CURRICULUM VITAE ABREVIADO (CVA)

IMPORTANT – The Curriculum Vitae cannot exceed 4 pages. Instructions to fill this document are available in the website.

Part A. PERSONAL INFORMATION

First name	TERESA		
Family name	IGLESIAS VACAS		
Gender (*)	Woman		
Social Security, Passport, ID number	15967877N		
e-mail	tiglesias@iib.uam.es	URL Web	www.iib.uam.es/persona?id=tiglesias
Open Researcher and Contributor ID (ORCID)		0000-0002-4326-9005	

(*) Mandatory

A.1. Current position

Position	Profesora de Investigación del CSIC		
Initial date	10/04/2023		
Institution	CSIC		
Department/Center	Neurological Diseases and Aging	Instituto de Investigaciones Biomédicas Sols-Morreale – IIBM – (CSIC-UAM)	
Country	SPAIN	Teleph. number	+34 915854487
Key words	Alzheimer, Huntington, Stroke, Epilepsy, Hydrocephalus, SINO Syndrome, Excitotoxicity, PRKD1, KIDINS220, AQP4, Neurodegeneration, Neuroprotection, Rare Disease		

A.2. Previous positions (research activity interuptions, indicate total months)

Period	Position/Institution/Country/Interruption cause
1987-1990	FIS Predoctoral Fellow, Pharmacology Dpt., Medical School, UCM, Madrid
1991-1992	MEC Postdoctoral Fellow, Antibióticos Farma SA, Madrid
1992-1994	Gobierno Vasco Postdoctoral Fellow, IIBm (CSIC-UAM), Madrid
1994-1995	CSIC Hired Scientist, IIBM (CSIC-UAM), Madrid
1995-1999	EMBO Postdoctoral Fellow & ICRF Hired Scientist – ICRF, London (UK)
1999-2000	CSIC Hired Scientist, IIBM, Madrid
2000-2021	Científica Titular del CSIC, IIBM, Madrid
2021-2023	Investigadora Científica del CSIC, IIBm, Madrid

Sexenios: 5 granted - last period 2018-2023

A.3. Education

PhD, Licensed, Graduate	University/Country	Year
Licensed in Biological Sciences	Universidad Autónoma de Madrid (UAM)	1986
PhD in Biological Sciences	Universidad Complutense de Madrid (UCM)	1991

Part B. CV SUMMARY

Dra. Iglesias has received **continuous funding as PI** by several Institutions in a **total of 18 research projects** (9 grants from the Spanish Government; 2 individual and 3 consortium biomedicine grants from “Comunidad de Madrid”; 4 CIBERNED collaborative projects) and **5 collaborative contracts with Companies**. She has also participated in one **European COST Action** (BM1001-ECMNET - WG3). Since 2008, she heads her group as PI in the Network Center for Biomedical Research in Neurodegenerative Diseases (CIBERNED, ISCIII).

She is main inventor of one **international licensed patent** (PCT/GB01/03977) and one recent International Patent application (PCT/EP2022/061794). She has developed **high peer review activity in national and international Evaluation Committees** (EU Commission & ERC grants, and COST actions; MINECO/MICINN, ANEP/AEI), as well as in **International Journals**. Recently, she has been named **European Review Panel Member of COST actions**, in the ES Medical and Health Sciences area (2020-). She is member of the **BT-CIEN Scientific Committee** (2015-), and **Coordinator of CIBERNED Scientific Training Program**, and **member of CIBERNED Steering Committee** (2015-).

