Olivier Malaise MD., PhD

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PROFESSOR

Osteoarticular side effects of immunotherapy Specialist

Has a Master in Rheumatology, a PhD in osteoarthritic area a Post-doctoral at the Institute of Regenerative medicine and biotherapies in Montpellier, France. He is member of the task force “immuno-rheumatology” of the Belgian Society of Rheumatology and Member of the task force “osteoporosis and bone disease” of the Belgian Society of Rheumatology. His scientific interest and current research area are the articular involvement of anti-cancer treatments, especially immunotherapies. Author and co-author of several publications in prestigious journals.

Full name: Olivier Malaise
Country: Belgium
Current Position:

2018 – present	Post-doctoral clinical master specialist at FNRS (Fonds National pour la Recherche Scientifique).
2018 – present	Clinical master specialist (Rheumatology department), University Hospital Center (CHU) in Liege, Belgium.
2018 – present	Member of the task force “immuno-rheumatology” of the Belgian Society of Rheumatology.
2018 – present	Member of the task force “osteoporosis and bone disease” of the Belgian Society of Rheumatology.

Education:

2018	Complementary master in Rheumatology, University of Liege, Belgium.
2018	Inter-university diploma in musculo-skeletal echography, University of Liege, Belgium.
2017	Inter-university diploma in inflammatory rheumatic disease, University of Montpellier, France.
2016	PhD thesis in Medical Science, University of Liege, Belgium “Leptin production by osteoarthritic synovial fibroblasts: role of GILZ”.
2016	Inter-university diploma in bone disease, University of Lill II, France.

2011	Master in Medical Science, University of Liege, Belgium.
2007	Baccalaureate in Medical Science, University of Liege, Belgium.

Experience:

2016 - 2017	Post-doctoral researcher: Research Scientist INSERM U1183 (Pr C Jorgenssen), Institute of Regenerative medicine and biotherapies (IRMB), CHU St Eloi, Montpellier, France.
2011 – 2016	Aspirate researcher at FNRS (Fonds National pour la Recherche Scientifique).

Scientific Publications:

Number of publications as author or co-author: 64

Recent scientific publications:

Background Osteoporosis (OP) is a serious and prevalent disease identified by Dual Energy X-ray Absorptiometry (DEXA) that can be performed in an ambulatory or a hospitalized population. Objectives Aims are: (1) to characterize patients who had DEXA in a university department of rheumatology to study its diagnostic efficacy; (2) to look after risk factors specific to our population; (3) to evaluate the efficiency of a non-systematic and opportunistic intra-hospital OP screening by comparing results for ambulatory and hospitalized patients in the different departments of the hospital. Methods From 2007 to 2012, 6406 initial DEXA from consecutive patients were prospectively encoded (1494 in 2007, 1158 in 2008, 1079 in 2009, 945 in 2010, 868 in 2011 and 862 in 2012) (identical DEXA). Results Cohort characteristics are the following: women 74.4%, mean age (± 1 standard deviation) 60.5 years (± 14.3), mean BMI 25.1 (± 5.1), history of cortico therapy in 30.5%, previous fracture in 29% and current hospitalization in 28.8% of cases. OP (at one of the 3 sites) was diagnosed in 22.3%, a stable value over the years, with, as a repartition, lumbar spine, femoral neck and total hip OP in 13.7%, 13.6% and 9.2% respectively. In 3.9%, OP was diagnosed at all the 3 sites.

Background Tissue accumulation of p16INK4A-positive senescent cells is associated with age-related disorders such as osteoarthritis (OA). These senescent cells induce a tissue loss of function through a particular secretory phenotype called SASP (senescence-associated secretory phenotype). Objectives Links between OA onset and cellular senescence remain poorly detailed. We wanted to determine the localization of articular senescent cells in in vivo OA mouse models and study the involvement of mesenchymal stem cells (MSC) senescence in OA pathogenesis. Methods Wild-type mice C57BL/6, SAMP8/R1 (senescence accelerated mouse-prone and resistant), transgenic p16INK4A +/Luc and p16INK4A Luc/Luc were used. Experimental OA was induced by intraarticular injections of collagenase (CIOA).