Together with Neurofilaments in SMA Mouse Models. **Biomolecules**. 2022 12(10):1524.

Bermedo-García F et al. Functional regeneration of the murine neuromuscular synapse relies on long-lasting morphological adaptations. **BMC Biol**. 2022 20(1):158.

Tabares L, Rizzoli SO. Editorial: Molecular Nanomachines of the Presynaptic Terminal. **Front Synaptic Neurosci**. 2022 14:941339.

Lopez-Manzaneda M et al. Presynaptic Mitochondria Communicate with Release Sites for Spatio-Temporal Regulation of Exocytosis at the Motor Nerve Terminal. **Front Synaptic Neurosci**. 2022;14:858340.

Lopez-Manzaneda M et al. Calcium is reduced in presynaptic mitochondria of motor nerve terminals during neurotransmission in SMA mice. **Hum Mol Genet.** 2021 30(8):629-643.

Tejero R et al. R-Roscovitine Improves Motoneuron Function in Mouse Models for Spinal Muscular Atrophy. **iScience.** 2020 23(2):100826.

Delezie J et al. BDNF is a mediator of glycolytic fiber-type specification in mouse skeletal muscle. **PNAS.** 2019;116(32):16111-16120.

Tejero R. et al. Maturation and heterogeneity of vertebrate motor synapses. **Current Opin. Physiol.** 2018; 4(1-6).

Bachiller S et al. HERC1 Ubiquitin Ligase Is Required for Normal Axonal Myelination in the Peripheral Nervous System. **Mol Neurobiol.** 2018 55(12):8856-8868.

Arumugam S et al. Smn-Deficiency Increases the Intrinsic Excitability of Motoneurons. **Front Cell Neurosci.** 2017. 11:269.

Lopez-Ortega E et al. CSP α , a Molecular Co-chaperone Essential for Short and Long-Term Synaptic Maintenance. **Front Neurosci.** 2017.11:39.

Tejero R et al. Synaptotagmin-2, and -1, linked to neurotransmission impairment and vulnerability in Spinal Muscular Atrophy. **Hum Mol Genet.** 2016. 25(21):4703-4716.

Rizzoli SO, Tabares L. Editorial: Molecular Nanomachines of the Presynaptic Terminal. **Front Synaptic Neurosci.** 2016. 8:27.

Cano R, Tabares L. The Active and Periaxial Zone Organization and the Functional Properties of Small and Large Synapses. **Front Synaptic Neurosci.** 2016. 8:12.

Wu YJ et al. Syntaxin 1B is important for mouse postnatal survival and proper synaptic function at the mouse neuromuscular junctions. **J Neurophysiol.** 2015. 114(4):2404-17.

Arnold AS et al. Morphological and functional remodeling of the neuromuscular junction by skeletal muscle PGC-1 α . **Nature Commun.** 2014. 5:3569.

Ruiz R et al. α -Synuclein A30P decreases neurodegeneration and increases synaptic vesicle release probability in CSP α -null mice. **Neuropharmacol.** 2014.76 Pt A:106-17.

Krieger F et al. Fast motor axon loss in SMARD1 does not correspond to morphological and functional alterations of the NMJ. **Neurobiol Dis.** 2013. 54:169-82.

Ackermann B et al. Plastin 3 ameliorates spinal muscular atrophy via delayed axon pruning and improves neuromuscular junction functionality. **Hum Mol Genet.** 2013. 22(7):1328-47.

Cano R et al. Structural and functional maturation of active zones in large synapses. **Mol Neurobiol.** 2013. 47(1):209-19.

Cano R et al The functional landscape of a presynaptic nerve terminal. **Cell Calcium.** 2012;52(3-4):321-6.

Torres-Benito L et al. SMN requirement for synaptic vesicle, active zone and microtubule postnatal organization in motor nerve terminals. **PLoS One.** 2011; 6(10):e26164.

Torres-Benito L et al. Synaptic defects in spinal muscular atrophy animal models. **Dev Neurobiol.** 2012;72(1):126-33. Review.

Ruiz R et al. Active zones and the readily releasable pool of synaptic vesicles at the neuromuscular junction of the mouse. **J Neurosci.** 2011;31(6):2000-8.

Tabares L, Betz WJ. Multiple functions of the vesicular proton pump in nerve terminals. **Neuron.** 2010;68(6):1020-2.

Ruiz R et al. Altered intracellular Ca²⁺ homeostasis in nerve terminals of severe spinal muscular atrophy mice. **J Neurosci.** 2010;30(3):849-57.

Simon CM et al. Ciliary neurotrophic factor-induced sprouting preserves motor function in a mouse model of mild spinal muscular atrophy. **Hum Mol Genet.** 2010; 19(6):973-86.

Gaffield MA et al. The spatial pattern of exocytosis and post-exocytic mobility of synaptotagmin in mouse motor nerve terminals. **J. Physiol.**, 587 (6):1187-1200, 2009.

Gaffield MA et al. Preferred sites of exocytosis and endocytosis colocalize during high but not lower frequency stimulation in mouse motor nerve terminals. **J. Neurosci.** 29(48):15308-16, 2009.

Ruiz R et al. Cysteine string protein- α is essential for the high calcium sensitivity of exocytosis in a vertebrate synapse. **Eur J. Neurosci.** 27(12):3118-3131, 2008.

Cabanes C et al. Neuroprotective effect of adult hematopoietic stem cells in a mouse model of motoneuron degeneration. **Neurobiol. Dis.** 26(2):408-18, 2007.

Tabares L et al. Monitoring Synaptic Function at the Neuromuscular Junction of a Mouse Expressing Synaptotagmin. **J. Neurosci.** 27:5448-5460, 2007.

Schmitz F*, Tabares* L et al. *equal contribution. CSP α -deficiency causes massive and rapid photoreceptor degeneration. **PNAS** 103(8):2926-31, 2006.

Moraleta JM et al. Adult stem cell therapy: Dream or reality? **Transplant Immunol.** 17(1):74-7, 2006.

Ruiz R et al Treatment with Trk C Agonist Antibodies Delays Disease Progression In Neuromuscular Degeneration (nmd) Mice. **Hum. Mol. Gen.** 14(13), 1825-1837, 2005.

Dernick G et al. Patch Amperometry -high resolution measurements of single vesicle fusion and release. **Nature Meth.** 2(9) 1-10, 2005.

Fernandez-Chacón R et al. CSP α Prevents Presynaptic Degeneration. **Neuron**, 42, 237-251, 2004.

Tabares L et al. Relationship between fusion pore opening and release during mast cell exocytosis studied with patch amperometry. **Biochem. Soc. Trans.** 31: 837-842, 2003.

Hernández-Díaz FJ et al. Estrogen Modulates α 1/ β -Adrenoceptor-Induced Signaling and Melatonin Production in Female Rat Pinealocytes. **Neuroend.** 73(2): 111-122, 2001.

Rey E et al. Dopamine induces intracellular Ca²⁺ signals mediated by α 1B-adrenoceptors in rat pineal cells. **Eu. J. Pharmacol.**, 430: 9-17, 2001.

Tabares L et al. Exocytosis of Catecholamine (CA)-containing and CA-free Granules in Chromaffin Cells. **J. Biol. Chem.**, 276: 39974-9, 2001.

Alés E et al. High calcium concentrations shift the mode of exocytosis to the kiss-and-run mechanism. **Nature Cell Biol.** 1, 40-44, 1999.