Her group "Novel Targets in Neurodegeneration and Neuroprotection" has been focused for many years on elucidating the role of PRKD1 and KIDINS220 in the pathophysiological processes of neurological and neurodegenerative diseases, searching for neuroprotective strategies. The group has developed multiple tools, cellular and animal models for PRKD1 and KIDINS220 studies along the years. Dr. T. Iglesias identified, cloned and characterized Kidins220 as the first PRKD1 substrate, (J. Biol. Chem., 2000; licensed International Patent PCT/GB01/03977). Her group has shown that Kidins220 is vital for neuronal differentiation and survival and that it is degraded in excitotoxicity and cerebral ischemia (J. Cell. Sci. 2009; J. Biol. Chem., 2010; Cell Death & Dis. 2019). They designed a neuroprotective strategy based on preventing the excitotoxic degradation of Kidins220 by a cell-penetrating peptide (Cell Death & Dis. 2015, National Patent application 201530118). They discovered an increase in KIDINS220, regulated by kinases and phosphatases, in the brain and cerebrospinal fluid of Alzheimer's disease patients, and its correlation with the pathological accumulation of tau/ptau. (Hum. Mol. Gen. 2013; J. Alzh. Dis, 2017), and identified alterations of KIDINS220 in Huntington's disease patients and mouse models (Brain Pathol. 2020). They were the first reporting PRKD1 function in neurons, discovering a new PDZ motif that regulates protein trafficking of Kidins220 to the neuronal surface (J. Biol. Chem., 2004, J. Biol. Chem., 2006), its regulation of nitric oxide synthases (PLosONE 2014; J Cell Sci 2014) and to demonstrate the neuroprotective role of PRKD1 through oxidative stress detoxifying mechanisms (Nat Comm. 2017; CIBERNED Young Investigator award). They have generated mice deficient in Kidins220 and Prkd1 in neurons (CKO) or in astrocytes (GKO), in which they have investigated how the elimination of these molecules impacts brain function. Using Kidins220 deficient mice they have unveiled dysfunctions in adult hippocampal neurogenesis and memory processes due to defects in survival of stem cells and neural progenitors (Cell Death & Dis. 2023), and identified the mechanisms by which Kidins220 regulates ventriculomegaly development (Mol Psychiatry 2021). This work has led to the application for an International Patent PCT/EP2022/061794 that opens perspectives for new therapies for KIDINS220-related diseases such as SINO syndrome (Genetics in Medicine 2024).

Research lines as Principal Investigator:
1) **The molecular mechanisms of excitotoxicity**, a type of neuronal death occurring in numerous neuropathologies and whose prevention may confer neuroprotection in a broad range of neurodegenerative conditions.
2) **PRKD1 role in epilepsy development**, using mouse models for its conditional deletion in neurons or in astrocytes that show significant changes in brain maturation, mitochondrial metabolisms and glutamatergic neurotransmission.
3) **The pathophysiological mechanisms associated with KIDINS220 deficiencies**. Using Kidins220-deficient mice, we have recently discovered a role of Kidins220 as a critical regulator of ventriculomegaly development and adult neurogenesis and memory. Nonsense variants in *KIDINS220* gene are associated with a novel rare syndrome characterized by spastic paraplegia, intellectual disability, nystagmus and obesity (SINO syndrome). SINO patients show ventriculomegaly that resembles that of Kidins220 deficient mice. We aim to investigate SINO syndrome etiopathology as part of an international collaborative framework.

