Supplementary Table 1. Inclusion and exclusion criteria for AD patients.

Inclusion criteria	1. An age range of 40–90 years. 2. Fulfill the core clinical criteria of the National Institute on Aging and the Alzheimer's Association workgroup for probable AD[1]. A. Insidious onset. B. Clear-cut history of worsening of cognition by report or observation. C. Cognitive deficits involve a minimum of two of the following cognitive domains: a. Amnestic presentation b. Non-amnestic presentations: language, visuospatial, executive dysfunction, and so on. D. Decreased [^{18}F]-FDG uptake on PET in the temporo–parietal cortex. E. Disproportionate atrophy on structural MRI in the medial, basal, and lateral temporal lobe, and the medial parietal cortex. 3. Patients who were diagnosed for the first time without the use of any AD-related medications, including anti-cholinesterase or NMDA receptor antagonists. 4. Right-handed participants.
Exclusion criteria	1. A current or known history of anti-cholinesterase medication or NMDA receptor antagonist medication. 2. A current or known history of major depression and/or other neuropsychiatric conditions such as psychosis. 3. Prominent features of the other neurodegenerative diseases such as dementia with Lewy bodies, frontotemporal dementia, Parkinson's disease, progressive supranuclear palsy, and so on. 4. Concomitant cerebrovascular disease, defined by a history of a stroke temporally related to the onset or worsening of cognitive impairment; or the presence of multiple or extensive infarcts or severe white matter hyperintensity burden. 5. Any neoplastic, chronic inflammatory, or noteworthy internal diseases that may result in cognitive deficits. 6. Presence of contraindications to MRI.

References

1. McKhann GM, Knopman DS, Chertkow H, et al. The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimers Dement 2011;7:263-269.

Supplementary Table 2. SUVRs of the cerebral region and hypometabolism rates in AD patients.

Region	Side	AD patients	Controls	t value	*p*-value	AD Hypometabolism, %
Executive subregion	L	1.09± 0.13	1.21 ± 0.10	4.32	<0.0001	9.92%
Executive subregion	R	1.13± 0.14	1.22± 0.11	3.78	0.0003	7.38%
DLPFC	L	1.13± 0.23	1.45± 0.12	7.09	<0.0001	22.06%
DLPFC	R	1.18± 0.24	1.43± 0.12	5.53	<0.0001	17.48%
vmPFC	L	1.18± 0.15	1.19± 0.10	0.62	0.53	-
vmPFC	R	1.14± 0.17	1.27± 0.11	4.04	0.0001	10.23%
ACC	L	1.19± 0.19	1.37± 0.15	4.85	<0.0001	13.14%
ACC	R	1.26± 0.17	1.32± 0.12	1.82	0.07	-
PCC	L	0.81± 0.12	1.05± 0.10	9.08	<0.0001	22.86%
PCC	R	0.97± 0.19	1.36± 0.14	10.05	<0.0001	28.68%
Precuneus	L	1.11± 0.19	1.51± 0.15	10.31	<0.0001	26.49%
Precuneus	R	1.01± 0.17	1.36± 0.13	10.25	<0.0001	25.74%
Temporal pole	L	0.68± 0.08	0.74± 0.06	3.74	0.0003	8.11%
Temporal pole	R	0.77± 0.09	0.84± 0.07	3.70	0.0004	8.33%
Temporal lobe	L	1.20± 0.19	1.42± 0.11	5.85	<0.0001	15.49%
Temporal lobe	R	0.95± 0.12	1.09± 0.11	5.50	<0.0001	12.84%
Anterior insula	L	1.24± 0.17	1.39± 0.16	4.21	<0.0001	10.79%
Anterior insula	R	1.27± 0.18	1.39± 0.15	3.32	0.001	8.63%

Abbreviations: ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; PCC, posterior cingulate cortex; vmPFC, ventromedial prefrontal cortex

Supplementary Table 3. Spatial coordinates and peak values of the striatal subregion showing significant differences in metabolism between AD patients and controls

Region	Side	Cluster Size	MNI coordinate	T value
Executive subregion	L	1799	-12 13 7	-8.12
Executive subregion	R	1410	12 13 7	-8.80
Caudal motor subregion	L	219	-26 -6 -1	-8.25
Caudal motor subregion	R	199	26 -6 -1	-8.78

All regions survived the most stringent whole-brain family-wise error (FWE) correction for multiple comparisons at $p < 0.05$ (minimum cluster size 100 voxels).

