BIOGRAPHICAL SKETCH

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NAME: Turkes, Emir

eRA COMMONS USER NAME (credential, e.g., agency login):

POSITION TITLE: Graduate Student

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE (if applicable)	END DATE MM/YYYY	FIELD OF STUDY
Boston University, Boston, MA, USA	BA	05/2015	Neuroscience
The University of Tokyo, Tokyo, Japan	MS	03/2018	Health Science
University College London, London, UK	PHD	09/2023 (expected)	Neuroscience

A. Personal Statement

I am a Ph.D. student at University College London under the supervision of Prof. Karen E. Duff. From February-September 2019 I was also a research technician under Prof. Duff at the Taub Institute at Columbia University Medical Center. My interest and experience is primarily in computational analysis. With Prof. Duff, this work has primarily focused on the unraveling of large publicly available single-cell transcriptomics datasets, such as those available from the Allen Institute. We have been using these datasets to investigate cell-type specific selective vulnerability to tauopathy. This work is both hypothesis testing and hypothesis generating; for instance, we test for correlations between cellular properties and relative vulnerability and then identify candidate genes/pathways that may drive the correlations. To facilitate these analyses, we have built custom workflows and software that may also be of general interest to the research community. My past research efforts were also computational in nature. As a Master's student at The University of Tokyo under Dr. Toshihiro Endo, I helped develop software for Intellicage, an automated mouse phenotyping system. The software was used to phenotype a novel oxytocin receptor KO mouse model of autism spectrum disorder. Also, towards the end of my undergraduate education, I volunteered and worked at the Boston University Speech Lab under Prof. Frank H. Guenther, where I analyzed resting-state fMRI data from the Human Connectome Project to help inform the DIVA model of speech production.

B. Positions and Honors

Positions and Employment

2015 - 2016 Research Assistant, Boston University Speech Lab, Boston, MA, USA
2019 - 2019 Technician B, Taub Institute for Research on Alzheimer's Disease and the Aging Brain, Columbia University Medical Center, New York, NY, USA

Other Experience and Professional Memberships

2018 - Member, Rotary International

Honors

2016 - 2018 Scholarship, Rotary Yoneyama Memorial Foundation

C. Contribution to Science

1. Computational approaches to investigating cell-type specific selective vulnerability to tauopathy

This work describes my current thesis research with Prof. Karen E. Duff. In tauopathies such as Alzheimer's Disease, it is known that certain cell types are preferentially affected by pathology over others. While a variety of factors are implicated in driving this, which range from morphological (e.g. cell size),

molecular (e.g. synaptic activity), and ontological (e.g. developmental origin) factors, more work is needed to understand their exact contributions. To test whether these factors correlate with cellular vulnerability, we turned to publicly available data, starting with single-cell transcriptomics. In contrast to many other studies exploring selective vulnerability using omics approaches, we have placed increased emphasis on the analysis of non-disease data. Using known markers of selectively vulnerable neurons, we were able to identify the respective cell-types in our non-disease datasets. We hypothesize that these cell-types exhibit transcriptomes that predispose them to vulnerability in tauopathy. Thus, our work has been to breakdown these transcriptomes into components that can be individually manipulated in model systems. For instance, using a custom gene ontology enrichment pipeline, we have found that vulnerable neurons are enriched in synaptic vesicle exocytosis activity and the formation of actin crosslinking. The two may act together in a plausible and previously reported mechanism wherein which synaptic vesicles become blocked by crosslinked actin and tau-associated microtubules, ultimately resulting in tauopathy. We are in the process of designing model systems for the validation of this and other mechanisms. In addition, the computational methods are being prepared for publication as general purpose software.

Poster Presentations:

a. **Turkes E.**, Duff K. (2020 February). *Cell Type Specific Selective Vulnerability to Pathological Tau in Alzheimer's Disease*. Poster presented at Tau 2020, Washington D.C., USA.

b. **Turkes E.**, Duff K. (2020 February). *Cell Type Specific Selective Vulnerability to Pathological Tau in Alzheimer's Disease*. Poster presented at UK-Japan Neuroscience Symposium, Edinburgh, UK.

c. **Turkes E.**, Duff K. (2019 October). *Transcriptomic signatures of cell-types that show differential vulnerability to tauopathy*. Poster presented at Connectome 2019, Birmingham, UK.

2. Development of automated mouse phenotyping tools and its application in a novel mouse model of autism spectrum disorder

For my Master's research, I helped develop computational tools for analysis using Intellicage, a mouse home cage system that enables automated phenotyping with minimal human intervention. This work was supervised by Dr. Toshihiro Endo and was also in collaboration with Phenovance LLC (Chiba, Japan) and NewBehavior (Zürich, Switzerland). Expanding upon the PyMICE library, I wrote a number of routines that aimed to infer exploratory and social behavior among mouse inhabitants. These routines were applied to a novel oxytocin receptor KO model of autism spectrum disorder, wherein which we focused on the early habituation period in Intellicage to study the interaction between genotype and environment. Our findings were that the KO mice exhibited phenotypes suggestive of increased anxiety and decreased preference formation, however the change was not significant across all comparisons. Further studies and additional computational analysis would be needed to confirm these findings. In addition to this work, we published an exhaustive review of all research to-date that have utilized the Intellicage system.

Publications:

a. Kiryk, A., Janusz, A., Zgnilicki, B., **Turkes, E.**, Knapska, E., Konopka, W., Lipp H., Kaczmarek, L. (2020). *Intellicage as a tool for measuring mouse behavior – 20 years perspective*. Behavioural Brain Research, 112620. https://doi.org/10.1016/j.bbr.2020.112620

Thesis:

b. **Turkes, E.** (2018). *A Never-ending Journey to Explore the Gene and Environment Interaction Upon Autism Spectrum Disorders*. Master's thesis. The University of Tokyo.

3. Updating the DIVA computation model of speech production using Human Connectome Project rs-fMRI data

Towards the end of my undergraduate degree, and for a short time after, I did fMRI research for the Boston University Speech Lab under Prof. Frank H. Guenther. The aim of this work was to better inform the DIVA

model of speech production, which while comprising a well-characterized network of brain regions, is poorly understood in terms of functional connections within the network. To further study the functional connectivity of the DIVA network, we analyzed resting-state functional MRI (rs-fMRI) from ~500 neurotypical adults collected as part of the Human Connectome Project. The results of this analysis suggested potential changes to the DIVA auditory target projections, most notably a decreased contribution of the ventral premotor cortex than previously assumed. These findings were presented in a poster session and other contributions are acknowledged in a textbook describing neural control of speech.

Poster Presentations:

a. **Turkes, E.**, Golfinopoulos, E., Guenther, F. H., & Tourville J. (2015, June). *Investigating Intrinsic Functional Connectivity Within the Speech Production Network*. Poster presented at NeuroHAM, Boston, MA.

Textbook Acknowledgements:

b. Guenther, F. H. (2016). *Neural Control of Speech*. Cambridge, MA: The MIT Press.

D. Additional Information: Research Support and/or Scholastic Performance