

Mapping ACC/ACA-associated circuitry in social motivation: projection strength and excitatory/inhibitory molecular proxy analysis

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Abstract. Social rejection can produce renewed affiliation, withdrawal, impaired self-regulation, and aggression. The Anterior Cingulate Cortex (ACC) has been repeatedly implicated in social rejection and social distress, but the circuit-level organization through which ACC may contribute to social motivational responses remains unclear. We used publicly available Allen Brain Atlas projection and gene-expression resources to examine ACC/Anterior Cingulate Area (ACA)-associated source regions and their excitatory/inhibitory molecular proxy profiles. Ipsilateral projection strength to ACA-related targets, including ACA, ACAd, and ACAv, was analyzed to identify candidate source regions. These regions were integrated with Allen reference gene-expression markers, using *Slc17a7* and *Slc17a6* as excitatory-related proxies and *Slc32a1* as an inhibitory-related proxy. Projection analysis identified a distributed set of candidate ACC/ACA-associated regions, with strong projection patterns from AM, local ACA subdivisions, and additional thalamic, cortical, and basal forebrain-related structures. Molecular proxy analysis showed that these candidate regions differed in their relative excitatory and inhibitory marker-expression profiles. These findings suggest that ACC/ACA is embedded in a distributed and molecularly heterogeneous network that may support the integration of social, affective, and regulatory information. This atlas-based framework provides candidate circuit targets for future studies investigating how ACC/ACA contributes to behavioral responses following social rejection.

Keywords: anterior cingulate cortex, social motivation, social rejection, brain connectivity, Allen Brain Atlas, excitatory/inhibitory balance

1. Introduction

Social motivation is a regulatory process that helps maintain social connection and respond to social deficit. The need to form and maintain social bonds is considered a basic human motivation, and disrupted social connection can affect emotional well-being, behavioral regulation, and interpersonal behavior [1]. Recent neuroscience models propose that social motivation operates through homeostatic mechanisms, in which deficient social contact recruits motivational circuits to restore social connection [2, 3]. Social rejection is a key context for studying this process because ostracism threatens basic social needs and can produce divergent outcomes, including renewed affiliation, withdrawal, impaired self-regulation, hostile cognition, or aggression

[4-7]. These outcomes suggest that rejection-related behavior depends on neural systems that integrate social threat, affective state, and regulatory demands.

The Anterior Cingulate Cortex (ACC) has been repeatedly implicated in social exclusion and social evaluation. Classic Cyberball work showed that social exclusion increased ACC activity and that this activity correlated with self-reported social distress, supporting the "social pain" account of rejection [8]. Subsequent neuroimaging, behavioral, pharmacological, and meta-analytic studies further supported the involvement of ACC-related regions in social rejection and pain-related processing [9-14]. However, later evidence suggests that ACC activity during rejection cannot be interpreted as a simple marker of social pain. Instead, rejection-related ACC activity may also reflect expectancy violation, salience, self-relevance, default-network engagement, or regulatory demand [15-18]. This broader view is consistent with theories implicating ACC in negative affect, cognitive control, effort-based decision-making, control allocation, and value-guided action [19-21]. Thus, ACC activity during rejection may reflect the integration of affective, motivational, and regulatory information rather than a single distress signal.

The anatomical and molecular organization of ACC-related circuitry remains unclear, limiting our understanding of how this system may support different behavioral responses to rejection. To address this gap, the present study used publicly available Allen Brain Atlas projection and gene-expression resources to examine ACC/anterior cingulate area (ACA)-associated circuitry [22, 23]. We analyzed projection strength to ACA-related targets, including ACA, ACAd, and ACAv, and integrated candidate source regions with Allen reference gene-expression markers. *Slc17a7* and *Slc17a6* were used as excitatory-related proxies, and *Slc32a1* was used as an inhibitory-related proxy. Because this study links human social-rejection theories with mouse atlas-derived anatomical and molecular resources, the analysis is intended as a translational, hypothesis-generating framework rather than direct behavioral evidence. By combining projection-strength analysis with marker-expression-based molecular proxies, this study provides an anatomical and molecular framework for understanding how ACC/ACA-associated circuitry may integrate social, affective, and regulatory information.

2. Methods

2.1. Data sources and study overview

This study used publicly available Allen Brain Atlas image, projection, and gene-expression resources to examine ACC/ACA-associated circuitry. Projection-related fluorescence images and projection-strength data were obtained from the Allen Mouse Brain Connectivity Atlas, a mesoscale viral-tracing and whole-brain imaging resource [23, 24]. Allen adult mouse gene-expression data were used for marker-expression-based molecular proxy analysis [22], and the Allen Mouse Brain Common Coordinate Framework served as the anatomical reference space [25].