Abbreviations: MNI, Montreal Neurological Institute.

Supplementary Table 4. Results of the partial Pearson's correlation analysis between the SUVR of the striatum and neuropsychiatric scores.

Region	Side	Digit span (forward)	Digit span (backward)	TMT A	TMT B	TMT B-A	Verbal Fluency Test	CDR
Striatum	L	-0.0728	0.3596	-0.1564	-0.3791	-0.2968	0.4491**	-0.1448
Striatum	R	0.1868	0.4436*	-0.1615	-0.2922	-0.3458	0.2975	-0.3189

*of statistical significance after false discovery rate [FDR]-corrected ($p < 0.05$);

**of statistical significance after false discovery rate [FDR]-corrected ($p < 0.01$)

Supplementary Table 5. Correlation between executive function and the striatal executive subregion or DLPFC in controls

Region	Side	Digit span (forward)		Digit span (backward)		TMT A		TMT B		TMT B-A		Verbal Fluenrcy Test	
		r	p-value	r	p-value	r	p-value	r	p-value	r	p-value	r	p-value
Executive subregion	L	-0.05	0.81	-0.32	0.08	0.14	0.48	0.25	0.16	0.24	0.24	-0.15	0.46
Executive subregion	R	-0.06	0.75	-0.32	0.08	0.18	0.35	0.14	0.44	0.11	0.57	-0.26	0.20
DLPFC	L	-0.01	0.96	-0.28	0.13	0.10	0.63	0.12	0.52	0.14	0.48	-0.12	0.54
DLPFC	R	0.01	0.95	-0.36	0.05	0.11	0.56	0.14	0.45	0.11	0.57	-0.07	0.72

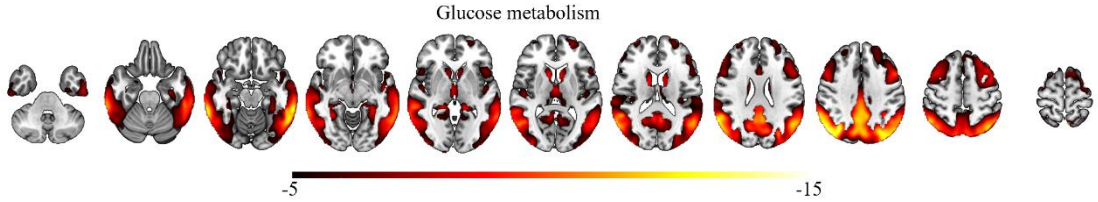
Supplementary Table 6. Results of the partial Pearson's correlation analysis between neuropsychiatric scores and the SUVR of the striatal subregion of striatal executive subregion and caudal motor subregion.

Region	Side	Digit span (forward)	Digit span (backward)	TMT-A	TMT-B	TMT B-A	Verbal Fluency Test
Executive subregion	L	0.0258	0.4744**	-0.1735	-0.5879**	-0.6707**	0.5467***
Executive subregion	R	0.1002	0.2783	-0.0614	-0.3215	-0.4246*	0.2690
Caudal motor subregion	L	0.1569	0.5128**	-0.1580	-0.4447	-0.5391*	0.5578***
Caudal motor subregion	R	0.2055	0.3729*	-0.1148	-0.2640	-0.3990	0.2572

