

The Computational Genomics Laboratory:

The overarching goal of my research program at Queen's University is to identify and characterize genomic factors that determine risk for multi-factorial diseases or modulate variable drug response. My lab is currently working on multiple disease models including asthma, chronic obstructive pulmonary disorder (COPD), sleep disordered breathing, as well as variable responses to new biologics for severe asthma. Specifically, we have developed pipelines for the analyses of various types of 'omics datasets to determine the interaction between genomic variants with environmental exposures and how these contribute to differences in gene expression, DNA methylation, and disease risk or drug efficacy. Please visit our website (Duanlab.ca) for more information and contact me if you are interested in one of the following projects: qingling.duan@queensu.ca.

CISC 499 Project 1:

One of the available projects aims to identify potentially causative variants, which could account for the previous correlations with COPD. This is an original project that will likely yield high impact results and a publication for the student. Although earlier genome-wide association studies (GWAS) have identified over 100 genetic associations with COPD, few have reported causative variants. This weakens the concept of genetic testing and precision medicine. The student will learn to apply various bioinformatics tools and software as well as work with large genomic databases such as from 1000 Genome Project and the UK Biobank to identify and characterize novel potentially causative variants correlated with COPD.

CISC 499 Project 2:

COPD is characterized by progressive inflammation and obstruction of the airways. It is the third leading cause of death worldwide, following heart disease and stroke. Major risk factors of this multifactorial, heterogeneous disease include environmental exposures such as tobacco smoke and air pollution but there is also a strong genetic component. Previous genome-wide association studies (GWAS) have identified dozens of genomic variations associated with lung function and COPD, however, the majority of genetic loci previously linked to COPD collectively account for only a small proportion of the phenotypic variation or heritability. The yet to be identified heritable risk of COPD is known as the "missing heritability", which may be explained in part by epigenomic modifications (e.g., DNA methylation, histone modifications). The aim of this project is to identify epigenomic variations correlated with COPD using both univariate and multivariate models.