For image processing, one representative Allen coronal image series containing ACC/ACA-associated fluorescent signal was selected for atlas-based regional analysis. This series included 139 raw coronal sections and was used to document the QuickNII-ilastik-Nutli workflow for segmentation and quantification. Projection analyses used Allen Brain Map API-derived metadata, including 2,408 non-failed Allen Mouse Connectivity SectionDataSet IDs after filtering relevant Mouse Connectivity products. Projection-strength values were extracted from ProjectionStructureUnionize records joined with experiment metadata, and `projection_energy` was used as the anatomical projection-strength metric.

The analysis included three linked components: atlas-based fluorescence signal quantification, ipsilateral projection-strength analysis to ACA-related targets, and integration of candidate ACC/ACA-associated source regions with Allen gene-expression markers to estimate excitatory/inhibitory molecular proxy profiles.

2.2. Atlas registration and fluorescent signal segmentation

Raw coronal fluorescence image series were registered to the Allen Mouse Brain Atlas using the QuickNII/QUINT workflow [26]. QuickNII aligned each section to corresponding atlas planes for region-based quantification, and registration quality was visually inspected using major anatomical landmarks, section orientation, and anterior-posterior position. Sections with poor alignment, severe tissue distortion, incomplete tissue coverage, or ambiguous anatomical correspondence were corrected when possible or excluded. The Figure 1/T1 series retained 139 registered sections for downstream segmentation and quantification.

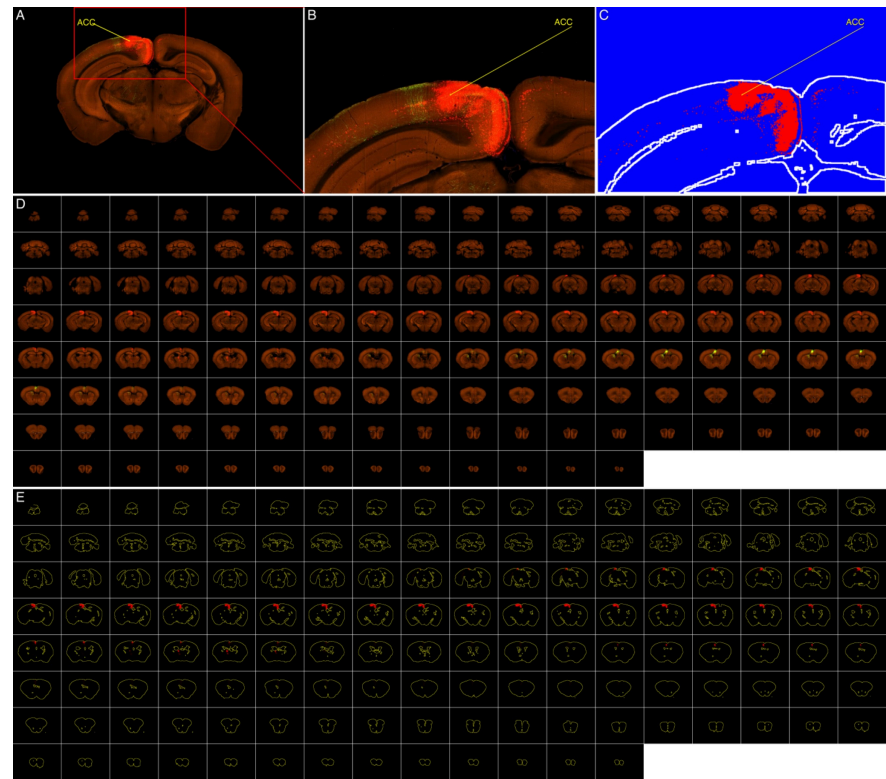


Figure 1. Segmentation and anatomical-context visualization of ACC/ACA-associated fluorescent signal for atlas-based quantification

Notes: (A) Representative raw coronal section showing ACC/ACA-associated fluorescent labeling. The red box indicates the magnified region shown in panel B, and the arrow marks the approximate ACC location. (B) Magnified medial cortical region showing fluorescent labeling near the ACC/ACA region. (C) ilastik segmentation example with an anatomical boundary derived from the corresponding raw image to show the relative tissue structure. Red indicates tracer-positive signal, and blue indicates background. (D) Raw coronal image series montage used for atlas-based regional analysis. (E) Corresponding segmentation-mask montage with anatomical boundaries derived from the raw image series, allowing the segmented signal to be interpreted relative to brain-section structure.

