Supplementary Materials for

Uncovering the biological basis of control energy: Structural and metabolic correlates of energy inefficiency in temporal lobe epilepsy

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Supplementary Methods

S1: Methodological consideration on intrinsic connectivity network (ICN) defined brain states

A “brain state”, or an activation state mentioned in this and other studies (9, 10, 13, 16), refers to a pattern of brain activity that can be coded as a vector of activity magnitudes across all brain regions. All brain regions will be active to a certain extent all the time; however, some will be more active than others during distinct cognitive tasks. The pattern of activation characteristic of a cognitive task is sometimes also referred to as the pattern of co-activation, to highlight the interesting dependence in activity between region pairs (35, 92–94). For example, during a working memory task, we can decompose the dynamic neural processes—from viewing a stimulus to pressing a button—into several brain states. When the subject views the stimulus, regions within the visual cortex are co-activated more than the rest of the brain; afterwards, activations in fronto-parietal regions become dominant, as these specifically subserve working memory; lastly, the motor system becomes more active to drive the button response. In each of these phases, a distinct system of brain regions (i.e., an ICN) is predominantly active, while the rest of the brain is relatively less active, as can be observed via fMRI (95).

Accordingly, our simulation mimics the transition from one brain state when a particular ICN is most active, to another brain state in which another ICN is most active, as an approximation of transitions between the empirically observed co-activation patterns (35, 92–94). Transitions between co-activation patterns characterized by selectively activated brain networks are commonly observed in fMRI studies. In our previous fMRI study (2), we used a clustering algorithm to show that transitions between representative co-activation patterns occur during both rest and an N-back working memory task. These transitions are influenced by task demands and meaningfully related to individual differences in behavior. Further, simulations showed that the control energy needed to facilitate such transitions is associated with chronological age (2). Using similar techniques, results from another group indicated that transitions between representative co-activation patterns are associated with cognitive performance in the presence of pathology (96). Such transitions can even be observed in fMRI studies performed on animal models, and are related to distinct levels of consciousness in the mouse brain (97).

In our study, the ICNs used to represent such co-activation patterns were obtained from an established parcellation of the brain into cognitive systems (38). It has been demonstrated that, regions belonging to such systems do not only co-synchronize during rest (37, 38), but also co-activate during different cognitive conditions (37, 39, 40). Using a mathematical simplification, we can further binarize these ICN-defined co-activation patterns by setting those regions that are relatively more active (i.e., regions within a specific ICN) to 1, and those that are relatively less active (i.e., the rest of the brain) to 0. In so doing, we consider activity in the less active regions as a mean-centered baseline, and the activity in the more active regions to be greater than that baseline by 1 arbitrary unit. This setting is analogous to
task fMRI analyses, where contrasts are commonly set between a condition of interest (1) and a baseline condition (0) (see Ref. (16)).

We have previously deployed such binarized ICNs as forms of simplified co-activation patterns in several studies (9, 10, 13, 16). Some previous studies have laid out the biological relevance of control energy estimated using binary brain states. For example, in one study, we demonstrated that variance in the control energy needed to attain a binary brain state centered on the frontal-parietal network was associated with executive function development in youth (16). Another independent group also adopted a similar strategy and found that the control energy needed to transition from a binary brain state centered on the DMN to another centered on the motor network was associated with aging (98). Importantly, consistent results have been reported even when random noise is introduced into the binary brain state, providing additional support to these observations (16).

In reality, there are many intermediate states when a partial ICN, or two or more ICNs, are active at the same time. Importantly, the linear nature of our NCT model permits us to write any transitions between any pair of such intermediate states in the form of linear combinations of transitions between single ICNs (9). Hence, we explored the average energy cost during simulated transitions among all the ICNs so that transitions between intermediate states could be simulated in the same process. This intuition is further verified, when we tested the average energy cost during simulated transitions among non-binary random brain states in which every region is activated at a random magnitude, and found almost identical results.

While this functional parcellation of ICNs has been derived from healthy subjects, it has recently been broadly applied to clinical populations, including epilepsy patients, to quantify the functional integrity of major cognitive systems. For example, this functional parcellation is frequently used to identify stratifying effects at the functional network level, e.g., for cortical atrophy patterns (99) and atypical functional connectome hierarchy (100) in TLE, disrupted functional gradients of the cortical connectome both in TLE (101) and generalized epilepsies (102), as well as associations between altered communication dynamics and cognitive deficits in TLE (34). This functional parcellation has also been used to localize large-scale functional networks within which the rapid spread of ictal activity arises (103), and to identify altered functional connectivity patterns important for epilepsy classification (104). Further, this functional parcellation provides the field map that has been used to quantify thalamocortical functional connectivity organization in TLE and generalized epilepsies (46, 105). Collectively, these past studies underscore the fact that this functional parcellation has substantially advanced our understanding of the pathological impacts of epilepsy on the functional organization of the brain.

Nonetheless, it is possible that TLE patients may present with atypical patterns of region allocation to large-scale functional networks. However, our approach to use generic ICNs detected previously was motivated by our goal to isolate the dynamical consequences of abnormal structural network topology; if we had used distinct ICNs for the two groups, our
results would have conflated the structural network mechanism with the separate mechanism of differences in brain states. Thus, employing different ICNs between groups would confound our interpretation of the control energy findings. Instead, by setting uniform brain states for patients and controls, we ensured comparability of control energy across groups. In a future study, it would be of interest to develop and rigorously validate a new methodological approach (and associated statistical inference methods) that would allow investigators to separately quantify the contributions of structural connectivity and distinct brain states on the estimated control energy in the context of group comparisons.

Taken together, ICN-defined binary brain states can be a useful tool in network control theory (NCT) studies. Importantly, our data and extensive prior studies underscore that this state transition framework can be used as a simple approximation of the empirical brain dynamics commonly observed via fMRI. In specific, by adopting the public functional parcellation of cognitive systems (i.e., ICNs) to define brain states in our study, we directly isolate the structural effects of interest while simultaneously ensuring a clean comparison with previous studies.
Supplementary Results

S1: Sensitivity Analyses on the impact of overall white matter health

In this section, we evaluated the impact of overall white matter health on our results reported in the main text. White matter lesions are commonly detected with specific T2-weighted imaging sequences in the form of white matter hyperintensity (WMH). In the absence of such data, here, we estimated global WMH volume with T1-weighted image using the Computational Anatomy Toolbox (CAT12). To evaluate the robustness of our original findings, we extracted estimates of WMH for each subject and regressed those estimates out of our control energy metrics. We then repeated our main analyses, and found consistent results at every step.

