Master	Title	Institute	Host Laboratory	Name of the PI	Supervisor	Description	Duration	e-mail
M1/M2	Caracterization of RUVBL 1/2, a new target in colorectal cancer	CRBM	Regulation of Gene Expression	Dominique HELMLINGER	Bérangère PRADET -BALADE	We recently found that the drug CB-6644 is cytotoxic in pre-clinical models that recapitulate a very aggressive subtype of colorectal cancer. CB-6644 inhibits the ATPase activity of RUVBL 1/2, which is part of chromatin remodelers and cytoplasmic chaperones. The Master student will use new tools that we develop in collaboration with chemists, for affinity-purifications and targeted degradation (PROTAC) of RUVBL 1/2, in transcriptomics and proteomics. These experiments will provide a comprehensive understanding of RUVBL 1/2 activity in colorectal cancer, an important question both for basic and translational biology.	6 months	berengere.pradet-balade@crbm.cnrs.fr
M1/M2	SAGA and TIP60 co-activators modulate enhancer-dependent gene expression in a context of MYC transcriptional addiction	CRBM	Regulation of Gene Expression	Dominique HELMLINGER	Peggy RAYNAUD	Transcription factors drive transcription initiation by recognizing consensus sites within promoters and enhancers, where they recruit cofactors to activate RNA polymerase II. Enhancers integrate signaling pathways to regulate transcription both temporally and spatially, and their reprogramming is now considered a key driver of tumorigenesis, providing phenotypic plasticity. We found that the SAGA and TIP60 transcriptional co-activators bind enhancers in colorectal cancer cells, and we are applying integrated functional genomic approaches to characterize their roles at these regions, with a focus on MYC-driven transcription addiction.	6 months	dhelmlinger@crbm.cnrs.fr peggy.raynaud@crbm.cnrs.fr
M2	Deciphering and targeting the collagen-Discoidin Domain Receptor signaling to enhance the therapeutic response in colorectal cancer	CRBM	Cancer cell Signaling	Serge ROCHE	Audrey SIRVENT	Several recent studies propose that the tyrosine kinase receptors DDR1 and DDR2 are important mediators of collagens-induced tumor-promoting effects, contributing to tumor progression, therapeutic resistance, and poor prognosis in colorectal cancer (CRC). This project aims to: (1) investigate the spatio-temporal dynamics of DDR1/2 signaling using TurboID-based proximity labeling combined to mass spectrometry; (2) characterize the role of DDR1/2 signaling in immune exclusion within the CRC tumor microenvironment and (3) evaluate whether pharmacological inhibition of DDR1/2 kinase activity using already clinically approved drugs developed for other targets can overcome resistance/enhance CRC therapeutic response. Methods: 2D & 3D cell culture, western-blots, immunoprecipitation, proteomics, multiplex flow cytometry and immunohistochemistry.	5 to 6 months	audrey.sirvent@crbm.cnrs.fr
M1/M2	PFAS and intestinal tumorigenesis: effect through the regulation of the tumor immune microenvironment	IRCM	Nuclear Signaling in Cancer	Vincent CAVAILLES	Vincent CAVAILLES	The main scientific question is to define how persistent pollutants (PFAS) influence the immune microenvironment of colorectal cancers (CRC), and what are the underlying molecular mechanisms in particular the role of tumor metabolites. We will employ a cocktail of different PFAS in order to provide a comprehensive understanding of their effects on the immune contexture of intestinal tumors using CRC cell lines, a genetically engineered mouse model and patient-derived tumor explants (PDTE).	6 months	vincent.cavailles@inserm.fr
M1/M2	Microbial metabolites in gut homeostasis and their potential as cancer therapeutics	IRCM	Epitranscriptomics & Cancer Adaptation	Alexandre DAVID	Kuldeep LAHRY	Microbial metabolite queuine (Q) is salvaged by mammals and site-specifically incorporated into a subset of cytoplasmic and mitochondrial tRNAs by the host tRNA-guanine transglycosylase (QTRT1/QTRT2). Queuosine modification at the wobble-position G34 stabilizes tRNAs and modulates mRNA decoding efficiency at defined codon sets. Although Q is dispensable for cell viability under standard culture conditions, hypomodification of Q-tRNAs correlates with proliferative disorders (including cancer) and has been implicated in development, differentiation, aging, and neuroprotection. Building on our recent unpublished findings (Zhang W*., Lahry K*., et al. Nature Cell Biology, in press), we have discovered that the upstream microbial precursor pre-queuosine1 (preQ1) exerts anti-proliferative activity in both human and mouse cells. In vitro and in vivo data show