Modelling Job's syndrome in zebrafish.
Contact: Prof. Francesco Argenton, e-mail: francesco.argenton@unipd.it

The Autosomal dominant hyper-IgE syndrome (AD-HIES) syndrome is a rare genetic disease that impairs bone metabolism, immune system response and wound healing capability. Patients are currently subjected only to symptomatic therapy and supportive care, because therapies against AD-HIES do not exist. Our project could clarify the molecular mechanisms behind AD-HIES pathogenesis in order to propose new therapeutic strategies (new pharmacological treatment and gene therapy approaches). Particularly, we want to better understand why AD-HIES patients develop bone abnormalities and why they have difficulties to regenerate their tissue after the injuries, evaluating the role of Vitamin D metabolism impairment in the determination of these symptoms.

Evaluation of epigenetic changes in skeletal muscle guiding the development of metabolic alterations and Type 2 Diabetes (T2D).
Contact: Prof. Stefano Cagnin, e-mail: stefano.cagnin@unipd.it

Skeletal muscle plays a key role in glucose homeostasis, being responsible for the uptake of the largest part of postprandial glucose from the blood. sAnk1.5 KO mice, which carry a deletion of 900 bp that eliminates part of a super enhancer region, show a reduced glucose tolerance. This project aims at performing an in-depth metabolic characterization of sAnk1.5 KO mice and identify epigenetic changes in skeletal muscles that result in altered glucose tolerance. Using chromosome conformation capture technologies (5-C, Hi-C) we expect to identify and characterize altered genomic DNA interactions caused by the deletion and correlate chromatin state with gene expression to better elucidate the involvement of skeletal muscle transcriptional profile in the development of T2D.

Deciphering host and tumor cell interactions and communications from the gene expressions of single cells.
Contact: Dott.ssa. Enrica Calura, e-mail: enrica.calura@unipd.it

Multiple reciprocal communications and interactions between cancer and non-malignant cells are involved in a variety of cancer processes. Computationally speaking, the detection of these communications is a network reconstruction problem, in which the expressed gene products represent the nodes and the interactions across proteins represent the edges. Single-cell gene expression data of multiple ecosystems of ovarian cancers - primary tumors and metastases, before and after, responder and non-responder to the therapies - will be analyzed and compared using existing software and proposing improved ones. These results will help in deciphering the tumor supportive communications and interactions and will improve the computational tools available for the biological community.

Metagenomic approaches and machine learning to explore variants as drivers for adaptation to ecological niches.
Contact: Prof. Stefano Campanaro, e-mail: stefano.campanaro@unipd.it

Encoded genes determine the metabolic and functional capabilities of a microbial species, therefore gene content drives microorganisms' adaptation to different ecological niches.
The aim of the project is to predict ecological niches from functional genes by combining genome-centric metagenomics and machine learning techniques. By leveraging data obtained from simulations run on microcosmos experiments, this study has the potential to provide a conceptual framework for the application of genomic data in ecology prediction.

Role of 3D organization and metabolism during differentiation of pluripotent stem cells.
Contacts: Prof. Graziano Martello, e-mail: graziano.martello@unipd.it

Pluripotent stem cells (PSCs) are routinely used for the study of diseases and cell differentiation, thanks to their capacity to give rise to a vast set of cell types and tissues. For instance, PSCs can generate astrocytes and neurons via standard 2D differentiation protocols, or generate brain organoids, composed of different cell types, via 3D differentiation. The aim of this project is to understand the impact of 2D vs 3D differentiation, looking at transcriptional and metabolic differences.

Dissecting the missing heritability of familial thyroid cancer.
Contacts: Prof. Giovanni Vazza, e-mail: giovanni.vazza@unipd.it

The genetics of familial forms of thyroid cancer is still unclear and currently there are no genetic tests for cancer risk assessment. The aim of this project is to assess the possible interplay and cooperation between germline and somatic mutations involved in the predisposition of familial thyroid cancer. Molecular genetics and functional studies will be integrated to gain insight into key genes and mutations modulating oncogenic signalling networks, which have the potential to be used as biomarkers or targets for future therapeutic approaches.