In specific, we found that temporal lobe epilepsy (TLE) patients required greater global optimal control energy (OCE) to activate the limbic network compared to healthy controls (HCs, Welch's $t_{108}=3.80, P_{corr}=0.002$). The global OCE needed to activate other intrinsic connectivity networks (ICNs) did not significantly differ between the two groups (Welch's $|t_{108}'s|<1.51, P_{corr}>0.642$). When we simulated the activation of left and right limbic ICN separately, we found again a significant hemisphere-by-group interaction [$F_{(2,107)}=10.76, P=6×10^{-5}$]. When comparing regional energy efficiency in supporting cross-ICN transitions, we found significantly elevated control energy consumption in TLE in the ipsilateral temporal pole ($t_{59}=4.89, P_{corr}=0.001$), inferior temporal gyrus ($t_{59}=4.71, P_{corr}=0.002$), amygdala ($t_{59}=5.15, P_{corr}=3×10^{-4}$), hippocampus ($t_{59}=4.87, P_{corr}=0.001$), parahippocampal gyrus ($t_{59}=4.26, P_{corr}=0.008$), fusiform gyrus ($t_{59}=4.86, P_{corr}=0.001$), and the isthmus of the cingulate gyrus ($t_{59}=4.09, P_{corr}=0.014$). Similar results were found for transitions between random states at the ipsilateral temporal pole ($t_{59}=4.29, P_{corr}=0.001$), inferior temporal gyrus ($t_{59}=4.33, P_{corr}=0.006$), amygdala ($t_{59}=3.49, P_{corr}=0.091$), hippocampus ($t_{59}=4.25, P_{corr}=0.009$), parahippocampal gyrus ($t_{59}=5.16, P_{corr}=3×10^{-4}$), fusiform gyrus ($t_{59}=5.12, P_{corr}=4×10^{-4}$), and isthmus of the cingulate gyrus ($t_{59}=4.75, P_{corr}=0.001$). When we tested associations between the LIs of control energy and glucose metabolism, we found significant correlations between the LIs at the amygdala ($R_{49}=-0.63, P_{corr}=6×10^{-6}$), hippocampus ($R_{49}=-0.60, P_{corr}=3×10^{-5}$), parahippocampal gyrus ($R_{49}=-0.49, P_{corr}=0.002$), and fusiform gyrus ($R_{49}=-0.39, P_{corr}=0.034$), marginally at the temporal pole ($R_{49}=-0.35, P_{corr}=0.075$), but not within the inferior temporal gyrus ($R_{49}=-0.34, P_{corr}=0.103$) or isthmus of cingulate gyrus ($R_{49}=-0.22, P_{corr}=0.587$). These results are highly consistent with our main findings, hence can support all the original conclusions in the main text. Therefore, changes to control energy metrics are mainly driven by changes to the dynamics occurring atop altered structural networks rather than being driven by overall white matter health.
**S2. Sensitivity analyses on the comparison between control energy and conventional descriptive connectomic metrics**

Conventional descriptive connectomic metrics, such as nodal degree and strength, can also provide information regarding local topology of the structural network. Accordingly, they may share similar cross-subject variances embedded in our control energy metrics. To provide further discrimination between these two types of metrics, in this section, we explored whether similar results can be produced with such conventional derivatives of the structural connectome, and whether control energy can provide unique information compared to them.

We began by replacing global OCE needed to activate each ICN with the connectivity strength of each ICN (i.e., sum of all edge weights connected to all regions within each ICN respectively), and compared the latter between TLE patients and HCs as we had in Figure 2 in the main text. Such network connectivity strength is an analog to nodal strength, but at the network level. Consistent with our control energy analyses, we first regressed out the same confounding factors used in the main text. We then conducted a permutation-based t-test on the so-obtained residuals, using the same approach as in our main analyses. Patients with TLE had lower limbic connectivity strength compared to HCs (Welch's $t_{108}=-3.24$, $P_{corr}=0.013$). The connectivity strength of other ICNs, however, did not significantly differ between groups ($Welch's \ |t_{108}'s|<1.48$, $P_{corr}>0.661$). These results confirm that limbic changes in control energy in TLE are paralleled by changes in connectivity strength, as expected based on the mathematical derivation of control energy metrics. We then assessed the specific contributions of limbic control energy and limbic connectivity strength in differentiating patients and HCs. We devised a logistic regression model using group as a binary dependent variable (HC or patient with TLE), and both limbic control energy and limbic connectivity strength as independent variables, entered in a stepwise fashion. Interestingly, control energy alone ($\beta=3.884$, $t=3.437$, $P=5.9\times10^{-4}$) was selected in the final model (Nagelkerke $R^2=0.160$, $\chi^2=14.016$, $P=1.8\times10^{-4}$), while connectivity strength was not included in the model, as it was not significant ($P=0.483$). This result suggests that control energy is more sensitive in highlighting difference between patients and HCs, rather than redundant.

Next, we replaced nodal minimal control energy (MCE) with nodal degree and strength, respectively, and performed the same statistical analyses as those presented in Figures 4 and 5 in the main text. Consistent with our control energy analyses, we first regressed out the same confounding factors used in the main text. We then conducted permutation-based analyses on the so-obtained residuals, using the same approach as in our main analyses.

1) We first probed group differences in nodal degree. Compared to controls, patients with TLE presented with greater degree in the ipsilateral putamen ($t_{59}=4.80$, $P_{corr}=8\times10^{-4}$) and lesser degree in the contralateral nucleus accumbens ($t_{59}=-3.92$, $P_{corr}=0.019$), but not in any of the ipsilateral temporo-limbic regions. The latter findings differ from our control energy findings (Table S2). We then used nodal degree as a nuisance covariate in all group comparisons of nodal control energy. These sensitivity analyses yielded virtually identical results to those of our
original analyses, as detailed in Table S2. Taken together, our findings demonstrate that the energetic differences between patients with TLE and controls was not driven by degree.

