Master	Title	Institute	Host Laboratory	Name of the PI	Supervisor	Description	Duration	e-mail
M1/M2	**Pathological role of cell division defects in kidney cancer**	CRBM	Centrosome, cilia and pathologies	Dr Bénédicte DELAVAL	Dr Bénédicte DELAVAL	Epithelial tubular organisation is a common feature of many tissues including the kidney. This epithelial organisation is disrupted in various pathologies and in particular in kidney cancer. Cell division plays a key role in maintaining the integrity of proliferative tissues. We have recently shown that defects in division orientation are observed during the cystic phases that precede the appearance of certain kidney cancers. We propose here to combine 3-dimensional cell culture microscopy approaches with the zebrafish as an in vivo model to characterise the contribution of perturbations in the geometry of cell division to the early phases of cellular disorganisation observed in kidney cancers.	5 months	benedicte.delaval@crbm.cnrs.fr
M1	**SLAP function in intestinal homeostasis and carcinogenesis**	CRBM	Tyrosine kinase signalling in human cancer	Dr Serge ROCHE	Dr Serge ROCHE	Using genetically modified mouse models, we want to address the function of the SLAP adaptor protein in intestinal homeostasis and response to chronic inflammation induced by DSS/to carcinogenesis induced by AOM/DSS. The internship student will participate to the genotyping and the characterization of the phenotype of our SLAP KO mice. In addition, we want to address if the level of SLAP can predict the sensitivity to various tyrosine kinase inhibitors using "organoids" derived from mice tumors or from tumor cell lines derived from patients. Methods: DNA/RNA extractions, PCR/Q-PCR, immunohistochemistry, western-blotting, culture of cell lines and organoïds.	> 2 months	audrey.sirvent@crbm.cnrs.fr serge.roche@crbm.cnrs.fr
M1/M2	**Vesicular trafficking in cell invasion**	CRBM	Cytoskeleton and membrane trafficking dynamics in cellular adhesion	Dr Anne BLANGY Dr Cécile GAUTHIER-ROUVIERE	Dr Daniel BOUVARD	Dysregulation of cell adhesion to the ECM is a major event associated with tumour progression. In this project, we aim at deciphering the emerging role of vesicular trafficking of late endosomes (LE) in normal and tumour cells. LE are intracellular vesicles that were originally identified to clear cellular component, but their role in cell signaling has recently been shed to light. In particular, we showed that a strong connection linked cell adhesion and integrin's signaling to their dynamic. Using cells derived from the osteogenic lineage (normal but also tumor cells also named osteosarcoma) we will address the role of proteins (flotillin, Lamtors) involved in LE dynamics as new regulators of cell migration/invasion. We will use state of the art cell imaging technics (FRAP, high speed videomicroscopy, etc…) to address how trafficking of LE affects cell migration machinery.	6 months	Daniel.bouvard@crbm.cnrs.fr
M1/M2	**How do overexpressed-flotillins disrupt vesicular trafficking in cancer cells to deregulate AXL? Identification of a potential target to improve the efficacy of anti-AXL antibodies.**	CRBM	Cytoskeleton and membrane trafficking dynamics in cellular adhesion	Dr Anne BLANGY Dr Cécile GAUTHIER-ROUVIERE	Dr Stéphane BODIN	Overexpression of flotillins is detected in a fraction of all solid cancers and participates in metastatic development. It promotes their oligomerisation and the formation of membrane microdomains initiating an endocytosis and vesicular trafficking pathway, named UFIT (Upregulated Flotillin Induced Trafficking pathway). This pathway favors the formation of non-degradative late endosomes with a recycling and signalling functions activating oncogenic signals. Recently we identified the tyrosine kinase receptor AXL as being a cargo of the UFIT pathway. AXL is overexpressed in many tumours and promotes invasion and resistance to therapy. We recently show that the UFIT pathway can participate in AXL stabilization and consequently to its overexpression.	4 months with potential possibility to extend to 5 months	stephane.bodin@crbm.cnrs.fr
