Neurobiology of Anger

Anger represents a pervasive and clinically consequential affective experience that frequently emerges across mental health settings. Although anger is a normative human emotion, problematic anger, defined as intense, frequent, or poorly regulated anger that produces functional impairment, poses substantial risks to psychological, interpersonal, and occupational well-being. Epidemiological data indicate that problematic anger is common in the general population; for example, a large-scale community study reported that approximately 7.8% of U.S. adults endorsed recurrent and intense episodes of inappropriate or uncontrolled anger associated with significant interpersonal or functional consequences (Okuda et al., 2015). Such findings underscore anger as a population-level concern rather than a niche clinical phenomenon. In clinical samples, the prevalence is markedly higher, with problematic anger strongly associated with aggression, intimate-partner conflict, self-injurious behavior, and workplace disruption (Palop Larrea et al., 2024; Richard, 2023).

A clear conceptual distinction between anger, aggression, and irritability/hostility is essential for precise assessment and treatment planning. Anger refers to the internal emotional experience that may or may not be expressed behaviorally. Aggression, by contrast, is a behavioral act, verbal or physical, characterized by intent to harm another person or object. Irritability and hostility reflect more stable trait-like predispositions toward annoyance, resentment, or negative beliefs about others. Contemporary empirical work highlights the importance of differentiating these constructs. Gröndal et al. (2023), for example, demonstrated that irritability, trait anger, and impulsivity, although conceptually related, exhibit distinct associations with subjective well-being, suggesting that each reflects a unique psychological and behavioral profile.

Clinically, anger dysregulation occurs across a diverse range of psychiatric conditions. Intermittent Explosive Disorder (IED) is characterized by recurrent behavioral outbursts of impulsive aggression, while Borderline Personality Disorder (BPD) often involves chronic and rapidly escalating anger that contributes to interpersonal instability and self-destructive behavior. Trauma-related disorders, particularly Posttraumatic Stress Disorder (PTSD), frequently include heightened anger, irritability, and aggressive impulses as part of broader dysregulation of threat detection and arousal. Additionally, anger features prominently in depression (particularly in mixed or irritable presentations), substance-use disorders, traumatic brain injury (TBI), and psychotic disorders (Kolla et al., 2023; Shafti et

al., 2023). Given its high prevalence and broad diagnostic distribution, anger is a critical domain in risk assessment, case formulation, and therapeutic intervention within mental-health practice.

Advances in affective neuroscience have substantially enriched contemporary understanding of anger, offering mental health providers a biologically grounded framework for conceptualizing and treating anger dysregulation. Central to this framework is a network of neural structures, including the amygdala, ventromedial prefrontal cortex (vmPFC), insula, anterior cingulate cortex (ACC), and striatum, that collectively govern threat detection, interoceptive experience, evaluative processing, and inhibitory control. A growing body of neuroimaging research demonstrates that individuals who exhibit heightened reactive anger or aggression often display increased amygdala reactivity, reduced functional connectivity between limbic and prefrontal regions, and diminished recruitment of prefrontal regulatory circuits during emotionally provocative tasks (Kolla et al., 2023; Richard, 2023). These findings highlight why clients frequently report that anger escalates "instantaneously" or feels "out of control," despite clear cognitive awareness of negative consequences.

For psychotherapists, conceptualizing anger as an emotion-regulation deficit rather than solely a behavioral problem offers a more nuanced and clinically actionable perspective. From a neurobiological standpoint, anger dysregulation often reflects an imbalance between bottom-up limbic activation and top-down regulatory mechanisms. Thus, interventions that strengthen prefrontal modulation (e.g., cognitive reappraisal, problem-solving skills, mindfulness-based attention training) or reduce physiological hyperarousal (e.g., paced breathing, HRV biofeedback, grounding techniques) align directly with underlying neural mechanisms. Psychoeducation grounded in these principles may help clients understand their symptoms, decrease self-blame, and enhance motivation for behavioral change.

Neurobiological knowledge also informs treatment selection and case conceptualization. Clients whose anger is rapid, impulsive, and behaviorally expressed may benefit most from structured cognitive-behavioral interventions emphasizing stimulus control, behavioral rehearsal, and inhibitory skill development. In contrast, clients with chronic, ruminative, or shame-laden anger may respond more effectively to mindfulness-based cognitive therapy (MBCT), acceptance- and commitment-based approaches, or schema therapy. Furthermore, digital interventions, including app-based emotion regulation programs, online CBT modules, and biofeedback-integrated tools—offer supplemental support for clients whose neural vulnerabilities impede consistent regulation in daily life.

Taken together, the neurobiology of anger provides a scientifically grounded framework that enhances clinical assessment, informs personalized intervention, and ultimately improves therapeutic outcomes. For psychotherapists, integrating neuroscientific insights is not merely an academic exercise; it is a practical strategy for understanding complex emotional processes and tailoring interventions to clients' intrinsic regulatory capacities.

Functional Models of Anger

From an evolutionary standpoint, anger is conceptualized as a functional emotion that evolved to signal goal obstruction, boundary violations, or social rank threats. Within evolutionary models, anger facilitates adaptive action tendencies that promote survival, including mobilizing energy, enhancing vigilance, and preparing the organism for assertive or defensive behavior (Sell et al., 2009). Unlike fear, which orients the organism toward avoidance, anger is approach-oriented, motivating confrontation or protection of valued resources. Contemporary neurobiological research supports this distinction, demonstrating that anger activates neural circuits associated with approach motivation and social dominance (Krämer et al., 2023).

Functional accounts also emphasize anger's role in social bargaining. When individuals perceive unfairness, exploitation, or invalidation, anger acts as a signaling mechanism to renegotiate interpersonal boundaries. This perspective is supported by findings that anger expression can increase social influence in negotiation contexts, particularly when status or fairness is at stake (Reyna et al., 2023). Clinically, however, once adaptive signaling becomes dysregulated or chronically activated, anger may transition from a functional response to a maladaptive emotion contributing to interpersonal conflict, health deterioration, and psychiatric morbidity.

As a result functional models provide an important foundation for understanding anger: it is not an inherently pathological emotion. Rather, pathology emerges when the intensity, duration, frequency, or behavioral consequences of anger exceed the demands of the situation or impair functioning. This conceptualization guides psychotherapists to assess not only the presence of anger, but its proportionality and contextual relevance.

Cognitive–Affective Models of Anger

Cognitive–affective theories propose that anger arises primarily from evaluative appraisals, particularly those involving perceived injustice, intentional harm, unfairness, or violation of expectations. According to appraisal theory, anger is triggered when an event is viewed as blameworthy, controllable by another person, or unjustified (Lazarus, 1991). These cognitive evaluations determine whether frustration escalates into anger and whether anger escalates into aggression.

A central construct within these models is hostile attribution bias, the tendency to interpret ambiguous social cues as intentionally harmful. Hostile attribution biases are robust predictors of anger and aggression across developmental stages and clinical populations (Vitiello et al., 2023). Recent evidence from cognitive bias modification studies demonstrates that altering such interpretive tendencies can reduce anger and aggressive behavior, providing empirical support for appraisal-based interventions (Ciesinski et al., 2023).

Rumination also plays a significant role in cognitive–affective models of anger. Anger rumination, the repetitive dwelling on anger-eliciting events, exacerbates physiological arousal, prolongs emotional activation, and increases aggressive impulses. Neurobiological studies show that rumination maintains amygdala activation and interferes with prefrontal regulatory processes (Richard, 2023). Clinically, anger rumination is a target in cognitive-behavioral therapy (CBT), dialectical behavior therapy (DBT), and mindfulness-based cognitive therapy (MBCT), which emphasize reappraisal, attentional shifting, and decentering.