2) Next, we considered nodal strength. Comparison of patients and controls highlighted lesser nodal strength in TLE patients, consistent with our control energy findings (Table S3). In all group comparisons of nodal control energy, we then covaried for nodal strength, and found that results were partially consistent with our original analysis. Specifically, we found group differences in control energy within the hippocampus, inferior temporal gyrus and fusiform gyrus that met the threshold for corrected significance (Table S3). Thus, while nodal strength is relevant to intergroup differences, it is not sufficient to fully account for our control energy findings.

3) Next, we tested associations of the laterality indices (LIs) of nodal degree and strength with glucose metabolism. Among the aforementioned regions in Tables S2 and S3, the LIs of nodal degree and glucose metabolism were not significantly correlated ($|R_{49}|<0.23$, $P_{corr}>0.534$). There were, however, significant correlations between LIs of glucose metabolism and nodal strength, which paralleled our findings of correlation analyses with control energy. Accordingly, we regressed out nodal strength from control energy, and re-estimated the associations between the LIs of glucose metabolism and control energy, and were able to largely replicate the correlational results reported in our main analyses, as detailed in Table S4.

4) Lastly, we evaluated whether the LI of control energy more accurately predicts the LI of glucose metabolism than the LI of nodal strength. For each of the above regions, we constructed a stepwise logistic regression model using the LI of glucose metabolism as the dependent variable, and both the LIs of control energy and nodal strength as independent variables. The results confirmed that control energy is a significant predictor of metabolic laterality, and suggested that strength and control energy may exhibit unique and, at times, complementary predictive power (Table S5). Specifically, in temporal pole and amygdala, the LI of control energy was the only successful predictor of metabolic LI. In the hippocampus and fusiform gyrus, both the LIs of control energy and nodal strength contributed to the prediction of metabolic LI; for the hippocampus, $beta$ values and $t$-statistics were higher for control energy than for strength. For the parahippocampal gyrus only, the LI of control energy was originally significant, but was then excluded by the model due to its high collinearity with the LI of nodal strength (VIF=3.327).

Taken together, these extensive sensitivity analyses indicate that control energy is not a redundant metric, and cannot be entirely explained by conventional descriptive connectomic metrics. Furthermore, all of our analyses demonstrate that control energy generally confers superior explanatory power compared to canonical nodal graph theoretical metrics.
S3. Sensitivity analyses on the comparison between control energy and controllability

Controllability metrics, such as average and modal controllability, are the first NCT metrics brought into neuroscientific inquires to describe the general ability of each region to control neural dynamics depending upon the full topology of the structural network (13). Controllability metrics and the control energy metrics used in this study are a family of internally-consistent metrics all derived from the same simplified noise-free linear and time-invariant model of network dynamics depicted in Eq. 1. They differ in that each summarizes a distinct aspect of that model applied in different contexts. The term controllability describes the general ability of each region to control neural dynamics depending upon the full topology of the structural network (13), and it is estimated in the scenario of controlling whole-brain network dynamics from a single region to achieve all possible brain state transitions (8). Importantly, controllability can be quantified by the infinite impulse response (average controllability) or the eigenvalues (modal controllability) of the A matrix; both quantifications assess all possible states, and if one instead wishes to model a specific transition between a single pair of states or a small number of states, then one needs a different metric (13). In this case, the metric of control energy becomes useful as it summarizes the control inputs $u(t)$ over a finite period of a simulated dynamic transition between two specific brain states. The control energy metric not only depends on the topology of the A matrix, but also on the specific time horizon ($0 \to T$), distance between the initial and final states ($x(T) - x(0)$), and the transition trajectory, as well as the location and number of nodes that are under control. In our study, we let all the brain regions to be controllable, which is more biologically plausible than controlling from a single region. The optimal control framework allows us to estimate the minimal energy costs to facilitate the designated brain state transitions while following the shortest trajectory. The approach relies on a key modeling assumption: that the brain operates as an efficient system that minimizes the expenditure of energy and the distance the system can traverse in state space. In Simulation II of our study, we pursued a hybrid approach in which we aimed to approximate all possible brain state transitions without constraining the trajectory length; this hybrid approach is made possible by the minimal control framework which allows the brain to move more freely across different intermediate states. As these descriptions and comparisons demonstrate, in contrast to the controllability metrics the control energy approach allows us to more flexibly model and describe the neural dynamics under specific state transition scenarios.

To further probe the distinctions between these NCT metrics at describing regional energetic efficiency, in this section, we first evaluated their associations, then test whether similar findings can be produced with average and modal controllability, and whether control energy can provide unique information compared to them.

In the main text, MCE during cross-ICN transitions was used to describe regional energetic efficiency. Accordingly, here we correlated the estimated MCE with both average and modal controllability, and whether control energy can provide unique information compared to them.

In the main text, MCE during cross-ICN transitions was used to describe regional energetic efficiency. Accordingly, here we correlated the estimated MCE with both average and modal controllability, and whether control energy can provide unique information compared to them.
demonstrated significant correlations between MCE and modal controllability, while none of them showed significant correlations between MCE and average controllability. Therefore, while the interpretation of the MCE can be analogous to controllability, these distinctions are more or less expected given the quite different factors these metrics are constructed to track and reflect. Nonetheless, they can still share cross-subject variances in our group-level analyses. We thus explored whether, after replacing control energy with controllability, we could reproduce results indicating that these NCT measures relate to brain metabolism. Consistent with our control energy analyses, we first regressed out the same confounding factors used in the main text. We then conducted permutation-based analyses on the so-obtained residuals, using the same approach as in our main analyses. We outline these sensitivity analyses below.

1) Compared to HCs, TLE patients had lower average controllability (Table S6) and greater modal controllability (Table 7), which largely paralleled our primary control energy findings. After regressing out either average or modal controllability before group comparisons of nodal control energy, we obtained results that were largely similar to those of our main analysis, as detailed in Tables S6 and S7.

2) When we tested the associations between LIs of glucose metabolism and average controllability among the aforementioned regions, we did not find any significant results (\(|R_s|<0.33, P_{corr}>0.136\). Notably, we found significant correlations between the LIs of glucose metabolism and modal controllability, which were similar to our findings in control energy. After regressing out modal controllability and then re-estimating associations between the LIs of glucose metabolism and control energy, we also largely reproduced the findings of our main analysis, as detailed in Table S8. Taken together, these results suggest that similar to control energy, modal controllability also relates to brain metabolism. However, the association between control energy and glucose metabolism cannot be fully accounted for by the association between modal controllability and glucose metabolism.

