LABORATORI D'ENGINYERIA GENÈTICA ANIMAL
FÀTIMA BOSCH TUBERT

PROFILE

Fatima Bosch is a Pharmacist (1980) and PhD in Biochemistry (1985) by the University of Barcelona. She conducted post-doctoral studies at Vanderbilt University (1985), Case Western Reserve University (1988-1990), and NCI-Frederick Cancer Research and Development Center (1991). She is currently Full Professor of Biochemistry and Molecular Biology (1999) and Director of the Center of Animal Biotechnology and Gene Therapy (2003) at the Universitat Autònoma Barcelona. She has been granted the Rey Juan Carlos I (1985), Francisco Grande Covián (1998), Narcís Monturiol (2002), Sant Jordi Cross (2005), Alberto Sols (2006) and ICREA Academia (2013) awards. She has been Founding member of the European Society of Gene and Cell Therapy (1992), President of the Spanish Society of Gene and Cell Therapy (2007-2009) and Vice-President of the European Association for the Study of Diabetes (2009-2012), member of the Gene Doping Expert Group of the World Anti-Doping Agency (2013-present) and member of the CIBER de Diabetes y Enfermedades Metabólicas Asociadas (CIBERDEM) (2008-present).

Her research focuses on studying the pathophysiological causes of diabetes mellitus using transgenic animal models and developing gene therapy approaches to this disease by in vivo genetic manipulation of tissuesusing viral vectors. She has also applied her know-how on gene transfer technologies to the development of gene therapies for severe inherited metabolic disorders such as Muccopolysaccharidoses to counteract neurologic and somatic pathology.

RESEARCH

RESEARCH INTERESTS/ STRATEGIC OBJECTIVES/ MAIN RESEARCH LINES

Our main strategic objective is to develop new gene therapy approaches for highly prevalent and rare metabolic and neurodegenerative diseases, with the ultimate goal to improve the quality of life for subjects affected by these diseases. Our studies focus on the pathophysiological causes of diabetes mellitus and its comorbidities and also of severe genetic storage diseases, generating transgenic animal models and developing gene transfer-based therapeutics.

LAB FEATURED PUBLICATIONS:

J. Rozman, B. Rathkolb, M. Oestereicher, C. Schütt, A.C. Ravindranath, S. Leuchtenberger, Sa. Sharma, M. Kistler, M. Willershäuser, R. Brommage, T. Meehan, J. Mason, H. Haselimashhadi, IMPC Consortium, Tertius Hough, AM. Mallon, S. Wells, L. Santos, C. Lelliott, J. White, T. Sorg, MF. Champy, L. Bower, C. Reynolds, A. Flenniken, S. Murray, L. Nutter, K. Svenson, D. West, G. TocchiniValentini, A. Beaudet, F. Bosch, R. Braun, M. Dobbie, X. Gao, Y. Herault, A. Moshri, B. Moore, K. Lloyd, C. McKerlie, H. Masuya, N. Tanaka, P. Flicek, H. Parkinson, R. Sedlacek, JK. Seong, LC. Wang, M. Moore, S. Brown, M. Tschoep, W. Wurst, M. Klingenspor, E. Wolf, J. Beckers, F. Machicao, A. Peter, H. StaigerHU. Häring, H. Grallert, M. Campillos, H. Maier, H. Fuchs, V. Gailus-Durner, T. Werner. Identification of genetic elements in metabolism by high-throughput mouse phenotyping. ***Nature Communications*** (2018) Jan 18;9(1):288

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V. Jimenez*, C. Jambrina*, E. Casana, V. Sacristan, S. Muñoz, S. Darriba, J. Rodó, C. Mallol,M. Garcia, X. León, S. Marcó, A. Ribera, I. Elias, A. Casellas, I. Grass, G. Elias, T. Ferré, S. Motas, S. Franckhauser, F. Mulero, M. Navarro, V. Haurigot, J. Ruberte and F Bosch. *Both authors contributed equally to this work. FGF21 gene therapy as treatment for obesity andinsulin resistance. ***EMBO Molecular Medicine*** (2018) Aug;10(8). pii: e8791

M. A. Muñoz-Lorente, P. Martínez, Á. Tejera, K. Whittemore, A. C. Moisés-Silva, F. Bosch, and M. A. Blasco. AAV9 mediated telomerase activation does not accelerate tumorigenesis in the context of oncogenic K-Ras-induced lung cancer. ***Plos Genetics*** (2018) Aug 16;14(8):e1007562.

A. Navarro-Romero , A.Vázquez-Oliver , M. Gomis-González, C.Garzón-Montesinos, R. Falcón-Moya, A. Pastor, E. Martín-García, N. Pizarro, A. Busquets-Garcia, JM. Revest, PV. Piazza, F. Bosch, M. Dierssen, R. de la Torre, A. Rodríguez-Moreno, R. Maldonado, A.Ozaita.Cannabinoid type-1 receptor blockade restores neurological phenotypes in two models for Down syndrome. ***Neurobiology of Disease*** 2019 May;125:92-106.

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