Title: Toward a Multimodal Neuroscience of Personality and Aggression: Integrating EEG, fMRI, and Computational Biomarkers of Externalizing Psychopathology

Authors: Helia Hajihassani[1], Parmis Barazesh[1], Nastaran Dadashi[1], Bita Amiri Havazeh[1], Mohammad Sadegh Fallahi[2], Romina Hajihassani[3], Aryana Avaei[4], Amir Ghaffari Jolfayi[5], Maedeh Jorfi[1], Mobina Salehi[1], Mohammad Reza Bigdeli[1,*]

1. *Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran.*
2. *Department of Neurosurgery, Tehran University of Medical Sciences, Tehran, Iran.*
3. *Faculty of Biological Sciences, University of Tehran, Tehran, Iran.*
4. *Department of Bioengineering, Imperial College London, London, UK.*
5. *Student Research Committee, Shahid Beheshti University of Medical Sciences, Tehran, Iran.*

***Corresponding Author**: Mohammad Reza Bigdeli, Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran. Email: bigdelimohammadreza@yahoo.com

To appear in: **Basic and Clinical Neuroscience**

Received date: 2026/04/28

Revised date: 2026/05/25

Accepted date: 2026/05/31

Please cite this article as:

Hajihassani, H., Barazesh, P., Dadashi, N., Amiri Havazeh, B., Fallahi, M.S., Hajihassani, R., et al. (In Press). Toward a Multimodal Neuroscience of Personality and Aggression: Integrating EEG, fMRI, and Computational Biomarkers of Externalizing Psychopathology. Basic and Clinical Neuroscience. Just Accepted publication Jul. 10, 2026. Doi: http://dx.doi.org/10.32598/bcn.2026.8578.1

DOI: http://dx.doi.org/10.32598/bcn.2026.8578.1

Abstract

Background: Personality traits associated with aggression and impulsivity have been consistently linked to externalizing behaviors and underlying neural differences. EEG and fMRI studies suggest that these traits are associated with altered activity and connectivity in emotion- and control-related brain networks. However, existing findings are heterogeneous across methods, tasks, and populations, limiting direct comparability.

Methodology: This paper presents a structured review of EEG and fMRI studies published between 2020 and 2026 that examine the neural correlates of aggressive personality traits. Literature was identified through targeted searches in PubMed, Scopus, and Web of Science using combinations of keywords related to aggression, personality traits, EEG, and fMRI. Studies were selected according to predefined inclusion criteria, including human neuroimaging studies focused on aggression-related personality traits, impulsivity, or externalizing behaviors. Eligible studies were grouped by imaging modality, experimental paradigm, population characteristics, and functional networks. The synthesis focuses on identifying convergent patterns across studies while distinguishing well-replicated findings from preliminary or inconsistent evidence

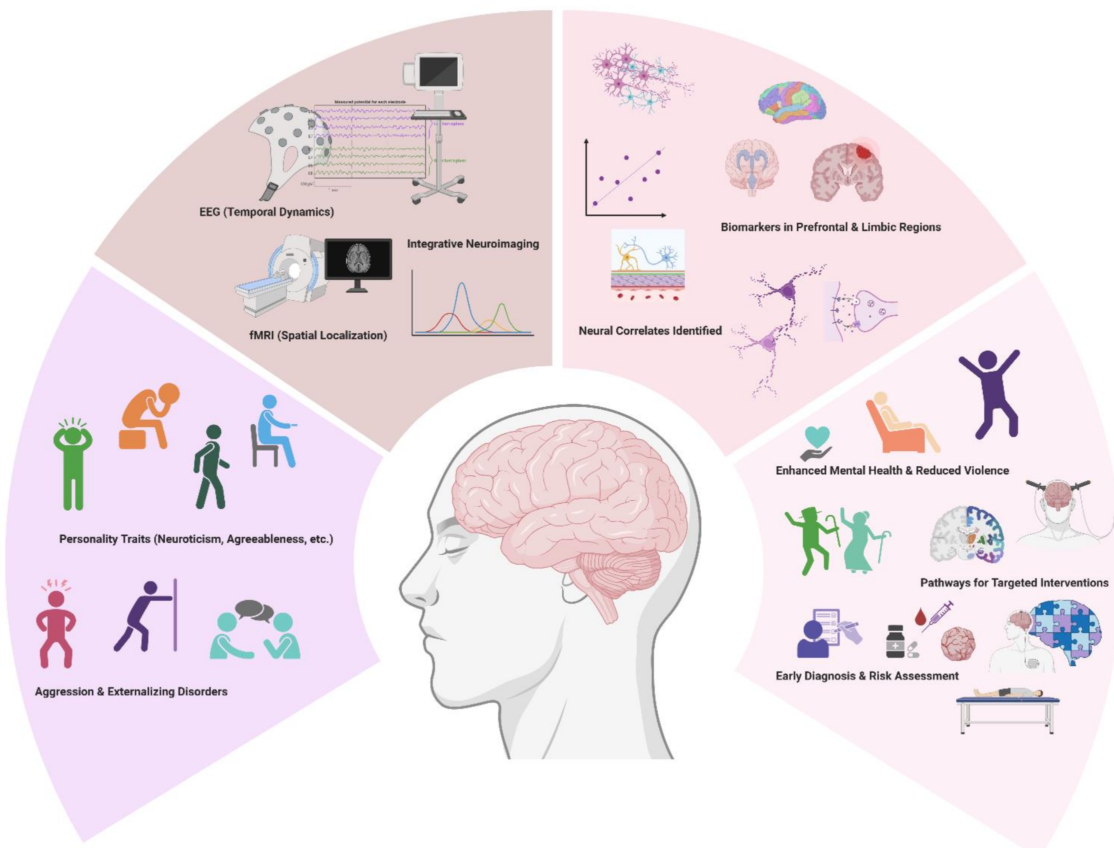
Results: Evidence across studies suggests that individuals with higher levels of aggressive traits show altered activity in prefrontal and limbic regions. EEG findings commonly report atypical frontal alpha asymmetry and alterations in the theta band, particularly in emotionally salient contexts. fMRI studies indicate disrupted functional connectivity within the amygdala–prefrontal circuitry and altered interactions across salience, default mode, and frontoparietal control networks. Despite these convergent patterns, variability in experimental paradigms, sample characteristics, and analytical approaches contributes to inconsistent findings across studies.

Conclusions: Aggression-related personality traits are associated with distributed neural alterations involving emotion regulation and cognitive control networks. While EEG and fMRI provide complementary insights into these mechanisms, current evidence is insufficient to establish robust clinical biomarkers. Future research should prioritize longitudinal designs, methodological standardization, and multimodal integration to improve interpretability and translational potential.

Keywords: Aggression, Personality Traits, EEG, fMRI, Functional Connectivity, Emotion Regulation, Neuroimaging

Graphical Abstract

Schematic overview of the neural mechanisms associated with aggression-related personality traits and externalizing behaviors. The graphical abstract illustrates the integration of EEG and fMRI findings, highlighting altered temporal dynamics, atypical frontal alpha asymmetry, theta-band alterations, disrupted prefrontal–limbic connectivity, and dysfunction across salience, default mode, and frontoparietal control networks. These multimodal neuroimaging findings collectively suggest impaired emotion regulation and cognitive control processes that may contribute to aggressive and impulsive behaviors. The figure also summarizes the potential translational implications of these neural markers for early risk assessment, targeted interventions, and biomarker development in aggression-related psychopathology. Created using BioRender.com.



1. Introduction

In recent years, the intersection of neuroimaging and personality research has revealed critical insights into the neural substrates of aggression and externalizing disorders. This paper synthesizes EEG and fMRI evidence on the neural correlates of aggression and impulsivity across clinical and subclinical populations, examining candidate neural markers and network-level mechanisms relevant to externalizing risk. Rather than providing a purely descriptive overview, it integrates dimensional personality models with multimodal neuroimaging findings to construct a network-level perspective on reactive and proactive aggression, while acknowledging methodological heterogeneity across tasks, samples, and analytic approaches.