3) Lastly, we tested whether control energy better tracks the relationship between energetic efficiency and glucose metabolism, compared to modal controllability. For each of our regions of interest (see main text and Table S8), we formulated a stepwise logistic regression model using the LI of glucose metabolism as the dependent variable, and both LIs of control energy and modal controllability as independent variables. For the temporal pole, amygdala, and parahippocampal gyrus, the LI of control energy was the identified as the exclusive predictor of the LI of glucose metabolism. For the hippocampus and fusiform gyrus, both LIs of control energy and modal controllability comparably contributed to predicting the LI of glucose metabolism. Only in the inferior temporal gyrus, the LI of modal controllability was the exclusive predictor of LI of glucose metabolism. These results are shown in Table S9.

In summary, controllability metrics, especially modal controllability can be used to largely reproduce the results to support our main claims, i.e., that energy efficiency estimated via NCT is associated with brain metabolism as tracked by PET. Nonetheless, control energy is more
sensitive to the effects of pathology (*i.e.*, differences between patients and controls) and is a better predictor of regional brain metabolism compared to controllability.
Table S1. Permutation-based nonparametric one-sample \( t \)-tests on laterality indices (LIs) of regional glucose uptake reveal significant ipsilateral hypometabolism in temporal lobe epilepsy patients.

| Lausanne Atlas Region Label | LI (mean±std) | \( t_{49} \) | \( P_{corr} \) | Cohen’s \( d \) |
|-----------------------------|---------------|--------------|----------------|---------------|
| lateralorbitofrontal_1      | -0.014±0.019  | -5.383       | 1×10\(^{-4}\) | -0.761        |
| rostralmiddlefrontal_1      | -0.005±0.01   | -3.678       | 0.023          | -0.520        |
| superiorfrontal_3           | -0.007±0.013  | -3.622       | 0.027          | -0.512        |
| isthmuscingulate_1          | -0.016±0.02   | -5.788       | 3×10\(^{-5}\) | -0.819        |
| supramarginal_1             | -0.014±0.024  | -4.226       | 0.005          | -0.598        |
| superiorparietal_2          | -0.011±0.017  | -4.492       | 0.002          | -0.635        |
| inferiorparietal_1          | -0.013±0.019  | -4.690       | 0.001          | -0.663        |
| precuneus_2                 | -0.017±0.036  | -3.401       | 0.049          | -0.481        |
| lateraloccipital_2          | -0.011±0.019  | -3.908       | 0.012          | -0.553        |
| fusiform_2                  | -0.016±0.019  | -6.196       | 1×10\(^{-5}\) | -0.876        |
| parahippocampal_1           | -0.018±0.021  | -5.981       | 2×10\(^{-5}\) | -0.846        |
| entorhinal_1                | -0.039±0.042  | -6.652       | 5×10\(^{-6}\) | -0.941        |
| temporalpole_1              | -0.045±0.049  | -6.608       | 5×10\(^{-6}\) | -0.935        |
| inferiortemporal_1          | -0.024±0.03   | -5.695       | 4×10\(^{-5}\) | -0.805        |
| inferiortemporal_2          | -0.013±0.017  | -5.452       | 1×10\(^{-4}\) | -0.771        |
| middletemporal_1            | -0.012±0.02   | -4.191       | 0.005          | -0.593        |
| middletemporal_2            | -0.026±0.027  | -6.795       | 2×10\(^{-6}\) | -0.961        |
| bankssts_1                  | -0.02±0.023   | -6.245       | 1×10\(^{-5}\) | -0.883        |
| superiortemporal_1          | -0.014±0.02   | -4.742       | 0.001          | -0.671        |
| insula_1                    | -0.024±0.039  | -4.845       | 0.002          | -0.634        |
| thalamusproper              | -0.008±0.013  | -4.228       | 0.005          | -0.598        |
| putamen                     | -0.005±0.008  | -4.830       | 0.001          | -0.683        |
| pallidum                    | -0.013±0.021  | -4.422       | 0.003          | -0.625        |
| accumbensarea               | -0.008±0.015  | -3.746       | 0.019          | -0.530        |
| hippocampus                 | -0.026±0.026  | -7.173       | 1×10\(^{-6}\) | -1.014        |
| amygdala                    | -0.028±0.031  | -6.423       | 9×10\(^{-6}\) | -0.908        |

* The LI is calculated as \( LI_i = \frac{Ipsilateral_i - Contralateral_i}{Ipsilateral_i + Contralateral_i} \), whereas a negative LI indicated lower ipsilateral compared to contralateral metabolism. Only regions with LIs significantly different from zero after multiple comparisons corrections are presented. Statistics including the \( t \)-value, corrected \( P \)-value (\( P_{corr} \)), and effect size (Cohen’s \( d \)) are depicted. The regions co-exhibiting ipsilateral abnormalities in energy profiles are highlighted in bold.
Table S2 shows statistics pertaining to comparisons of deviation scores (against data from healthy controls) of control energy and nodal degree, which were determined by a permutation-based one sample $t$-test. $P_{\text{corr}}$, $P$ values were corrected for multiple comparisons by controlling the family-wise error rate.

