

Personal Statement

I am Menna Arafat, a physician and bioinformatician working on computational modeling of biological systems, with a focus on multi-omics integration and machine learning. Over the past five years, I have contributed to projects across genomics, transcriptomics, epigenomics, proteomics, and metabolomics, using both bulk and single-cell datasets. My main interest is in developing frameworks that characterize the molecular architecture of disease and identify therapeutic vulnerabilities.

Much of my work has focused on multi-omics integration, pathway communication, and regulatory network inference. I have developed frameworks to detect molecular signatures associated with disease states, model regulatory programs, and prioritize potential therapeutic targets.

Two recent studies published in *Springer Nature* and *Cells* reflect this direction. In one, I carried out a complete metabolomic analysis of colorectal cancer and used weighted network modeling to identify metabolite modules associated with tumor phenotype. In the other, I modeled metabolomic and proteomic profiles to define subtype-specific network modules in rhabdomyosarcoma. In these projects, I highlighted coordinated molecular patterns associated with disease phenotype that conventional differential analyses overlook.

Building on this, I developed CrosstalkX, an R package that infers pathway interactions using protein–protein interactions and mutual information. The tool infers nonlinear dependencies among pathways and transcription factors and provides a reproducible approach for detecting high-order interaction patterns relevant to complex biological processes.

Looking ahead, I aim to study probabilistic machine learning and dynamical systems modeling through deep learning–based representation models. I am particularly interested in deep learning and diffusion-based models that map complex biological states into low-dimensional latent spaces. These representations provide a principled way to examine how biological states evolve over time, how perturbations propagate through regulatory networks, and how disease trajectories diverge from normal physiology, and more importantly, help identify interventional points where targeted perturbations could shift a diseased system toward a more stable or healthy state. My goal is to develop structured latent-variable models, graph diffusion methods, and generative frameworks that describe disease as an evolving process and inform strategies for therapeutic intervention in complex conditions such as cancer.