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	María José		
Family name	Martínez Díaz-Guerra		
Gender (*)		Birth date (dd/mm/yyyy)	
Social Security, Passport, ID number			
e-mail	Mariajose.martinez@uclm.es	URL Web	
Open Researcher and Contributor ID (ORCID) (*)		Researcher ID M-2855-2014 Orcid code 0000 0003 3843 3912	

(*) Mandatory

A.1. Current position

Position	Catedrática de Bioquímica y Biología Molecular		
Initial date	21-04-2017		
Institution	Facultad de Medicina. Universidad Castilla-La Mancha		
Department/Center	Departmento Química Inorgánica, Orgánica y Bioquímica		
Country	Spain	Teleph. number	605892683
Key words	Notch, macrophage, inflammation		

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

Period	Position/Institution/Country/Interruption cause
2002-2017, 1999-2002	Profesor Titular and Profesor asociado UCLM, Spain
1993-1999 1991-1993 1987-1990	Investigador contratado CSIC Spain Postdoctoral Fellow Ministerio de Educación y Ciencia. Institute Cochin de Genetique Moleculaire. France Predoctoral fellow Fondo Investigaciones Sanitarias. Spain

A.3. Education

PhD, Licensed, Graduate	University/Country	Year
Pharmacy	Universidad Complutense de Madrid	1986
PhD Pharmacy (Biochemistry)	Universidad Complutense de Madrid	1990

(Include all the necessary rows)

Part B. CV SUMMARY *(max. 5000 characters, including spaces)*

I am currently Professor of the Department of Biochemistry and Molecular Biology at the Faculty of Medicine of the University of Castilla-La Mancha (UCLM), which I joined in 1999. I teach Biochemistry and Immunology in the 2nd year of the degree and Immunology in the 3rd

year of the degree of Biotechnology. I started my scientific activity at the Instituto de Bioquimica (CSIC-UCM) studying the mechanism implicated in macrophage activation under the direction of Dr Lisardo Boscá. I did a three-year postdoctoral stay at the Cochin Institute of Molecular Genetics in Paris, during which I studied glucose-induced gene expression changes in glycolysis enzymes. After I joined University of Castilla-La Mancha, in 1999-2000, I have been PI of six national grants funded by the Instituto Carlos III and the Ministry of Science and Innovation, and four regional grants from the Consejería de Sanidad of Castilla-La Mancha, which allowed me to organize a research group and to

establish a line of research that has been maintained for the last twenty years. We aimed to study the role of the NOTCH receptors in the activation of macrophages and the development of the inflammatory response, in which we continue to work on. In our first publication we demonstrated that NOTCH1 receptor was relevant in Toll4 and interferon-gamma signaling in macrophages by increasing its cytotoxic and proinflammatory activity (J. Immunol. 2006). These new role of NOTCH1 receptors in macrophage activation and M1 polarization initiated a novel line of research with great international repercussion, that is still being carried out by numerous researchers around the world, as evidenced by the fact that our first work, in which we described these processes in 2006 has been cited multiple times. We also showed that part of the NOTCH signaling effect in proinflammatory macrophage activation was mediated by the NFkB transcription factor (Eur.J.Immunol 2009), and that membrane proteins that inhibited NOTCH signaling as DLK1, limits macrophage proinflammatory activity (Eur.J.Immunol 2015). We have confirmed the proinflammatoy role of NOTCH1 as we observed that the absence of NOTCH1 in murine myeloid cells attenuated the development of an experimental autoimmune encephalomyelitis by affecting Th1 and Th17 priming (Eur.J.Immunol.2017).

There are still many unknown aspects of the regulatory role of NOTCH receptors in macrophage activation, in this sense, we have recently shown that the NOTCH3 receptor also plays an important function in the proinflammatory activation of macrophages through the potentiation of NFkB activity (Sci Rep., 2020). Lastly, we have observed that the NOTCH4 receptor, which is induced late during macrophage proinflammatory activation, exerts an inhibitory role in this process, essentially limiting IFN-γ signaling (Front. Immunol.2021) and that its expression is important in M2 macrophage polarization induced by IL13 (Int Immunol. 2023). We are interested in continuing this line of research, analyzing the role that the NOTCH4 receptor may play in the activity and function of M2 macrophages and its relation to the development of pathologies in which M2 macrophage activity is relevant.

Throughout these years I have contributed to the training of several students directing final degree and master's projects as well as doctoral theses. I have also participated in the dissemination programs of the Castilla-La Mancha University to explain our scientific activity to high school students.