| Ipsilateral regions                | Control Energy (original results) | Degree | Control Energy (covaried for Degree) |
|------------------------------------|-----------------------------------|--------|--------------------------------------|
|                                    | $t$  | $P_{\text{corr}}$ | $t$  | $P_{\text{corr}}$ | $t$  | $P_{\text{corr}}$ |
| Temporal Pole                     | 5.40 | $1\times10^{-4}$ | -1.88 | 0.985 | 5.15 | $3\times10^{-4}$ |
| Inferior Temporal                 | 5.03 | $5\times10^{-4}$ | -0.52 | 1   | 5.52 | $9\times10^{-5}$ |
| Amygdala                          | 6.01 | $1\times10^{-5}$ | -2.13 | 0.917 | 5.84 | $3\times10^{-5}$ |
| Hippocampus                       | 5.24 | $2\times10^{-4}$ | -1.71 | 0.997 | 5.48 | $1\times10^{-4}$ |
| Parahippocampal                   | 4.54 | 0.003            | -2.03 | 0.955 | 4.45 | 0.004            |
| Fusiform                          | 4.93 | $7\times10^{-4}$ | -3.45 | 0.086 | 5.79 | $3\times10^{-5}$ |
| Isthmus of Cingulate              | 4.17 | 0.011            | 2.16  | 0.905 | 4.02 | 0.018            |
Table S3 shows statistics pertaining to comparisons of deviation scores (against data from healthy controls) of control energy and nodal strength, which were determined by a permutation-based one sample $t$-test. $P_{\text{corr}}$, $P$ values were corrected for multiple comparisons by controlling the family-wise error rate; $P_{\text{unc}}$, uncorrected $P$ values for reference only.

| Ipsilateral regions          | Control Energy (original results) | Strength | Control Energy (covaried for Strength) |
|------------------------------|----------------------------------|----------|----------------------------------------|
|                              | $t$  | $P_{\text{corr}}$ | $t$  | $P_{\text{corr}}$ | $t$  | $P_{\text{unc}}/P_{\text{corr}}$ |
| Temporal Pole                | 5.40 | $1\times10^{-4}$ | -4.39 | 0.005 | 2.53 | 0.014/0.719 |
| Inferior Temporal            | 5.03 | $5\times10^{-4}$ | -4.89 | 0.001 | 5.43 | $1\times10^{-6}/1\times10^{-4}$ |
| Amygdala                     | 6.01 | $1\times10^{-5}$ | -4.10 | 0.015 | 3.12 | 0.003/0.241 |
| Hippocampus                  | 5.24 | $2\times10^{-4}$ | -4.23 | 0.009 | 4.01 | $2\times10^{-4}/0.018$ |
| Parahippocampal              | 4.54 | 0.003          | -5.03 | $5\times10^{-4}$ | 2.32 | 0.024/0.876 |
| Fusiform                     | 4.93 | $7\times10^{-4}$ | -5.52 | $9\times10^{-5}$ | 5.24 | $2\times10^{-6}/2\times10^{-4}$ |
| Isthmus of Cingulate         | 4.17 | 0.011          | -5.62 | $6\times10^{-5}$ | 0.76 | 0.453/1 |
Table S4  Associations between LIs of glucose metabolism with nodal strength and control energy (with and without regressing out nodal strength), that were determined by a permutation-based product-moment correlation; $P_{\text{corr}}$, $P$ values were corrected for multiple comparisons by controlling the family-wise error rate.

| Ipsilateral regions       | Control Energy (original results) | Strength | Control Energy (covaried for Strength) |
|---------------------------|----------------------------------|----------|----------------------------------------|
|                           | $R$     | $P_{\text{corr}}$ | $R$   | $P_{\text{corr}}$ | $R$   | $P_{\text{corr}}$ |
| Temporal Pole             | -0.37   | 0.049             | 0.22  | 0.602             | -0.39 | 0.034             |
| Inferior Temporal         | -0.34   | 0.096             | 0.59  | 5×10^{-5}          | -0.32 | 0.143             |
| Amygdala                  | -0.62   | 8×10^{-6}         | 0.41  | 0.023             | -0.52 | 0.001             |
| Hippocampus               | -0.60   | 3×10^{-5}         | 0.54  | 3×10^{-4}          | -0.46 | 0.005             |
| Parahippocampal           | -0.50   | 0.002             | 0.60  | 3×10^{-5}          | -0.36 | 0.066             |
| Fusiform                  | -0.39   | 0.036             | 0.45  | 0.007             | -0.33 | 0.108             |
| Isthmus of Cingulate      | -0.22   | 0.551             | 0.21  | 0.668             | -0.03 | 1                 |
Table S5 Stepwise linear regression models revealing the contributions of the LIs of control energy (CE) and nodal strength (Str) in predicting the LI of glucose metabolism (Glu). $\beta$ values presented here are standardized coefficients.

| LI of:          | Final model                  | Statistics for the final model | Statistics for each independent variable |
|-----------------|------------------------------|-------------------------------|------------------------------------------|
| Temporal Pole   | Glu $\sim \beta_1 \times$ CE | $R^2=0.140$, $F(1,48)=7.810$, $P=0.007$ | $\beta_1=-0.374$, $t=-2.795$, $P=0.007$, VIF=1 |
| Inferior Temporal| Glu $\sim \beta_1 \times$ Str | $R^2=0.347$, $F(1,48)=25.457$, $P=7\times10^{-6}$ | $\beta_1=0.589$, $t=5.046$, $P=7\times10^{-6}$, VIF=1 |
| Amygdala        | Glu $\sim \beta_1 \times$ CE | $R^2=0.385$, $F(1,48)=29.990$, $P=2\times10^{-6}$ | $\beta_1=-0.620$, $t=-5.476$, $P=2\times10^{-6}$, VIF=1 |
| Hippocampus     | Glu $\sim \beta_1 \times$ CE+$\beta_2 \times$ Str | $R^2=0.440$, $F(2,47)=18.483$, $P=1\times10^{-6}$ | $\beta_1=-0.436$, $t=-3.499$, $P=0.001$, VIF=1.305, $\beta_2=0.332$, $t=2.660$, $P=0.011$, VIF=1.305 |
| Parahippocampal | Glu $\sim \beta_1 \times$ Str | $R^2=0.363$, $F(1,48)=27.409$, $P=4\times10^{-6}$ | $\beta_1=0.603$, $t=5.235$, $P=4\times10^{-6}$, VIF=1 |
| Fusiform        | Glu $\sim \beta_1 \times$ CE+$\beta_2 \times$ Str | $R^2=0.333$, $F(2,47)=11.743$, $P=7\times10^{-5}$ | $\beta_1=-0.363$, $t=-3.046$, $P=0.004$, VIF=1.003, $\beta_2=0.428$, $t=3.589$, $P=0.001$, VIF=1.003 |
| Isthmus of Cingulate | No variable entered. | | |
Table S6 shows statistics pertaining to comparisons of deviation scores (against data from healthy controls) of control energy and average controllability, which were determined by a permutation-based one sample \( t \)-test. \( P_{\text{corr}} \), \( P \) values were corrected for multiple comparisons by controlling the family-wise error rate.

