Dr. **Oscar Zaragoza Hernández** has devoted his entire scientific career to microbiology, and especially to the biology of fungi that cause disease in humans. He carried out the thesis in the laboratory of Dr. Juana María Gancedo (Institute of Biomedical Research, CSIC, Madrid). His thesis was focused on the molecular mechanisms that regulate carbon catabolite repression in the model yeast Saccharomyces cerevisiae. During this time, he also began under the tutelage of Dr. Carlos Gancedo, to develop a parallel investigation focused on the yeast Candida albicans, which is the main cause of candidemia in humans. This alternative line allowed the discovery of the trehalose synthesis pathway as antifungal target in pathogenic yeasts. His definitive orientation into Clinical Mycology took place in 2001, when he joined the laboratory of Dr. Arturo Casadevall (Albert Einstein College of Medicine, New York), which is one of the groups with the highest international recognition in fungal pathogens. In this laboratory, the work of Dr. Zaragoza focused on the mechanisms of virulence and host adaptation of pathogenic fungi, taking the yeast Cryptococcus neoformans as a model. This period was very successful and as a consequence, he published more that thirty articles in scientific journal. In 2006, Dr. Zaragoza joined the Mycology Laboratory of the National Centre for Microbiology of the Instituto de Salud Carlos III thanks to the award of a "Ramón y Cajal" contract. Currently, and since 2018, he hold the Scientific Investigator position at this Institution.

2.- Brief introduction to Dr. Zaragoza´s research field

His research is focus mainly on pathogenic yeast (Candida and Cryptococcus). The work of Dr. Zaragoza focused on his stage as PI in two main areas: Investigation of the mechanisms of action of antifungals and resistance, and research of the adaptation processes of fungi to the host environment. The group of Dr. Zaragoza demonstrated that the antifungal Amphotericin B induces accumulation of free radicals in the cell, which is an important fungicidal mechanism for this molecule. (Microbes Infect. 2011, 13:457-67; Antimicrob Agents Chemother. 2014, 58:6627-38). These results have clinical relevance, since they open the door to new therapeutic strategies that help improve the use of this antifungal.

The other main research line is focused in the study of the adaptation mechanisms of pathogenic yeast to the host. One of the models that best illustrates this ability to adapt is the yeast Cryptococcus neoformans, which is acquired by inhalation, and has a special ability to adapt and survive in the lung. Our group described in 2010 a new mechanism that explains how this yeast adapts to the lung, which involves a massive increase in the size of the blastoconidia and the formation of abnormally large cells, called giant cells or titan cells (PLoS Pathogens, 2010, 6:e1000945). This work has had a great impact in the field. In addition, the group has become a reference in the field of fungal morphogenesis, and has been invited to write reviews in some of the most prestigious journals (Current Opinion in Microbiology, 2013, 16: 409; Semin. Cell. Dev. Biol., 2016, 57: 100-109, Plos Pathogens, 2018, 14(5):e1007007, Cellular Microbiology, 2016, 18(1):111-24; Curr Top Microbiol Immunol. 2018; doi: 10.1007/82_2018_145). This research line has allowed the identification of new factors involved in cryptococcal morphogenesis by using NGS techniques, such as whole genome sequencing or RNAseq.

- Multidisciplinary research and promotion of use of alternative models

Dr. Zaragoza´s research is performed using multidisciplinary approaches (microbiology, cellular biology, molecular biology, genomics and immunology). Furthermore, he has actively participated in the development and implementation of research models that reduce the bioethical impact of animal experimentation, to investigate virulence of pathogenic fungi. He has participated in multiple studies in which vertebrate animals are replaced by invertebrates, mainly lepidoptera and nematodes (Virulence, 2015, 6:66-74.; Virulence. 2014, 15;5:454-6; Antimicrob Agents Chemother. 2013, 57:4769-81; PLoS One. 2013, 8:e60047; Med Mycol. 2013, 51:461-72; PLoS One. 2011,6:e24485).