Fluorescent tracer-positive signal was segmented from background using an ilastik-based pixel-classification workflow [27]. Signal-positive and signal-negative pixels were manually labeled on representative sections to train the classifier, which was then applied across the image series to generate section-level segmentation masks. Segmentation quality was visually inspected by comparing raw fluorescence images with corresponding masks, and sections with poor signal-background separation, excessive background classification, or tissue artifacts were manually checked before downstream processing.

The ilastik output shown in Figure 1 represents the signal-extraction stage rather than final regional quantification or molecular E/I analysis.

2.3. Regional quantification using Nutil

QuickNII registration files and ilastik segmentation masks were processed with Nutil as part of the QUINT workflow for atlas-based quantification of labeled signal in rodent brain sections [28, 29]. Nutil assigned segmented tracer-positive signal to Allen atlas-defined brain regions and generated regional measures, including object count, object pixels, object area, and load, which were summarized by Allen structure acronym and anatomical hierarchy. Weak, ambiguous, fiber-like, or artifact-prone signal was interpreted cautiously and was not treated as definitive anatomical input without supporting projection evidence. This step summarized the anatomical distribution of segmented fluorescent signal but was not used to calculate excitatory/inhibitory molecular proxy values.

2.4. Analysis of ipsilateral projections to ACA-related targets

Ipsilateral projection patterns to ACA-related targets were analyzed using Allen Mouse Brain Connectivity Atlas projection data [23, 24]. ACA-related targets were defined by Allen atlas labels and included ACA, ACAd, and ACAv. The primary analysis focused on ipsilateral entries, while contralateral outputs were retained for supplementary comparison. Projection entries were filtered to include non-failed Allen Mouse Connectivity SectionDataSet IDs, and side-specific projection matrices were generated from non-injection target rows with lateralized hemisphere assignments. Injection hemisphere was inferred from hemisphere-specific injection unionize rows, targets in the same hemisphere were classified as ipsilateral, and non-lateralized summary rows were excluded.

Projection entries were grouped by injection structure acronym and ACA-related target region. Mean projection_energy across experiments was used as the projection-strength metric, and projection strength across ACA, ACAd, and ACAv was averaged to rank candidate ACA-projecting regions. The top 10 regions were visualized with horizontal and target-specific grouped bar plots, while broader ipsilateral projection organization was shown with a top 50 heatmap. Because ACAv and ACAd can also appear as injection structures, ACA-related injection sites were interpreted as local or intra-ACA projection patterns rather than extra-ACA source regions.

2.5. Allen reference excitatory and inhibitory proxy mapping

Allen adult mouse gene-expression data were used to generate excitatory and inhibitory molecular proxy maps across atlas-defined brain regions [22]. Excitatory-related signal was estimated using Slc17a7 and Slc17a6, corresponding to VGLUT1- and VGLUT2-associated expression [30], and inhibitory-related signal was estimated using Slc32a1, corresponding to VGAT-associated GABAergic expression [31]. Excitatory and inhibitory proxies were calculated as

$$E_{proxy} = Slc17a7_{expression} + Slc17a6_{expression} \quad (1)$$

$$I_{proxy} = Slc32a1_{expression} \quad (2)$$

Marker-expression values were mapped onto Allen atlas-defined coronal sections and visualized across twelve representative anterior-posterior levels. Stabilized E/I and I/E log-ratio maps were calculated using a pseudocount-based approach to reduce instability from low denominator values:

$$stabilized_EI_logratio = \log_2 \frac{E_{proxy} + \alpha}{I_{proxy} + \alpha} \quad (3)$$

$$\text{stabilized_IE_logratio} = \log_2 \frac{I_{\text{proxy}} + \alpha}{E_{\text{proxy}} + \alpha} \quad (4)$$

where $\alpha = 3.2859$. Display values were clipped to $[-3, 3]$, and low-signal or invalid regions were masked to avoid artificially inflated ratios. These values were used as marker-expression-based molecular proxies and not directly measure synaptic excitation, synaptic inhibition, cell-type proportions, or neural activity.