M1/M2	**Mechanisms regulating the dynamics of osteoclast cyloskeleton as targets against osteoporosis**	CRBM	Cytoskeleton and membrane trafficking dynamics in cellular adhesion	Dr Anne BLANGY	Dr Anne BLANGY	Osteoclasts hyperactivity causes osteoporosis, a major public health problem, and is associated with bone metastases. We study the molecular mechanisms controlling cytoskeleton dynamics to allow osteoclast adhesion on bone and bone resorption. Our proteomic and transcriptomic data identified new candidate proteins controlling bone resorption. Our general scientific approach is to decipher the signaling pathways taken by candidate proteins to influence cytoskeleton dynamics in osteoclasts and bone resorption activity. Techniques used are cell culture, RNA interference, CRISPR, cutting edge fluorescence microscopy approaches.	Up to 6 months	anne.blangy@crbm.cnrs.fr
M1/M2	**How flotillin upregulation generates exosomes in cancer cells**	CRBM	Cytoskeleton and membrane trafficking dynamics	Dr Cécile GAUTHIER ROUVIERE	Dr Sylia CHEHADE	Metastasis formation is under the control of small extracellular vesicles called exosomes, that are lipid-enriched structures containing proteins and nucleic acids which are released by live cells. These exosomes modify the cell environement and support cell invasion and the formation of the pre-metastatic niche. The group works on proteins named flotillins, that are upregulated in many cancers, which is associated to metastasis formation. The group showed that upregulated flotillin derails the cellular membrane trafic to secrete exosomes and the student will participate to the elucidation of the mechanisms.	5 months	Sylia.chehade@crbm.cnrs.fr cecile.gauthier@crbm.cnrs.fr
M1/M2	**Control of chromosome segregation and genome stability by the ubiquitin ligase SCFGrr1**	CRBM	Mitotic regulation of chromosome partitioning and cell division	Dr Simonetta PIATTI	Dr Simonetta PIATTI	Chromosome instability is a common feature of cancer cells and is characterised by a gain or loss of chromosomes during mitosis, referred to as aneuploidy. Changes in chromosome number can lead to the abnormal expression of oncogenes or tumour suppressors, and be instrumental for tumour onset and development. Furthermore, it can confer a selective advantage that could be exploited by cancer cells to exceedingly proliferate under challenging conditions. Although accurate chromosome segregation is critical for enome stability, our understanding of the mechanisms underlying this process is still fragmentary. Our lab studies how balanced chromosome segregation is achieved, using the budding yeast Saccharomyces cerevisiae as model system. In particular, we are trying to decipher the mechanism of action of the conserved protein kinase Mps1, which is essential for accurate chromosome segregation and also triggers the "Spindle Assembly Checkpoint", the surveillance device that delays mitotic progression until all chromosomes are correctly attached to spindle microtubules. Through a genetic screen, we have recently identified a ubiquitin ligase called SCFGrr1 as an antagonist of Mps1 in both chromosome segregation and the Spindle Assembly Checkpoint. Our preliminary data suggest that SCFGrr1 opposes the function of Mps1 by promoting the degradation of an unknown substrate. Aim of this project is to identify the critical target of SCFGrr1 in the control of chromosome segregation, using multiple approaches that involve genetics, cell biology, biochemistry and mass spectrometry. Altogether, the project will contribute to our understanding of the mechanisms ensuring genome stability in eukaryotes.	6 months	simonetta.piatti@crbm.cnrs.fr
