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He completed his post-doctoral training at Harvard Medical School, then joined Aix-Marseille University as professor at the Centre d'Immunologie de Marseille-Luminy (CIML) in 1993 before becoming its director from 2008 to 2017. He is also one of the founders of Marseille-Immunopôle, an immunology cluster created in 2014 linking fundamental and therapeutic research, innovation and industrial development on the Aix-Marseille metropole.
Eric Vivier's work focuses on innate immunity and in particular Natural killer and other innate lymphoid cells, at Ciml, at AP-HM and at Innate-Pharma. Professor Vivier has published over 350 scientific articles and is on the list of the most cited researchers:

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Harnessing innate immunity in cancer therapy and beyond

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Abstract

New therapies that promote antitumor immunity have been recently developed. Most of these immunomodulatory approaches have focused on enhancing T-cell responses, either by targeting inhibitory pathways with immune checkpoint inhibitors, or by targeting activating pathways, as with chimeric antigen receptor T cells or bispecific antibodies. Although these therapies have led to unprecedented successes, only a minority of patients with cancer benefit from these treatments, highlighting the need to identify new cells and molecules that could be exploited in the next generation of immunotherapy. Given the crucial role of innate immune responses in immunity, harnessing these responses opens up new possibilities for long-lasting, multilayered tumor control. We will present innovative anti-tumor therapies based on the manipulation of the innate immune system. In addition, given the urgent need for effective treatments for pneumonia in patients with COVID-19, the elucidation of the immune responses that occur during the course of COVID-19 could lead to the repurposing of approved immunomodulatory drugs and candidate drugs that have already been tested in clinical trials. Along this line, we will present our results indicating the association of COVID-19 inflammation with activation of the C5a–C5aR1 axis.