Cognitive–affective models therefore highlight that anger is not merely a reaction to external stimuli but a consequence of interpretive processes that shape emotional experience. This understanding guides clinicians toward interventions that modify maladaptive appraisals and cognitive patterns contributing to anger dysregulation.

Dimensional Models: Trait and State Anger

Dimensional frameworks conceptualize anger along two core dimensions: state anger (the momentary emotional experience) and trait anger (a predisposition to experience frequent or intense anger across situations). Trait anger reflects stable emotional reactivity shaped by temperament, neurobiology, and developmental experiences. Research consistently links high trait anger with heightened baseline arousal, reduced prefrontal inhibition, and increased limbic sensitivity (Tonnaer et al., 2023). Such individuals demonstrate faster physiological activation and slower recovery following provocation.

State anger, by contrast, represents the acute experience of anger in response to specific circumstances. State anger is influenced by situational factors, interpersonal dynamics, and contextual triggers. Although state anger may be transient, recurrent episodes elevate the risk for aggression, relationship conflict, and emotional dysregulation.

Additional constructs within dimensional models include anger expression, anger suppression, and anger control. Anger expression refers to the outward communication or behavioral discharge of anger, whereas anger suppression reflects internalized inhibition without expression. Both patterns confer risks: expression may lead to aggression or

interpersonal harm, while suppression is associated with somatic complaints, depression, and passive-aggressive behavior (Gröndal et al., 2023).

Dimensional models also incorporate anger rumination and hostility, which contribute uniquely to anger-related pathology. These multidimensional conceptualizations allow clinicians to assess anger across behavioral, cognitive, and emotional domains, informing treatment decisions. For example, clients with high trait anger may require lifelong emotion-regulation skill development, while those with primarily state-based anger may benefit from trigger identification and situational coping strategies.

Anger Versus Related Constructs

Anger intersects with several related constructs, fear, irritability, hostility, and contempt—yet differs in important ways. Differentiating these experiences is essential for accurate diagnosis and effective intervention. Anger and fear both involve heightened arousal but differ in motivational direction. Fear activates withdrawal and avoidance systems, while anger activates approach and confrontation. Neurobiologically, fear is associated with amygdala-hypothalamus-periaqueductal-gray pathways oriented toward defensive withdrawal, whereas anger engages a partially overlapping network with greater involvement of the insula and striatal regions linked to approach behavior (Krämer et al., 2023). Clinically, mislabeling fear as anger (or vice versa) may obscure trauma responses or mask underlying anxiety that requires targeted intervention.

Irritability, on the other hand, is characterized by a lowered threshold for negative emotional responses, including anger, frustration, or annoyance. It represents a broader affective instability rather than a discrete emotion. In mood disorders, particularly major depression and mixed bipolar states, irritability may be more prominent than anger per se. Neuroimaging studies show that irritability correlates with heightened limbic reactivity similar to anger but is more strongly associated with chronic negative affect and mood dysregulation (Palmier-Claus et al., 2023).

Hostility is a different construct, a cognitive-attitudinal construct involving cynicism, mistrust, and negative beliefs about others' intentions. Hostility often precedes anger but can persist independently of situational triggers. High hostility is associated with cardiovascular morbidity and long-term interpersonal dysfunction (Palop Larrea et al., 2024). While anger is episodic and affective, hostility is persistent and cognitive.

When examining the concept of contempt, contempt involves moral or interpersonal superiority and is expressed through disdain rather than confrontation. Although contempt may co-occur with anger, particularly in relationship conflict, it is associated with different interpersonal consequences, including withdrawal and relational erosion.

Differentiating these constructs enhances diagnostic precision and guides psychotherapists toward interventions tailored to the client's specific emotional and cognitive patterns.

Core Neural Circuitry of Anger

Limbic Structures

The limbic system constitutes the foundational neural substrate of anger, mediating the detection of threat, the generation of affective arousal, and the encoding of emotionally salient memories. Among its components, the amygdala plays a central role. The amygdala is responsible for rapidly appraising environmental cues, detecting potential threats, and mobilizing physiological and behavioral responses. In the context of anger, the amygdala facilitates approach-oriented defensive aggression, distinguishing anger from fear-based avoidance responses. Contemporary neuroimaging research consistently demonstrates heightened amygdala reactivity in individuals who exhibit impulsive aggression, elevated trait anger, or rapid anger escalation (Tonnaer et al., 2023). Moreover, aberrant amygdala activation is often accompanied by reduced connectivity with prefrontal regulatory regions, suggesting a mechanism by which bottom-up emotional reactivity overwhelms top-down control processes (Kolla et al., 2023).

Another crucial limbic region in anger processing is the insula, particularly the anterior insula, which integrates interoceptive sensations with subjective emotional awareness. The insula contributes to the experience of anger by mapping bodily states, such as increased heart rate, heat sensations, or muscular tension, onto conscious awareness. Functional MRI studies reveal that anger provocation reliably activates the anterior insula, reflecting its role in processing violation of social norms, disgust-based anger, and moral outrage (Krämer et al., 2023). The insula also contributes to aggression through its involvement in pain processing and empathy; deficits in insular activation may reduce sensitivity to others' distress, increasing the likelihood of retaliatory behavior (Bertsch et al., 2023).

The hippocampus plays a more indirect role by encoding contextual memory and linking current experiences to previously stored emotionally charged events. In anger dysregulation, particularly in populations with trauma histories, the hippocampus may facilitate rapid activation of anger in situations reminiscent of past threat or violation. Impairments in hippocampal functioning, commonly observed in individuals with PTSD or severe early adversity, contribute to difficulties distinguishing present cues from past experiences, thereby heightening reactive anger (Liberzon & Abelson, 2016). Collectively,

these limbic structures form the neural foundation of anger reactivity, linking threat detection and affective arousal with somatic and contextual processing.

Prefrontal and Cingulate Regions

Top-down regulation of anger depends heavily on the integrity and functioning of the prefrontal cortex (PFC) and anterior cingulate cortex (ACC). These regions orchestrate inhibitory control, emotion regulation, decision-making, and evaluation of long-term consequences, acting as counterweights to limbic-driven reactivity.

The ventromedial prefrontal cortex (vmPFC) is particularly important for modulating anger and aggression. This region integrates emotional information, supports valuation and moral judgment, and inhibits maladaptive behavioral responses. Lesion studies demonstrate that damage to the vmPFC often results in heightened irritability, impulsive aggression, and impaired social judgment—an effect famously illustrated in classic neuropsychological cases. Contemporary imaging studies extend these findings, revealing that reduced vmPFC activation and weakened amygdala–vmPFC connectivity are associated with reactive aggression and anger regulation deficits (Kolla et al., 2023). These findings suggest that vmPFC dysfunction compromises the recalibration of emotional responses, allowing rapid limbic activation to drive behavior.

The dorsolateral prefrontal cortex (dlPFC) contributes to cognitive control, working memory, and reappraisal processes. During anger provocation, the dlPFC supports the restructuring of cognitive appraisals, enabling individuals to reinterpret triggering situations, reduce emotional intensity, and inhibit aggressive impulses. Individuals with impaired executive functioning, due to ADHD, TBI, chronic stress, or psychiatric disorders, often show deficits in dlPFC recruitment and, correspondingly, difficulty implementing anger-regulation strategies (Panasiti et al., 2023). Therapeutic interventions that target dlPFC-dependent processes, such as cognitive reappraisal or metacognitive training, often improve anger regulation by strengthening prefrontal engagement.