M1/M2	**Gene expression regulation by chaperones in colorectal cancer cells**	CRBM	Regulation of gene expression	Dr Dominique HELMLINGER	Dr Bérengère PRADET BALADE	HSP90, R2TP and TTT are essential for the proliferation of both normal and cancer cells. This chaperone machinery controls an essential step in gene expression by assembling newly synthesized proteins into active complexes, such as the SAGA and TIP60 transcription co-activators. We recently discovered that R2TP and TTT are implicated in colorectal tumor maintenance. The goal of this internship is to characterize how R2TP and TTT regulate SAGA and TIP60 biogenesis in cancer cells. For this, we will use a combination of proteomics and single-molecule imaging in colorectal cancer cell lines. We expect this work to eventually lead to the design of specific inhibitors of R2TP and TTT for cancer treatment.	6 months	pradet@crbm.cnrs.fr dhelmlinger@crbm.cnrs.fr

M1/M2	**Deregulated replication and cancer: Roles of HPV16-E7 oncoprotein and RB tumor suppressor inactivation**	IGMM	DNA Replication, Genome Instability & Cell Ident	Dr Etienne SCHWOB	Dr Vjekoslav DULIC	"High-risk" human papillomaviruses (HPV) are responsible for 5% of all human cancers, including cervical carcinomas. The HPV-16-E7 oncoprotein (E7), which inactivates RB tumour suppressor, has been identified as the main contributor to carcinogenesis. The aim of this project is to decipher the mechanisms whereby RB inactivation by viral oncoproteins compromises genome stability in human cells. Using an inducible RB inactivation model and E7-expressing cells we seek to identify key events responsible for deregulated replication leading to genotoxic stress and chromosome instability at the early and decisive stages of tumour initiation.	5 months	vjekoslav.dulic@igmm.cnrs.fr
M1/M2	**Developing organoïd cultures from brain tumors**	IGF	Brain plasticity, stem cells and diffuse low-grade gliomas	Dr Jean-Philippe HUGNOT	Dr Jean-Philippe HUGNOT	Gliomas are incurable brain tumors. Our research team, located at the IGF focuses on IDH1 mutant tumors that mainly affect young adults (18-30 years). There is no good model for these tumors which poses a real problem for the development of biomarkers of progression and innovative therapies. The development of cultures in the form of 3D organoids is a recent advance in the field and the internship project aims at implementing this mode of 3D cultures to form glioma "tumoroids". These cellular structures will be studied according to several modalities: tumoral cell diversity, interactions of tumor cells with their microenvironment, canonical signalling pathways (Notch) and cancer stem cell genes.	M1: 2 months M2: 6 months	jean-philippe.hugnot@umontpellier.fr
M1/M2	**Role of micro-RNAs modifications dynamic in the regulation of cancer stem cell properties**	IGF	Signaling, Plasticity and Cancer	Dr Julie PANNEQUIN	Dr Chris PLANQUE	These last three years, chemical modifications of RNA emerged as a novel key variable in gene expression control and fine-tuning of biological functions. In a tumoral context, these modifications favor tumor evolution, dissemination, acquisition of resistance to therapy and disease relapse. For now, the presence and the role of chemical modifications in mature micro-RNAs (miRNAs) have been poorly studied, even though these molecules are involved in the regulation of gene expression essential to the maintenance of cancer stem cell (CSC) phenotype, particularly in colon cancer. This innovative project proposes to identify and quantify by mass spectrometry miRNAs chemical modifications from cell populations with cancer stem properties. Thus, miRNA modifications will be sought from models of primary colorectal cancer cultures established in the laboratory and treated by chemotherapy or from cell populations sorted according to their ALDH activity, a functional and metabolic marker of CSCs. In addition to their potential use as biomarkers, these modifications could open up new therapeutic perspectives through the targeting of the enzymatic actors involved in their regulation. This innovative project stems from an ongoing study of SMART (Spectrométrie de masse des Modifications des ARN Totaux) from Montpellier and is conducted within the framework of the «EPITRAN» COST that promotes the development of epitranscriptomic in Europe. The main techniques used during the internship by the student will be the following : molecular biology (RNA extraction from cells and samples), cell biology (cell culture, functional tests), cytometry, mass spectrometry and bioinformatics.	6 months	chris.planque@igf.cnrs.fr alexandre.david@igf.cnrs.fr