that preQ1 is bioavailable in plasma and tissues and becomes incorporated into host tRNAs. Mechanistically, preQ1 incorporation leads to selective depletion of its cognate tRNA species, driving translational repression that disproportionately impacts housekeeping gene expression. We further identify IRE1 RNase activity as a mediator of ribosome-associated, selective degradation of preQ1-modified tRNAs, independent of canonical unfolded protein/stress signaling. This Master's project will dissect molecular and cellular mechanisms by which the Q/preQ1 axis couples the microbiota to host physiology programs, with emphasis on intestinal epithelial homeostasis and cancer progression. The host team focuses on the contribution of RNA modifications on cancer cell adaptation using genetic studies in living cells and animals, together with RNA mass spectrometry, next-generation sequencing and AI-based approaches.	6 months	alexandre.david@inserm.fr kuldeep.lahry@inserm.fr
M1	Harnessing the epitranscriptome to identify new players involved in chemoresistance in ovarian cancer.	IRCM	Epitranscriptomics & Cancer Adaptation	Alexandre DAVID	Stanislas QUESADA	High-grade serous ovarian carcinomas (HGSOC) are the most lethal gynecological malignancies. Due to the absence of symptoms in early stages, 70% of cases are diagnosed at an advanced FIGO stage III/IV. For these advanced stages, there are currently no predictive markers for neoadjuvant chemotherapy effectiveness, as the known indicators of response (e.g., KELIM, CRS) are by definition only available after treatment. As such, we sought to characterize this specific population through epitranscriptomic analysis. Indeed, over the past three years, chemical modifications of RNA have emerged as a new epigenetic layer involved in all stages of gene expression regulation and controlling key biological processes. This Master residency will rely on two aspects: 1/ Biomarker discovery through cutting edge technology for predictive purpose 2/ functional analysis of candidate genes involved in épitranscriptome metabolism, as a new approach to decipher new drivers of chemoresistance.	6 months	Stanislas.quesada@icm.unicancer.fr Alexandre.david@inserm.fr
M1/M2	Investigating the Therapeutic Efficacy of an Anti-SCARB1 Antibody in Renal Cell Carcinoma	IRCM	Metabolism and sarcomas	Laetitia LINARES	Benjamin FOURNEAUX	As part of our ongoing research on therapies targeting tumor metabolic dependencies, our team has recently developed a novel antibody against SCARB1, a key cholesterol transporter implicated in the progression of clear cell renal cell carcinoma (ccRCC). This internship project aims to evaluate the in vitro efficacy and specificity of this antibody using a range of techniques, including immunofluorescence, flow cytometry, western blotting, and cell viability assays. We are looking for motivated Master's level students (M1 or M2) with a strong interest in cellular biology, oncology, and biotherapeutics. This internship offers a hands-on opportunity to contribute to translational cancer research in a dynamic and collaborative academic environment.	5/6 months	benjamin.fourneaux@inserm.fr laetitia.linares@inserm.fr
M1/M2	Understanding liposarcoma-muscle crosstalk	IRCM	Metabolism and sarcomas	Laetitia LINARES	Gilles GADEA	Liposarcomas are rare cancers of the adipose tissue which are resistant to all currently available therapies. Only large and radical surgery can cure a limited number of patients. Liposarcomas are characterized by MDM2 amplification. Our team has shown that MDM2 protein can control amino acid metabolism, making liposarcomas dependent on serine. More recently, the team showed that this dependence is such that it forces the establishment of a crosstalk between the tumor and the skeletal muscles, which become serine supplier for tumor growth. In this context, this project proposes to study the molecular basis of dialogue, more specifically the role of extracellular vesicles in muscle rewiring.	6 months	gilles.gadea@inserm.fr

Master	Title	Institute	Host Laboratory	Name of the PI	Supervisor	Description	Duration	e-mail