The anterior cingulate cortex (ACC) plays a dual role in anger regulation. The dorsal ACC facilitates conflict monitoring and error detection, enabling individuals to detect discrepancies between their goals (e.g., maintaining calm) and their emotional impulses (e.g., escalating anger). The rostral/ventral ACC contributes to emotional appraisal and modulation. In individuals with PTSD, BPD, and chronic irritability, ACC hypoactivation is frequently observed during emotional tasks, suggesting a diminished ability to detect emotional conflict or implement adaptive regulation strategies (Palmier-Claus et al., 2023). Conversely, hyperactivation of the dorsal ACC has been associated with anger rumination,

reflecting increased cognitive fixation on conflict, threat, or perceived injustice (Richard, 2023).

Collectively, prefrontal and cingulate regions function as the brain's regulatory control centers for anger. When these systems operate effectively, anger is modulated, contextualized, and behaviorally directed in adaptive ways. When compromised, dysregulated anger, marked by impulsivity, rumination, and aggression, emerges as a core clinical symptom.

Subcortical and Brainstem Systems

Beyond limbic and prefrontal regions, anger expression relies on subcortical and brainstem structures that mediate autonomic arousal, motor readiness, and defensive responses.

The hypothalamus is a central integrator of anger-related physiological arousal. It orchestrates autonomic and endocrine responses, including activation of the sympathetic nervous system and release of stress hormones, to prepare the organism for confrontation. Stimulation of specific hypothalamic nuclei induces aggressive behavior in animal models, and human neuroimaging studies implicate hypothalamic activity in anger expression and retaliatory impulses (Geniole et al., 2019). Dysregulation of hypothalamic output, often associated with chronic stress or trauma, increases physiological reactivity and lowers the threshold for anger escalation.

The periaqueductal gray (PAG), located in the midbrain, plays a critical role in coordinating fight-or-flight behaviors. The PAG receives direct projections from the amygdala and hypothalamus and modulates defensive aggression. Activation of the dorsal PAG is associated with active defensive responses, including aggressive posturing and vocalization. Dysregulation of PAG circuitry is implicated in trauma-related irritability and exaggerated reactivity, contributing to the intensity and rapid onset of anger episodes (Keay & Bandler, 2017).

The basal ganglia, including the striatum, also contribute to anger via their roles in reward processing, motivation, and action selection. Retaliation and aggressive behaviors can be reinforcing, activating striatal reward circuits that perpetuate maladaptive anger patterns. Functional MRI studies show that acts of revenge or punishment activate ventral striatal regions, suggesting that anger-driven behavior may engage reward pathways in ways that make such behavior self-reinforcing (Chester, 2017). Individuals with heightened striatal responsiveness may therefore be more vulnerable to retaliatory aggression.

Together, these subcortical and brainstem structures form a fast-response system that prepares the body for action. While essential for survival, overactivation or insufficient

inhibition of these pathways can contribute to chronic anger, impulsive aggression, and difficulty modulating physiological arousal.

Network Perspective

Modern neuroscience conceptualizes anger not merely as the product of discrete brain regions, but as emerging from interactions among large-scale neural networks. Two overarching network, the induction network and the control network, play opposing but complementary roles in anger processing.

The induction network includes the amygdala, insula, PAG, and hypothalamus. These structures generate rapid affective responses to perceived threat, injustice, or goal obstruction. Activation of this network initiates physiological arousal, aggressive impulses, and emotional intensity. Individuals with anger dysregulation often show hyperreactivity of the induction network, reflecting a low threshold for threat perception or heightened sensitivity to provocation (Richard, 2023).

On the other hand, the control network includes the vmPFC, dlPFC, ACC, and parietal regions. This network manages inhibitory control, cognitive reappraisal, and conflict monitoring. When functioning effectively, the control network reduces emotional reactivity, integrates contextual information, and generates appropriate behavioral responses.

Dysregulated anger frequently results from imbalanced connectivity between induction and control networks. Meta-analytic evidence indicates that individuals with reactive aggression exhibit stronger amygdala responses combined with reduced prefrontal modulation, a pattern suggested to underlie impulsive, emotionally driven aggression (Tonnaer et al., 2023). Similarly, reduced vmPFC–amygdala connectivity has been observed in individuals with BPD, PTSD, and IED, reflecting impaired integration of emotional cues with regulatory processes (Kolla et al., 2023).

The network model has significant implications for psychotherapy. Interventions that enhance prefrontal engagement, such as cognitive reappraisal, mindfulness, or metacognitive training, strengthen the control network. Interventions that reduce physiological arousal, such as paced breathing, HRV biofeedback, or grounding, reduce induction-network activation. Thus, treatment can be conceptualized as recalibrating the dynamic balance between bottom-up emotional induction and top-down regulation.

Neurochemistry and Physiological Correlates of Anger

Monoamines and Aggression

Anger and aggression are shaped by complex neurochemical processes, with monoamine systems, particularly serotonin, dopamine, and norepinephrine, playing

central regulatory roles. Among these, serotonin (5-HT) has been the most extensively studied. Serotonergic dysfunction is strongly associated with impulsive aggression, a finding consistently supported across neuroimaging, cerebrospinal fluid (CSF), and pharmacological challenge studies (Dahlgren et al., 2023). Reduced 5-HT synthesis and impaired binding in prefrontal regions are associated with decreased inhibitory control and heightened reactivity to provocation. Selective serotonin reuptake inhibitors (SSRIs) have demonstrated reductions in irritability and impulsive aggression across various clinical populations, further supporting a serotonergic mechanism in anger regulation (Coccaro & Lee, 2023). Importantly, serotonin's effects are not uniform; while low serotonergic functioning increases vulnerability to reactive aggression, high serotonergic tone may contribute to perseverative or ruminative anger, underscoring the complexity of this neuromodulatory system.

Dopamine also plays a meaningful role in anger, particularly in the rewarding and motivational aspects of aggressive behavior. Retaliatory aggression activates dopaminergic regions of the ventral striatum, highlighting that anger-driven acts can be intrinsically reinforcing (Chester, 2017). Elevated dopamine reactivity is associated with novelty seeking, impulsivity, and heightened sensitivity to perceived provocation (Bertsch et al., 2023). Dopaminergic dysregulation is especially prominent in neurodevelopmental conditions (e.g., ADHD) and in substance use disorders, wherein stimulant-induced dopamine surges lower thresholds for anger and aggressive outbursts.

Norepinephrine (NE), the primary neurotransmitter of the sympathetic nervous system, modulates arousal, vigilance, and stress responses. Elevated noradrenergic activity contributes to physical symptoms of anger, including increased heart rate, sweating, and muscle tension, by driving sympathetic activation and narrowing attentional focus to perceived threats (Gerlicher et al., 2023). Hypernoradrenergic states are especially common in PTSD, where exaggerated startle responses and hypervigilance prime individuals for rapid anger escalation. Pharmacologic agents that reduce noradrenergic signaling, such as propranolol, have been shown to decrease physiological arousal and dampen anger reactivity in select populations.