M1/M2	**Genome manipulation by CRISPR/Cas9 to explore functional interactions between microRNAs and their targets**	IGH	Systemic impact of small regulatory RNAs	Dr hervé SEITZ	Dr Isabelle BUSSEAU Dr Hervé HEITZ	The project aims at using CRISPR/Cas9/HDR technology to introduce in vivo small sequence changes on the seed of a miRNA of interest, as well as, on a compensatory basis, on its complementary sites in one or more of its target RNAs, to question their function in development and disease. This approach is suitable for application to any miRNA in a wide variety of organisms. The project will suit a candidate willing to use a variety of complementary technologies that are part of the team's areas of expertise: bioinformatics, molecular biology, cell cultures and/or Drosophila genetics	To be discussed with candidate.	Isabelle.busseau@igh.cnrs.fr Herve.seitz@igh.cnrs.fr
M1/M2	**Role of proteins involved in epithelial integrity and polarity, regulated by (de)phosphorylation by Syk or PTPN13, in mammary tumour invasion**	IRCM	Signaling of tumor invasion	Dr Peter COOPMAN	Dr Marion PETER	In the context of breast cancer, we are studying the signalling pathways controlled by the tyrosine kinase Syk and the tyrosine phosphatase PTPN13, which we have shown to be tumour suppressors. The student will characterise new effectors of Syk and PTPN13, involved in the maintenance of epithelial integrity and polarity. The function of these Syk and PTPN13 target proteins, the consequences of their (de)phosphorylation and their contributions to epithelial integrity will be studied in particular by advanced imaging approaches : confocal and two-photon microscopy, FRET/FLIM.	M1: 2 to 6 months M2: 5 to 6 months	marion.peter@inserm.fr
M1/M2	**Proteomic and phenotypic study of melanoma sensitivity to protein kinases inhibitors**	IRCM	Signaling of tumor invasion	Dr Peter COOPMAN	Dr Romain LARIVE	Metastatic melanoma is resistant to classical chemotherapies, but highly sensitive to drugs targeting the proteins kinases of the canonical signaling pathway MAPK. Nevertheless, melanoma cells acquire resistance to these new treatments, by various cellular mechanisms and molecular plasticity of cell signaling. Using our quantitative phosphoproteomic data, we modelize the intracellular molecular signaling of melanoma cells that are sensitive or resistant to MAPK inhibitors. During his/her internship, the student trainee will evaluate the value of these mathematical models to predict the sensibility of melanoma cells to new combinations of inhibitors in order to link cell sensitivity to the inhibitors (cellular phenotype) to intracellular signaling dynamic.	2 to 6 months	romain.larive@umontpellier.fr
M1/M2	**Search for new therapeutic targets in KRAS-associated signalling pathways in lung adenocarcinoma**	IRCM	Signaling of tumor invasion	Dr Peter COOPMAN	Dr Gilles FREISS	Within the theranostic biomarkers of lung cancer KRAS gene mutations are characterised by their frequency and the difficulty in developing effective inhibition strategies. We seek to identify novel signalling pathways interconnected with KRAS pathways. We have identified two signalling proteins, the tyrosine kinase Syk and the tyrosine phosphatase PTPN13 that appear to be specifically involved in lung adenocarcinoma tumorigenesis. We aim to identify the signalling networks linking KRAS, SYK and PTPN13 from interactome and (phospho)proteome analyses combined with bioinformatics studies. To this end, cell and mouse models of KRAS mutated lung cancers expressing or not PTPN13 or SYK have been developed or are under development. These models will allow us to evaluate the effects of the expression of these two enzymes on the aggressiveness of KRAS mutated lung tumours and to study the interactions between their signalling pathways and that of KRAS.	6 months	gilles.freiss@inserm.fr

