A low dimensional structure of behavioral and anatomical correlates in aphasia syndromes

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INTRODUCTION

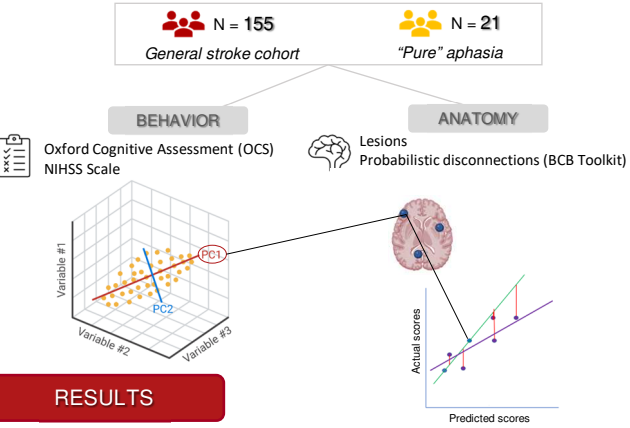
Post-stroke cognitive deficits have traditionally been explained by a **modular view**, where focal lesions disrupt distinct cortical regions, producing selective impairments.

However, population-level neuroimaging and modeling studies show that deficits often cluster together, reflecting disruption of distributed large-scale networks rather than isolated modules.

In language, **low-dimensional approaches** reveal graded combinations of phonological and semantic impairments, challenging classical categorical aphasia subtypes.

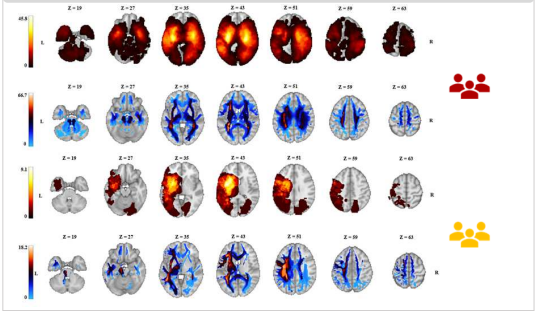
AIM: To assess whether post-stroke language deficits reflect discrete modular syndromes or a low-dimensional, network-based structure by comparing pure aphasia cases with a broader stroke cohort.

METHODS

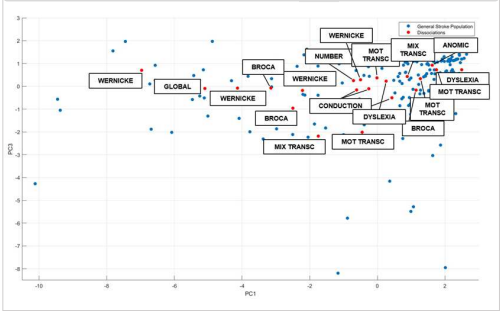


RESULTS

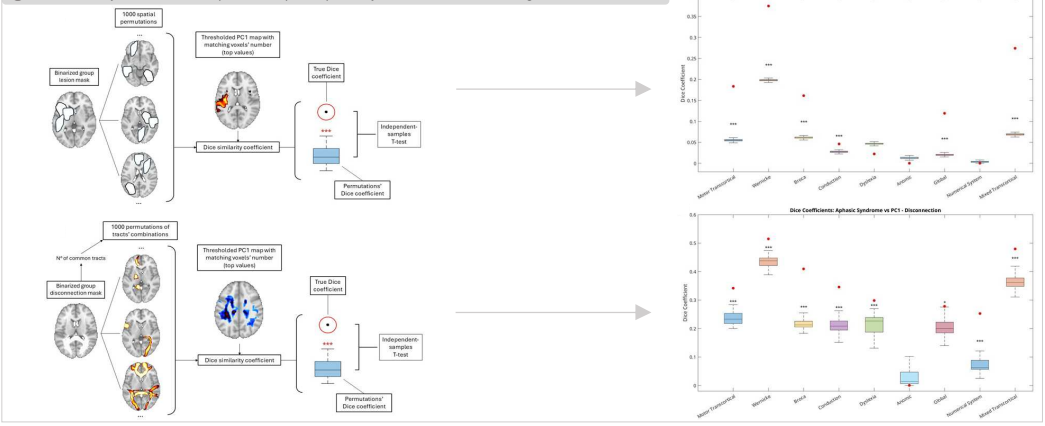
1 «Pure» syndromes showed mainly left hemisphere lesions and bilateral disconnections



2 «Pure» aphasia syndromes' behavioral values were not outliers



3 Lesion anatomy and disconnection patterns of «pure» aphasia syndromes were similar to the general stroke cohort



CONCLUSIONS

Classical aphasia syndromes seem to reflect graded variations along **a continuum of language-cognitive deficits**, supported by distributed cortical and white matter network disruptions.

Clinical implications: More accurate representation of the heterogeneity observed in post-stroke language impairments than traditional categorical classifications. It is the most likely sensitive method for monitoring patients in rehabilitation settings and tracking functional recovery.