Part C. RELEVANT MERITS *(sorted by typology)*

C.1. Publications *(see instructions)*

1.- María José Romero de Ávila, Susana López-López, Aarón García-Blázquez, José Javier García-Ramírez, Eva M. Monsalve (AC), and María José M. Díaz-Guerra (AC) (2024) **RND3 potentiates proinflammatory activation through NOTCH signaling in activated macrophages.** J.Immunol. Res. (accepted, in press).

2.- Susana López-López, María José Romero de Ávila, María Julia González-Gómez, José Javier García-Ramírez (AC), and María José M. Díaz-Guerra (AC)(2023) **NOTCH4 potentiates the IL-13 induced genetic program in M2 alternative macrophages through the AP1 and IRF4-JMJD3 axis**. Int Immunol**.** .35(10):497-509.

3.- Susana López-López, María José Romero de Ávila, Natalia Carolina Hernández de León, Francisco Ruiz-Marcos, José Javier García-Ramírez, Eva M. Monsalve (AC) and María José M. Díaz-Guerra (AC). (2021) **NOTCH4 Exhibits Anti-Inflammatory Activity in Activated Macrophages by Interfering With Interferon-γ and TLR4 Signaling**. Front Immunol. 12:734966.

4.- López-López S, Monsalve EM, Romero de Ávila MJ, González-Gómez J, Hernández deLeón N, Ruiz-Marcos F, Baladrón V, Nueda ML, García-León MJ, Screpanti I, Felli MP, Laborda J, García-Ramírez JJ, Díaz-Guerra MJM (AC). (2020) **NOTCH3 signaling is essential for NF-κB activation in TLR-activated macrophages.** Scientific reports, 10(1):14839.

5.- Miriam Fernández, Eva M. Monsalve, Susana López-López, Pedro Tranque, María José M. Díaz-Guerra (AC). (2017) **Notch1 signaling in myeloid cells modulates the development of experimental autoimmune encephalomyelitis**. Eur. J. Immunol. 2017 47(12):2090-2100.

6.- Ruiz-García A, López-López S, García-Ramírez JJ, Baladrón V, Ruiz-Hidalgo MJ, López-Sanz L, Ballesteros Á, Laborda J, Monsalve EM, Díaz-Guerra MJ. (AC) (2016) **The Tetraspanin TSPAN33**

Controls TLR-Triggered Macrophage Activation through Modulation of NOTCH Signaling. J. of Immunol. ;197(8):3371-3381.

7- Julia M. González, Almudena Ruiz-García, Eva Monsalve, J. Laborda, María J.M. Díaz-Guerra (AC) and M. J. Ruiz-Hidalgo (AC).(2015) **DLK1, a new inflammatory inhibitor by interfering Notch1 signaling in Toll activated macrophages**. Eur J Immunol (9):2615-27.

8.- Almudena Ruiz-García, Eva Monsalve, Samuel Rivero, Jorge Laborda, Ramón Bartrons, María José M. Díaz-Guerra (AC) (2011) **Cooperation of adenosine with Toll-4 receptor agonists leads to increased glycolytic flux through the enhanced expression of PFKFB3 gene**. J Biol Chem.286(22):19247-58.

9.- Eva Monsalve, Almudena Ruiz-García, Victoriano Baladrón, María José Ruiz-Hidalgo, Beatriz Sánchez-Solana, Samuel Rivero, José J. García-Ramírez Antonio Rubio, Jorge Laborda and María José M. Díaz-Guerra (AC) (2009) **Notch1 upregulates LPS-induced macrophage activation by increasing NF-kB activity**.. Eur J Immunol.;39(9):2556-70.

10.- Monsalve E., Pérez M A, A Rubio, Ruiz-Hidalgo MJ, Baladrón V, García-Ramírez JJ, Gómez J C., Laborda J .and Díaz-Guerra MJM. (AC) (2006) **Notch-1 upregulation and signaling following macrophage activation modulates gene expression patterns known to affect antigen presenting capacity and cytotoxic activity**. J. Immunol (9) 5362-73.

C.2. Congress, indicating the modality of their participation (invited conference, oral presentation, poster)

C.3. Research projects, indicating your personal contribution. In the case of young researchers, indicate lines of research for which they have been responsible.