2.6. Integrated analysis of ACC/ACA projection strength and excitatory/inhibitory molecular proxies

Candidate ACC/ACA-associated source regions were identified from Allen projection data and matched to Allen reference gene-expression data using atlas structure labels. For each source region, projection strength to ACA, ACAd, and ACAv was averaged to generate a combined ACC/ACA projection measure, and source regions were ranked by combined mean projection strength. Projection-ranked regions were matched to gene-expression records by exact structure acronym when available; otherwise, parent-level values were estimated by aggregating matched child or layer-specific entries where appropriate. Regions without valid matches were marked as missing rather than estimated from unrelated structures.

Excitatory and inhibitory molecular proxies were calculated as described above, and stabilized E/I values were used to classify regions as excitation-leaning (> 0.25), inhibition-leaning (< -0.25), or relatively balanced (-0.25 to 0.25). Integrated visualizations compared anatomical projection strength with molecular proxy profiles. Combined projection strength was log-transformed using $\log_{1p}$ for scatter plot visualization, and heatmap features were normalized by column-wise z-scoring to compare projection-strength measures, marker-expression values, E/I proxy values, I/E proxy values, and stabilized E/I log-ratios across candidate source regions. Projection-strength, fluorescence-segmentation, and molecular-proxy analyses were interpreted as related but distinct layers of evidence.

2.7. Data processing and visualization

All quantitative summaries and visualizations were generated from Allen-derived projection, segmentation, or marker-expression outputs using Python-based analysis scripts. Data processing used pandas and NumPy, and figures were generated using matplotlib and seaborn. Some image validation and image-size checks used PIL/Pillow. Projection-strength plots were used to rank candidate source regions, grouped bar plots were used to compare target-specific projection patterns across ACA, ACAd, and ACAv, and heatmaps were used to visualize projection and marker-expression features across candidate regions. Scatter plots were used to compare log-transformed projection strength with stabilized E/I proxy values. These analyses were descriptive and exploratory, and no causal or behavioral inference was made from projection strength or marker-expression proxy values alone.

3. Results

Because ACC/ACA-related circuitry has been implicated in social motivation and behavioral responses to social rejection, the first goal of the analysis was to characterize the anatomical organization of regions associated with ACC/ACA signal. Specifically, we aimed to identify where ACC/ACA-associated input-related signals were distributed across the brain and to connect this anatomical information with projection-strength and molecular-proxy analyses. To do this, we first examined an Allen-derived coronal fluorescence image series containing ACC/ACA-associated tracer signal and processed it through an atlas-based segmentation and quantification workflow. This image-based analysis provided a visual and anatomical

reference for identifying tracer-positive signals across sections before integrating projection and gene-expression data.

ACC/ACA-associated fluorescent signal was extracted from the representative Allen coronal image series using an ilastik-based segmentation workflow and was then prepared for atlas-based regional quantification (Figure 1). The raw image showed prominent tracer-associated labeling in the medial cortical region corresponding approximately to ACC/ACA, and the magnified panel highlighted the local distribution of fluorescent signal near the annotated ACC region (Figure 1A–B). The segmentation example separated tracer-positive signal from background, and the added anatomical boundary helped show the position of segmented signal relative to the surrounding tissue structure (Figure 1C). Across the full coronal image series, the raw montage showed that tracer-associated signal was not confined to a single section but appeared across multiple anterior-posterior levels (Figure 1D). The corresponding segmentation-mask montage preserved the spatial pattern of tracer-positive signal while adding anatomical boundaries derived from the raw image series, making the segmented signal easier to interpret relative to brain-section structure (Figure 1E). These image-derived results supported downstream regional quantification, but segmentation alone was interpreted cautiously and was not treated as direct evidence of excitatory/inhibitory molecular profile or functional connectivity.

After establishing the image-based anatomical reference, we next used Allen Mouse Brain Connectivity Atlas projection data to examine ipsilateral projection strength to ACA-related targets. This analysis focused on ACA, ACAd, and ACAv because these atlas-defined targets correspond to the ACC/ACA-related region of interest in the present study. Projection data were extracted from Allen connectivity records, filtered to non-failed Mouse Connectivity experiments, organized by injection structure and target region, and summarized using mean projection_energy as the projection-strength metric. The purpose of this step was to identify candidate source regions with strong anatomical projection relationships to ACA-related targets, with primary emphasis on ipsilateral projections.

Ipsilateral projection analysis identified a distributed set of candidate regions projecting to ACA-related targets rather than a single dominant anatomical source (Figure 2).

