Marc DALOD, PhD

Group leader: Dendritic cells and antiviral defence

Background

1996-2000	PhD work at the Cochin Hospital in Paris under supervision of Dr. J.-G. Guillet, on the characterization of anti-HIV-1 CD8 T cells responses
2000-2002	post-doctoral fellow in the laboratory of Pr. C.A. Biron (Brown University, Providence, RI, USA) on innate antiviral immunity
2003	CNRS tenure researcher position
2004	Team leader at the CIML
2015-2017	CIML deputy director

Awards

2007	CNRS bronze medal
2011-2017	Grantee from the European Research Council (ERC Starting Grant “SystemsDendritic”)

Impact of research

Total 66 publications in peer-reviewed international Journals, 31 as major author [(co-)last or (co-)corresponding or 1st]
Impact of research based on ISI web of knowledge:
H index of 35
Total citation counts of 5.190
7 highly cited papers in the last 10 years
Included in the top 1% most cited researchers in Immunology worldwide

Membership

Member of IUIS Nomenclature Sub-Committee for Monocytes and Dendritic Cells in Blood
http://www.iuisonline.org/pages/nomenclature-members.htm

Main achievements

During my PhD, I investigated whether anti-HIV-1 CD8 T cells responses harbored specific alterations which could contribute to their failure to prevent disease development in most patients. I observed in particular that acute antiHIV-1 CD8 T cell responses were delayed and blunted (Dalod et al. *J. Clin. Invest.* 1999). Thus, I decided to study innate antiviral immunity and its role in CD8 T cell activation.

As a post-doctoral fellow, I identified and characterized for the first time mouse plasmacytoid dendritic cells (pDC) as the major producers of IFN-I and IL-12 in vivo during murine cytomegalovirus (MCMV) infection (Dalod et al. *J Exp Med.* 2002; Dalod et al. *J Exp Med*. 2003).

My team at CIML has developed the first unifying model of DC subsets across tissues and species

(Guilliams et al. *Eur. J. Immunol.* 2010; Crozat et al. *J. Immunol.* 2011; Vu Manh et al. *Front. Immunol.* 2015a). I designed an original strategy based on comparative genomics to seek for potential equivalences between DC subsets across species and for their putative ontogenetic relationships with other immune cells (Robbins et al. *Genome Biol.* 2008; Crozat at al. *Immunol. Rev.* 2010; Contreras et al. *J. Immunol.* 2010; Vu Manh et al. *J. Immunol.* 2014; Dutertre et al. *J. Immunol.* 2014; Vu Manh et al. *Front. Immunol.* 2015a; Vu Manh et al. *Front. Immunol.* 2015b).

In our 2008 landmark comparative genomics study of mouse and human leukocytes, we demonstrated that all mouse spleen and human blood DC subsets share a core gene signature and constitute a specific leukocyte family distinct from other myeloid cells including *in vitro* derived GM-CSF DC. We identified human BDCA3 DC as putative equivalents to mouse CD8α DC. Since this study, BDCA3 DC have taken center stage. In 2010, simultaneously to 3 other reports, we demonstrated that, like mouse CD8α DC, human BDCA3 DC are more efficient than other human DC subsets for cross-presentation of cell-associated antigens, and that both cell types specifically express the chemokine receptor XCR1 (Crozat et al. *J Exp Med*. 2010). In collaboration with CIPHE, we have developed a zoo of reporter and conditional knock-out mice to decipher the molecular mechanisms regulation the functions of mouse XCR1+ DC. We have also optimized protocols for *in vitro* differentiation of relatively high numbers of *bona fide* human XCR1+ DC and their functional characterization. This system will enable genetic and pharmacological manipulation of human XCR1+ DC to examine to which extent their functions and their molecular regulation are conserved with those of mouse XCR1+ DC.

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