The BIG news is our Annual Meeting…

Genes, Brain and Behavior, May 22-May 25, University of Galway, Galway, Ireland

Meeting Updates

Preliminary Meeting Program

https://www.conftool.pro/ibangs2023/sessions.php

Annual Meeting Registration

Registration pricing through May 8th (Includes Lunch, Snacks, Opening Reception and Banquet)

Regular Member $500.00  
Student/Postdoc Member $350.00  
Regular Non Member $650.00  
Student/Postdoc Non Member $450.00

Accommodations and Travel Information
https://www.ibangs.org/faqs-galway

Late Breaking Abstract Submission  
Deadline April 14th


Letter from the President and President-Elect

Judy Grisel and Karla Kaun

As an organisation IBANGS is committed to supporting and disseminating research in the public interest, and therefore aligned with the highest research and ethical standards. IBANGS’ commitment to these standards embraces the belief that advancing knowledge benefits from an inclusive, welcoming and cooperative community of investigators with diverse backgrounds, experiences, methodologies and interests.

We hope you agree, and invite you to consider ways to help broaden membership and participation in our society.

If you are a principal investigator, please encourage your mentees and other colleagues to join the society, and to attend the annual meeting in Galway. The presenters are diverse in career stage, gender, and international representation. As usual, there will be a wide range of model organisms represented and a breadth of timely topics.

If you are a trainee or new to the society, please reach out to contribute your ideas and let us know how we can help. IBANGS is a wonderful venue for those studying the molecular substrates of behaviour in humans or model organisms to exchange ideas, make new friends, and develop meaningful collaborations. Toward those ends, the
meeting in Galway will begin with a workshop on functional interpretation of GWAS data, followed by a networking event. Child care stipends are available.

Travel Awards 2023

Congratulations to ALL of this year’s Travel Awardees. 27 Graduate Students, 7 Postdoctoral associates and 3 Junior Faculty. Whoa!

Special mention to the Awardees that were selected to give an oral presentation in Galway

From left to right
**Tariq Brown**, Graduate student, Brown University, “Alcohol-Induced Alternative Splicing of Dopamine-2 Receptors in Drosophila Memory Circuits”

**Hayley Thorpe**, Graduate student, University of Guelph. “Cell Adhesion Molecule 2 and cannabinoids: A point of physiological and behavioural drug use vulnerability”

**Merideth Loth**, Postdoctoral Associate, University of Colorado, Boulder. “Elucidating the cellular basis of dopamine and oxytocin interactions underlying pair bonding”

**Rajani Maiya**, Louisiana State University, Junior Faculty, “Transcriptional signatures of social-stress escalated alcohol consumption”

THANK YOU to our sponsors for helping to support trainee attendance at the meeting: Curam, Future Neuro, Irish Society of Human Genetics Stoelting, Vector Builder, Wiley and Zantiks

THANK YOU to the members that donate money to student travel awards when they renew their society membership: Zoe Bichler, Camron Bryant, Mary-Anne Enoch, Catherine Fernandes, Judy Grisel, John Hernandez, Megan Mulligan, Richard Radcliffe, Mark Rutledge-Gorman, Hee-Sup Shin, and Cheryl Williams

Committee News
Annie Park, Postdoctoral Researcher, University of Oxford joins the DEI Committee
http://www.cnscb.ox.ac.uk/people/annie-park/

Upcoming Virtual Seminar

Michael Miles, MD, PhD
April 12th, 2023
12:00 pm EDT
Zoom TBA
https://pharmtox.vcu.edu/about/our-team/michael-f-miles-md-phd.html

Member News

Camron Bryant, has been promoted to full Professor at Boston University. https://sites.bu.edu/bryantlab/

Cathy Kaczorowski has moved her lab to the University of Michigan. https://kaczorowski.lab.medicine.umich.edu/