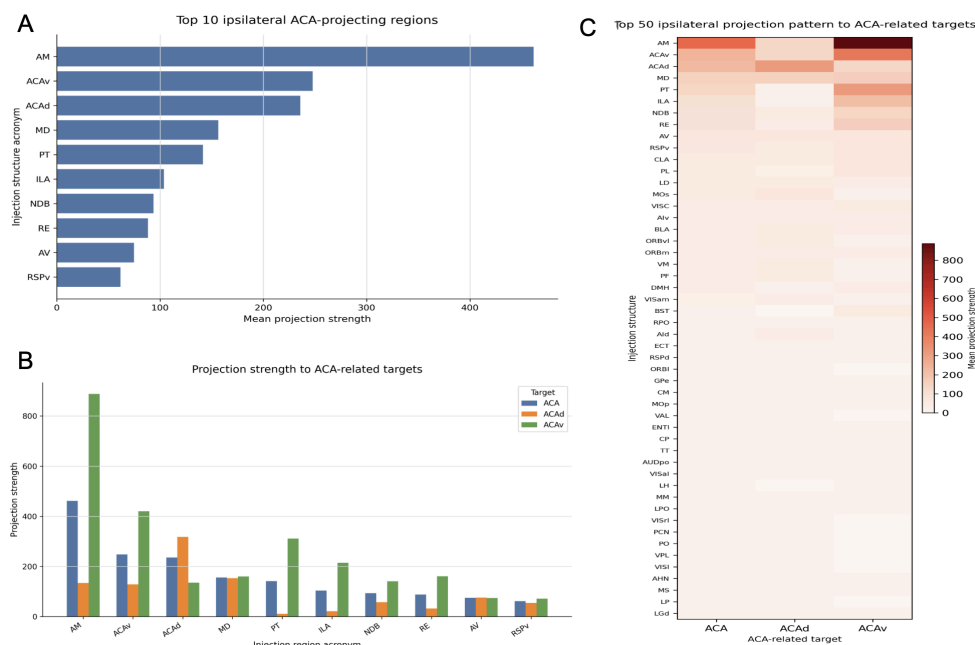


Figure 2. Ipsilateral projection strength to ACA-related target regions

Notes: (A) Top 10 ipsilateral ACA-projecting regions ranked by mean projection strength. Injection regions are shown on the y-axis, and mean projection strength is shown on the x-axis. AM showed the strongest projection strength, followed by local ACA-related regions and additional thalamic, cortical, and basal forebrain-related structures. (B) Projection strength from the top 10 injection regions separated by ACA-related target subdivision, including ACA, ACAd, and ACAv. AM showed especially strong projection to ACAv, while local ACA subdivisions showed strong intra-ACA projection patterns. (C) Heatmap of the top 50 ipsilateral projection patterns to ACA-related targets. Rows represent injection structures, columns represent ACA-related targets, and darker red indicates stronger mean projection strength.

AM showed the strongest mean ipsilateral projection strength, followed by local ACA subdivisions and additional thalamic, cortical, and basal forebrain-related structures, including ACAv, ACAd, MD, PT, ILA, NDB, RE, AV, and RSPv (Figure 2A). Target-specific analysis showed that projection strength varied across ACA subdivisions. AM projected especially strongly to ACAv, whereas local ACA-related injection structures showed strong projection patterns within ACA-related subdivisions, consistent with local or intra-ACA connectivity rather than exclusively long-range input (Figure 2B). The top 50 ipsilateral projection heatmap further showed that projection strength was unevenly distributed, with a small group of high-ranking regions showing stronger projection values and many additional regions showing weaker projection patterns (Figure 2C). Together, these results suggest that ACA-related targets receive anatomically heterogeneous ipsilateral input from thalamic, cortical, and basal forebrain-related sources.

To examine whether candidate ACC/ACA-associated source regions also differed in molecular profile, we next analyzed Allen adult mouse gene-expression data using excitatory- and inhibitory-related marker-expression proxies. Slc17a7 and Slc17a6 were used to estimate excitatory-related proxy signals, whereas Slc32a1 was used to estimate inhibitory-related proxy signals. These marker-expression values were mapped across atlas-defined coronal sections and used to calculate stabilized E/I and reciprocal I/E log-ratio maps. This analysis was intended to provide a reference-level molecular proxy layer, not a direct measure of synaptic excitation, inhibition, cell-type composition, or neural activity.