M1/M2	Inter-organ communications during cancer-associated cachexia	IRCM	Epithelial growth and cancer	Alexandre DJIANE	Charles GEMINARD	Cachexia is a systemic disorder frequently associated with cancer, characterised by severe weight loss, muscle and adipose tissue atrophy. These profound metabolic changes result in general weakness and cachexia is responsible for the death of 30% of cancer patients. Despite its clinical importance, there are currently no robust biomarkers and therapeutic strategies are mostly ineffective. Using drosophila and mouse models of cachexia we have identified the molecules secreted by the tumours, and the other organs (liver, adipose...) potentially contributing to tissue wasting. We currently work on two main axes: i) we have shown that the CREBRF/REPTOR transcription factor is required for adipose tissue wasting and we seek to identify the transcriptional and metabolic programs they control. ii) using blocking antibodies in mouse models, we could demonstrate the role of a family of ligands in adipose tissue atrophy, thus reverting cachexia, and we seek to better understand their mode of action.	4/6 months	alexandre.djiane@inserm.fr charles.geminard@inserm.fr
M1/M2	Investigating a new level of crosstalk between the p53 and the Hippo pathway in cancers	IRCM	Epithelial growth and cancer	Alexandre DJIANE	Lisa HERON-MILHAVET	Hippo and p53 represent two tumour suppressor pathways frequently affected in cancers. In the laboratory, we have uncovered new physical interactions between members of the Hippo and p53 pathways. The objective of the project is to molecularly characterize these new levels of cross-talk and give insights into their functional implications in human cancer cell lines.	4/5 months	Lisa.heron-milhavet@inserm.fr
M2	MelanoPredict : How to predict the unpredictable with the rare melanoma cells that will resist to therapy ?	IRCM	Signaling of tumor invasion	Peter COOPMAN	Romain LARIVE	La prise en charge du mélanome avancé a été révolutionnée depuis 15 ans grâce à la découverte de l'oncogène BRAF-V600 (~45 % des cas), permettant le développement d'inhibiteurs ciblant la voie MAPK (MAPKi), et grâce à l'immunothérapie avec le développement d'anticorps inhibiteurs de points de contrôle immunitaire. Ces deux approches ont permis une amélioration spectaculaire du pronostic des patient·es, mais l'efficacité des inhibiteurs MAPK est limitée par l'apparition très fréquente de résistances acquises. L'efficacité des traitements anticancéreux repose sur l'individualisation des traitements. Pour les MAPKi, aucun biomarqueur ne permet de prédire la durée de réponse au traitement. Notre hypothèse est que le réseau de signalisation intracellulaire MAPK est au cœur de la résistance du mélanome aux MAPKi. Nous avons développé une approche de phospho-protéomique quantitative sur cellule unique pour analyser l'hétérogénéité intratumorale, et nous avons récemment identifié une sous-population minoritaire préseque des paraspeckles en conditions physiologique ou pathologique. Paraspeckles are nuclear membraneless organelles formed by phase separation of their constituents and they are altered in several diseases like cancers. To explore the consequences of association of such nuclear organelles with genomic sequences for 3D genome organization and gene expression, the team has developed the RD-HRS method (Lecouvreur et a	5/6 months	romain.larive@umontpellier.fr
M1/M2	Search for upstream signaling pathways that activate the Syk tyrosine kinase and mediate its tumor suppressor and drug response activities in breast cancer	IRCM	Signaling of tumor invasion	Peter COOPMAN	Peter COOPMAN Marion PETER	We first discovered that the protein kinase Syk acts as a tumor suppressor in breast cancer (Nature, 2000). Our research has mainly focused on the role of Syk downstream effectors in cancer. Conversely, this internship aims to identify upstream signaling pathways that activate Syk in breast cancer cells, which currently remain unknown. Using (phospho)proteomic analyses on a differential interactome, comparing WT with mutant Syk forms that no longer localize nor interact at the sub-membrane level, we identified a unique and promising candidate protein that will be studied in detail by cancer cell biology assays (proliferation, invasion, cytotoxicity), immunocytochemistry and biochemistry.	2 to 6 months (Master 1) 5 to 6 months (Master 2)	peter.coopman@inserm.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	Peter COOPMAN	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 by different approaches, including advanced imaging techniques.	2 to 6 months (Master 1) 5 to 6 months (Master 2)	marion.peter@inserm.fr