M1/M2	**Metabolic analysis of effector and regulatory γδ T cell populations and impact on anti-tumor immune response**	IRCM	Immunity and Cancer team	Dr Virginie LAFONT	Dr Ghita CHABAB	Cellular metabolism comprises a series of interconnected biochemical pathways that use energy-rich molecules to produce ATP either by oxidative phosphorylation (OXPHOS) or by performing glycolysis. T cell activation is generally linked to a metabolic switch from OXPHOS to glycolysis. While naive T cells rely on OXPHOS to maintain energy demand; activated T cells engage increased glycolysis and glutaminolysis consuming massive amount of glucose and glutamine, to generate their functions. In contrast, the metabolic profile of regulatory T cells relies on OXPHOS and fatty acid oxidation (FAO) to support their survival and differentiation. γδ T cells participate to the immune response against many tumors through their direct cytotoxic activity against cancer cells and their capacity to regulate the biological functions of other immune cells. Nevertheless, their presence in the tumor microenvironment has also been associated with poor prognosis in several cancers suggesting that γδ T cells may also display pro-tumoral activities. Accordingly, we recently described that γδ T cell subsets expressing CD73 display regulatory functions through the production of immunosuppressive molecules, such as IL-10, adenosine and the chemotactic factor IL-8. In parallel, we showed that in human breast and ovarian tumors, γδ T cells were present and that ~20% of tumor-infiltrating γδ T cells expressed CD73 and displayed suppressive functions. The project will aim at providing the characterization and comparison of metabolic programs used by CD73- (effector) and CD73+ (regulatory) γδ T cell subsets with the final goal to identify mechanisms able to boost the anti-tumor immune response. The metabolic program of γδ T cells will be analyzed by an innovative protocols: the SCENITH, a flow cytometry-based method to functionally profile energy metabolism with single cell resolution. From these data, new therapeutic approaches could be proposed to improve the anti-tumoral functions of effectors γδ T cells.	M1: 2-4 months M2: 5-6 months	Virginie.lafont@inserm.fr
M1/M2	**Role of autophagy in the dialogue between neurons and cancer cells in colorectal cancer**	IRCM	Tumor microenvironnement and resistance to treatment	Dr Andrei TURTOI	Dr Sophie PATTINGRE	The presence of perineural invasions within tumors is a sign of the aggressiveness of the tumor and resistance to treatments. Autophagy, that allows the lysosomal degradation of intracellular material, is frequently activated in cancer, favoring the cancer cell survival during stress. The aim of this project is to study the role of autophagy, in the dialogue between neurons and cancer cells, in the formation of perineural invasions and their pro-tumor functions.	6 months	sophie.pattingre@inserm.fr
M1/M2	**Targeting the breast tumor microenvironment with anti-cathepsin D hydrophilic Antibody Drug Conjugates (ADC) with high Drug Antibody Ratio (DAR)**	IRCM	Breast Cancer, microenvironment and Immunotargeting	Dr Emmanuelle LIAUDET COOPMAN	Dr Valérie LAURENT-MATHA	Few targeted therapies are available for recurrent and treatment-resistant breast cancer. The aspartyl protease, cathepsin D (Cath-D), a poor prognostic marker is overexpressed and hypersecreted by different subtypes of breast cancer, both hormono-dependent (HR+) expressing and not expressing HER2 (HER2+/-), and triple-negative (TNBC, HR-/HER2-). Works of the team have shown that Cath-D secreted in the tumour microenvironment displays oncogenic actions. With the support of Labex MAbImprove, the immunotargeting of secreted Cath-D with human anti-Cath-D (F1) antibodies (Ab) led to a significant decrease of tumour growth in vivo in nude mice xenografted with the TNBC line, MDA-MB-231, and with PDXs (Ashraf*, Mansouri* et al., JITC, 2019). Hypersecreted Cath-D is thus a new target opening new therapeutic perspectives. In view of the growing interest of ADCs (Antibody Drug Conjugate) in the