1.1. Understanding Aggression and Externalizing Psychopathology

Aggression and violence remain significant global health concerns, contributing to psychological morbidity, healthcare costs, and societal disruption. Understanding their multifaceted origins requires an integrated approach, especially considering neural and psychological mechanisms (1). At the neurobiological level, research suggests that aggressive behavior is associated with altered connectivity between brain systems responsible for emotional salience and cognitive control, dysregulated limbic-prefrontal circuits, and stress-related and neurotransmitter stress and neurotransmitter systems influencing threat perception and impulse control (2). Externalizing liability encompasses a broad spectrum of disinhibition, including aggression, antisocial behavior, rule-breaking, and impulsivity. While substance misuse frequently co-occurs within this spectrum, emerging dimensional models distinguish shared liability factors from disorder-specific mechanisms (3). Importantly, contemporary models differentiate reactive aggression (impulsive, threat-driven, affectively charged) from proactive aggression (instrumental, goal-directed, and less emotionally reactive), as these subtypes are associated with partially dissociable neural circuits involving limbic reactivity and prefrontal regulatory control (4, 5).

Thus, comprehensive neuroimaging reviews are critical for clarifying their heterogeneous neural correlates and identifying candidate neural markers and mechanistic targets. Aggression frequently co-occurs with personality traits such as impulsivity and low agreeableness, assessed via validated models like the Big Five (6). Framing aggression within dimensional personality constructs allows for a transdiagnostic perspective that links trait-level antagonism and disinhibition to large-scale brain network dysfunction rather than to categorical diagnoses alone.

1.2. Personality Models and Their Links to Aggressive Behavior

Personality comprises emotional, cognitive, and conceptual systems that shape how a person reacts to the world. The Five Factor Model (FFM) encompasses extraversion, agreeableness, conscientiousness, neuroticism, and openness. Notably, the predictive relevance of personality traits appears to be driven not by broad domains but by facet-level nuances, as reported across multiple independent samples. Item-level facets reflecting antagonism, impulsive sensation-seeking, and affective dysregulation are more strongly associated with aggression and externalizing behavior across independent samples. This convergence across studies suggests that personality facets provide a more biologically and behaviorally meaningful framework for linking individual differences to neural markers of aggression than traditional domain-level models (7-9). Importantly, these facets appear to align more closely with neural systems implicated in threat reactivity, reward sensitivity, and cognitive control, thereby facilitating circuit-level interpretations of personality–aggression associations.

Studies indicate that personality traits are modest but significant predictors of violence and aggression. Low agreeableness and conscientiousness, alongside high neuroticism, have been associated with elevated levels of aggression across populations At the neural level, low agreeableness and antagonism have been associated with altered default mode and salience network connectivity. In contrast, high neuroticism is frequently linked to heightened limbic reactivity and reduced prefrontal regulatory engagement—patterns relevant to reactive forms of aggression (10).

Eysenck's PEN model (psychoticism, extraversion, neuroticism) links high psychoticism to violent crime and recidivism (11). As summarized conceptually in Figure 1, major personality dimensions may be mapped onto broad neural systems involved in emotional reactivity, inhibitory control, and social-cognitive processing. Boduszek et al. found that elevated psychoticism in violent recidivists predicted criminal cognition with substantial explanatory value using EPQ-R scores (12-14). From a neurobiological perspective, psychoticism has been conceptually associated with reduced frontotemporal integration and atypical prefrontal modulation of affective impulses, providing a theoretical bridge between trait-level disinhibition and network-level dysfunction observed in externalizing populations. Together, dimensional

(FFM) and trait-biological (PEN) frameworks converge in suggesting that aggression-related personality features are best understood as distributed network phenomena rather than localized brain abnormalities.

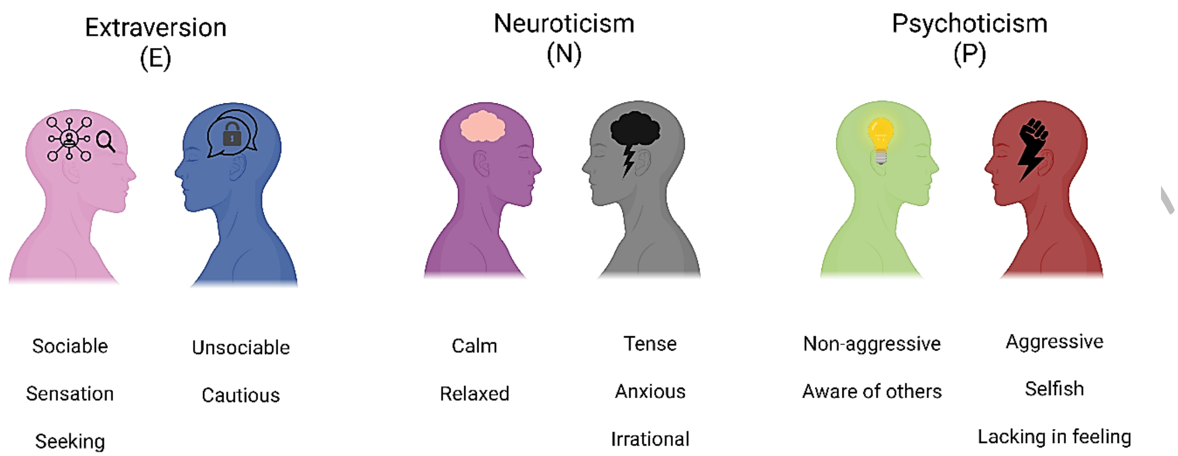


Figure 1. Conceptual representation of Eysenck's PEN personality dimensions and their putative associations with large-scale neural systems involved in emotional reactivity, social cognition, reward processing, and cognitive control. The figure summarizes how psychoticism, extraversion, and neuroticism may relate to distributed patterns of limbic activity, prefrontal regulation, and socioemotional processing relevant to aggression and externalizing behavior. The illustration is intended as a theoretical neurobiological framework rather than a direct anatomical mapping of specific circuits and was created with BioRender.com**.**

1.3. The Neurobiological Foundations: EEG and fMRI in Personality Research

Advances in neuroimaging enable non-invasive mapping of brain activity linked to personality and aggression. EEG captures millisecond-scale neural processes critical for rapid emotional and cognitive responses, while fMRI enables precise localization of brain activity during complex behaviors. Rather than indexing isolated regional activation, contemporary research increasingly conceptualizes personality–aggression associations as network-level phenomena observable across complementary temporal and spatial scales, with EEG characterizing oscillatory dynamics (e.g., alpha asymmetry, theta modulation) and event-related components associated with inhibitory control and affective reactivity, which are particularly relevant to impulsive and reactive aggression. In contrast, fMRI delineates functional connectivity within and between large-scale systems—including salience, frontoparietal control, and default mode networks—that are implicated in trait antagonism, disinhibition, and emotional dysregulation.

Because EEG reflects electrical field potentials that are subject to source-localization ambiguity, and fMRI relies on indirect hemodynamic (BOLD) coupling, convergent interpretation across modalities may strengthen mechanistic inference. Accordingly, integrating temporal electrophysiological markers with spatial connectivity patterns may enable a more comprehensive characterization of aggression-related network dysfunction. These complementary characteristics provide the rationale for the multimodal framework adopted in this paper (1, 15).

This paper synthesizes EEG oscillatory markers and fMRI connectivity patterns within shared large-scale brain networks, including the salience, frontoparietal control, and default mode networks. It also differentiates neural signatures associated with reactive versus proactive aggression and evaluates the translational relevance of EEG-informed fMRI and connectome-based predictive modeling, while acknowledging current validation constraints. Organizing the literature around convergent circuit-level mechanisms rather than isolated regional findings aims to clarify where the evidence is consistent, where it remains heterogeneous, and which methodological gaps must be addressed before clinical biomarker implementation becomes feasible.