| Ipsilateral regions         | Control Energy (original results) | Average Controllability | Control Energy (covaried for Av. Control) |
|-----------------------------|-----------------------------------|-------------------------|------------------------------------------|
|                             | \( t \)                           | \( P_{\text{corr}} \)   | \( t \)                                   | \( P_{\text{corr}} \)                           | \( t \)                                   | \( P_{\text{corr}} \)                           |
| Temporal Pole               | 5.40                              | 1\( \times \)10\(^{-4} \)| -1.39                                   | 1                                                | 5.29                                   | 2\( \times \)10\(^{-4} \)|                          |
| Inferior Temporal           | 5.03                              | 5\( \times \)10\(^{-4} \)| -6.12                                   | 5\( \times \)10\(^{-6} \)                       | 6.10                                   | 7\( \times \)10\(^{-6} \)|                          |
| Amygdala                    | 6.01                              | 1\( \times \)10\(^{-5} \)| -2.08                                   | 0.869                                            | 5.95                                   | 1\( \times \)10\(^{-5} \)|                          |
| Hippocampus                 | 5.24                              | 2\( \times \)10\(^{-4} \)| -6.90                                   | 1\( \times \)10\(^{-6} \)                       | 4.14                                   | 0.012                                                |
| Parahippocampal             | 4.54                              | 0.003                    | -5.49                                   | 5\( \times \)10\(^{-5} \)                       | 3.78                                   | 0.039                                                |
| Fusiform                    | 4.93                              | 7\( \times \)10\(^{-4} \)| -5.83                                   | 1\( \times \)10\(^{-5} \)                       | 5.43                                   | 1\( \times \)10\(^{-4} \)                           |
| Isthmus of Cingulate        | 4.17                              | 0.011                    | -6.37                                   | 2\( \times \)10\(^{-6} \)                       | 2.64                                   | 0.632                                                |
Table S7 shows statistics pertaining to comparisons of deviation scores (against data from healthy controls) of control energy and modal controllability, which were determined by a permutation-based one sample t-test. $P_{corr}$, $P$ values were corrected for multiple comparisons by controlling the family-wise error rate; $P_{unc}$, uncorrected $P$ values for reference only.

| Ipsilateral regions   | Control Energy (original results) | Modal Controllability | Control Energy (covaried for Mod. Control) |
|-----------------------|----------------------------------|-----------------------|-------------------------------------------|
|                       | $t$ | $P_{corr}$ | $t$ | $P_{corr}$ | $t$ | $P_{unc}/P_{corr}$ |
| Temporal Pole         | 5.40 | $1\times10^{-4}$ | 4.31 | 0.005 | 2.97 | 0.004/0.355 |
| Inferior Temporal     | 5.03 | $5\times10^{-4}$ | 4.14 | 0.009 | 4.86 | 1$\times10^{-5}$/0.001 |
| Amygdala              | 6.01 | $1\times10^{-5}$ | 3.75 | 0.035 | 4.84 | 1$\times10^{-5}$/0.001 |
| Hippocampus           | 5.24 | $2\times10^{-4}$ | 5.02 | $4\times10^{-4}$ | 3.73 | $4\times10^{-4}$/0.045 |
| Parahippocampal       | 4.54 | 0.003 | 3.57 | 0.062 | 2.70 | 0.009/0.596 |
| Fusiform              | 4.93 | $7\times10^{-4}$ | 3.12 | 0.224 | 5.04 | $5\times10^{-6}$/5$\times10^{-4}$ |
| Isthmus of Cingulate  | 4.17 | 0.011 | 3.94 | 0.018 | 1.84 | 0.071/1 |
Table S8 Associations between the LIs of glucose metabolism with modal controllability and control energy (with and without regressing out modal controllability), which were determined by a permutation-based product-moment correlation. $P_{corr}$, $P$ values were corrected for multiple comparisons by controlling the family-wise error rate.

| Ipsilateral regions     | Control Energy (original results) | Modal Controllability | Control Energy (covaried for Mod. Control) |
|-------------------------|----------------------------------|-----------------------|-------------------------------------------|
|                         | $R$ | $P_{corr}$ | $R$ | $P_{corr}$ | $R$ | $P_{corr}$ |
| Temporal Pole           | -0.37 | 0.049 | -0.18 | 0.820 | -0.41 | 0.019 |
| Inferior Temporal       | -0.34 | 0.096 | -0.63 $\times 10^{-5}$ | 0.302 |
| Amygdala                | -0.62 $\times 10^{-6}$ | 0.48 | 0.003 | -0.36 | 0.065 |
| Hippocampus             | -0.60 $\times 10^{-5}$ | 0.51 | 0.001 | -0.39 | 0.035 |
| Parahippocampal         | -0.50 | 0.002 | -0.47 | 0.004 | -0.41 | 0.021 |
| Fusiform                | -0.39 | 0.036 | -0.46 | 0.006 | -0.32 | 0.137 |
| Isthmus of Cingulate    | -0.22 | 0.551 | -0.18 | 0.787 | -0.04 | 1 |
Table S9 Stepwise linear regression models revealing the contributions of the LIs of control energy (CE) and modal controllability (Mod) in predicting the LI of glucose metabolism (Glu). β values presented here are standardized coefficients.