treatment of recurrent cancer, we are developping an anti-Cath-D ADC with a high DAR 8 (Drug to Antibody Ratio), hydrophilic, coupled via a cleavable arm to a topoisomerase I inhibitor, Deruxtecan, using the same technology as those used for the development of the ADC "Enhertu" approved as third line treatment in recurrent HER2+ breast cancer. As Cath-D is internalised by different tumour cells (epithelial cells and fibroblasts), as well as the Cath-D/ADC-anti-Cath-D complex, a strong tumour regression should be induced. This approach is original because the target is both secreted and associated with cell membranes from tumor and stromal cells, contrary to available ADCs which are only directed against membrane receptors. This anti-Cath ADC should strongly amplify the therapeutic impact of the observed naked Ab. The objectives of this master's degree course are to study the cytotoxic activity of this anti-Cath-D ADC in vitro in comparaison with a control ADC in the MDA-MB231 and Sum159 TNBC cell lines, and the hormone-resistant (HR+) MCF-7-LCC2 line. We will test the effect of anti-cath-D ADC on cell survival in two-dimensional (2D) and three-dimensional (3D) culture in spheroid models. Internalization experiments of the ADC/CathD complex will be performed on cancer cells and stromal cells. The immuno-targeting of the massively secreted Cath-D in the breast tumour microenvironment with a human ADC-anti-Cath-D biomedicine should open up new treatment perspectives for patients resistant to currently available treatments with fewer side effects than conventional chemotherapy.	4 to 6 months	valerie.laurent2@umontpellier.fr
M1/M2	**Transcriptional Regulation of the inflammatory phenotype in Ulcerative Colitis**	IRCM	Nuclear signaling and cancer	Dr Vincent CAVAILLES	Dr Marion LAPIERRE Dr Vincent CAVAILLES	This project aims to study the impact of the transcription factor RIP140 on the inflammatory process in the intestinal mucosa. The objective is to clarify its biological role in the inflammatory response and to study its impact on signaling pathways deregulated during inflammation. This will be addressed by in vitro approaches on cancer cell lines and in vivo using transgenic mouse models, and validated on biopsies from patients with chronic inflammatory bowel diseases.	2 à 6 months	marion.lapierre@inserm.fr
M1	**Study of the functional interactions between the transcriptional coregulators RIP140 and LCoR in breast carcinogenesis**	IRCM	Nuclear signaling and cancer	Dr Vincent CAVAILLES	Dr Stépahn JALAGUIER	Breast cancer, the most common cancer in women, involves many players including transcription factors. Among the latter, we have demonstrated that the RIP140 and LCoR proteins play a major role in breast carcinogenesis. The aim of the internship will be to decipher the functional interactions between the two transcription factors in breast cancer models.	2 mois	Stephan.jalaguier@inserm.fr
M1/M2	**Transcriptional control of the tumor immune ecosystem**	IRCM	Nuclear signaling and cancer	Dr Vincent CAVAILLES	Dr Marion LAPIERRE Dr Vincent CAVAILLES	The transcriptional coregulator RIP140 is involved in key steps of colorectal carcinogenesis. Our results clearly indicate that RIP140 controls the remodeling of the immune microenvironment of these tumors. Indeed, histological analysis of the colon of RIP/APCKOint mice shows important differences in particular concerning tertiary lymphoid structures when Rip140 is no longer expressed in intestinal epithelial cells. The objectives of this internship are therefore to study the effect of RIP140 on the remodeling of the immune microenvironment of intestinal tumors by deciphering the underlying signaling pathways and by clarifying its role in vivo in the antitumor immune response.	6 months	vincent.cavailles@inserm.fr