Part C. RELEVANT MERITS

C.1. Selected Publications

1- Alstrup M*, Cesca F*, Krawczun-Rygmaczewska A, López-Menéndez C, Pose-Utrilla J, Castberg FC, Ortved Bjerager M, Finnila C, Fagerberg C, Engelund MB, Kibæk M, Kruer M, Bakhtiari S, Padilla-Lopez S, Manwaring L, Keren B, Afenjar A, Santorelli F, Shillington A, Glassford M, Vezain M, Martinovic J, Stevens C, Gowda VK, Srinivasan VM, Thiffault I, Pastinen T, Baranano K, Lee A, Granadillo J, Keegan CE, Matthews N, Saugier-Veber P, **Iglesias T*** & Østergaard E (**2024**) (*Corresponding Authors) "Refining the phenotype of SINO syndrome: A comprehensive cohort report of 14 novel cases"
Genet. Med. (GIM) DOI: 10.1016/j.gim.2024.101219 . **IF: 6,6 - D1**
2- del Puerto A, Lopez-Fonseca C, Simón-García A, Martí-Prado B, Barrios-Muñoz AL, Pose-Utrilla J, López-Menéndez C, Alcover-Sanchez B, Cesca F, Schiavo G, Campanero MR, Fariñas I, **Iglesias T*** & Porlan E* (**2023**) (*Co-corresponding and Co-Senior) "Kidins220 sets the threshold for survival of neural stem cells and progenitors to sustain adult neurogenesis"
Cell Death Dis. Aug 4;14(8):500. doi: 10.1038/s41419-023-05995-7. **IF: 9,685- Q1**
3- Picó S, Parras A, Santos-Galindo M, Pose-Utrilla J, Castro M, Fraga E, Hernández IH, Elorza A, Anta H, Wang N, Martí-Sánchez L, Belloc E, García-Esparcia P, Garrido JJ, Ferrer I, Macías-García D, Mir P, Artuch R, Pérez B, Hernández F, Pérez-Cerdá C, Navarro P, López-Sendón JL, **Iglesias T**, Yang XW, Méndez R & Lucas JJ (**2021**) "CPEB alteration and aberrant transcriptome-polyadenylation unveil a treatable SLC19A3 deficiency in Huntington's disease".
Science Transl Med doi: 10.1126/scitranslmed.abe7104 **IF: 16.304 - D1**
4- Del Puerto A, Pose-Utrilla J, Simón-García A, López-Menéndez C, Jiménez AJ, Porlan E, SM Pajuelo L, Cano-García G, Martí-Prado B, Sebastián-Serrano A, Sánchez-Carralero MP, Cesca F, Schiavo G, Ferrer I, Fariñas I, Campanero MR & **Iglesias T** (**2021**) "Kidins220 deficiency causes ventriculomegaly via SNX27-retromer-dependent AQP4 degradation"
Mol Psychiatry* doi:10.1038/s41380-021-01127-9. **IF2019: 12.384 - D1**
***Publication related to the international Patent application **PCT/EP2022/061794**
5- Sebastián-Serrano A, Simón-García A, Belmonte-Alfaro A, Pose-Utrilla J, Santos-Galindo M, del Puerto A, García-Guerra L, Hernández IH, Schiavo G, Campanero MR, Lucas JJ & **Iglesias T** (**2020**) "Differential regulation of Kidins220 isoforms in Huntington´s disease".
Brain Pathol doi: 10.1111/bpa.12761. **IF2018: 6.155 - D1**
6- López-Menéndez C, Gamir-Morralla A, Luján R, Díaz-Guerra M & **Iglesias T**. (**2019**) "Excitotoxic targeting of Kidins220 to the Golgi apparatus precedes calpain-cleavage of Rap1-activation complexes"
Cell Death Dis. Jul 11;10(7):535**.** **IF2018: 5.959 - Q1**
7- Pose-Utrilla J*, García-Guerra L, Del Puerto A, Martín A, Jurado-Arjona J, De León-Reyes NS, Gamir-Morralla A, Sebastián-Serrano A, García-Gallo M, Kremer L, Fielitz J, Ireson C, Pérez-Álvarez MJ, Ferrer I, Hernández F, Ávila J, Lasa M, Campanero MR, & **Iglesias T** (**2017**) "Excitotoxic inactivation of constitutive oxidative stress detoxification pathway in neurons can be rescued by PKD1". ***Young investigator award CIBERNED 2018"- 3000€**
Nat. Comm. Dec 22;8(1):2275. doi: 10.1038/s41467-017-02322-5 **IF2016: 12.124 - D1**.
8- Gamir-Morralla A, López-Menéndez C, Ayuso-Dolado S, Tejeda GS, Montaner J, Rossell A, **Iglesias T*** & M. Díaz-Guerra* **(**(*Co-corresponding and Co-Senior) **(2015)** "Development of a neuroprotective peptide that preserves survival pathways by preventing Kidins220/ARMS calpain-processing induced by excitotoxicity"
Cell Death Dis**.** doi: 10.1038/cddis.2015.307. **IF 2014: 5,014 Q1**
9- López-Menéndez C, Gamir-Morralla A, Jurado-Arjona J, Higuero AM, Campanero MR, Ferrer I, Hernández F, Ávila J, Díaz-Guerra M & **Iglesias T (2013)** "Kidins220 accumulates with tau in human Alzheimer's Disease and related models: modulation of its calpain-processing by GSK3b/PP1 imbalance"
Hum. Mol. Gen**.** 22: 466-482 **IF2012: 7,692 - D1**

C.2. Congress

44 Guest lectures and over 75 participations in national and international meetings. (Highlighted Invited conferences)

- "Molecular Targets for Ventriculomegaly and Neurodegeneration" **Global Summit on Neurodegenerative Diseases NEURO 2020/22. Salamanca**. June 21-14, **2022**
- "Novel Cellular and Molecular Mechanisms of Hydrocephalus". Symposium "Neuron and Glia Biology". **XIX Congress of the Spanish Society of Cell Biology (SEBC)**". Boadilla del Monte (Madrid). October 26– 29, **2021**