| LI of: | Final model | Statistics for the final model | Statistics for each independent variable |
|--------|-------------|-------------------------------|-------------------------------------|
| Temporal Pole | Glu $\sim \beta_1 \times$ CE | $R^2=0.140$, $F(1,48)=7.810$, $P=0.007$ | $\beta_1=-0.374$, $t=-2.795$, $P=0.007$, VIF=1 |
| Inferior Temporal | Glu $\sim \beta_1 \times$ Mod | $R^2=0.391$, $F(1,48)=30.823$, $P=1 \times 10^{-6}$ | $\beta_1=-0.625$, $t=-5.552$, $P=1 \times 10^{-6}$, VIF=1 |
| Amygdala | Glu $\sim \beta_1 \times$ CE | $R^2=0.385$, $F(1,48)=29.990$, $P=2 \times 10^{-6}$ | $\beta_1=-0.620$, $t=-5.476$, $P=2 \times 10^{-6}$, VIF=1 |
| Hippocampus | Glu $\sim \beta_1 \times$ CE + $\beta_2 \times$ Mod | $R^2=0.407$, $F(2,47)=16.151$, $P=5 \times 10^{-6}$ | $\beta_1=-0.451$, $t=-3.382$, $P=0.001$, VIF=1.412, $\beta_2=-0.269$, $t=-2.018$, $P=0.049$, VIF=1.412 |
| Parahippocampal | Glu $\sim \beta_1 \times$ CE | $R^2=0.250$, $F(1,48)=15.973$, $P=2 \times 10^{-4}$ | $\beta_1=-0.5$, $t=-3.997$, $P=2 \times 10^{-4}$, VIF=1 |
| Fusiform | Glu $\sim \beta_1 \times$ CE + $\beta_2 \times$ Mod | $R^2=0.361$, $F(2,47)=13.280$, $P=3 \times 10^{-5}$ | $\beta_1=-0.391$, $t=-3.349$, $P=0.002$, VIF=1, $\beta_2=-0.459$, $t=-3.936$, $P=3 \times 10^{-4}$, VIF=1 |
| Isthmus of Cingulate | No variable entered. | |


Table S10. Label correspondence between the symmetrically modified Lausanne Atlas and the original Lausanne Atlas (76). Each region is assigned to one of the intrinsic connectivity networks (ICNs) (38) based on spatial overlap between the Lausanne Atlas and the Schaefer Atlas (77).

| Original Lausanne Atlas | Symmetrically modified Lausanne Atlas | ICN |
|-------------------------|--------------------------------------|-----|
| **Index**               | **Region Label**                     |     |
| 1                       | R_lateralorbitofrontal_1             |     |
| 2                       | R_lateralorbitofrontal_2             |     |
| 3                       | R_parsorbitals_1                     |     |
| 4                       | R_frontalpole_1                      |     |
| 5                       | R_medialorbitofrontal_1              |     |
| 6                       | R_medialorbitofrontal_2              |     |
| 7                       | R_parstriangularis_1                 |     |
| 8                       | R_parsopercularis_1                  |     |
| 9                       | R_rostralmiddlefrontal_1             |     |
| 10                      | R_rostralmiddlefrontal_2             |     |
| 11                      | R_superiorfrontal_1                  |     |
| 12                      | R_superiorfrontal_2                  |     |
| 13                      | R_superiorfrontal_3                  |     |
| 14                      | R_superiorfrontal_4                  |     |
| 15                      | R_caudalmiddlefrontal_1              |     |
| 16                      | R_precentral_1                       |     |
| 17                      | R_precentral_2                       |     |
| 18                      | R_precentral_3                       |     |
| 19                      | R_paracentral_1                      |     |
| 20                      | R_rostralanteriorcingulate_1         |     |
| 21                      | R_caudalanteriorcingulate_1          |     |
| 22                      | R_posteriorcingulate_1               |     |
| 23                      | R_isthmuscingulate_1                 |     |
| 24                      | R_postcentral_1                      |     |
| 25                      | R_postcentral_2                      |     |
| 26                      | R_supramarginal_1                    |     |
| 27                      | R_supramarginal_2                    |     |
| 28                      | R_superiorparietal_1                 |     |
| 29                      | R_superiorparietal_2                 |     |
| 30                      | R_superiorparietal_3                 |     |
| 31                      | R_inferiorparietal_1                 |     |
| 32                      | R_inferiorparietal_2                 |     |

ICNs: LIM - Limbic, DMN - Default Mode Network, SAL/VAN - Salience/Vanilloid, CONT - Cingulum, DAN - Dorsal Attention Network, VIS - Visual Network.
|   |   |   |   |
|---|---|---|---|
| 33 | R_inferiorparietal_3 | 31 | R_inferiorparietal_3 |
| 34 | R_precuneus_1 | 32 | R_precuneus_1 |
| 35 | R_precuneus_2 | 33 | R_precuneus_2 |
| 36 | R_cuneus_1 | 34 | R_cuneus_1 |
| 37 | R_pericalcarine_1 | 35 | R_pericalcarine_1 |
| 38 | R_lateraloccipital_1 | 36 | R_lateraloccipital_1 |
| 39 | R_lateraloccipital_2 | 37 | R_lateraloccipital_2 |
| 40 | R_lateraloccipital_3 | 38 | R_lateraloccipital_3 |
| 41 | R_lingual_1 | 39 | R_lingual_1 |
| 42 | R_lingual_2 | 40 | R_lingual_2 |
| 43 | R_fusiform_1 | 41 | R_fusiform_1 |
| 44 | R_fusiform_2 | 42 | R_fusiform_2 |
| 45 | R_parahippocampal_1 | 43 | R_parahippocampal_1 |
| 46 | R_entorhinal_1 | 44 | R_entorhinal_1 |
| 47 | R_temporalpole_1 | 45 | R_temporalpole_1 |
| 48 | R_inferiortemporal_1 | 46 | R_inferiortemporal_1 |
| 49 | R_inferiortemporal_2 | 47 | R_inferiortemporal_2 |
| 50 | R_middletemporal_1 | 48 | R_middletemporal_1 |
| 51 | R_middletemporal_2 | 49 | R_middletemporal_2 |
| 52 | R_bankssts_1 | 50 | R_bankssts_1 |
| 53 | R_superiortemporal_1 | 51 | R_superiortemporal_1 |
| 54 | R_superiortemporal_2 | 52 | R_superiortemporal_2 |
| 55 | R_transversetemporal_1 | 53 | R_transversetemporal_1 |
| 56 | R_insula_1 | 54 | R_insula_1 |
| 57 | R_insula_2 | 55 | R_insula_2 |
| 58 | R_thalamusproper | 56 | R_thalamusproper |
| 59 | R_caudate | 57 | R_caudate |
| 60 | R_putamen | 58 | R_putamen |
| 61 | R_pallidum | 59 | R_pallidum |
| 62 | R_accumbensarea | 60 | R_accumbensarea |
| 63 | R_hippocampus | 61 | R_hippocampus |
| 64 | R_amygdala | 62 | R_amygdala |
| 65 | L_lateralorbitofrontal_1 | 63 | L_lateralorbitofrontal_1 |
| 66 | L_lateralorbitofrontal_2 | 64 | L_lateralorbitofrontal_2 |
| 67 | L_parsorbitalis_1 | 65 | L_parsorbitalis_1 |
| 68 | L_frontalpole_1 | 66 | L_frontalpole_1 |
| 69 | L_medialorbitofrontal_1 | 67 | L_medialorbitofrontal_1 |
| 70 | L_parstriangularis_1 | 68 | L_parstriangularis_1 |
| 71 | L_parsopercularis_1 | 69 | L_parsopercularis_1 |