Collectively, monoamine systems influence anger through their effects on arousal, motivation, inhibition, and reward processing. Serotonin primarily modulates inhibition, dopamine shapes motivational salience and reinforcement, and norepinephrine amplifies sympathetic arousal. These systems interact dynamically, and dysregulation in any one system can shift the emotional and behavioral expression of anger.

Stress Systems and the HPA Axis

Stress physiology is deeply intertwined with anger expression, particularly through the functioning of the hypothalamic-pituitary-adrenal (HPA) axis. Acute anger activates both the sympathetic-adrenal-medullary system and the HPA axis, resulting in rapid release of catecholamines followed by cortisol secretion. Cortisol is essential for mobilizing energy and restoring homeostasis; however, chronic activation of the HPA axis can lead to maladaptive physiological and emotional outcomes. Individuals with chronic anger or aggressive tendencies often exhibit altered cortisol profiles. For example, atypical cortisol awakening responses and blunted diurnal cortisol slopes have been observed in individuals with persistent irritability or impulsive aggression (Ramos et al., 2023). These atypical patterns may reflect chronic stress load, impaired recovery after anger episodes, or dysregulated feedback within the HPA axis.

In PTSD, a condition frequently marked by anger dysregulation, HPA axis disturbances are well documented. Some studies report lower basal cortisol, while others identify exaggerated cortisol reactivity to trauma reminders. Regardless of directionality, these findings suggest impaired stress regulation, which may predispose individuals to rapid anger mobilization when confronted with cues of threat or injustice (Palmier-Claus et al., 2023). From a physiological standpoint, anger is associated with heightened cardiovascular activation. Increased blood pressure, elevated heart rate, and reduced heart rate variability (HRV) are common responses to anger provocation. Reduced HRV, a marker of compromised parasympathetic functioning, is particularly relevant, as it reflects diminished capacity for flexible emotional regulation. Interventions such as HRV biofeedback and paced breathing directly target this physiological dimension and are increasingly used in anger treatment to support autonomic regulation (Lehrer & Gevirtz, 2023).

Overall, dysregulation in the HPA axis contributes to heightened emotional volatility, prolonged arousal, and vulnerability to anger dysregulation. Understanding these biological underpinnings is essential in developing integrated psychotherapeutic approaches.

Hormonal and Inflammatory Markers

Beyond monoamines and stress hormones, several other biological systems influence anger, including sex hormones and inflammatory pathways. Testosterone, often implicated in aggression, exerts complex effects that depend on social context, dominance motivation, and emotion-regulation capacity. Recent studies emphasize that testosterone does not universally promote aggression; instead, it increases sensitivity to status threats and enhances approach motivation, which may heighten anger reactivity in individuals with

poor regulatory control (Geniole et al., 2020). The interaction between testosterone and cortisol, known as the dual-hormone hypothesis, has gained empirical traction. High testosterone coupled with low cortisol predicts aggressive behavior more robustly than testosterone alone (Montoya et al., 2023).

Oxytocin, commonly viewed as an affiliative hormone, also plays context-dependent roles in anger and aggression. Under conditions of perceived threat to close relationships, oxytocin can amplify reactive aggression and intensify anger responses toward out-group individuals (Shamay-Tsoory & Abu-Akel, 2016). These findings complicate simplistic narratives about oxytocin as universally prosocial.

Emerging research highlights the importance of inflammatory processes in irritability, aggression, and affective dysregulation. Systemic inflammation, indexed by markers such as C-reactive protein (CRP) or interleukin-6 (IL-6), has been associated with chronic irritability, anger attacks, and aggression, particularly in individuals with mood disorders (Dougherty et al., 2023). Inflammation may increase anger reactivity through effects on neurotransmitter metabolism, neural plasticity, and limbic-prefrontal connectivity. For example, inflammatory cytokines reduce serotonin availability and alter dopamine signaling, thereby impacting both inhibition and reward-related anger pathways.

These biological insights suggest several directions for psychotherapy integration, such as incorporating psychoeducation about the physiological contributors to anger, addressing lifestyle factors that modulate inflammation (e.g., sleep, diet, exercise), and exploring interdisciplinary treatment approaches involving primary care or psychiatry.

Somatic and Autonomic Signatures of Anger

Anger manifests not only as a subjective emotional state but also as a constellation of somatic and autonomic responses that prepare the body for action. Understanding these physiological signatures can help clinicians design more targeted interventions.

Anger provocation reliably increases sympathetic nervous system activity, leading to elevated heart rate, increased blood pressure, and heightened muscular tension. These bodily responses contribute to the subjective feeling of anger, often described as "hot," "tight," or "energized." Research shows that individuals with chronic anger have elevated baseline sympathetic tone, which may reduce their threshold for emotional reactivity (Gerlicher et al., 2023).

Perhaps more important clinically is heart rate variability (HRV), a measure of parasympathetic modulation. Low HRV is associated with emotional rigidity, poor stress recovery, and heightened vulnerability to anger outbursts. Studies demonstrate that individuals with low HRV exhibit greater amygdala reactivity and weaker prefrontal control

during anger-inducing tasks, suggesting a direct physiological pathway to dysregulation (Lehrer & Gevirtz, 2023). Consequently, HRV biofeedback, paced breathing, and mindfulness-based interventions are increasingly used to support autonomic flexibility and reduce anger intensity.

Another important somatic correlate is facial electromyography (EMG), particularly increased activation of the corrugator supercilii muscle, associated with frowning, during anger. Although primarily a research tool, facial EMG underscores the embodied nature of anger and its integration of cognitive, affective, and motor processes (Krämer et al., 2023).

Somatic awareness is therefore crucial in psychotherapy for anger. Clients frequently lack awareness of the early physiological cues of anger escalation; interventions such as interoceptive training, body scans, and somatic grounding help cultivate recognition of these cues, thereby increasing opportunity for early regulation.

Developmental and Individual Differences

Anger and aggression have substantial heritable components, reflecting the complex interplay between genetic predispositions and environmental influences. Twin studies estimate that approximately 40–50% of the variance in aggressive and irritability-related traits is attributable to genetic factors (Porsch et al., 2023). Genome-wide association studies (GWAS) and candidate-gene approaches have identified several polymorphisms associated with anger reactivity, impulsivity, and aggression-related phenotypes. Among the most well-studied is the monoamine oxidase A (MAOA) gene, which encodes an enzyme critical for the metabolism of monoamines. Low-activity MAOA variants (MAOA-L) are linked with greater amygdala reactivity, reduced prefrontal regulation, and heightened susceptibility to impulsive aggression, although only in combination with adverse developmental environments (Whelan et al., 2023).

Beyond MAOA, polymorphisms in serotonin transporter genes (e.g., 5-HTTLPR), dopamine receptors (e.g., DRD4), and noradrenergic genes have been associated with variation in anger proneness and emotional volatility (Coccaro & Lee, 2023). These genetic influences rarely exert direct effects; instead, they interact with environmental experiences to shape neural circuitry. Individuals with genetically mediated differences in amygdala responsiveness or prefrontal control may be more sensitive to interpersonal stressors, more reactive to provocation, or less capable of modulating anger.

Importantly, the field has shifted from searching for single "aggression genes" to understanding polygenic risk, multifactorial combinations of small genetic effects. Polygenic models explain how individuals vary in anger thresholds, stress reactivity, and vulnerability to dysregulation. These insights underscore the importance of

biopsychosocial formulations that integrate neurobiological predispositions with learning history, trauma exposure, and current environmental demands.