1.4. Dimensional Personality Models: From Theory to Neural Correlates

Eysenck's extraversion, neuroticism, and psychoticism are core biologically informed personality dimensions shaped by gene–environment interactions. Neural correlates of these dimensions have been examined in EEG and fMRI research, with findings suggesting that extraversion is linked to enhanced activation in reward-related circuits, neuroticism to hyperactivity in limbic regions such as the amygdala, and psychoticism to atypical connectivity within fronto-temporal networks, including prefrontal and temporal regions (8). At a systems level, extraversion has been linked to mesolimbic reward circuitry and ventral striatal–prefrontal interactions; neuroticism to heightened salience-network responsivity and altered amygdala–prefrontal coupling; and psychoticism to reduced frontotemporal integration and diminished regulatory control over affective and social processing networks.

Although later psychodynamic and sociocultural extensions emphasized developmental and contextual modulation of personality structure, contemporary neuroscience primarily operationalizes these dimensions through measurable network-level activity and connectivity patterns (16). Together, the dimensional models highlight how personality reflects both neural mechanisms and sociocultural modulation, bridging theoretical constructs with measurable brain activity. This dynamic is illustrated in **Figure 2**.

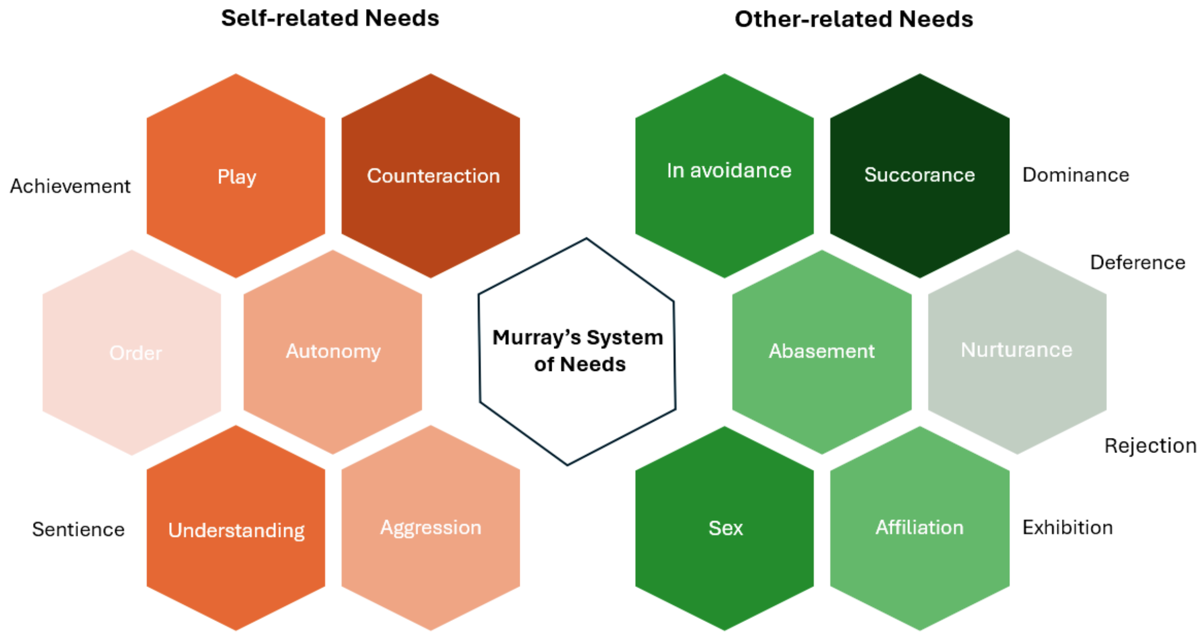


Figure 2. Conceptual representation of Murray's system of needs, illustrating self-related and other-related motivational domains that contribute to personality organization and behavioral regulation. The framework highlights how motivational constructs relevant to aggression, affiliation, dominance, autonomy, and emotional regulation may interact with broader personality dimensions discussed in contemporary neurobiological models. The illustration is intended to provide a theoretical context for understanding personality-related behavioral tendencies and does not represent direct neuroanatomical or functional brain mappings.

2. Emerging Tools: EEG Markers of Personality and Externalizing Traits

Recent advances in EEG and fMRI have enabled increasingly refined investigation into neural correlates of personality and aggression. EEG and fMRI together help identify brain circuits involved in aggression, providing mechanistic insights into large-scale network dysfunction underlying externalizing behaviors. While these approaches may inform future risk stratification frameworks, their direct clinical application remains preliminary (17, 18).

Beyond classic spectral analysis, newer EEG approaches now examine functional brain connectivity, revealing the dynamic architecture of brain networks linked with personality and aggression. In particular, studies have shown that reduced frontal alpha power and context-dependent alterations in theta-band dynamics are associated with impulsivity and aggression-related traits, suggesting candidate electrophysiological correlates for externalizing tendencies.

The incorporation of artificial intelligence (AI) and machine learning, including deep learning methods such as convolutional neural networks (CNNs), has expanded analytical capacity for detecting multivariate EEG patterns related to emotion regulation and aggression risk profiling. Machine learning extracts signal features from EEG to classify affective states and potentially identify individuals at higher risk for externalizing behavior (17, 19). However, many predictive models rely on cross-validation within limited samples, and external replication remains necessary to establish generalizability and avoid overfitting.

These methodological developments contribute to hypothesis generation and model refinement, although claims of reliable clinical biomarkers should be interpreted cautiously. Wearable EEG technology now enables realistic, non-laboratory monitoring, thereby enhancing ecological validity and potential clinical applications (20).

2.1. fMRI in Aggression and Personality Research: Mapping the Functional Brain

fMRI measures brain activity by detecting changes in blood oxygenation level-dependent (BOLD) signal, providing high spatial resolution for examining distributed neural activity during affective and cognitive processes. Structural MRI has linked brain morphology to personality/aggression, but advances in acquisition protocols, higher field strengths, and analytical methods have expanded the capacity to characterize functional brain networks (21). These technical advances have improved the identification of networks implicated in aggression, particularly altered amygdala–prefrontal coupling associated with emotion regulation, orbitofrontal and dorsolateral prefrontal contributions to impulse control, and interactions within large-scale salience and frontoparietal control networks involved in threat evaluation and behavioral regulation. In parallel, connectome-based predictive modeling (CPM) uses resting-state fMRI data to forecast individual behavioral profiles, bridging population-level findings with neural signatures of personality and externalizing traits at the individual level (22). Although CPM represents an important step toward individualized inference, predictive performance varies across samples, and independent external validation remains essential before clinical translation. **Figure 3** provides a conceptual overview of how fMRI-based approaches, including task-based activation, resting-state connectivity, and connectome-based predictive modeling, can be used to investigate neural systems associated with personality traits, aggression, and externalizing behavior. (15, 23).

