The Institute for Behavioral Genetics (IBG)

### The Institute

**Mission**

IBG is a research institute located on the campus of the University of Colorado Boulder. It is affiliated with the University of Colorado Denver and operates as a joint program between the two institutions. IBG has a strong focus on understanding the genetic and environmental influences on human behavior and health.

**Research**

IBG is a resource for top faculty in the world for their work on behavioral genetics and related fields. IBG supports research through grants and fellowships, and it is home to a DNA repository of 40,000 samples for research on behavior.

**Training**

IBG provides strong training in a primary academic discipline, accomplished by requiring students to obtain a genetic research on behavior. Data collection and analysis are conducted in close partnership with the Colorado Learning Disabilities Research Center, and the Colorado Learning Disabilities Research Center and the IBG share many resources for research and training programs.

**Facility**

IBG houses cutting-edge equipment for research on behavior, including a DNA repository of 40,000 samples for research on behavior.

**Staff**

Approximately 22 postdoctoral fellows, research associates, and senior research assistants are employed at IBG.

**Faculty, Researchers & Students**

Faculty, researchers, and students from IBG have made significant contributions to the study of genetics-related research on behavior. This has resulted in a strong interdisciplinary research environment, which has led to the identification of novel genes, the discovery of new biological processes, and the development of new treatments for genetic disorders.

### Fiscal Impact

- **Revenues:**
  - ~$24,000,000 in external research grants
  - ~$30,000,000 in external research contracts

- **Expenses:**
  - ~$9,600,000 in total costs
  - ~$15,000,000 for researchers, staff, and students

- **Net Income:**
  - ~$5,400,000

### Scientific Impact

- **50+ peer-reviewed scientific papers annually**
- **13 papers published in Science by IBG faculty**
- **7 Faculty Fellows cited more than 1000 times each in 2016**

### Societal Impact

- **Drug Abuse**
  - Center on Antisocial Drug Dependence
  - Biology of Narcotic Abuse
  - Biology of Alcoholism

- **Aging & Neurodegenerative Diseases**
  - Molecular Genetics of Aging
  - Alzheimer’s Disease
  - Down Syndrome
  - Alzheimer’s Disease
  - Behavioral & Neurodegenerative Disorders

- **Learning Disabilities**
  - Executive Functions Brain Imaging
  - Mental Health

### Research Highlights

- **Genetics of Personality:**
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Health:**
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Development:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Disease:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Behavior:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Substance Abuse:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Cognitive Function:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Neurodegeneration:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Neuroendocrine Function:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Neuroplasticity:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Neurotrophic Factors:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Pain:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Psychopathology:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Schizophrenia:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Substance Abuse:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Stress:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Tobacco:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)

- **Genetics of Visuospatial Function:**
  - Showed link between RCAN1 overexpression and age-related neurodegeneration in C. elegans. (Johnson et al., 2017)
  - Discovered a mutation in the gene CACNA1C in individuals with autism. (Bowland et al., 2016)
  - Identified a causal role for the cytokine IL-17 in the manifestation of autism-related phenotypes in offspring exposed prenatally to maternal inflammation. (Wong et al., 2016)