Early Adversity, Trauma, and Attachment

Early childhood experiences profoundly shape the neural systems governing anger. Trauma, inconsistent caregiving, and attachment disruptions are associated with alterations in amygdala-prefrontal circuitry, heightened physiological reactivity, and chronic emotion-regulation difficulties (Marusak et al., 2023). Children exposed to maltreatment often show hypervigilance, rapid threat detection, and difficulty downregulating emotional arousal, patterns that persist into adulthood and contribute to anger dysregulation.

Trauma-related changes in the amygdala include increased volume and heightened reactivity, while chronic stress exposure is associated with reduced hippocampal volume, impairing contextual modulation of emotional responses (Heberlein et al., 2023). These neurobiological alterations contribute to the common clinical presentation of anger in trauma survivors, who may experience sudden and intense anger in response to cues that resemble past threat or injustice. In PTSD, deficits in prefrontal regulatory regions and the ACC further exacerbate anger, irritability, and aggressive impulses (Palmier-Claus et al., 2023).

Attachment experiences also exert powerful influences on anger development. Insecure attachment styles, particularly anxious and disorganized attachment, are associated with increased anger expression, heightened sensitivity to rejection, and difficulty regulating interpersonal emotions. Neuroimaging studies suggest that attachment insecurity contributes to reduced prefrontal activation during social conflict and increased activation of limbic and salience networks (De Winter et al., 2023). Insecurely attached individuals may interpret ambiguous interpersonal cues as threatening or rejecting, creating fertile ground for anger escalation.

Conversely, secure attachment promotes development of robust regulatory capacities. Through consistent and responsive caregiving, children learn to modulate physiological arousal, integrate emotional experiences, and develop reflective functioning, skills that support adaptive anger expression later in life. Thus, early adversity and attachment shape both neural architecture and psychological processes that contribute to anger-related vulnerabilities.

Sex and Gender Differences

Although anger is often stereotypically associated with men, empirical findings reveal a more nuanced picture. Sex differences in anger expression likely reflect a combination of

neurobiological, hormonal, and sociocultural influences rather than innate emotional disparities.

Neuroimaging research demonstrates sex-based variation in the neural circuits underlying emotional processing. Women often show heightened activation in limbic structures, including the amygdala and insula, during negative emotional stimuli, while men tend to show greater activation in prefrontal and parietal control regions during anger provocation (Mohammadi et al., 2023). These differences may relate to divergent emotion-regulation strategies: men may engage more in suppression or cognitive control, whereas women may experience more internalized anger or somatic distress.

Hormonal influences also play a significant role. Testosterone is linked with dominance behavior, threat sensitivity, and approach motivation, which may contribute to anger-related behaviors in men. However, testosterone alone does not predict aggression; instead, its effects depend on social context and cortisol levels (Montoya et al., 2023). Women, on the other hand, may exhibit heightened anger responses during hormonal fluctuations, such as in premenstrual dysphoric disorder (PMDD), where estrogen-progesterone interactions modulate serotonergic and GABAergic pathways associated with irritability and emotional lability (Osman et al., 2023).

Socialization patterns further moderate anger expression. Boys are often encouraged to express anger outwardly, whereas girls may be discouraged from open anger expression, leading to internalized anger, irritability, or relational aggression. Over time, socialization may shape neural and behavioral patterns, influencing how men and women experience, express, and regulate anger.

Understanding sex and gender differences is clinically relevant, as it guides tailored interventions. For example, emotion-regulation skills or interpersonal strategies may need to be adapted to account for gendered socialization, trauma exposure patterns, or hormonal influences on mood and anger.

Neurodevelopmental and Neurocognitive Factors

Neurodevelopmental conditions, cognitive capacity, and executive functioning significantly influence anger regulation. Attention-deficit/hyperactivity disorder (ADHD), characterized by impulsivity, emotional dysregulation, and executive functioning deficits, is strongly associated with anger difficulties. Neural models suggest that individuals with ADHD exhibit impaired prefrontal control and heightened amygdala reactivity, contributing to rapid anger escalation and difficulty inhibiting aggressive impulses (Shaw et al., 2023). Treatments that target executive skills, such as behavioral interventions, stimulants, or

mindfulness-based programs, often improve anger outcomes by strengthening regulatory capacity.

Traumatic brain injury (TBI) is another major contributor to anger dysregulation. Damage to prefrontal regions disrupts inhibitory control, social judgment, and emotion regulation. Individuals with TBI often show irritability, impulsive anger, and reduced frustration tolerance; these symptoms reflect both neural damage and alterations in white-matter connectivity that impede efficient communication between regulatory and limbic systems (Dabek et al., 2023).

Autism spectrum disorder (ASD) presents unique challenges in anger regulation. Sensory sensitivities, social communication differences, and difficulty interpreting social cues can lead to intense frustration and anger. Neurobiologically, ASD is associated with atypical functioning in salience and interoceptive networks, including the insula, ACC, and amygdala (Uljarević et al., 2023). Interventions for individuals with ASD often require structured routines, sensory accommodations, and explicit teaching of emotion-recognition and coping strategies.

More broadly, executive functioning, including working memory, cognitive flexibility, and inhibitory control, predicts anger regulation capacities across clinical and nonclinical populations. Poor executive functioning impairs individuals' ability to reappraise situations, shift attention away from provocations, and inhibit retaliatory impulses. Psychotherapeutic interventions that strengthen these skills, such as cognitive remediation or DBT distress-tolerance training, can improve anger outcomes by enhancing top-down regulatory processes.

<div align="center">Anger Across Diagnoses: Neurobiological Phenotypes</div>

Intermittent Explosive Disorder (IED) and Impulsive Aggression

Intermittent Explosive Disorder (IED) is the diagnostic condition most centrally defined by recurrent anger outbursts and impulsive aggression. Neurobiological models of IED highlight severe deficits in prefrontal inhibitory control, excessive limbic reactivity, and dysregulated autonomic responses. Functional neuroimaging studies consistently show hyperactivation of the amygdala during anger provocation combined with reduced activation in the ventromedial and dorsolateral prefrontal cortices, suggesting impaired top-down regulation of emotional impulses (Coccaro et al., 2023). Additionally, individuals with IED exhibit reduced amygdala–prefrontal connectivity, indicating a weakened regulatory pathway that normally inhibits impulsive anger (Tonnaer et al., 2023).

IED is also associated with abnormalities in serotonergic functioning, including decreased 5-HT2A receptor binding and blunted serotonergic responsivity during

provocation tasks (Coccaro & Lee, 2023). These disturbances align with the well-established role of serotonin in impulse control and aggression inhibition. Autonomic hyperarousal is another defining feature of IED. Individuals with IED display heightened sympathetic activation, lower heart rate variability (HRV), and prolonged physiological arousal after anger episodes (Lee et al., 2024), suggesting difficulties achieving physiological recovery.

Clinical trials reinforce these neurobiological findings. Cognitive-behavioral therapy (CBT) targeting anger triggers, cognitive appraisal, and impulse regulation is one of the most effective treatments for IED. In randomized trials, CBT significantly reduces aggression frequency and intensity compared to supportive therapy or treatment as usual (McCloskey et al., 2023). These improvements likely arise from strengthening prefrontal cognitive control mechanisms and enhancing executive functioning, consistent with neurobiological models. Pharmacologic interventions, particularly SSRIs, also attenuate impulsive aggression in IED, further supporting its serotonergic basis (Coccaro & Lee, 2023).