Member Publications

Contributed by Fred Wolf

"Distinct Forms of Ethanol Tolerance in Flies"
Caleb Larnerd, Pratik Adhikari, Ashley Valdez, Alexander Del Toro, and Fred W. Wolf
While it is not surprising that ethanol use leads to tolerance—a common physiological response to many drugs—what is surprising is that distinct forms of ethanol tolerance—acute, rapid, and chronic—are conserved across species, even in the fly Drosophila melanogaster. But the molecular and circuit underpinnings of these early forms of behavioural plasticity remain largely unknown. This week, Larnerd et al. dissect three forms of ethanol tolerance in flies that depended on the pattern of initial ethanol exposure. Repeating a sedating exposure 4 h later, after the initial dose was metabolized, gave rise to rapid tolerance, which mimics the tolerance seen in humans engaged in binge drinking. Flies chronically exposed to low levels of ethanol developed chronic tolerance, in which flies resisted ethanol sedation, similar to maintenance drinking in people with alcohol use disorder (AUD). In a repeated ethanol exposure, flies received an inebriating, but not sedating, dose of ethanol for 20 min/d for 4 d. That produced repeated tolerance, characterized by reduced sensitivity and a slight aversion to ethanol. The researchers examined the molecular roots of this neural plasticity, focusing on immediate early gene transcription factors and the structure of chromatin. Interestingly, the three forms of tolerance were distinct, each inducing different factors. The researchers traced plasticity encoding to histone deacetylation by multiple histone deacetylases including the sirtuin Sirt1, which promoted rapid tolerance but inhibited chronic tolerance. They further distinguished the forms of tolerance anatomically by mapping them to distinct circuits of the mushroom body, the learning and memory center in the fly brain. Critically, the researchers found that rapid and chronic tolerance are molecularly and temporally similar to intermediate-term and long-term memory, respectively. However, ethanol appears to create a unique form of long-term memory. This description of multiple, distinct forms of ethanol tolerance point to complex neural plasticity that forms long-term memory-like states, which may form a basis for the development of AUD.

Contributed by Clyde Francks

“Genetic architecture of the white matter connectome of the human brain”
Zhiqiang Sha, Dick Schijven, Simon E. Fisher, and Clyde Francks
https://www.science.org/doi/10.1126/sciadv.add2870

White matter tracts form the structural basis of large-scale brain networks. We applied brain-wide tractography to diffusion images from 30,810 adults (U.K. Biobank) and
found significant heritability for 90 node-level and 851 edge-level network connectivity measures. Multivariate genome-wide association analyses identified 325 genetic loci, of which 80% had not been previously associated with brain metrics. Enrichment analyses implicated neurodevelopmental processes including neurogenesis, neural differentiation, neural migration, neural projection guidance, and axon development, as well as prenatal brain expression especially in stem cells, astrocytes, microglia, and neurons. The multivariate association profiles implicated 31 loci in connectivity between core regions of the left-hemisphere language network. Polygenic scores for psychiatric, neurological, and behavioral traits also showed significant multivariate associations with structural connectivity, each implicating distinct sets of brain regions with trait-relevant functional profiles. This large-scale mapping study revealed common genetic contributions to variation in the structural connectome of the human brain.

Summary article (free registration):
https://www.genomeweb.com/genetic-research/large-scale-study-reveals-genetics-white-matter-connections-human-brain#.ZBrJofbMJOQ

Contributed by Manal Tabbaa

“Mouse population genetics phenocopies heterogeneity of human Chd8 haploinsufficiency”
Manal Tabbaa, Allison Knoll, and Pat Levitt
https://doi.org/10.1016/j.neuron.2023.01.009

Preclinical models of neurodevelopmental disorders typically use single inbred mouse strains, which fail to capture the genetic diversity and symptom heterogeneity that is common clinically. We tested whether modeling genetic background diversity in mouse genetic reference panels would recapitulate population and individual differences in responses to a syndromic mutation in the high-confidence autism risk gene, CHD8. We measured clinically relevant phenotypes in >1,000 mice from 33 strains, including brain and body weights and cognition, activity, anxiety, and social behaviors, using 5 behavioral assays: cued fear conditioning, open field tests in dark and bright light, direct social interaction, and social dominance. Trait disruptions mimicked those seen clinically, with robust strain and sex differences. Some strains exhibited large effect-size trait disruptions, sometimes in opposite directions, and—remarkably—others expressed resilience. Therefore, systematically introducing genetic diversity into models of neurodevelopmental disorders provides a better framework for discovering individual differences in symptom etiologies.
Featured in Spectrum News:

Please note that all members can share their recent publications with colleagues via the Newsletter and also via Twitter. Just tag us! @IBANGStweets

Watch for nominee and voting information for the IBANGS Executive Committee Elections. An email will be sent to the membership in the coming days.

Happy Spring To All