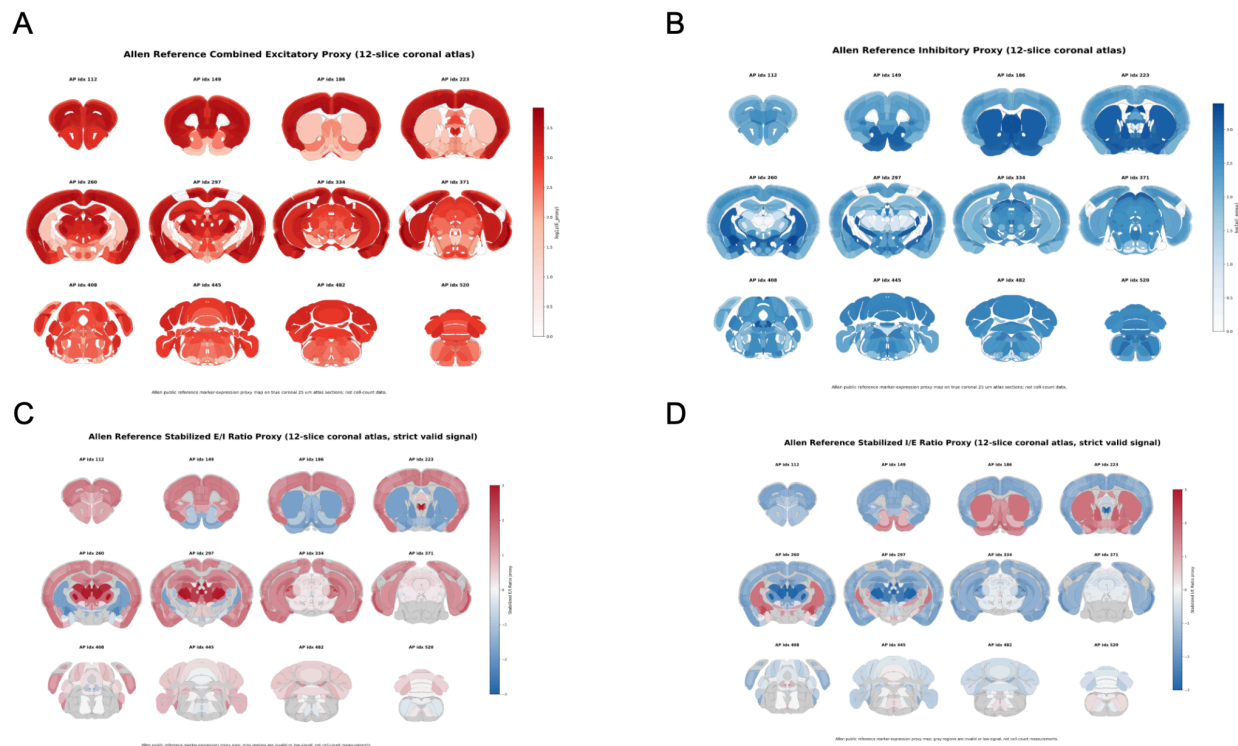


Figure 3. Allen reference excitatory and inhibitory marker-expression proxy maps

Notes: (A) Twelve representative coronal atlas sections showing the combined excitatory proxy generated from Slc17a7 and Slc17a6 expression. Warmer red intensity indicates higher excitatory-related proxy signal. (B) Corresponding inhibitory proxy map generated from Slc32a1 expression. Blue intensity indicates higher inhibitory-related proxy signal. (C) Stabilized excitatory/inhibitory (E/I) ratio proxy map. Red indicates relatively higher excitatory proxy values, blue indicates relatively higher inhibitory proxy values, and gray indicates low-signal or invalid regions masked to reduce unstable ratio interpretation. (D) Stabilized inhibitory/excitatory (I/E) ratio proxy map, showing the reciprocal directional comparison.

Allen reference excitatory and inhibitory marker-expression proxy maps showed distinct spatial distributions across coronal atlas sections (Figure 3).

The combined excitatory proxy map generated from Slc17a7 and Slc17a6 expression showed broad regional variation in glutamatergic marker-associated signal (Figure 3A). The inhibitory proxy map generated from Slc32a1 expression showed a different spatial pattern, indicating that excitatory- and inhibitory-related marker expression were not uniformly distributed across the same anatomical regions (Figure 3B). Stabilized E/I and reciprocal I/E ratio maps further highlighted regional differences in relative marker-expression profiles, while low-signal or invalid regions were masked to reduce unstable ratio interpretation (Figure 3C–D). These results provided the molecular-proxy framework used to interpret candidate ACC/ACA-associated source regions identified from the projection analysis.