Overall, IED represents a phenotype of reactive, impulsive anger characterized by rapid emotional escalation, impaired inhibitory control, and deficient serotonergic modulation. These features distinguish IED from disorders involving chronic or ruminative anger and underscore the importance of interventions that target cognitive control and autonomic regulation.

Anger in PTSD and Complex Trauma

Anger is a core symptom cluster in Posttraumatic Stress Disorder (PTSD), often arising from hypervigilance, moral injury, betrayal trauma, and dysregulated threat responses. Neurobiologically, PTSD is characterized by hyperresponsivity of the amygdala, reduced hippocampal volume, and hypoactivation of the prefrontal cortex, especially the ventromedial and anterior cingulate regions (Heberlein et al., 2023). These alterations create a system primed for rapid threat detection but impaired in differentiating real from perceived danger, conditions that foster anger, irritability, and aggressive impulses.

In PTSD, anger often emerges from moral injury, where trauma violates one's core values, or from appraisals of injustice associated with interpersonal or systemic betrayal. Neurobiological models propose that anger in PTSD is maintained by heightened activation of the salience network (amygdala, insula) and deficient engagement of cognitive-control networks (ACC, prefrontal regions) during emotionally charged contexts (Palmier-Claus et al., 2023). This imbalance contributes to the characteristic explosive anger and emotional volatility in trauma survivors.

Complex trauma adds additional layers of dysregulation. Individuals exposed to chronic childhood maltreatment may exhibit persistent hypervigilance, rapid anger escalation, and difficulty modulating emotional arousal. Neurodevelopmental impacts of early trauma, such as reduced connectivity between the amygdala and prefrontal cortex, shape lifelong patterns of anger dysregulation (Marusak et al., 2023).

Trauma-focused interventions (e.g., EMDR, trauma-focused CBT) often reduce anger by resolving trauma memories, recalibrating threat detection systems, and strengthening emotion-regulation networks. However, research indicates that treating PTSD alone does not reliably improve chronic anger, and additional anger-focused interventions may be required (van der Veen et al., 2023). Skills-based modalities (DBT, MBCT, HRV biofeedback) can complement trauma therapy by directly targeting physiological arousal and emotional regulation deficits.

Borderline Personality Disorder (BPD) and Chronic Anger

Borderline Personality Disorder (BPD) is characterized by emotional instability, interpersonal sensitivity, and intense, rapidly shifting anger. Unlike the impulsive aggression of IED, anger in BPD is often chronic, relationally driven, and intertwined with shame, abandonment fears, and identity dysregulation.

Neurobiologically, BPD is associated with heightened amygdala reactivity, reflecting exaggerated sensitivity to perceived rejection or interpersonal threat. At the same time, individuals with BPD exhibit reduced activation in prefrontal control regions, including the dorsolateral prefrontal cortex and ACC, during emotional tasks (Kolla et al., 2023). This neural profile suggests a core deficit in integrating emotional cues with cognitive regulation strategies, making anger difficult to modulate.

A distinguishing feature of BPD anger is its affective lability, reflecting rapid oscillations between intense anger, fear, shame, and despair. This pattern is supported by altered functioning of the insula and default mode network (DMN), which contribute to disrupted self-referential processing and unstable identity (Uddin et al., 2023). These disturbances amplify interpersonal sensitivity, leading to misinterpretation of ambiguous cues as hostile or rejecting.

Emotion-regulation deficits in BPD have strong biological underpinnings as well. Reduced gamma-aminobutyric acid (GABA) signaling, impaired oxytocin modulation, and increased HPA-axis reactivity contribute to heightened emotional arousal and impulsivity (Palmier-Claus et al., 2023). These neurochemical vulnerabilities worsen under interpersonal stress, increasing anger dysregulation.

Dialectical Behavior Therapy (DBT), the gold-standard treatment for BPD, is particularly effective in reducing anger intensity and frequency. DBT strengthens prefrontal regulatory functioning by teaching distress tolerance, mindfulness, and interpersonal skills. Neuroimaging studies suggest that DBT enhances activation in prefrontal and ACC regions, contributing to improved emotional regulation (Schaefer et al., 2023). Schema therapy and mentalization-based therapy (MBT) also reduce anger by improving reflective functioning and addressing maladaptive interpersonal schemas.

Psychosis, Mood Disorders, and Substance Use Disorders

Anger and aggression in psychotic disorders may arise from paranoid ideation, threat misperception, command hallucinations, or impaired cognitive control. Neurobiologically, psychotic-spectrum conditions involve disruptions in salience processing, with excessive dopamine activity contributing to aberrant threat attribution. Individuals with schizophrenia may show heightened amygdala activation to neutral stimuli, reflecting impaired discrimination of emotional cues (Fett et al., 2023). When coupled with executive dysfunction, these perceptual distortions increase the likelihood of anger-driven behaviors.

CBT for psychosis has demonstrated moderate reductions in aggression and anger by addressing misinterpretations, paranoid thinking, and arousal (Ballesteros et al., 2023). Antipsychotic medications reduce anger by modulating dopaminergic overactivity, further confirming dopamine's role in aggression within psychosis.

Mood Disorders

Anger and irritability are frequently observed in major depressive disorder (MDD), especially in irritable depression, where anger serves as a manifestation of negative affect and emotional pain. Neuroimaging research suggests that individuals with irritable depression exhibit heightened amygdala and insula activation, combined with diminished prefrontal recruitment during emotional tasks (Mohammadi et al., 2023). These findings parallel those observed in BPD and PTSD, reinforcing the role of emotion-regulation deficits across affective disorders.

In bipolar disorder, anger is especially prominent during mixed states and manic episodes, where dysregulated dopaminergic activity and heightened physiological arousal amplify irritability and explosive anger. Functional studies reveal abnormalities in limbic-prefrontal networks similar to those in impulsive aggression, although driven by distinct mood-related mechanisms (Palmier-Claus et al., 2023).

Substance Use Disorders

Substance use significantly alters neural mechanisms governing anger, particularly through effects on reward, inhibition, and arousal systems. Alcohol reduces prefrontal inhibitory control and increases aggression by potentiating GABAergic inhibition and dampening glutamatergic signaling. Stimulants such as methamphetamine and cocaine increase dopaminergic activity, heightening irritability, impulsivity, and reactive anger. Chronic substance use also impairs serotonergic functioning, increasing vulnerability to anger dysregulation (Coccaro & Lee, 2023).

Treatment for substance-related anger often requires integrated approaches addressing both emotion regulation and substance use. CBT for substance use, DBT skills, and relapse prevention strategies help reduce anger by restoring regulatory capacity and decreasing physiological instability.

Assessment and Case Formulation of Anger Dysregulation

Assessment of anger dysregulation requires a multimodal, integrative approach that captures the cognitive, affective, behavioral, physiological, and interpersonal dimensions of anger. Unlike symptoms that can be quantified solely through self-report, anger is deeply contextual and shaped by situation-specific appraisals, relational dynamics, and neurobiological factors. Clinicians must therefore gather information through structured and unstructured interviews, validated assessment instruments, behavioral observations, and collateral reports when appropriate. A thorough clinical interview should explore the onset, intensity, frequency, duration, and functional impact of anger episodes. Particular attention should be given to antecedent conditions, including perceived injustice, interpersonal triggers, cognitive interpretations, and physiological cues preceding escalation. Individuals often describe increasing muscular tension, heat sensations, or narrowed attentional focus prior to explosive anger; yet many remain unaware of these early signs. Exploring both overt expressions of anger and subtler forms, such as passive-aggressive behavior, simmering resentment, or suppressed irritability, provides a more complete clinical picture.