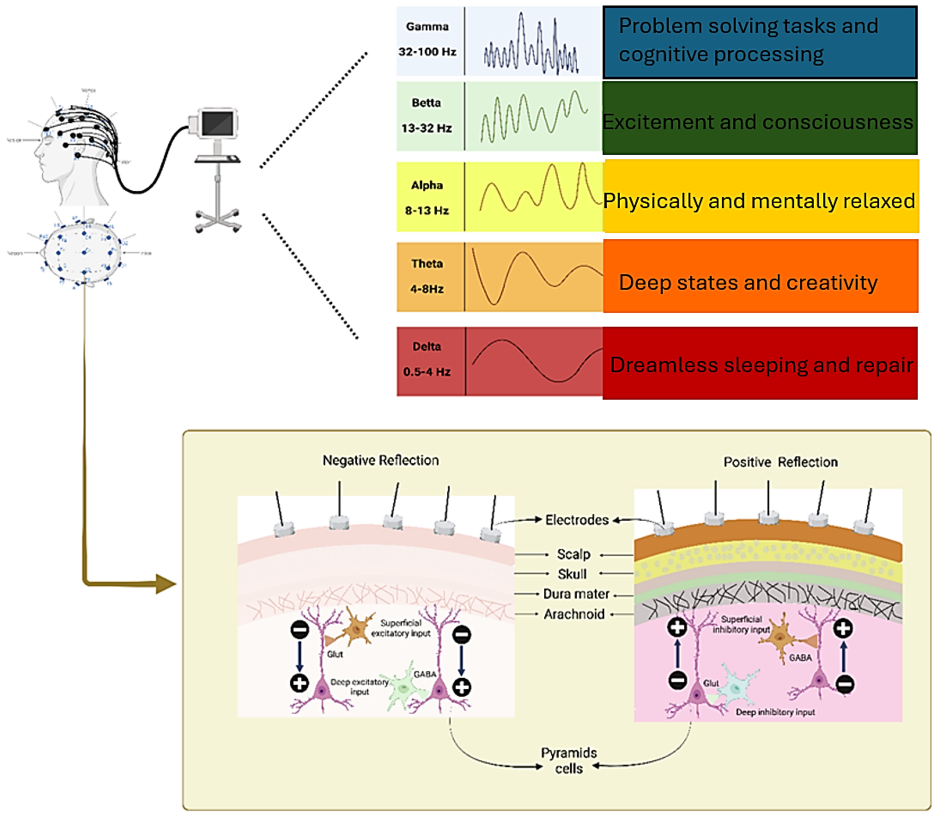


Figure 3. Conceptual overview of EEG signal acquisition and electrophysiological features commonly investigated in personality and aggression research. The illustration summarizes scalp EEG recording, canonical frequency bands (delta, theta, alpha, beta, and gamma), and neural oscillatory processes associated with emotional reactivity, cognitive control, inhibitory processing, and affective regulation. Electrophysiological markers such as frontal alpha asymmetry, theta-band modulation, and event-related components are frequently examined as candidate correlates of impulsivity, aggression, and externalizing behavior. The lower panel schematically illustrates the contributions of excitatory and inhibitory cortical activity to scalp-recorded EEG signals. Created with BioRender.com.

2.2. EEG-fMRI Fusion: A New Frontier in Spatiotemporal Brain Imaging

Since 1997, combining EEG and fMRI has emerged as an important multimodal approach for investigating brain function. The fusion leverages EEG's temporal and fMRI's spatial strengths for a more comprehensive spatiotemporal characterization of brain function at the network level. One method can now inform the interpretation of the other, enabling access to neurophysiological information that would be difficult to capture through unimodal analysis (24).

Simultaneous recordings, paired with advanced analytics, are increasingly implemented in specialized research settings. In particular, EEG-informed fMRI enables emotion- - and impulse-related EEG markers (such as event-related potentials or alpha power fluctuations) to guide the identification of BOLD activity corresponding to aggression and emotional regulation networks (25, 26). Such approaches facilitate temporally informed modeling of hemodynamic responses within distributed control and salience networks.

Research has shown an association between BOLD signal fluctuations and EEG alpha activity (27). However, the strength and direction of these associations vary across paradigms, frequency bands, and analytic strategies.

Concurrent EEG-fMRI has also helped elucidate mechanisms of neurovascular coupling, linking electrophysiological activity to BOLD fluctuations. It is vital to examine changes in arousal and sleep, as EEG alterations often precede or follow changes in fMRI connectivity (28). Although this evidence is not specific to aggression, it demonstrates how EEG-fMRI can clarify state-dependent changes in brain networks relevant to emotion regulation and behavioral control. When the two modalities are combined within a unified analytic framework, they provide a detailed, time-resolved representation of the neural mechanisms underpinning complex behaviors such as aggression and emotional dysregulation. **Figure 4** illustrates the fMRI/BOLD component of this multimodal framework, which can be integrated with temporally precise EEG markers in EEG-informed fMRI analyses.

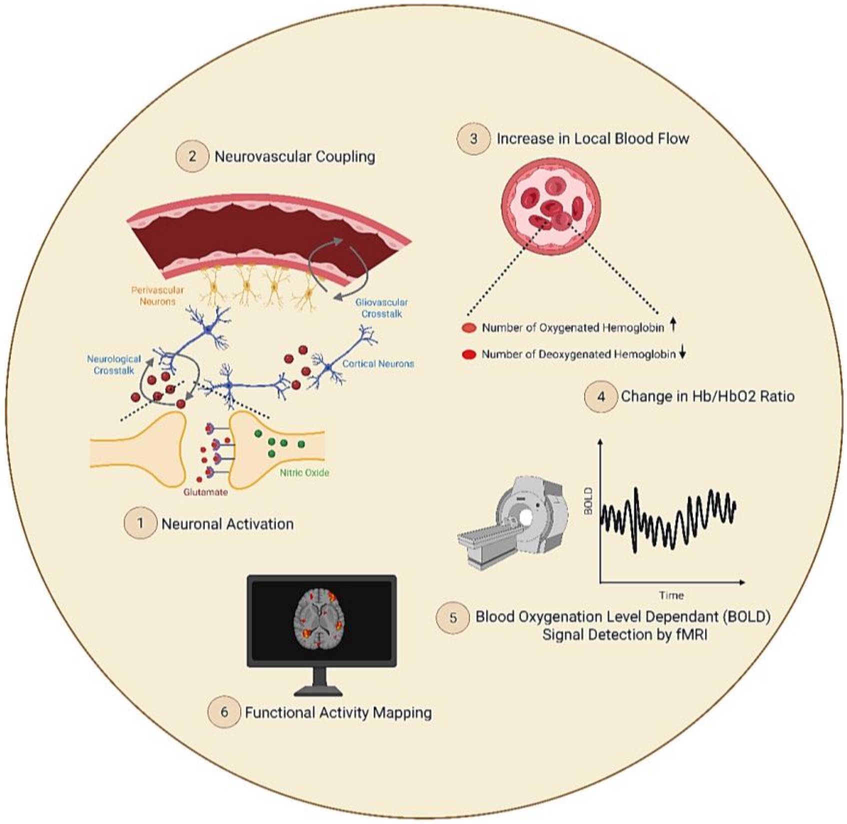


Figure 4. Schematic overview of fMRI signal generation and BOLD-based functional brain mapping. The figure illustrates the sequence from neuronal activation and neurovascular coupling to local blood-flow changes, altered oxyhemoglobin/deoxyhemoglobin ratios, and BOLD signal detection. In the context of personality and aggression research, this process provides the basis for mapping activity and connectivity within prefrontal–limbic and large-scale control networks. Created with BioRender.com.

2.3. Decoding Personality and Aggression through EEG

Personality traits and aggression manifest in EEG patterns such as altered frontal alpha asymmetry, context-dependent theta-band alterations, and reduced beta coherence, which have been linked to impulsivity, disinhibition, and aggression-related traits across affective and externalizing research paradigms (29) (30).

EEG is valuable for identifying neural signatures linked to various psychiatric conditions. Specifically, beyond general psychiatric assessment, EEG research on externalizing phenotypes has revealed these patterns, associated with impulsivity, disinhibition, and aggression. These findings support a temporally resolved model in which rapid oscillatory dynamics and event-related responses reflect variability in inhibitory control and affective reactivity (31, 32). Importantly, effect sizes and directionality of oscillatory findings vary across tasks, developmental stages, and analytic pipelines, underscoring the need for cautious interpretation of EEG markers as trait-level indicators of aggression.

2.4. EEG Candidate Biomarkers: Toward Early Detection of Externalizing Disorders

Although early studies reported limited associations between resting-state EEG power and personality traits, more recent findings indicate stronger, context-dependent results that remain heterogeneous across frequency bands and analytical approaches. While some studies failed to identify robust trait-specific EEG markers (Korjus et al., 2015), newer research has highlighted context-dependent associations involving frontal alpha asymmetry, theta dynamics, and large-scale network connectivity, particularly in relation to emotional regulation and externalizing behaviors (33, 34).

