Hugues de Thé

Born January 18, 1959 in Marseille,
French citizen, married, 4 children
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Degrees

- MD: University of Paris V (1989)
- PhD: University of Paris VI (1990)

Professional experience

- Residency, Paris Hospitals, Medical Research (1984-1988)
- MD/PhD student, post-doc (INSERM U163) P. Tiollais, Pasteur Institute (1985-1991)
- Assistant professor, CNRS UPR 043, Hospital St. Louis, Paris (1991-1993)
- Associate professor, University of Paris, Hospital St. Louis, Paris (1993-1995)
- Professor of molecular biology, University of Paris (1995-2014)
- Head, CNRS/University of Paris VII Research Unit, Hospital St. Louis, Paris (1995-2019)
- Attending physician, St. Louis Hospital, Paris (1995-)
- Professor at the Collège de France (2014-)

Scientific advisory boards (selected)

- Adviser to the director of INSERM (1997-2001)
- President scientific council of ARC (2003-2005)
- Bettencourt Schuller foundation, president of the scientific advisory board (2014-)

Honours and awards (selected)

- Prix Rosen (Fondation for Medical Research) 1999
- Prix Mergier-Bourdeix (French Academy of Science) 2004
- Member of EMBO (2004)
- Prix Griffuel, ARC (2010)
- Prix Claude Bernard, City of Paris (2010)
- French Legion of Honour (2010)
- Senior grant European Research Council (ERC) 2011
- Member French Academy of Science (2011)
- Foreign cooperation award, Chinese Office Science & Technology Awards (2011)
- Ernest Beutler award, American Society of Hematology (2016)
- Sjoberg Prize, Swedish Royal Academy of Sciences (2018)
- Senior grant European Research Council (ERC) 2018
- Member, the National Academy of Medicine (USA) 2020

Hugues de Thé M.D. Ph.D. is Professor of molecular oncology at the Collège de France and physician at Hospital St. Louis, Paris. After making significant contributions to Retinoic Acid signaling during his MD/PhD training, he played a key role in the discovery of the PML/RARA oncoprotein, the driver of acute promyelocytic leukemia. Ever since, he has investigated how PML/RARA drives leukemogenesis and the mechanisms underlying the exquisite clinical response

to RA and arsenic. This led him to address issues of transcriptional control, cell biology, proteolysis and mouse modeling. In particular, he established the key role of therapy-induced PML/RARA degradation and PML nuclear bodies in APL response, crafting the physio-pathological bases for definitive curative regimens, first established in mice and subsequently in patients.