Psychometrically validated measures contribute meaningfully to the assessment process. Instruments such as the State–Trait Anger Expression Inventory-2 (STAXI-2) quantify state anger, trait anger, anger expression, anger suppression, and anger control. These dimensions allow clinicians to differentiate between individuals prone to chronic anger versus those who experience episodic but intense episodes. Other widely used measures include the Buss–Perry Aggression Questionnaire, which evaluates physical aggression, verbal aggression, anger, and hostility, and the Anger Rumination Scale, which assesses the cognitive perseveration that often prolongs anger states. These instruments demonstrate utility across both clinical and community samples and correlate strongly

with neurobiological markers of anger, including amygdala reactivity and reduced prefrontal regulation (Tonnaer et al., 2023). Incorporating these tools enables clinicians to track progress across treatment, benchmark symptom severity, and identify domains requiring targeted intervention.

Behavioral observation remains essential, particularly when clients minimize or lack awareness of their anger patterns. Subtle cues such as increased vocal intensity, agitation, clenched fists, or abrupt shifts in facial expression may emerge during emotionally evocative topics. At times, avoidance or emotional numbing may mask underlying anger, especially among individuals with trauma histories or internalized anger schemas. Collateral reports from partners, family members, or workplace supervisors may provide invaluable insight into interpersonal dynamics and consequences that clients fail to recognize or report. In cases involving potential violence, self-harm, or risk to others, gathering collateral information becomes not only beneficial but ethically necessary.

Formulation of anger dysregulation should integrate biological predispositions, cognitive appraisals, emotional processes, behavioral patterns, interpersonal schemas, and environmental contexts into a coherent conceptual model. At the neurobiological level, clinicians should consider whether patterns of anger reflect heightened limbic reactivity, impaired prefrontal control, autonomic hyperarousal, or neurodevelopmental factors such as ADHD or TBI. Cognitive contributors often include hostile attribution bias, black-and-white thinking, personalization, and catastrophic interpretations of interpersonal events. Emotionally, many individuals experience rapid transitions from vulnerable affective states, such as shame, fear, or sadness, into anger, which functions as a protective secondary emotion. Understanding this emotional layering helps clinicians address the underlying distress rather than solely the outward expression of anger.

Behavioral patterns also play a critical role in case formulation. Recurrent avoidance of conflict may contribute to anger buildup, while aggressive outbursts may provide immediate emotional relief that reinforces maladaptive expression. Clients who ruminate on perceived slights or injustices often experience prolonged anger episodes driven by sustained physiological arousal and cognitive fixation. Interpersonal dynamics further shape anger responses. In attachment-disrupted individuals, anger may emerge in the context of perceived abandonment or rejection, while in individuals with narcissistic traits, anger may reflect threats to self-esteem or identity. Identifying these relational schemas helps tailor interventions to the core drivers of dysregulation.

Environmental and sociocultural factors add additional layers to formulation. High-stress environments, inconsistent parenting, workplace coercion, or ongoing trauma exposure may serve as chronic triggers. Cultural norms surrounding anger expression, such as

gendered expectations, familial communication styles, or societal stigmatization, also influence how anger manifests. Clinicians must assess whether anger is contextually disproportionate, culturally mediated, or adaptive within specific circumstances. For example, anger in response to ongoing injustice or systemic oppression requires a different formulation approach than anger emerging in intimate relationships or low-stakes interpersonal contexts.

Risk assessment is an indispensable component of anger evaluation. Anger dysregulation can contribute to heightened risk of interpersonal violence, property destruction, self-directed harm, and legal or occupational consequences. Risk assessment should explore not only prior aggressive acts but also current ideation, access to weapons, substance use, stress load, and co-occurring psychiatric symptoms such as paranoia or impulsivity. Acute risk may be signaled by sudden changes in behavior, escalating interpersonal conflicts, or loss of inhibitory control. Chronic risk may involve longstanding patterns of explosive anger, poor impulse regulation, or antisocial behaviors. Clinicians should integrate structured tools (e.g., HCR-20 for violence risk in forensic contexts) with clinical judgment, recognizing that anger must be assessed within the broader context of personality traits, trauma history, and situational pressures.

Finally, an effective case formulation synthesizes these multidimensional assessment findings into a guiding narrative that informs treatment planning. A comprehensive formulation identifies primary mechanisms driving anger (e.g., limbic hyperreactivity, cognitive distortions, attachment wounds, executive function deficits), maintenance factors (e.g., rumination, reinforcement cycles, relational instability), and protective factors (e.g., insight, social support, coping skills). The formulation should provide a clear rationale for chosen interventions, for example, selecting DBT for individuals with high emotional reactivity and poor distress tolerance, CBT for those with cognitive distortions and misinterpretations, or mindfulness-based approaches for those with rumination-driven anger. Formulation also guides pacing, indicating whether preparatory skills (e.g., autonomic regulation, grounding) must precede trauma-focused or cognitive restructuring work. Ultimately, a well-developed formulation not only clarifies the clinical picture but also enhances collaboration, motivation, and therapeutic coherence for both client and clinician.

Psychotherapy Interventions Informed by Anger Neurobiology

Cognitive-behavioral approaches represent the most empirically supported class of psychotherapeutic interventions for anger dysregulation. Their strong evidence base reflects a close alignment with the underlying neurobiology of anger, particularly the circuits involving cognitive appraisal, threat interpretation, executive functioning, and

inhibitory control. Cognitive-behavioral therapy (CBT) directly targets the cognitive and behavioral mechanisms that maintain maladaptive anger responses, including hostile attribution biases, ruminative processing, misinterpretation of interpersonal cues, and impulsive action tendencies. These mechanisms correspond to neurobiological findings showing heightened amygdala activation, reduced prefrontal regulation, and altered connectivity between limbic and executive networks in individuals with chronic or impulsive anger (Tonnaer et al., 2023).

At its core, CBT for anger begins with psychoeducation about the nature of anger as an emotion of perceived violation or goal obstruction, illustrating how anger arises through a combination of physiological arousal, cognitive meaning-making, and behavioral impulses. Psychoeducation supports normalization and fosters client engagement by framing anger as a modifiable neurobiological and psychological process. Clients learn that heightened limbic reactivity and rapid amygdala responses can produce instantaneous anger, but that cognitive framings, such as assumptions about intention, fairness, or threat, determine the intensity and direction of anger. This psychoeducational foundation is critical for helping clients reappraise their symptoms and motivates the acquisition of regulatory strategies.

A central focus of CBT interventions for anger is cognitive restructuring, which addresses maladaptive thoughts that amplify emotional arousal. Clients are taught to identify automatic thoughts that occur during escalating anger, such as beliefs that others are intentionally disrespectful, unjust, or malicious. These cognitions often reflect hostile attribution biases, which are well-documented in anger-prone individuals (Vitiello et al., 2023). Cognitive restructuring aims to evaluate evidence for and against these interpretations, generate alternative explanations, and reduce the moral intensity or personal significance assigned to triggering events. Neuroimaging studies support the utility of this approach, showing that cognitive reappraisal increases activation in the dorsolateral prefrontal cortex (dlPFC) and ventromedial prefrontal cortex (vmPFC), while reducing amygdala reactivity during anger provocation (Kolla et al., 2023). This enhanced top-down control is consistent with the neural mechanisms underlying effective anger regulation.