Collectively, these findings suggest that EEG candidate electrophysiological markers of personality are not uniform but may emerge under specific task conditions, developmental stages, or network-level analyses rather than simple spectral power measures. EEG neurofeedback targeting theta/alpha/gamma bands has been explored as a potential intervention to enhance emotional regulation; however, the evidence remains mixed and often derives from small or heterogeneous samples. In this context, theta power is more consistently associated with anxiety and other internalizing features, whereas externalizing behaviors such as aggression are typically related to reduced frontal alpha asymmetry and decreased beta synchronization (35). These distinctions underscore that oscillatory markers may reflect partially overlapping but dissociable neural processes across internalizing and externalizing dimensions. Clarifying this distinction helps prevent conflation between internalizing and externalizing processes when interpreting EEG markers of personality.

2.5. Brain Network Dynamics and Functional Connectivity in Aggression

Recent EEG studies focus on functional connectivity, revealing real-time interactions that illustrate how brain networks coordinate during emotion regulation and cognitive control. Resting-state EEG and fMRI studies indicate that specific components of the default mode network (DMN), particularly the posterior cingulate cortex (PCC) and medial prefrontal cortex (mPFC), are differentially associated with personality traits. For example, resting-state connectivity within DMN regions has been linked to trait-level variation in conscientiousness and social functioning (36). In contrast, agreeableness was associated with posterior cingulate alpha-band activity at rest, reflecting trait-related modulation of self-referential and social cognitive processing. Moreover, irregular connectivity between the DMN and the salience network has been observed in aggression-prone individuals, suggesting potential alterations in network switching between internally oriented (DMN) and externally oriented (salience/frontoparietal) systems. However, findings remain heterogeneous across samples and analytic methods.

Recent advances in computational modeling and deep learning have expanded the analytical capacity of EEG data. For example, machine learning can extract distinguishing features from EEG data, supporting multivariate classification approaches to externalizing traits in research settings (37). However, predictive accuracy varies substantially across datasets, and cross-study generalizability remains limited.

These approaches improve the identification of cross-frequency coupling patterns, coherence measures, and connectivity metrics linked to inhibitory control and emotional regulation deficits. **Table 1** summarizes key EEG candidate markers of connectivity-related abnormalities (e.g., abnormal theta–beta coupling, reduced frontal connectivity) across antisocial/externalizing populations.

Table 1. Candidate EEG Markers Associated with Antisocial and Externalizing Disorders

EEG Biomarker(s)	Study Sample	Key Finding	References
Midfrontal theta & reduced P300	Adults with externalizing traits (ASPD, CD, ADHD, SUD)	Reduced P3 amplitude and midfrontal theta activity were associated with impulsivity/disinhibition	(38)
Theta/beta ratio (TBR)	Patients diagnosed with ADHD (aged 8–18 years)	Elevated frontal TBR in ADHD with abnormal CPT-3 scores; inconsistent associations with behavioral indices	(39)
Prolonged No-Go P3 latency	Violent mentally disordered offenders vs. controls	Prolonged P300/N2 latency suggests cognitive control deficits	(40)
Low resting alpha power	Violent offenders with psychopathic traits	The psychopathy lifestyle facet was correlated with lower alpha power, interpreted as cortical hyperarousal	(41)

2.6. Neural Circuits of Aggression: Key Insights from fMRI Studies

Most structural MRI studies use voxel-based morphometry (VBM) to compare gray matter concentrations. While some findings remain inconsistent—for instance, decreases or increases in posterior cingulate volume have yielded mixed associations with agreeableness—meta-analytic approaches have clarified key structural trends.

Meta-analyses show recurrent volumetric reductions in executive prefrontal and limbic areas among people who engage in aggressive or antisocial behavior (42-44). For example, reduced volumes in the amygdala and prefrontal cortex have been frequently reported, with reactive aggression often linked to heightened limbic reactivity and altered amygdala–prefrontal coupling, and proactive aggression more frequently associated with prefrontal regulatory alterations (45-47).

fMRI, whether task-based or resting-state, contributes to these insights by demonstrating how specific personality characteristics affect the processing of emotional and provocative stimuli. For instance, limbic involvement is reflected in emotional reactions, while different parts of the prefrontal cortex manage cognitive control (48). In addition, convergent fMRI evidence points to dysregulated communication between limbic threat-reactivity regions—primarily the amygdala and insula—and prefrontal regulatory circuits, including the mPFC and anterior cingulate cortex, as a recurring neural signature of aggression (26, 49).

Functional connectivity (FC) findings suggest that aggression is characterized by a network-level phenotype rather than a single-region abnormality. In pediatric clinical samples with clinically significant aggressive behavior, resting-state analyses have reported increased global connectivity of the amygdala alongside reduced fronto-limbic coupling, with aggression severity tracking lower connectivity in ventromedial prefrontal and dorsal anterior cingulate regions (50). Complementing this developmental evidence, task-based fMRI work and mechanistic syntheses of reactive aggression consistently implicate disrupted coordination between limbic threat-reactivity systems such as the amygdala and insula, and prefrontal regulatory circuitry, such as the medial prefrontal cortex and anterior cingulate cortex, aligning aggression with impaired top-down control under provocation (51, 52).

Connectivity patterns also differ by aggression subtype. In healthy adults, trait proactive aggression has been linked to weaker posterior cingulate cortex (PCC)-centered functional connectivity with distributed default mode and control-related regions, consistent with altered self-referential and regulatory network integration in goal-directed aggression (53). More broadly, recent large-scale meta-analytic syntheses localize aggression-related abnormalities across interacting systems, including the salience, frontoparietal, and default mode networks, supporting a transdiagnostic network model spanning emotion processing, valuation, and cognitive control (54, 55).

Overall, resting-state and task-based fMRI findings converge on altered prefrontal–limbic–cingulate circuitry. However, the direction and magnitude of these effects vary across developmental stage, clinical status, aggression subtype, and analytic approach. These findings suggest that aggression-related traits are characterized by coordinated dysfunction across emotion-processing and regulatory networks rather than isolated regional abnormalities (56).

3. Connectome-Based Predictive Modeling (CPM)

CPM uses resting-state functional connectivity to predict individual differences in behavior and cognition, moving beyond group trends to individualized patterns. This method can link distinct brain network profiles to cognitive capabilities or aggression risk and has been shown to predict executive function scores and memory performance. In neuropsychiatric conditions like ADHD, deviations from typical connectivity in the salience network and DMN offer a potential research framework for characterizing brain–behavior variation (22, 57, 58).

CPM, along with other multivariate modeling approaches, contributes to the development of brain–behavior prediction frameworks; however, predictive accuracy varies substantially across datasets, and independent external validation remains essential before clinical translation (59).

Recent applications of CPM in personality and aggression research have identified network signatures asso ciated with externalizing traits, reduced frontoparietal connectivity, and enhanced limbic recruitment at rest, correlating with impulsivity and poor behavioral control. While such individualized connectivity profiles may inform future risk stratification models, most findings derive from cross-validated samples, and replication across independent cohorts is necessary to establish robustness and generalizability (60).

4. Multimodal Neuroimaging Approaches: The EEG-fMRI Synergy

The integration of EEG and fMRI has become a key multimodal approach in research on aggression and externalizing disorders, combining EEG's millisecond temporal resolution with fMRI's high spatial specificity. Simultaneous EEG–fMRI recordings enable direct mapping of fast electrophysiological dynamics, such as oscillations and event-related potentials, onto large-scale brain networks. Recent studies applying this approach demonstrate that aggressive phenotypes are characterized by disrupted coordination across distributed neural systems rather than isolated regional abnormalities. In a task-based EEG–fMRI study, Schauer et al. showed that patients with borderline personality disorder exhibited reduced frontal theta responses to loss feedback and abnormal coupling between theta activity and dorsolateral prefrontal cortex (dlPFC) BOLD responses, indicating impaired recruitment of prefrontal control mechanisms during aversive outcome processing. These findings illustrate how EEG-informed fMRI can identify both the timing and anatomical locus of network dysfunction underlying impulsive aggression (61).