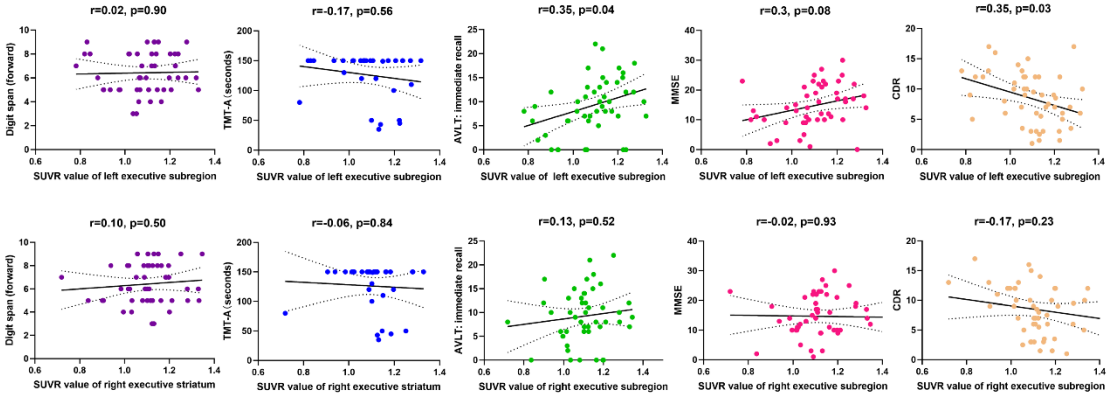
*of statistical significance after false discovery rate [FDR]-corrected ($p < 0.05$);

**of statistical significance after false discovery rate [FDR]-corrected ($p < 0.01$)

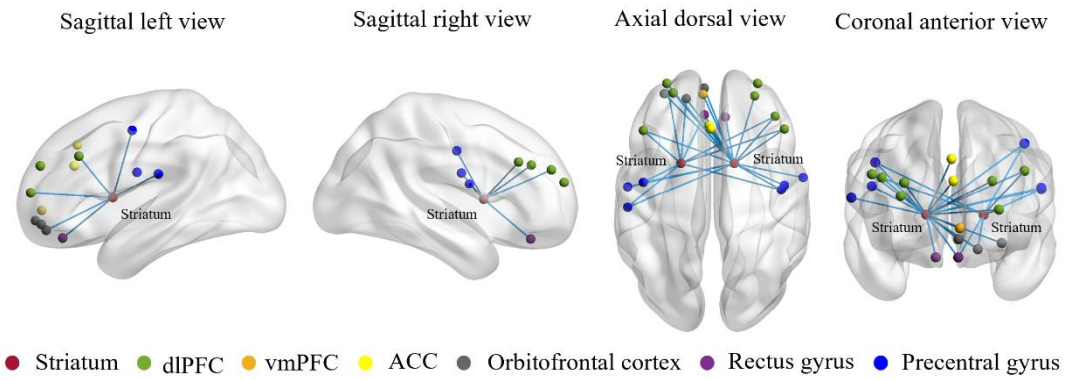
***of statistical significance after false discovery rate [FDR]-corrected ($p < 0.001$)



Supplementary Figure 1. Regions of hypometabolism in AD. Compared to controls, AD patients demonstrated widespread and bilateral hypometabolism involving parietal, temporal, and to a lesser extent frontal regions, as well as the subcortical nuclei including the caudate, putamen, and thalamus. T values are color-coded in a red-yellow color gradient to highlight the differences for subjects with AD patients < controls. Data were analyzed at a height threshold of $p < 0.001$ and were cluster-level corrected for FWE at $p < 0.05$.



Supplementary Figure 2. Correlations between the striatal executive subregion and neuropsychological scores in patients with AD. There was no significant correlation between the SUVR value of the striatal executive subregion and Digit span (forward), or TMT A. Scatter plots show that the SUVR value of the left striatal executive subregion was significantly correlated with AVLT (immediate recall) and CDR score; however, this was not the case for the SUVR of the striatal right executive subregion. Region and scatterplot colors, purple: digit span (forward), blue: TMT A, green: AVLT: immediate recall, red: MMSE, orange: CDR



Supplementary Figure 3. Compared to the controls, AD patients showed decreased metabolic connectivity between the striatum and DLPFC. Furthermore, there were weaker connections between the striatum and the ventromedial prefrontal cortex, anterior cingulate cortex, orbitofrontal cortex, rectus gyrus, and precentral gyrus. Abbreviations: ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; vmPFC, ventromedial prefrontal cortex