M1	**Transcriptional control of tumor hypoxia-induced responses by RIP140 in colon cancer**	IRCM	Nuclear signaling and cancer	Dr Vincent CAVAILLES	Dr Catherine TEYSSIER	Hypoxia, which corresponds to a decrease in the level of oxygen in tissues, promotes the development and progression of cancers, including colon cancers. HIF(hypoxia inducible factors)-1 and especially HIF-2 play important roles in colon cancer. However, the precise mechanisms by which HIF-1 and HIF-2 lead to distinct cellular responses still need to be defined. Our project aims to define the basis of the interference between hypoxia and RIP140, a transcription coregulatory that acts as as a tumor suppressor in colon cancer and interacts with HIF. Our institute possesses a brand new hypoxia station that allows the incubation of cell and organoids samples under controlled O2 pressure. The overall objective of our project is to identify a new regulator of tumoral hypoxia in colon cancer. The internship will benefit of available tools and cellular models, along with the established expertise of the team in transcription and cancer. The intern will also take advantage of the well-equipped and thought-provoking en coupled via a cleavab	2 months	Catherine.teyssier@inserm.fr

M1/M2	**Detection and characterization of the RIP140 mutation in patients with microsatellite instable colorectal cancer**	IRCM	Nuclear signaling and cancer	Dr Audrey CASTET-NICOLAS	Dr Marion LAPIERRE	In colorectal cancer (CRC) with microsatellite instability (MSI), a truncative mutation of RIP140 (RIPMSI) exerts a dominant negative effect and is associated with a significant decrease in the survival of patients. The RIPMSI mutation thus represents a new potential prognosis/predictive marker. The goal is to further characterize this mutant and develop new detection techniques. We will set up its detection on circulating cell-free DNA (cfDNA) from blood samples and by immunohistochemistry on tissue sections using a specific anti-RIPMSI antibody. We will compare the sensibility and specificity of these techniques and validate the correlation with patient survival.	6 months	audrey-castet@chu-montpellier.fr
M1	**Development of therapeutic proteins data search tools for IMGT/mAb-DB feeding**	IMGT	IMGT®, the international ImMunoGeneTics information system®	Pr Sofia KOSSIDA	Dr Taciana MANSO Pr Sofia KOSSIDA	The international ImMunoGeneTics information system® (IMGT®, http://www.imgt.org) characterizes genes and alleles of the antigen receptors, immunoglobulins (IG) and T cell receptors (TR) since 1989. IMGT/mAb-DB the IMGT database for monoclonal antibodies (mab) and other therapeutic proteins with clinical indications, is a unique resource containing comprehensive therapeutic metadata. IMGT/mAb-DB data are extracted from WHO-INN Proposed and Recommended lists and the amino acid sequences are annotated by IMGT experts. In this project, the student will develop bioinformatics tools to harvest some information from WHO-INN database for INN data and other official websites for therapeutic metadata to feed IMGT/mAb-DB.	2 months	sofia.kossida@igh.cnrs.fr
M1/M2	**The mitochondrial network, a reflection of cellular "health status": Applications in Oncology**	ISEM	Isem, équipe EVAS	Dr Mylène WEILL	Dr Sophie CHARASSE Dr Abdel AOUACHERIA	The abundance, morphology and dynamics of mitochondria allow a reading critical of the internal cellular state. The maintenance of structurally integrated and metabolically active is a condition sine qua non to the proper functioning of cells and survival and "good health" of organisms (Aouacheria et al., 2017). In response to various intracellular and extracellular, mitochondria adapt their number, shape, position, shape, connectivity and their movement. Cells containing tubular mitochondria or elongated, networked and energy-producing are generally viable and perform their functions. Conversely, stressed cells often have mitochondria fragmented, isolated, with low membrane potential and mitochondrial functions compromises. This «MITOMATIQUE» project proposes to analyze and quantify mitochondrial networks, by confocal microscopy screening in: •Various cancer versus normal cell types •Different tumour stages (tumour progression, cell invasion) •Test the efficacy of new therapeutic molecules (single or combined).	5 mois	sophie.charrasse@umontpellier.fr