My laboratory, in the Center for Cell Analysis and Modeling at the University of Connecticut Health Center, works on the molecular basis for fragile X disorders. These include: fragile X syndrome, the major single gene cause of mental retardation and autism in young boys, fragile X tremor ataxia syndrome, a major cause of tremor, ataxia and cognitive decline in older males, and fragile X premature ovarian insufficiency, a major cause of early menopause in females. All of these disorders are caused by expansions of CGG repeat sequences in the FMR1 gene, which are found in 1/130 females and 1/300 males in the general population. Approximately 20,000 Connecticut residents carry such expansions and are therefore at risk for fragile X disorders.

Using advanced biochemical, biophysical and cell biological experiments my laboratory has discovered a common biochemical mechanism for fragile X disorders. This mechanism could potentially be targeted to develop therapeutic strategies for ameliorating fragile X disorders. However we have not been able to develop such strategies because the very high fringe benefit rate at UCHC precludes hiring sufficient research staff with advanced biochemical training.

Reducing the fringe benefit rate at UCHC would allow us to develop therapeutic strategies for fragile X disorders that could potentially benefit 20,000 Connecticut residents.

John H. Carson, Ph.D.
Professor
Department of Molecular Biology
Center for Cell Analysis and Modeling
University of Connecticut Health
263 Farmington Avenue
Farmington, CT 06030
johnherbertcarson@gmail.com