M2	Analysis of cytokine and metabolic signatures in sebaceous tumors in relationship with tumor malignancy and the Mismatch repair system. Analyse des signatures cytokinique et métabolique dans les tumeurs sébacées en lien avec la malignité des tumeurs et le système Mismatch repair.	IRCM	Molecular Oncogenesis	Laurent LE CAM	Eric FROUIN Matthieu LACROIX	The project of this M2 internship aims to better understand the tumorigenesis of human sebaceous tumors and in particular the deficiency of the mismatch repair system, and whether this carcinogenesis pathway is linked to lipid metabolism or modifies the immune microenvironment of these tumors. During this internship, the student will learn to use various technologies: Tissue-micro-array fabrication, microscopic analyses, RNA and DNA extractions, pangenomic analyses, cell culture. Part of the internship will take place within the Department of Pathological Anatomy of the Nîmes University Hospital.	6 months	eric.frouin@chu-nimes.fr
M2	Deciphering the Metabolic and Epigenetic Heterogeneity in IDH-mutant Acute Myeloid Leukemia	IRCM	Molecular Oncogenesis	Laurent LE CAM	Lucille STUANI	The focus of this internship is to investigate how the metabolic adaptations induced by IDH mutations in Acute Myeloid Leukemia (AML) and in particular related or not to the production of the oncometabolite 2-HG, are impacting epigenetic changes and vice versa, affecting tumor heterogeneity and response to therapies, in particular IDH mutant inhibitors.	6 months	Lucille.stuani@inserm.fr
M2	Rôle des paraspeckles dans l'organisation 3D et l'expression du génome	IGMM	Genome organization and epigenetic control	Thierry FORNE	Thierry FORNE	Les paraspeckles sont des organites nucléaires sans membrane formés par séparation de phase de leurs constituants et ils sont altérés dans plusieurs maladies dont certains cancers. Afin d'explorer les conséquences de l'association de ces organites avec la chromatine pour l'organisation 3D et l'expression du génome, l'équipe a développé la méthode RD-HRS (Lecouvreur et al., sous presse : https://hal.science/hal-04711849v1). Cette méthode permet un profilage global des séquences d'ADN et d'ARNs associées à l'ensemble des corps nucléaires, sans savoir lesquelles correspondent à un corps nucléaire donné. Le but du projet sera d'adapter l'approche de marquage par hybridation-proximité (HyPro) (Yap et al., 2021, Mol. Cell 82, 463-478) à la méthode RD-HRS pour biotinyler les ARN et l'ADN génomique à proximité du long ARN non codant NEAT1_2 spécifique des paraspeckles dans une lignée de fibroblastes bien caractérisées (cellules IMR-90). Le séquençage des ADN/ARN biotynylés permettra de comparer la composition spécifique des paraspeckles en conditions physiologique ou pathologique. Paraspeckles are nuclear membraneless organelles formed by phase separation of their constituents and they are altered in several diseases like cancers. To explore the consequences of association of such nuclear organelles with genomic sequences for 3D genome organization and gene expression, the team has developed the RD-HRS method (Lecouvreur et al., in press: https://hal.science/hal-04711849v1). This method allows global profiling of DNA sequences and RNAs associated with all nuclear bodies, without knowing which sequences correspond to a given nuclear body. The aim of the proposed project will be to adapt the Hybridization-Proximity (HyPro) labelling approach (Yap et al. 2021, Mol. Cell 82, 463-478) to the RD-HRS method to biotinylate the RNAs and genomic DNA in the vicinity of the paraspeckles-specific NEAT1_2 long non-coding RNA in the well-characterised IMR-90 fibroblasts. Sequencing of biotinylated DNA/RNAs will then allow to compare the specific composition of paraspeckles in physiological or pathological conditions.	5/6 months	thierry.forne@igmm.cnrs.fr

Master	Title	Institute	Host Laboratory	Name of the PI	Supervisor	Description	Duration	e-mail
M1/M2	Development of PROTACs for a targeted inhibition of SUMOylation in cancer cells	IGMM	SUMOylation and myeloid leukemias	Guillaume BOSSIS	Olivier COUX	SUMOylation is a post-translational modification that modifies protein function and fate in a variety of ways and is consequently involved in the regulation of most cellular processes. G. Bossis's team (IGMM) has shown that SUMOylation (1) plays a critical role in Acute Myeloid Leukemias (AML) response to therapies (chemotherapies, differentiation therapies and epigenetic therapies) and (2) is an interesting therapeutic target in AML. The goal of the project is to develop new strategies to inhibit SUMOylation, based on PROTACs (*) targeting SUMO enzymes. (*) PROTACs are bifunctional molecules able to provoke the degradation of their target (see Alabi & Crews (2021) J Biol Chem. 296, 100647)	To be discussed with applicants	olivier.coux@cnrs.fr guillaume.bossis@igmm.cnrs.fr