Resting-state investigations further support a network-level model of aggression. Using high-density EEG, Kleinert and Nash demonstrated that individuals with higher trait aggression exhibit reduced temporal stability of intrinsic brain network states, reflected by shorter EEG microstate durations and faster switching between large-scale configurations. EEG microstates correspond to fMRI networks (ACC, insula, frontoparietal), aligning with inefficient organization in externalizing populations (31). Consistently, connectome-based fMRI studies in youth with conduct problems report reduced global efficiency and weakened subcortical hub connectivity in individuals with more severe antisocial behavior. Together, these results suggest that trait aggression is associated with unstable intrinsic network dynamics, observable across both electrophysiological and hemodynamic modalities.

Simultaneous EEG–fMRI has also been instrumental in clarifying the neural timing of inhibitory control deficits relevant to aggression. Single-trial EEG-informed fMRI studies during response inhibition tasks reveal that variability in EEG markers of control, such as the NoGo P3 component, maps onto differential engagement of fronto-striatal circuits. Schmüser et al. identified distinct neurophysiological phenotypes in healthy adults, showing that individuals with attenuated P3 responses exhibited hyperactivation of the inferior frontal cortex and the putamen, along with greater impulsivity and behavioral variability (62). This pattern suggests inefficient recruitment of inhibitory networks when normal electrophysiological control signals are diminished. Collectively, multimodal findings support a distributed network framework involving control, salience, and limbic systems; however, direct evidence for improved diagnostic specificity or treatment personalization remains limited and requires prospective validation (54).

EEG-fMRI integration enhances neurobiological characterization. However, simultaneous EEG–fMRI remains technically demanding and is primarily implemented in specialized research settings. Its clinical utility for aggression-related assessment, therefore, remains preliminary, although it may be valuable for studying dynamic states such as arousal, sleep, and rapid cognitive transitions (24).

Across studies from our search, EEG–fMRI integration in aggression/externalizing most often uses ERP-informed GLMs, time–frequency EEG-informed regressors, or data-driven EEG phenotypes mapped onto BOLD responses, and it repeatedly implicates fronto-cingulo-insular/frontoparietal control networks and their coupling with limbic/striatal systems. Methodological factors (EEG feature selection, model integration) shape the direction/localization of the effect. EEG-informed fMRI allows researchers to model BOLD signal changes in relation to specific electrophysiological events, thereby providing a framework for investigating neurovascular coupling and the dynamic interaction between electrical and hemodynamic signals (63). This capacity is further enhanced by layer-specific laminar EEG-fMRI methods, which link scalp oscillations to fMRI signals specific to cortical layers. Although still methodologically specialized and primarily applied in high-field research environments, this approach offers a promising strategy for examining laminar-specific contributions to information processing within and across cortical circuits (64). However, laminar EEG–fMRI applications in personality and aggression research remain limited, and their translational implications are yet to be established. **Table 2** summarizes task-based and resting-state EEG–fMRI studies examining inhibitory-control and aggression-related neural dynamics. Across methodologies, convergent trends suggest associations between reduced P3/theta indices and altered frontal–striatal or cingulo-insular connectivity, supporting a distributed-network model of externalizing behavior. Importantly, only a limited number of simultaneous EEG–fMRI studies have directly examined aggression phenotypes. In contrast, most investigations focus on inhibitory control, impulsivity, or broader transdiagnostic processes relevant to externalizing behavior.

Table 2. Selected Simultaneous and EEG-Informed EEG–fMRI Studies Relevant to Aggression and Externalizing Phenotypes

Sample	Aggression/externalizing construct	Paradigm	Integration method	Main finding	References
Healthy adults	Trait aggression (questionnaire)	Resting-state EEG	EEG microstate analysis	Higher aggression was associated with lower microstate stability (gender-moderated)	(31)
BPD patients vs. healthy controls	Impulsivity/impulsive aggression	Reward/feedback task	Simultaneous EEG–fMRI; theta-modulated BOLD	Group×valence interaction: controls show stronger theta-dlPFC BOLD to losses (opposite in BPD)	(65)
38 healthy adults; Nogo-IC+ (n=21) vs Nogo-IC− (n=17)	Impulse-control phenotypes; Nogo-IC− higher ADHD risk	Go/NoGo	Single-trial EEG/fMRI; EEG-driven phenotype	Nogo-IC−: reduced P3, hyperactivation IFG/insula/putamen, greater RT variability	(62)
23 healthy young adults (subset EEG-fMRI n≈17)	Sequential inhibitory control (externalizing-relevant)	Flanker/NoGo	Simultaneous EEG–fMRI; ERP–BOLD	Sequential inhibition stages were linked to control-network engagement	(66)
Healthy adults	Contextual stopping (transdiagnostic)	Contextual stopping	EEG-informed fMRI (time–frequency)	Overlapping inhibition effects across frequencies, frontal involvement	(67)
Healthy adults	Trial-to-trial variability (externalizing heterogeneity)	Prior probability/decision-making	EEG-informed fMRI (single-trial CNV)	Captured fluctuations invisible in fMRI-only	(68)
15 healthy adults	EEG regressor effects on networks	Visual choice RT/target detection	Single-trial ERP-informed fMRI; GLM/event-duration	P3 amplitude/latency effects vary by regressor type	(69)

4.1. Translating Imaging to Psychiatry: Applications for Diagnosis and Intervention

These combined methods have enabled the identification of neurophysiological changes during reward processing in disorders such as borderline personality disorder and have supported the detection of neural correlates of impulse control in regions such as the putamen and inferior frontal cortex. Multimodal techniques provide a more comprehensive picture, linking dynamic neural states with behavior and offering potential value for future diagnostic and prognostic research, (61).

Studies emphasize the accelerating use of EEG-fMRI and related techniques to improve psychiatric diagnosis and intervention, highlighting progress in identifying candidate biomarkers and refining neurofeedback, neuromodulation, and neurostimulation therapies (70). For instance, EEG-informed neurofeedback has been explored as a strategy to modulate fronto-limbic activity associated with impulsive aggression. However, effect sizes vary, and long-term durability remains under investigation. In contrast, fMRI-guided rTMS has been applied to prefrontal targets implicated in regulatory control, with preliminary evidence suggesting modulation of behavioral outcomes in selected samples.

Multimodal neuroimaging serves as an adjunctive tool and mechanistic guide for intervention design in aggression/impulse-control pathologies, though limited by cost, complexity, and need for standardized protocols (71, 72).

4.2. Toward Candidate Biomarker-Informed Psychiatry: Multimodal Imaging in Action

Multimodal research on candidate biomarkers in aggression and externalizing increasingly combines EEG-derived markers (e.g., oscillatory power, ERP indices, and connectivity features) with MRI measures (task activation, resting-state connectivity, and structural indices), using multimodal fusion frameworks that range from asymmetric fusion (one modality constraining another) to symmetric/data-driven fusion, after dimensionality reduction. (73, 74).

Such approaches facilitate the identification of crossmodal neural signatures that link cognitive control and emotion-regulation networks to aggressive or impulsive traits, potentially informing hypothesis-driven targets for neuromodulation and biofeedback strategies. However, clinical efficacy remains under ongoing evaluation.

In the broader EEG/neuropsychiatric ML literature, the most frequently used model families remain support vector machines, regularized regression (e.g., elastic net/LASSO), and tree-based methods (e.g., random forests), with deep learning (CNNs) increasingly applied to neuroimaging/EEG classification, benchmarked against standard ML (75, 76).

Across these frameworks, fusion models that integrate temporal EEG dynamics with spatial connectivity metrics have been shown to enhance multivariate prediction of aggression-related traits and decision-making variability in research samples. However, performance gains are often modest and dependent on preprocessing pipelines, feature selection strategies, and sample characteristics.

When prediction frameworks are used, performance is typically assessed via cross-validation, with external replication being crucial to ensure reliable translation of biomarker findings to clinical practic e and prevent overfitting or inflated effect sizes (22).

4.3. Brain Training and Self-Regulation: EEG-fMRI Neurofeedback Advances

EEG and, more recently, fMRI have been used to study neurofeedback. This therapeutic approach aims to facilitate voluntary modulation of brain activity and self-regulatory processes. Preliminary research suggests that EEG-based neurofeedback may reduce impulsivity and aggressive behavior, particularly in contexts such as substance use disorders (77). Arnold et al. (2013) found similar ADHD symptom improvements in active and sham neurofeedback groups, suggesting non-specific/placebo effects and the need for methodological rigor (78).