TITULO DEL PROYECTO: **NOTCH3 and NOTCH4, two NOTCH receptors with a new role in the control of macrophage activation and inflammation.**
ENTIDAD FINANCIADORA: Plan Nacional PID2019-109421RB-I00
CUANTÍA SUBVENCIÓN: 121.000 €
DURACIÓN: Desde 1-6-2020 hasta 31-12-2023.
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra y José Javier García Ramirez**. Facultad de Medicina . Universidad Castilla-La Mancha.

TITULO DEL PROYECTO: **Nuevas dianas en el control de la inflamación: RND3 y LHX2, dos genes modulados por NOTCH.**
ENTIDAD FINANCIADORA: Consejería de Educación, Cultura y Deportes de Castilla-La Mancha
CUANTÍA SUBVENCIÓN:109.900 €
DURACIÓN: Desde 1-1-18 hasta 31-12-2021.
INVESTIGADOR PRINCIPAL**: José Javier García Ramirez y Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha.

TITULO DEL PROYECTO: **Nuevas dianas moleculares de los receptores Notch en la inflamación. Análisis en modelos animales y en procesos inflamatorios.**
ENTIDAD FINANCIADORA: Instituto Carlos III PI15/00991
CUANTÍA SUBVENCIÓN: 92.565,00 €
DURACIÓN: Desde 1-1-2016 hasta 31-12-2018.
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha.

TITULO DEL PROYECTO: **Estudio de las bases moleculares de la actividad antiinflamatoria de los receptores Notch. Análisis en modelos animales y en procesos inflamatorios agudos.**
ENTIDAD FINANCIADORA: Instituto Carlos III PI12/01546
CUANTÍA SUBVENCIÓN: 110.715,00 €

DURACIÓN: Desde 1-1-2013 hasta 31-12-2015.
INVESTIGADOR PRINCIPAL**: Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha.

TITULO DEL PROYECTO: **Estudio de la función de los receptores Notch en la activación clásica y alternativa de los macrófagos. Análisis en modelos celulares, animales y en procesos inflamatorios crónicos.**
ENTIDAD FINANCIADORA: Instituto Carlos III PI09/1624
CUANTÍA SUBVENCIÓN: 148.830,00 €
DURACIÓN: Desde 1-1-2009 hasta 31-12-2012.
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha.

TITULO DEL PROYECTO: **Estudio de la función del receptor Notch-1 en el macrófago. Desarrollo de modelos animales para establecer su implicación en procesos inflamatorios.**
ENTIDAD FINANCIADORA: Instituto Carlos III PI060449.
CUANTÍA SUBVENCIÓN: 86.900,00 €
DURACIÓN: Desde 2006 hasta 2009
INVESTIGADOR PRINCIPAL**: Mª José Martínez Díaz-Guerra.** Facultad de Medicina . Universidad Castilla-La Mancha

TITULO DEL PROYECTO: **Papel de las proteínas Notch en la activación del macrófago y su transformación en célula espumosa**
ENTIDAD FINANCIADORA: Instituto Carlos III PI030766
CUANTÍA SUBVENCIÓN: 86.595,00 €
DURACIÓN: Desde 2002 hasta 2005
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha

TITULO DEL PROYECTO: **Estudio de la función de la adenosina y los receptores A2 en la activación alternativa del macrófago y la resolución de los procesos inflamatorios.**
ENTIDAD FINANCIADORA: Consejería de Ciencia y Tecnología de Castilla-La Mancha
CUANTÍA SUBVENCIÓN: 100.000,00€
DURACIÓN 2009-2011
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra**. Facultad de Medicina, Universidad Castilla-La Mancha

TITULO DEL PROYECTO: **Estudio de la función del receptor Notch-1 en el control de la activación del macrófago. Implicación en procesos inflamatorios crónicos.**
ENTIDAD FINANCIADORA: Consejería de Sanidad de Castilla-La Mancha SAN06-015.
CUANTÍA SUBVENCIÓN: 114.632,00 €
DURACIÓN: Desde 28-9-2006 hasta 30-12-2008.
INVESTIGADOR PRINCIPAL: **Mª José Martínez Díaz-Guerra**. Facultad de Medicina . Universidad Castilla-La Mancha

C.4. Contracts, technological or transfer merits, Include patents and other industrial or intellectual property activities (contracts, licenses, agreements, etc.) in which you have collaborated. Indicate: a) the order of signature of authors; b) reference; c) title; d) priority countries; e) date; f) Entity and companies that exploit the patent or similar information, if any