M1/M2	SUMOylation & epigenetics in AML blood cancer	IGMM	SUMOylation and myeloid leukemias	Guillaume BOSSIS	Denis TEMPÉ	Our team has validated SUMOylation as a therapeutic target in a preclinical model of Acute Myeloid Leukemia and identified the transcriptional and epigenetic changes regulated by it. Our current objectives are (i) to identify the proteins involved in this regulation by CRISPR and ATAC-seq screens and (ii) to set up a cellular model for modulating SUMO activity at specific genomic locations. The 6-month internship will contribute to the progress of this project by combining these two approaches in order to validate screen hits and to understand SUMOylation's direct involvement in transcriptional and epigenetic control.	6 months	denis.tempe@igmm.cnrs.fr
M1/M2	Investigating DNA Replication Dynamics in Genome Stability and Cancer	IGMM	DNA replication, genome instability and cell identity	Etienne SCHWOB	Cyril RIBEYRE	DNA replication is a highly regulated process influenced by numerous constraints that affect genome stability and cellular responses to cancer therapies. Our lab investigates key aspects of this regulation, with a particular focus on replication origin control and the replication of heterochromatin. We employ a range of experimental techniques—including microscopy, DNA combing, CUT&RUN, and ChIP-seq—to explore these mechanisms at both molecular and genomic levels. The internship project will center on one of these research areas, depending on the student's interests and ongoing work in the lab.	6 months	cyril.ribeyre@igmm.cnrs.fr
M2	Exploration of PTK7-expressing fibroblasts in colon pathologies	IGMM	Inflammation and cancer	Michael HAHNE	Bénédicte LEMMERS	Protein Tyrosine Kinase 7 (PTK7) receptor expression is associated with bad prognosis in colorectal cancer (CRC) and has been accordingly suggested as a drug target. Our immunohistological analysis of mouse colons displayed PTK7 expression in both colonic epithelial cells (CEC) and fibroblasts and we therefore have generated cell type specific knock-out mice, deleting PTK7 in either CEC or different colonic fibroblast subsets. During his or her internship, the student will explore these mouse models using up to date approaches.	6 months	benedicte.lemmers@igmm.cnrs.fr
M2	Impact of 5-Fluorouracil on cell plasticity and drug tolerance in colorectal cancer	IGF	Signalization, plasticity and cancer	Julie PANNEQUIN	Laura JENTSCHEL	We recently discovered that 5-FU is incorporated into ribosomal RNA, leading to translational reprogramming in colorectal cancer cells. Our translatome profiling revealed a drug-tolerant state marked by increased translation of genes linked to senescence, metabolism, and epigenetic regulation—features associated with tumor plasticity and relapse. This project investigates how these changes contribute to 5-FU resistance, with the goal of identifying novel therapeutic vulnerabilities in persister cancer cells and prevent tumor relapse.	5/6 months	Laura.jentschel@igf.cnrs.fr julie,pannequin@igf.cnrs,fr
M1/M2	Ferroptosis inducers: an adjuvant strategy to target persister cells and prevent tumor recurrence in BRAF^V600E-mutated colorectal cancer?	IGF	Signalization, plasticity and cancer	Julie PANNEQUIN	Jean-Marc PASCUSSI	Colorectal cancer (CRC), particularly in its metastatic form, is among the most common and deadly cancers worldwide, with the BRAFV600E mutation—present in 8–15% of CRC cases—being associated with poor prognosis and high recurrence rates. These aggressive and treatment-resistant tumors may harbor a higher number of persister cells, which are central to therapy resistance and relapse, and appear to rely heavily on iron metabolism, making them susceptible to ferroptosis, an iron-dependent form of cell death. Targeting ferroptosis in combination with current therapies may offer a promising strategy to eliminate these resistant cells and improve outcomes for patients with BRAFV600E-mutant CRC.	6 months	Jean-marc.pascussi@inserm.fr
M1/M2	Optimizing bicomponent pore forming toxins for CXCR4-targeted cancer cell killing	IGF	Signalization, plasticity and cancer	Julie PANNEQUIN	Jean-Marc PASCUSSI	Pore forming toxins (PFTs) are a class of proteins often produced by pathogenic bacteria as virulence factors. These proteins fold as soluble monomers that subsequently recognize cell membranes and oligomerize into transmembrane pores. These structures alter membrane permeability, leading to multiple cellular responses including cell death. In theory, PFTs could be engineered into a new paradigm of Paul Ehrlich's "magic bullet" idea, which may translate into a new class of targeted therapies for cancer. However, natural toxins recognize specific lipids, sugars or protein receptors that in most cases are not relevant cancer targets. Here