Emerging EEG-fMRI-guided neurofeedback continues to link EEG frequency bands (theta, alpha, gamma) to DMN activity for customized ADHD treatments (78-80). Such approaches remain primarily experimental and are often limited to small samples or feasibility trials.

Although direct evidence that mindfulness-based neurofeedback (mbNF) reduces aggression remains limited, its modulatory effects on cognitive control and emotion regulation networks suggest indirect therapeutic potential. Real-time fMRI, EEG-informed neurofeedback, rTMS, and mindfulness-based neurofeedback may target prefrontal–limbic connectivity relevant to emotional regulation and impulse control. However, effect sizes, durability of response, and comparative efficacy relative to standard treatments require further controlled evaluation (81, 82).

These approaches may inform mechanistically guided interventions for disorders characterized by aggression and emotional dysregulation, pending large-scale replication and standardized outcome metrics (83). **Table 3** summarizes EEG-fMRI neurofeedback studies examining changes in prefrontal-limbic connectivity and self-regulation.

Notably, most multimodal neurofeedback studies to date focus on emotion regulation or motor rehabilitation, with relatively few trials directly targeting aggression or externalizing phenotypes; consequently, extrapolation to impulsive aggression should be made cautiously.

Table 3. Recent EEG–fMRI Neurofeedback and Emotion Regulation Studies

Modality / Interventions	Study sample	Key findings	Reference
Bimodal EEG–fMRI NF targeting M1/SMA vs motor imagery	Chronic stroke survivors, n=30 (NF n=15, MI n=15)	NF showed greater upper-limb gains, a higher responder rate, and motor-system re-engagement	(84)
Theta EEG NF during simultaneous fMRI (active vs sham)	Healthy volunteers, n=18	Active NF: stronger frontal-theta asymmetry, tighter EEG–BOLD coupling, left DLPFC involvement	(85)
Frontal alpha EEG–fMRI NF (positive affect upregulation)	32 healthy adults (EEG-NF vs sham)	Real NF increased frontal alpha + BOLD in prefrontal/parietal/limbic (1.9%), enhanced connectivity	(17)
Simultaneous rtfMRI+EEG NF (positive-emotion, real vs sham)	MDD patients: n=16 real, n=8 sham	Real NF: ↑ amygdala activity, ↑ amygdala–rACC connectivity, EEG asymmetry shift, improved mood	(86)

4.4. From Biomarkers to Brain Stimulation: The Emerging Role of rTMS

By identifying MRI and EEG candidate markers that could predict rTMS treatment response in depression, Klooster et al.'s 2024 study suggests a shift toward personalized neuromodulation based on individual neuroimaging profiles (87). While the direct application of rTMS for aggression is an area of active research, the principle of using neuroimaging to guide and personalize such interventions is a key theme for future therapeutic developments (88). Emerging data indicate that prefrontal rTMS has been associated with modulation of top-down regulation in the dorsolateral prefrontal and cingulo-insular networks. Neuroimaging-informed approaches may help refine stimulation targets; however, standardized predictive biomarkers for aggression-specific neuromodulation have not yet been established.

Translating neural targets into validated biomarkers and effective therapies demands rigorous scientific validation across extensive, reproducible studies (89). While the enthusiasm surrounding AI/ML and novel neurofeedback protocols is understandable, the field must be committed to rigorous, controlled studies to ensure that new interventions are safe and genuinely effective (90).

4.5. Longitudinal Perspectives and Developmental Trajectories

As aggression and related personality traits are dynamic constructs that evolve throughout life, particularly during formative years such as adolescence, understanding their neurological roots requires a developmental perspective (91). While helpful for discovering new connections, cross-sectional studies capture only a moment in time. Neuroimaging studies that follow individuals over extended periods have a distinct advantage for studying the association between changes in brain structure or function and the emergence, persistence, or reduction of aggressive behaviors and personality traits. Such designs may help clarify potential early neural correlates, developmental pathways, and sensitive periods for intervention (92).

A growing body of evidence points to reactive aggression (RA) as a transdiagnostic risk signal for a variety of mental disorders, characterized by impulsive, affect-driven responses to perceived threats. Persistence from childhood into adolescence is often linked to more severe and persistent forms of psychopathology (52).

Longitudinal neuroimaging projects increasingly examine amygdala–prefrontal circuit development during threat and reinforcement learning. Developmental models suggest that heightened limbic responsivity combined with delayed prefrontal maturation may contribute to the persistence and escalation of reactive aggression. To characterize individual variation in these neurodevelopmental processes, such studies often sample adolescents across a broad spectrum of emotional reactivity (93)

Supporting these endeavors, additional longitudinal studies are examining the paths taken by proactive (planned, instrumental) and reactive aggression from adolescence into young adulthood, along with the correlations between these paths and variables like impulsivity, internalizing emotions, and callous-unemotional trait scores (94). According to a study, proactive aggression was still significantly predicted by the degree of CU characteristics, even after accounting for reactive aggression. This suggests that various forms of aggression have distinct developmental connections. Studies like these highlight the need to classify aggressive behaviors to learn about their distinct causes and neurological characteristics (95, 96).

Longitudinal research supports a developmental-risk framing, showing that neural markers measured in adolescence can predict later externalizing outcomes. In EEG work, reduced P3/P300 amplitude has been linked to broad externalizing liability and is often interpreted as an endophenotypic indicator of disinhibition across development (97). Prospective fMRI studies similarly show that mid-adolescent activation during response inhibition predicts subsequent substance use, consistent with a developmental pathway in which weaker recruitment of inhibitory-control circuitry precedes later risk behavior (98). Beyond task activation, longitudinal resting-state findings indicate that higher externalizing is associated with stronger amygdala–ACC and amygdala–OFC functional connectivity across adolescence into young adulthood, linking developmental changes in emotion-control circuitry to externalizing trajectories (99). Finally, longitudinal affective fMRI evidence suggests that amygdala reactivity can prospectively relate to later antisocial outcomes, reinforcing the idea that multiple developmental neurobiological routes may underlie distinct aggression phenotypes.

By integrating longitudinal neuroimaging with behavioral phenotyping, this line of research may inform more developmentally sensitive prevention and intervention models, although translation into scalable clinical tools remains an ongoing challenge (100).

Future work should prioritize integrating EEG/fMRI biomarkers with longitudinal behavioral data to build predictive developmental models of externalizing risk and resilience.

5. Methodological Approach

This review used a structured, literature search to synthesize contemporary research on personality traits, aggression, and multimodal neuroimaging, including EEG and fMRI. Electronic databases, including PubMed, Scopus, and Web of Science, were searched for peer-reviewed articles published primarily between 2020 and 2026, with older foundational studies included only when necessary to define core theoretical models or methodological concepts.

Search queries combined keywords and Boolean operators such as ("EEG" OR "fMRI") AND ("personality" OR "traits") AND ("aggression" OR "externalizing" OR "violence"). Additional search terms included "impulsivity," "reactive aggression," "proactive aggression," "functional connectivity," "frontal alpha asymmetry," "theta," "P3," "amygdala," "prefrontal cortex," and "connectome-based predictive modeling." Reference lists of key articles were also screened to identify additional relevant publications.

Priority was given to studies that employed validated psychometric instruments, clearly defined aggression constructs (e.g., reactive and proactive aggression), and established neuroimaging methodologies. Studies were included if they: (1) examined aggression, impulsivity, externalizing behavior, or aggression-related personality traits; (2) used EEG, fMRI, structural MRI, or multimodal EEG–fMRI methods; (3) involved human participants; and (4) reported neural activity, connectivity, or electrophysiological findings relevant to personality or aggression. Studies were excluded if they were animal studies, conference abstracts, case reports, non-English publications, or did not report neuroimaging findings directly relevant to aggression, impulsivity, or externalizing traits. Both task-based and resting-state paradigms were considered, with particular attention to large-scale brain network findings, electrophysiological markers, and multimodal integration frameworks.

