

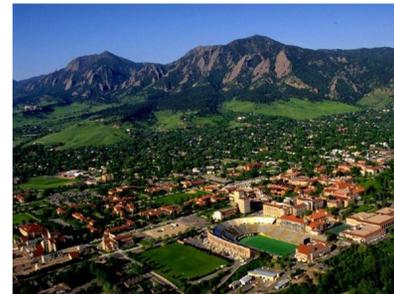
Patients with Schizophrenia Show Aberrant Patterns of Basal Ganglia Activation: Evidence from ALE Meta-Analysis

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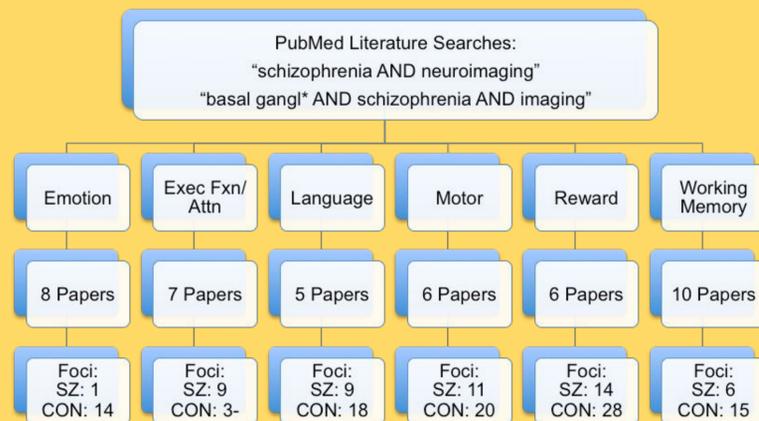
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Introduction

- Evidence demonstrates basal ganglia abnormalities exist across the psychosis spectrum¹⁻⁶
- The basal ganglia and its respective cortical projections are implicated in behaviors often impaired in psychotic individuals⁷
- Here, our goal was to understand the functional topography and activation differences of the basal ganglia in healthy adults and individuals with schizophrenia (SCZ)
- We predicted decreased activation across domains in SCZ individuals when compared to controls

Methods



- Two searches for functional studies involving task domains of emotion, executive function/attention, language, motor, reward, and working memory
- All analyses were conducted using BrainMap GingerALE 2.3.5 (<http://brainmap.org>)
- All maps: uncorrected $p < .001$ as the cluster-forming threshold, FDR $p < .05$ for cluster-level inference, 5,000 threshold permutations
- Group contrasts/conjunctions: uncorrected $p < .05$, 10,000 p-value permutations, minimum cluster size of 50mm^3 , minimum cluster size of 100mm^3 for pooled comparison

Results

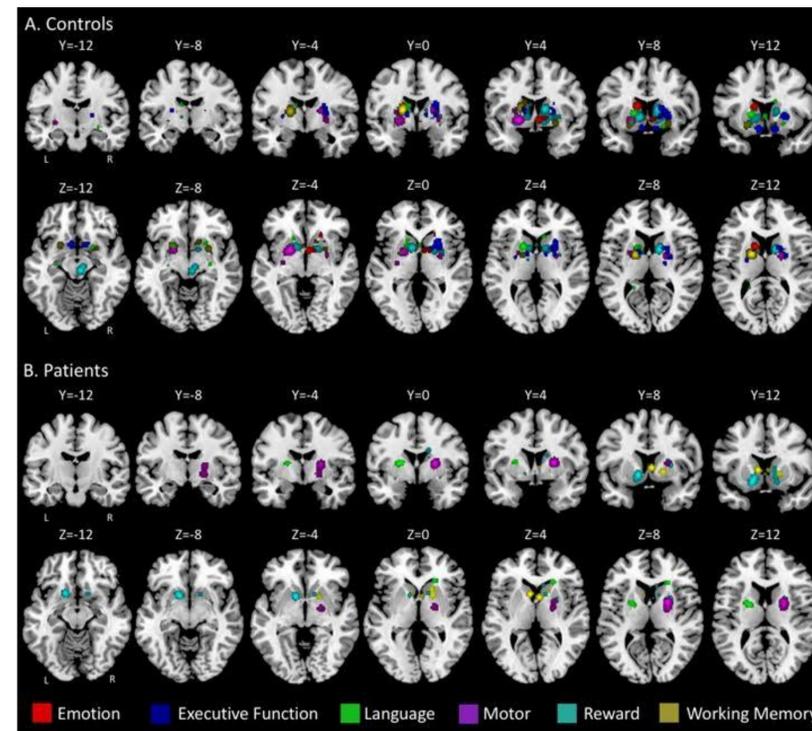


Figure 1. Maps of task domains for controls and SCZ

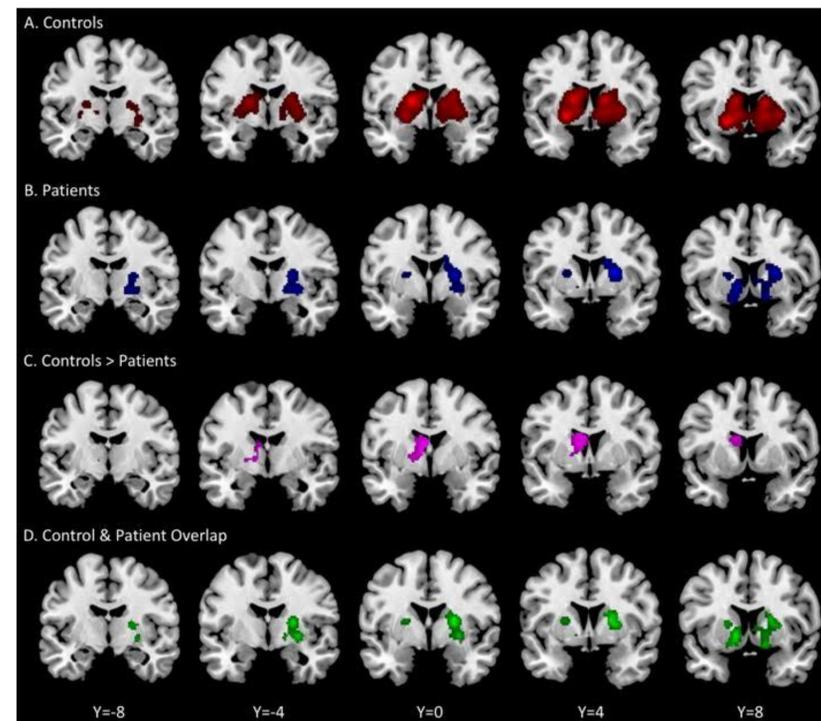


Figure 2. Maps when collapsing foci across all domains for controls and SCZ

Summary

- Across task domains, SCZ patients exhibited significantly fewer activated basal ganglia foci relative to healthy controls
- Decreased activation was most notable in the domains of emotion and executive function, where SCZ patients did not exhibit any significant responses
- Activations were weaker compared to controls in studies where patients did show basal ganglia activity
- We suggest the basal ganglia deficits shown here may contribute to negative symptoms, positive symptoms, cognitive dysfunction, and motor dysfunction seen in patients with SCZ
- The aberrant basal ganglia activation observed in this study provides insight into the role of fronto-subcortical circuits in schizophrenia, and may be explained in relation to the implications of the dopamine hypothesis of SCZ

References

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This work was supported by NIH R01MH094650 (V.A.M) and NIH F32MH102898 (J.A.B.)