we propose to use Staphylococcus aureus (SA) bicomponent PFTs as scaffolds to engineer synthetic toxins that specifically target cancer cells or the associated tumour microenvironment. Several members of this PFT family naturally recognize subsets of human chemokine receptors including CCR2, CCR5, CXCR1, CXCR2 and ACKR1. Using recently developed computational design approaches in combination with cell-based assays, we have established the proof of concept for manipulating the lytic activity of bicomponent PFTs through reengineering of receptor binding specificity. We will use this methodology to engineer toxins that show specific activity toward CXCR4, and fine-tune the self-assembling properties of the synthetic toxins to control their lytic activity thresholds with high precision . We will investigate the impact of the designed toxins in the context of colorectal cancer and establish a proof-of-concept for the potential therapeutic use of synthetic bicomponent PFTs (aim 3). This research will enable the development of a new generation of self-targeted protein-based therapeutics for cancer.	6 months	Jean-marc.pascussi@inserm.fr
M2	Establishing an in vitro 3D co-culture model to study the crosstalk between murine colorectal cancer cells and sensory neurons	IGF	Signalization, plasticity and cancer	Julie PANNEQUIN	Caroline BONNANS Nicolas DIGLERIA-PILLARD	The aim of the Master student project will be to study the crosstalk between murine colorectal cancer cells and sensory neurons in an in vitro 3D co-culture model by using tumoroids. The student will work on finding the best culture conditions for optimum viability and interaction (Objective 1). The student will measure tumoroid size and neurite number and length to be able to determine whether cancer cells and sensory neurons could impact their respective growth (Objective 2).	6 months	caroline.bonnans@igf.cnrs.fr
M1	Development of a first-in-class prodrug-drug conversion system to target highly specific Multiple Myeloma cells	IGH	Epigenome modifications and genomic instability in normal and malignant B cells	Jérôme MOREAUX	Malik LUTZMANN	Multiple Myeloma is a non-curable blood cancer of antibody producing cells (plasmocytes) that did not exit the cell cycle but continue to proliferate, producing toxic amounts of useless antibodies and suppressing normal hematopoiesis. This project aims to target these cancer cells in a completely new, highly specific approach by reconstituting only in the presence of intracellular antibodies (thus only in Multiple Myeloma cells) an enzyme that converts a non-toxic prodrug into a cytotoxic drug.	6 months	Malik.lutzmann@igh.cnrs.fr
M1/M2	Assembly of snRNP U5, a key component of the spliceosome	IGH	Cell biology of RNA	Edouard BERTRAND	Céline VERGHEGGEN Séverine BOULON	The laboratory is involved In studying the action mechanism of R2TP chaperone, working with HSP90 in cellular machinery assembly. U5 snRNP that is part of the spliceosome is assembled by R2TP. If spliceosome is not correctly assembled, this leads to alterations involved in human diseases. By using Alphafold3, we have predicted interaction domains between U5 components and R2TP and its cofactors. We will evaluate how the expression of punctual mutants of this interaction can affect assembly and localization of snRNP U5 in human cells.	6 months	celine.verheggen@igh.cnrs.fr severine.boulon@igh.cnrs.fr

Master	Title	Institute	Host Laboratory	Name of the PI	Supervisor	Description	Duration	e-mail
M2	Analyse biophysique et fonctionnelle de granules d'ARN: Exemple des granules germinaux chez la drosophile Biophysical and functional analysis of RNA granules: example of Drosophila germ granules	IGH	mRNA Regulation and development	Martine SIMONELIG	Anne RAMAT	Membraneless condensates are thought to play a key role in organizing biological reactions within the cell. However, the relationships between the organization of condensates and their functions are poorly understood. RNA granules are a specific class of condensates. The project aims at analyzing RNA granules in the Drosophila germline, to decipher the links between biophysical properties, organization and functions of membraneless condensates. The project involves innovative approaches to purify RNA granules and single molecule imaging to record ongoing translation. **Methods and approaches:** Drosophila molecular genetics; optogenetics; RNA biology; single molecule imaging: smFISH, SunTag; live imaging. **Key-words**: mRNA regulation; Phase separation; RNA granule; Translational regulation; Single molecule imaging	6 months	Martine.Simonelig@igh.cnrs.fr