Titles and abstracts were first screened for relevance, followed by full-text evaluation of studies meeting the initial criteria. Key information was extracted regarding study design, sample characteristics, neuroimaging modality, behavioral or personality measures, experimental paradigm, and main neural findings. As illustrated in **Figure 5,** the final synthesis included 112 studies after duplicates were removed and non-relevant articles were excluded.

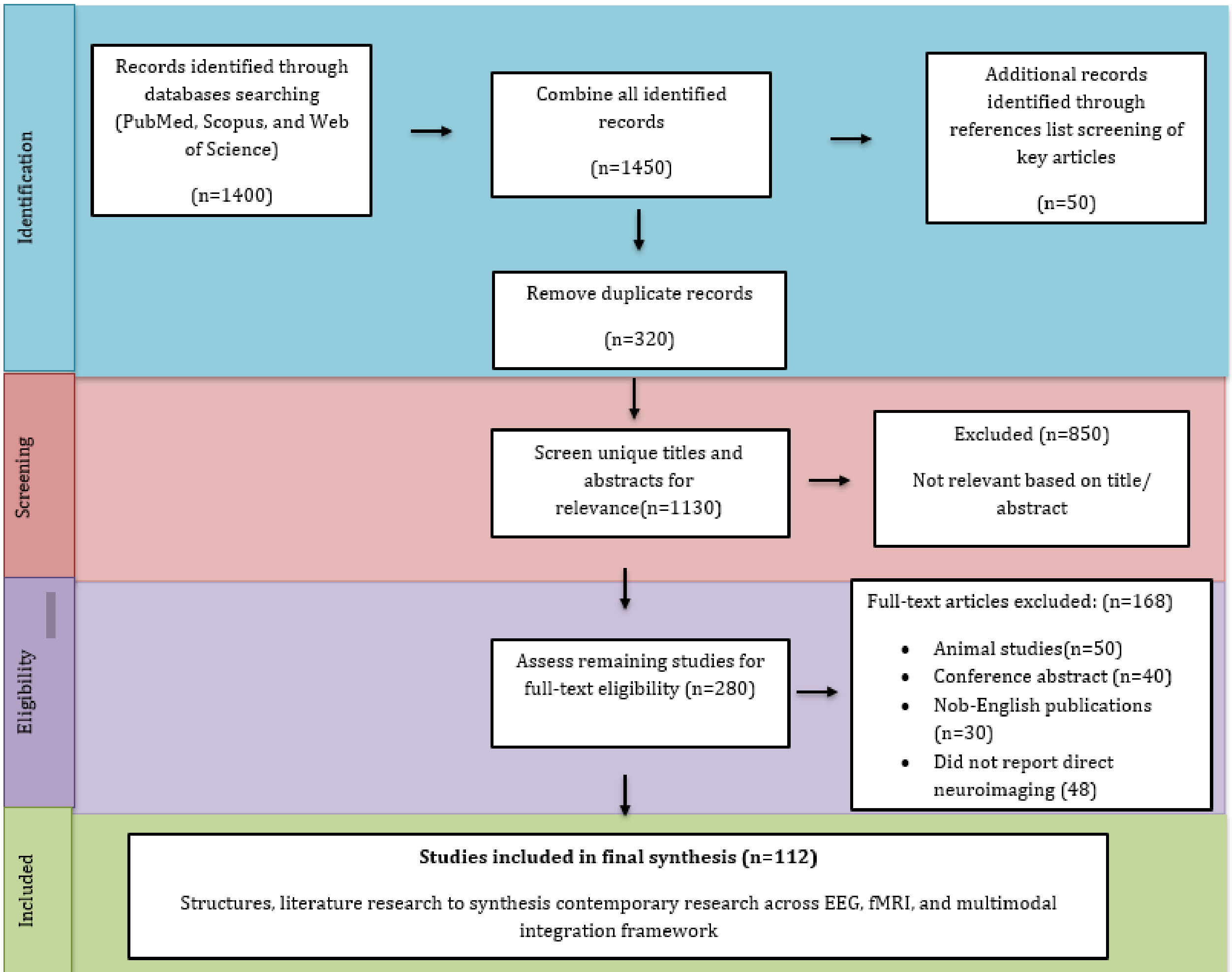


Figure 5. Flow diagram of the literature search and study selection process. The flowchart details the identification of records from databases and reference lists, followed by the screening and eligibility phases, resulting in a final inclusion of 112 studies for synthesis.

This concept-driven synthesis identifies convergent patterns, methodological heterogeneity, and translational directions across EEG, fMRI, and multimodal predictive modeling research.

6. Conclusions and Discussions

This paper synthesizes recent advances in multimodal EEG–fMRI research and highlights network-level mechanisms of externalizing psychopathology. Given the complexity of personality, multidimensional models that integrate affective, behavioral, and neuroimaging data may improve construct validity and mechanistic interpretation.

Current evidence suggests partially dissociable neural patterns across reactive and proactive aggression subtypes, although these distinctions remain preliminary and require further replication.AI/ML and CPM have demonstrated the feasibility of individual-level predictions from brain connectivity patterns, although external validation remains necessary before clinical translation (101, 102).

However, key methodological gaps persist. EEG offers temporal precision, but source localization is limited by volume conduction (103). In fMRI, the BOLD signal is an indirect readout shaped by hemodynamics, and variability in the hemodynamic response can reduce temporal specificity and complicate comparisons across individuals and studies (104-106).

Multimodal EEG-fMRI integration can partially address these limitations by mapping rapid electrophysiological dynamics onto precise spatial networks (107). Nevertheless, the reviewed evidence remains heterogeneous across populations, experimental paradigms, preprocessing pipelines, and analytic strategies. Therefore, well-replicated findings, such as altered prefrontal–limbic connectivity and atypical frontal EEG dynamics, should be distinguished from more preliminary findings involving predictive modeling, laminar EEG–fMRI, and clinical biomarker applications.

Recent work emphasizes integrating neuroimaging data with dimensional personality assessments, which may refine mechanistic models and inform hypothesis-driven intervention strategies, pending robust validation (108-110).

Future research must prioritize standardization of acquisition/analysis protocols to improve reproducibility and reduce methodological variability (111). Moreover, diversifying samples across age, sex, ethnicity, and SES is essential to ensure clinical relevance. (112). With rigorous methods and validation, multimodal neuroimaging holds promise for future risk stratification frameworks and mechanistically informed interventions, although current evidence is not yet sufficient for routine clinical implementation.

7. Abbreviation

ACC – Anterior Cingulate Cortex
ADHD – Attention-Deficit/Hyperactivity Disorder
AI – Artificial Intelligence
ASPD – Antisocial Personality Disorder
BOLD – Blood-Oxygen-Level-Dependent
CNN – Convolutional Neural Network
CPM – Connectome-Based Predictive Modeling
CU – Callous–Unemotional
DMN – Default Mode Network
dlPFC – Dorsolateral Prefrontal Cortex
EEG – Electroencephalography
EPQ-R – Eysenck Personality Questionnaire
ERP – Event-Related Potential
FC – Functional Connectivity
FFM – Five Factor Model
fMRI – Functional Magnetic Resonance Imaging
GLM – General Linear Model
IFG – Inferior Frontal Gyrus
mbNF – Mindfulness-Based Neurofeedback
MI – Motor Imagery
ML – Machine Learning
M1 – Primary Motor Cortex
NF – Neurofeedback
OFC – Orbitofrontal Cortex
PA – Proactive Aggression
PCC – Posterior Cingulate Cortex
PFC – Prefrontal Cortex
P300 / P3 – Positive Deflection at ~300 ms (ERP Component)
RA – Reactive Aggression
rACC – Rostral Anterior Cingulate Cortex
rTMS – Repetitive Transcranial Magnetic Stimulation
RT – Reaction Time
SMA – Supplementary Motor Area
SUD – Substance Use Disorder
TBR – Theta/Beta Ratio
VBM – Voxel-Based Morphometry

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