Finally, projection-strength and molecular-proxy information were integrated to examine whether anatomically strong candidate source regions differed in their relative excitatory/inhibitory marker-expression profiles. Candidate source regions were ranked by combined mean projection strength to ACA, ACAd, and ACAv, then matched to Allen gene-expression records by atlas structure labels. When exact source-region matches were not available, parent-level or layer-aggregated values were used where appropriate, and unmatched regions were treated as missing rather than inferred from unrelated structures. This integrated analysis allowed anatomical projection strength and marker-expression proxy values to be compared across the same candidate ACC/ACA-associated source regions.

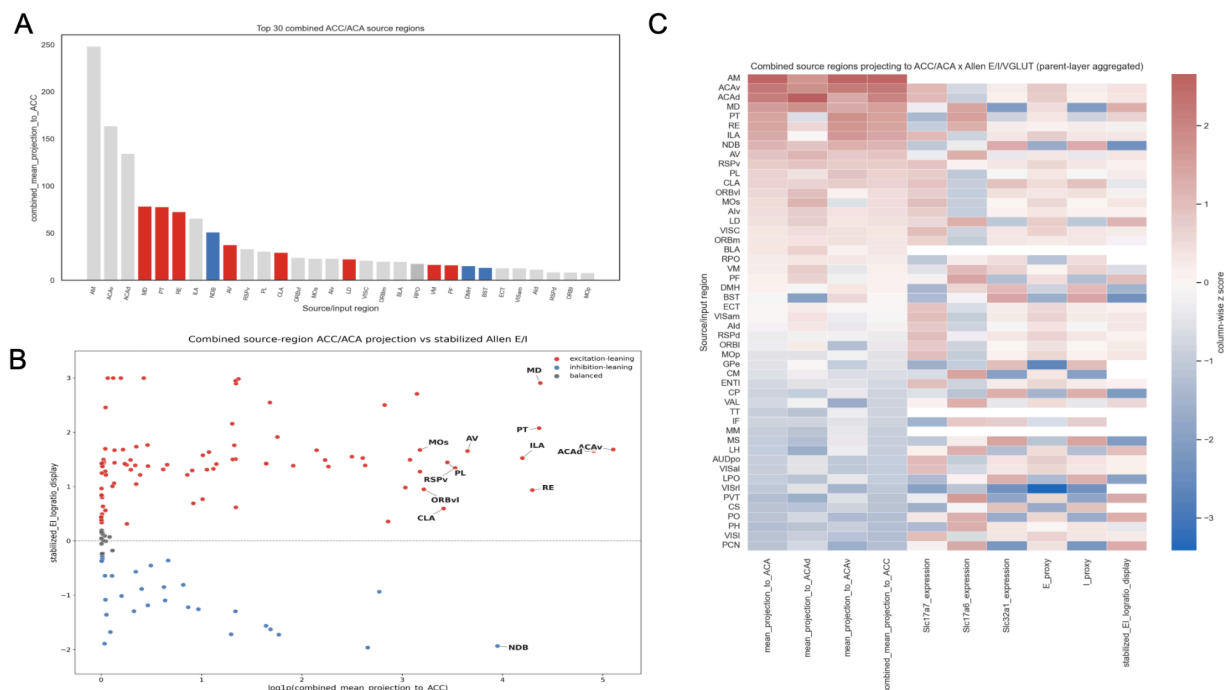


Figure 4. Integrated projection strength and excitatory/inhibitory molecular proxy analysis of ACC/ACA-associated source regions

Notes: (A) Top 30 candidate source regions ranked by combined mean projection strength to ACA, ACAd, and ACAv. AM showed the strongest combined projection signal, followed by local ACA-related regions and additional thalamic, cortical, and basal forebrain-related structures. (B) Scatter plot comparing log-transformed combined projection strength with stabilized E/I proxy values across source regions. Red indicates excitation-leaning regions, blue indicates inhibition-leaning regions, and gray indicates relatively balanced regions. (C) Heatmap integrating projection-strength measures, Allen marker-expression values, and E/I proxy features across candidate source regions. Values are displayed as column-wise z scores, with red indicating relatively higher values and blue indicating relatively lower values within each feature.

The integrated analysis again identified AM as the strongest combined candidate source region, followed by local ACA-related regions and additional thalamic, cortical, and basal forebrain-related structures, including MD, PT, RE, ILA, NDB, AV, and RSPv (Figure 4A). The scatter plot comparing log-transformed combined projection strength with stabilized E/I proxy values showed that candidate source regions varied along both dimensions: some regions had relatively strong projection strength and excitation-leaning proxy values, whereas others showed weaker projection strength, inhibition-leaning profiles, or relatively balanced E/I proxy values (Figure 4B). The integrated heatmap further showed heterogeneity across projection-strength measures, *Slc17a7* and *Slc17a6* expression, *Slc32a1* expression, E/I proxy values, I/E proxy values, and stabilized E/I log-ratios (Figure 4C). Overall, these results suggest that ACC/ACA-associated circuitry is distributed, unevenly weighted, and molecularly heterogeneous, supporting the interpretation that ACC/ACA may integrate information from multiple anatomical and molecularly distinct source systems.