DAN = Default Mode Network, DMN = Default Mode Network, VIS = Visual System, SMN = Salience Network, SAL/VAN = Salience/Ventral Attention Network, SUB = Subcortex
|   | L_rostralmiddlefrontal_1  |   | L_rostralmiddlefrontal_1  | CONT |
|---|--------------------------|---|--------------------------|------|
| 72| L_rostralmiddlefrontal_2  |   | L_rostralmiddlefrontal_2  | CONT |
| 73| L_rostralmiddlefrontal_3  |   | L_rostralmiddlefrontal_3  | DMN  |
| 74| L_superiorfrontal_1       |   | L_superiorfrontal_1       | DMN  |
| 75| L_superiorfrontal_2       |   | L_superiorfrontal_2       | DMN  |
| 76| L_superiorfrontal_3       |   | L_superiorfrontal_3       | DMN  |
| 77| L_superiorfrontal_4       |   | L_superiorfrontal_4       | SAL/VAN |
| 78| L_caudalmiddlefrontal_1   |   | L_caudalmiddlefrontal_1   | DAN  |
| 79| L_precentral_1            |   | L_precentral_1            | SMN  |
| 80| L_precentral_2            |   | L_precentral_2            | SMN  |
| 81| L_precentral_3            |   | L_precentral_3            | SMN  |
| 82| L_precentral_4            |   | L_precentral_4            | SAL/VAN |
| 83| L_paracentral_1           |   | L_paracentral_1           | SMN  |
| 84| L_rostralanteriorcingulate_1 |   | L_rostralanteriorcingulate_1 | DMN |
| 85| L_caudalanteriorcingulate_1 |   | L_caudalanteriorcingulate_1 | SAL/VAN |
| 86| L_posteriorcingulate_1    |   | L_posteriorcingulate_1    | CONT |
| 87| L_isthmuscingulate_1      |   | L_isthmuscingulate_1      | DMN  |
| 88| L_postcentral_1           |   | L_postcentral_1           | SMN  |
| 89| L_postcentral_2           |   | L_postcentral_2           | SMN  |
| 90| L_postcentral_3           |   | L_postcentral_3           | SMN  |
| 91| L_supramarginal_1         |   | L_supramarginal_1         | SAL/VAN |
| 92| L_supramarginal_2         |   | L_supramarginal_2         | CONT |
| 93| L_superioparietal_1       |   | L_superioparietal_1       | DAN  |
| 94| L_superioparietal_2       |   | L_superioparietal_2       | DAN  |
| 95| L_superioparietal_3       |   | L_superioparietal_3       | VIS  |
| 96| L_inferioparietal_1       |   | L_inferioparietal_1       | DAN  |
| 97| L_inferioparietal_2       |   | L_inferioparietal_2       | DMN  |
| 98| L_precuneus_1             |   | L_precuneus_1             | DMN  |
| 99| L_precuneus_2             |   | L_precuneus_2             | DMN  |
|100| L_lineus_1                |   | L_lineus_1                | VIS  |
|101| L_pericalcarine_1         |   | L_pericalcarine_1         | VIS  |
|102| L_lateraloccipital_1      |   | L_lateraloccipital_1      | VIS  |
|103| L_lateraloccipital_2      |   | L_lateraloccipital_2      | VIS  |
|104| L_lingual_1               |   | L_lingual_1               | VIS  |
|105| L_lingual_2               |   | L_lingual_2               | VIS  |
|106| L_fusiform_1              |   | L_fusiform_1              | VIS  |
|107| L_fusiform_2              |   | L_fusiform_2              | LIM  |
|108| L_parahippocampal_1       |   | L_parahippocampal_1       | LIM  |
|109| L_entorhinal_1            |   | L_entorhinal_1            | LIM  |
|110| L_entorhinal_1            |   | L_entorhinal_1            | LIM  |
|   | L_temporalpole_1 |   | L_temporalpole_1 | LIM |
|---|-----------------|---|-----------------|-----|
| 111 | L_inferiortemporal_1 | 105 | L_inferiortemporal_1 | LIM |
| 112 | L_inferiortemporal_2 | 107 | L_inferiortemporal_2 | DAN |
| 113 | L_middletemporal_1 | 108 | L_middletemporal_1 | DMN |
| 114 | L_middletemporal_2 | 109 | L_middletemporal_2 | DMN |
| 115 | L_bankssts_1 | 110 | L_bankssts_1 | DMN |
| 116 | L_superiortemporal_1 | 111 | L_superiortemporal_1 | SMN |
| 117 | L_superiortemporal_2 | 112 | L_superiortemporal_2 | DMN |
| 118 | L_transversetemporal_1 | 113 | L_transversetemporal_1 | SMN |
| 119 | L_insula_1 | 114 | L_insula_1 | SMN |
| 120 | L_insula_2 | 115 | L_insula_2 | SAL/VAN |
| 121 | L_thalamusproper | 116 | L_thalamusproper | SUB |
| 122 | L_caudate | 117 | L_caudate | SUB |
| 123 | L_putamen | 118 | L_putamen | SUB |
| 124 | L_pallidum | 119 | L_pallidum | SUB |
| 125 | L_accumbensarea | 120 | L_accumbensarea | SUB |
| 126 | L_hippocampus | 121 | L_hippocampus | LIM |
| 127 | L_amygdala | 122 | L_amygdala | LIM |
| 128 | brainstem | 123 | brainstem | Not assigned |

* The brainstem is excluded from the analysis due to insufficient coverage in some subjects. Abbreviations: VIS, visual network; SMN, somatomotor network; DAN, dorsal attention network; SAL/VAN, salience/ventral attention network; CONT, executive control network; DMN, default mode network; SUB, subcortical network.
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