**INTRODUCTION**

- Tics can be idiopathic, presenting as focal symptoms of Tourette Syndrome (TS), or secondary to neural pathologies e.g., focal brain lesions.
- Neuroimaging studies implicate widespread cortical and subcortical abnormalities in TS. However, due to neuroanatomical heterogeneity, it remains unclear which brain regions are key to TS symptomatology.
- Likewise, tic-inducing lesions can occur throughout the brain, suggesting that tic symptoms may better localize to a neural network rather than a single brain region.
- 'Coordinate network mapping' (CNM) and 'lesion network mapping' (LNM) can be used to localize neuroimaging findings and symptom inducing brain lesions to a common brain network, respectively.

**AIMS**

1. To localize heterogeneous structural neuroimaging findings in TS to a network of brain regions
2. Identify a common network for tics based on idiopathic and secondary tic symptoms

**NETWORK MAPPING TECHNIQUES**

- **Coordinate Network Mapping**
  - Spherical seeds (4mm) were generated at each coordinate from the 7 studies, and pooled together to create a combined seed for each study.
  - Regions functionally connected to each study’s combined seed were identified using a normative connectome (n = 1000)^1^, generating a connectivity map for each study.
  - Positive and negative connectivity maps from each lesion were overlapped to represent the network map for TS, thresholded to only include voxels common to ≥ 6/7 (86%) studies.

- **Lesion Network Mapping**
  - Lesions were hand-traced onto a standard 2 x 2 x 2 MNI152 brain atlas using FSLeyes (version 6.0.4).
  - Regions functionally connected to each lesion location were identified using a normative connectome (n = 1000)^2^, generating a connectivity map for each lesion.
  - Positive and negative connectivity maps from each lesion were overlapped to represent a network map for tic-inducing lesions, thresholded to only include voxels common to ≥ 17/20 (85%) lesions.

**RESULTS**

**CNM**
- Heterogeneous neuroimaging findings in patients with TS localized to a common network, involving structures within the cortico-basal-ganglia-thalamo-cortical circuit (see figure A).

**LNM**
- Tic-inducing lesions were reported in heterogeneous locations, however, mapped to a common network (see figure B). Our findings are largely consistent with those previously published by Ganos and colleagues.

**CONCLUSIONS & FUTURE DIRECTIONS**

- Seemingly inconsistent structural neuroimaging findings in patients with TS map to a common network involving regions previously associated with tic onset and premonitory urge.
- This network identified in TS and a network for tic-inducing lesions converge in the thalamus, caudate, putamen, GPe and occipital lobe, revealing a sub-network which may mediate tics as shared symptoms between idiopathic and acquired tics.
- Brain regions involved in this sub-network are consistent with common neuromodulation targets for tics.

**NEXT STEPS**
- Further examination of the relationship between intrinsic functional connectivity patterns in this network and tic symptoms severity.

**REFERENCES**

1. Ricketts et al. (2019). Current developmental disorders reports, 6
2. Darby et al. (2019). Brain, 142
5. Ganos et al. (2022). Brain
7. Baldemann et al. (2016). Brain stimulation, 9