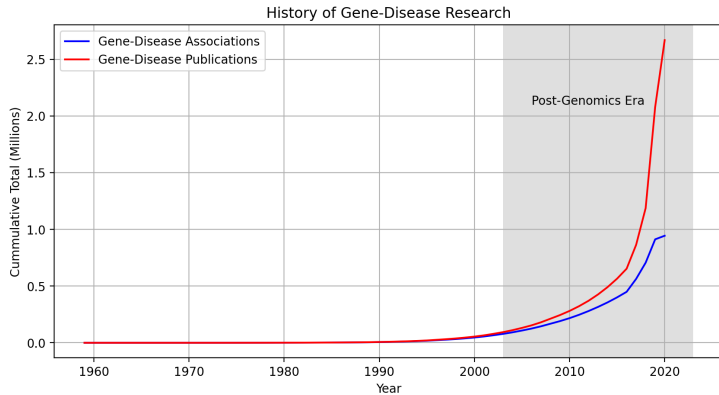


# BioXplore: Interactive Exploration of Biological Knowledge Networks

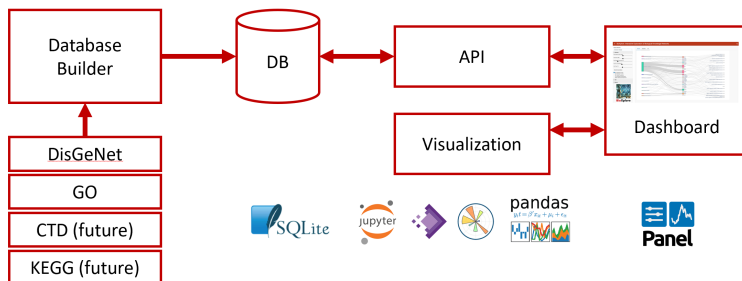
Seamus Coyne, Sriya Vuppala; John Rachlin

## BACKGROUND



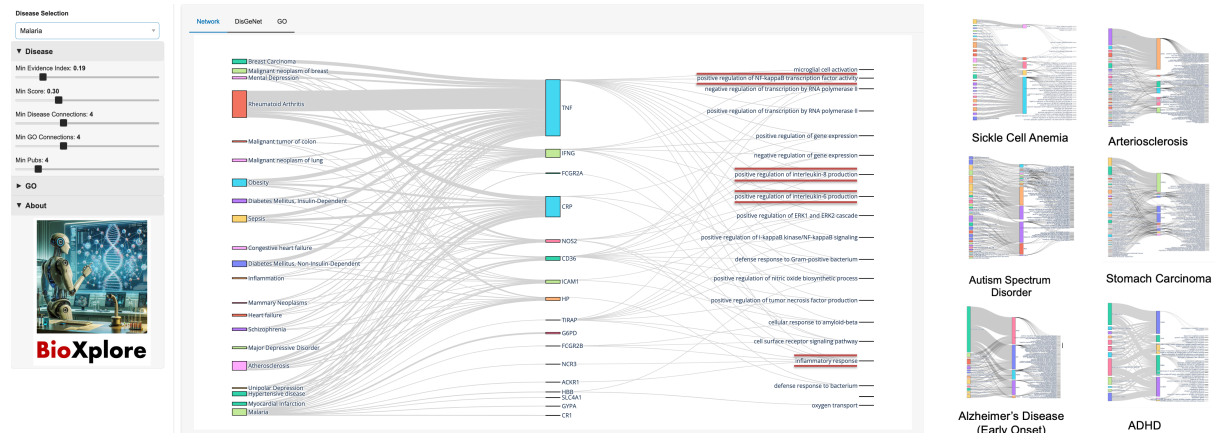
**Figure 1. Motivation.** Sequencing of the human genome gave rise to increased identification of gene-disease associations and a new wave of biomedical research aimed at understanding the genetic underpinnings of disease. Networks are a widely used framework for modeling relationships between genes, diseases, and functional annotations, though their inherent complexity can make interpreting visualizations difficult (1). BioXplore is an interactive dashboard for mining biomedical knowledge. It integrates data from DisGeNET (2, 3), GO (4), and NCBI (5).

## METHODS



**Figure 2. BioXplore Architecture.** The BioXplore framework includes data integration tools an API, visualization libraries, and an interactive user interface for exploring biological networks.

## PRODUCT



**Figure 3. BioXplore Dashboard.** The BioXplore interactive dashboard (above left) allows one to navigate disease/gene association networks based on selected evidence thresholds. Sampled networks (above right) demonstrate varying complexity in the interconnection between diseases and genes.

## FINDINGS

### TNF

**Figure 3. Explaining the Co-occurrence of Malaria and Type 2 Diabetes.** Studies have reported the co-occurrence of malaria and type II diabetes in developing countries. Moreover, evidence suggests that the two diseases are linked via the inflammation process (6). BioXplore has identified multiple genes that malaria and type 2 diabetes have in common and corroborates the inflammation-mediated link between the diseases. TNF plays an especially prominent role in malarial infection. Infected erythrocytes activate macrophages and natural killer cells, subsequently producing TNF- $\alpha$ , which upregulates the transcription of TCF4 (a type II diabetes marker). TCF4, in turn, interferes with insulin signaling and adipocyte lipid metabolism (7). As a proinflammatory cytokine, TNF- $\alpha$  is able to activate the NF- $\kappa$ B pathway. As an inducible transcription factor, NF- $\kappa$ B initiates the inflammation by positive regulation of interleukin 8 and interleukin 6 production (8).

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