4. Discussion

The present study used publicly available Allen Brain Atlas projection and gene-expression resources to examine ACC/ACA-associated circuitry as an anatomical and molecular framework for social motivation and behavioral responses to social rejection. ACA-related targets received projections from a distributed set of cortical, thalamic, and basal forebrain-related regions, and these candidate source regions differed in excitatory/inhibitory marker-expression proxy profiles. Together, these findings suggest that ACC/ACA-related circuitry is not organized around a single input source or molecular profile, but instead reflects a heterogeneous network that may support the integration of affective, motivational, and regulatory information.

Projection analysis identified AM as the strongest ipsilateral projecting region to ACA-related targets, especially ACAv, while local ACA-related regions likely reflected intra-ACA connectivity rather than long-range extra-ACA input. Additional candidate source regions included MD, PT, RE, ILA, NDB, AV, and RSPv. This distributed projection pattern is consistent with broader theories of cingulate and frontal cortical function, which emphasize roles in affective integration, cognitive control, effort-based decision-making, control allocation, value-guided behavior, social cognition, and adaptive action selection [19-21, 32-36]. Rather than functioning as an isolated "social pain" region, ACC/ACA may operate as an integrative circuit node that combines thalamic, cortical, and modulatory signals.

This interpretation may help explain why ACC activity during social rejection has been difficult to assign to a single psychological process. Classic and subsequent work linked ACC activity to social distress and supported the social pain account of rejection [8, 11, 12]. However, later work suggests that rejection-related ACC activity may also reflect salience, expectancy violation, self-relevance, default-network engagement, or regulatory demand rather than pain-like distress alone [15-18]. The present atlas-based findings fit this broader interpretation: convergent input from thalamic, prefrontal, cortical, and basal forebrain-related regions may allow ACC/ACA to integrate multiple social and motivational signals rather than encode a single distress marker.

The molecular proxy analysis added a second interpretive layer by showing that candidate ACC/ACA-associated source regions differed in relative excitatory and inhibitory marker-expression profiles. Some regions were excitation-leaning, whereas others were inhibition-leaning or relatively balanced, suggesting that projection strength alone is insufficient to describe ACC/ACA circuit organization. Regions with comparable anatomical projection strength may differ in molecular profile and may therefore contribute differently to ACC/ACA processing. These findings may also be relevant to divergent behavioral outcomes after rejection, including renewed affiliation, withdrawal, aggression, impaired self-regulation, or broader mental health risk [4-6, 37, 38]. Although indirect, the combined projection and molecular-proxy framework provides testable hypotheses about how different ACC/ACA-associated input systems may contribute to social motivation.

Several limitations should be considered. This study used publicly available atlas, projection, and gene-expression datasets rather than newly collected behavioral or physiological data, so the results cannot establish causal function. Allen projection-strength values reflect anatomical tracing and quantification methods rather than direct functional synaptic strength, and local ACA-related projection patterns may reflect neighboring or intra-ACA connectivity. In addition, E/I values were based on marker-expression proxies, including *Slc17a7*, *Slc17a6*, and *Slc32a1*, and do not directly measure synaptic excitation, inhibition, cell-type proportions, or neural activity. Finally, because the theoretical framing draws partly from human social rejection literature whereas the atlas analyses are based on mouse brain resources, cross-species interpretation should remain hypothesis-generating rather than definitive [39, 40]. Future studies using behavioral models, cell-type-specific tracing, electrophysiology, calcium imaging, optogenetic or chemogenetic manipulation, and spatial transcriptomics could directly test whether candidate regions such as AM, MD, ILA, NDB, or RSPv causally influence ACC/ACA activity during social approach, withdrawal, or aggression.

5. Conclusion

In summary, this study suggests that ACC/ACA-related targets are embedded in a distributed and molecularly heterogeneous set of source regions. By combining projection-strength analysis with excitatory/inhibitory marker-expression proxies, this work provides a compact circuit-level framework for understanding how ACC/ACA-associated circuitry may contribute to social motivation and behavioral responses to rejection.

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