Introduction

Impaired brain glucose consumption is a possible trigger of Alzheimer’s disease (AD). Animal models help characterize each contributor to the cascade independently. Here we report a comprehensive longitudinal study of functional connectivity, white matter microstructure and cerebral glucose metabolism in a rat model of sporadic Alzheimer’s disease.

Methods

Animals (N=17) underwent a bilateral icv-injection of either streptozotocin (3 mg/kg, STZ group, N=9) or buffer (CTL group, N=8). MRI: Rats were scanned at 4 timepoints following surgery (Figure 1), on a 9.4 T Bruker scanner. PET: Rats were injected with 30 mCi FDG and imaged at 12/16/30/44 days after injection. FDG-PET and SPGR (3D T1-weighted) images were acquired. Region of interest (ROI) analysis was performed to identify temporal and spatial alterations in functional connectivity, microstructure and cerebral glucose metabolism.

Results

FDG-PET highlighted reduced SUV in STZ rats, in a more stable pattern over time (Figure 4). Regions with reduced SUV broadly agreed with regions of WM degeneration (Figure 3). Diffusion and kurtosis tensors were calculated and axonal loss at 13 weeks. The sample size is likely too small at 21 weeks (Figure 1).

Discussion and Conclusions

Our study highlights the importance of whole-brain analysis of brain imaging and metabolic data, as well as the potential of FDG-PET to evaluate early changes in vivo. It also supports the use of hybrid matrices to study functional connectivity in pathological models.

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References

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10. Diffusion and kurtosis tensors were calculated 19,20, and axonal loss at 13 weeks. The sample size is likely too small at 21 weeks (Figure 1).
11. SUV and 26 ROIs were segmented. SUV 18, and 26 ROIs were segmented. SUV