- "*PKD1 protects from oxidative stress: Good news for neurons*". Symposium "Cellular Neurobiology" **XVIII Congress of the Spanish Society of Cell Biology (SEBC)**". Badajoz. October 15-18, **2019**
- "Novel Molecular Mechanisms involved in Hydrocephalus Pathogenesis" **VII International Congress on Research and Innovation in Neurodegenerative Diseases** (CIIIEN). Valencia. September 18-22**, 2019**.
- **"**Phosphatases and kinases unbalance in acute and chronic neurodegeneration". **FEBS advanced lecture course** - Europhosphatases: From Molecular Mechanisms to System-wide Responses. Debrecen, Hungary. June 11–16, **2019.**
- "Kidins220/ARMS accumulation in Alzheimer´s Disease: Role of phosphorylation on its proteolysis by calpain" **Current trends in Biomedicine. Workshop "Proteases at work"** Baeza. October 20-22**, 2014**
- **"**Kindins220/ARMS function in neuronal survival and neurodegeneration" **9th International Conference for Neurons and Brain Diseases - ASND 2014**. Madrid. July 14-16, **2014**

C.3. Selected Research projects, (as Principal Investigator Last 10 years).

1- PID2023-153284OB-I00 "Enfermedades Neurológicas Relacionadas con PRKD1 y KIDINS220: Nuevos Modelos de Enfermedad para Identificar Mecanismos Patológicos, Dianas y Estrategias Terapéuticas" 01/09/2024-31/08/2028 Funds: 412.500 €+FPI contract
2- CIBERNED 2022/03 cooperative project "Analysis of thiamine deficiency in Huntington's disease as a biomarker of progression and for evaluation of therapeutic response" 01/2023-12/2024; PI - Group funds: 24.000 €
3- PID2020-115218RB-100 "Neuroprotection Strategies and Molecular Mechanisms Related to Kidins220 and Protein Kinase D Dysfunction" 09/2021 – 12/2024 – Funds: 290.400 €
4- CIBERNED 2018/06 cooperative project "Targeting CPEB-dependent impaired mitochondrial metabolism and synaptic and stem cell function in Huntington's disease" 01/2019-12/2020; Group funds: 48.800 €
5- SAF2017-88885-R "Molecular mechanisms underlying brain damage and neurodegeneration induced by Kidins220 defficiency or by selective PKD1 deletion in neurons and astrocytes". 01/2018 - 08/2021; Funds: 242.000 €+FPI contract
6- B2017/BMD3700- NEUROMETAB-CM. Comunidad de Madrid consortium. "Metabolic basis of Neurodegeneration" 01/2018- 06/2022. Group funds: 117.056 €
7- CIBERNED 2015-2/06 cooperative project "Molecular mechanisms of brain and muscle stem cell function in aging and neurodegeneration" 03/2016 - 11/2018; Group funds: 70.000 €
8- SAF2014-52737-P "Participation and regulation of PKD and Kidins220 in molecular processes & signaling pathways of Neurodegeneration" 01/2015-07/2018. Funds: 193.600 €
9- CIBERNED 2013/07 cooperative project "Role of GSK-3 β in the cortical circuits alterations found in Alzheimer´s disease". PI & coordinator. 10/2013 – 12/2015; Group funds: 63.200€.
10- P2010/BMD-2331 NEURODEGMODELS. Comunidad de Madrid consortium. "Signaling networks and effector pathways in cellular and animal models of neurodegenerative diseases". 01/2012 – 06/2016. Group funds: 69.000 €
11- SAF2011-26233. "Participation of PKD and its substrate, Kidins220, in the molecular pathways of neuronal survival and neurodegeneration" 01/2012-12/2014; Funds: 205.700 €

C.4. Contracts, technological or transfer merits

C.4.2. Patents

1. Inventors: **T Iglesias**, MR Campanero, J Pose-Utrilla, A Simón, C López-Menéndez, L Sánchez-Miranda, and y AM del Puerto. Title: "Methods and compositions for the treatment of disorders characterized by a KIDINS220 dysfunction in a subject". **PCT/EP2022/061794**. National Phases. Countries: Europe & USA. Priority date: May 2021. Owner entity: CSIC/UAM/CIBERNED. Under licensing negotiation.
2. Inventors: G Schiavo, **T Iglesias**. "Novel compounds and methods (in particular, related to the physiological substrate for PKD/PKCμ and methods of use: Rat and human Kidins220 polynucleotide and polypeptides sequences and antibodies". Owner entity: ICRT (London, UK). Priority date: 2001. International Patent **PCT/GB01/03977**; Re No US 60/230,449. **Licensed**.