Another key component of CBT is identification of anger triggers through functional analysis or "chain analysis." Clients examine the antecedents, internal states, behaviors, and consequences associated with anger episodes. This process highlights specific interpersonal cues, sensory triggers, or cognitive vulnerabilities that precipitate dysregulation. By mapping the sequence of events, clients gain insight into early warning signs, such as muscle tension, rapid heartbeat, or narrowed attention, that signal rising arousal. Early intervention at these points can prevent escalation, and behavioral

experiments can be used to help clients test alternative responses. Functional chain analysis aligns with neuroscientific models emphasizing the importance of early detection of physiological arousal in preventing full-scale activation of limbic networks (Gerlicher et al., 2023).

Behavioral interventions complement cognitive strategies by targeting the actions and physiological reactions associated with anger. Skills such as problem-solving, assertive communication, and exposure-based rehearsal are integral to CBT protocols. Problem-solving training helps clients slow down impulsive responses, consider multiple solutions, evaluate consequences, and select adaptive behaviors. Assertiveness training addresses common patterns of passive avoidance or aggressive confrontation by teaching clients to express needs clearly and respectfully without escalation. These behavioral strategies strengthen prefrontal networks implicated in planning, decision making, and response inhibition.

Physiological regulation techniques are also essential within CBT frameworks. Clients often learn relaxation skills, such as diaphragmatic breathing, progressive muscle relaxation, and grounding exercises, which reduce sympathetic activation and lower physiological arousal. Such techniques help recalibrate autonomic functioning and increase heart rate variability (HRV), thereby enhancing regulatory capacity (Lehrer & Gevirtz, 2023). By modulating bottom-up arousal, these methods allow cognitive strategies to be more effective and reduce the timing gap between trigger and response, decreasing the likelihood of impulsive anger.

Anger-focused CBT has demonstrated significant effectiveness across multiple clinical trials and populations. For individuals with Intermittent Explosive Disorder (IED), CBT reduces the frequency and intensity of aggressive outbursts, improves inhibitory control, and enhances emotion-regulation skills. A recent randomized controlled trial demonstrated that a 12-session anger-focused CBT protocol produced greater reductions in aggression than supportive therapy, with effects maintained at follow-up (McCloskey et al., 2023). In correctional and forensic populations, CBT-based anger management programs consistently reduce violent behaviors, institutional infractions, and recidivism (Ballesteros et al., 2023). These findings underscore the broad applicability of CBT across clinical contexts and severity levels.

The use of imaginal and in vivo exposure techniques within CBT further strengthens clients' regulatory skills. Exposure to anger-provoking memories or hypothetical scenarios allows clients to practice cognitive reappraisal, behavioral alternatives, and physiological regulation in controlled conditions. Repeated exposure helps weaken the associative links between triggers and maladaptive responses, reducing the salience of anger cues over

time. Neurobiological research supports the use of exposure as a mechanism for recalibrating amygdala responsiveness and increasing regulatory network activation (Marusak et al., 2023). Importantly, exposure techniques are carefully tailored to avoid reinforcing aggressive scripts and instead emphasize the mastery of nonaggressive coping strategies.

In addition to traditional in-person CBT, internet-based CBT (iCBT) has emerged as an effective and accessible intervention for anger dysregulation. Therapist-supported digital programs offer structured modules on cognitive restructuring, coping skills, and behavioral experiments. Recent trials show that iCBT reduces anger severity and improves emotion regulation among adults with maladaptive anger, with effect sizes comparable to face-to-face therapy (Bjureberg et al., 2023). Digital delivery enhances accessibility for individuals reluctant to seek in-person treatment or those living in underserved areas. Importantly, iCBT appears to be particularly effective when programs include real-time feedback, interactive exercises, and personalized guidance.

Together, cognitive-behavioral interventions form an integrated system of strategies that target both the psychological and neurobiological roots of anger dysregulation. By modifying cognitive appraisals, strengthening prefrontal control, interrupting behavioral patterns, and reducing physiological arousal, CBT provides a comprehensive and empirically supported framework for treating anger across diverse clinical populations.

Mindfulness, Acceptance, and Affect-Labeling Interventions

Mindfulness-based, acceptance-based, and affect-labeling interventions offer a complementary set of strategies for treating anger dysregulation, targeting mechanisms that differ in meaningful ways from traditional cognitive-behavioral approaches. Whereas CBT emphasizes reappraisal and cognitive restructuring, mindfulness and acceptance-based treatments focus on cultivating nonjudgmental awareness, reducing emotional avoidance, and altering one's relationship to internal experiences. These approaches align with contemporary neuroscientific models of anger, particularly those emphasizing limbic hyperreactivity, attentional narrowing, and reduced prefrontal modulation. By increasing awareness of bodily cues, decoupling automatic appraisals from behavioral impulses, and decreasing experiential avoidance, mindfulness and acceptance practices enhance emotion-regulation capacity and disrupt habitual anger-driven responses.

Mindfulness-based interventions draw heavily from practices that cultivate deliberate attention to present-moment experience, including breath awareness, body scanning, open-monitoring meditation, and nonreactive observation of thoughts and emotions. One of the primary mechanisms by which mindfulness reduces anger involves attenuating

automaticity. Anger episodes often unfold rapidly because attentional processes narrow around perceived threats or injustices. Mindfulness broadens attentional scope, allowing individuals to observe the early physiological and cognitive signals of anger—such as muscle tension, rising heat, or escalating hostile thoughts, before full emotional activation occurs. Neuroimaging studies show that mindfulness reduces amygdala activation and increases activation of regulatory regions, including the ventromedial prefrontal cortex (vmPFC) and anterior cingulate cortex (ACC), thereby supporting more flexible and adaptive responses (Krämer et al., 2023).

Another mechanism by which mindfulness improves anger regulation involves decentering, the ability to observe internal experiences from a psychological distance rather than becoming fused with them. Decentering facilitates a shift from "I am angry" to "I am noticing anger sensations," which weakens the automatic impulse toward reactive behavior. This shift aligns with neural findings showing enhanced connectivity between prefrontal and limbic regions during mindfulness practice, supporting greater integration between emotional and cognitive networks (Farb et al., 2023). In the context of anger, decentering reduces the intensity of emotional arousal by altering the interpretive lens through which anger-eliciting stimuli are viewed.

Mindfulness-based cognitive therapy (MBCT) and mindfulness-based stress reduction (MBSR) have demonstrated significant benefits for individuals with high anger, irritability, or emotional reactivity. MBCT integrates cognitive strategies with mindfulness to reduce the internal amplification processes that sustain anger, such as rumination. Rumination is particularly relevant for anger dysregulation, as individuals may repeatedly dwell on perceived wrongs or injustices, thereby maintaining elevated autonomic arousal and prolonging emotional distress. Mindfulness disrupts these ruminative cycles by teaching individuals to shift attention away from repetitive, threat-oriented thinking and toward present-moment somatic experience (Bjureberg et al., 2023). Structural and functional imaging studies support these clinical findings, showing reductions in default mode network (DMN) activity, associated with rumination, and increases in attentional network activation following mindfulness training (Marusak et al., 2023).

Acceptance and Commitment Therapy (ACT) represents another influential approach that operates on complementary principles. ACT conceptualizes anger not as a problem to eliminate but as an internal experience to be approached with openness and curiosity. Central to ACT is experiential acceptance, the willingness to allow anger sensations, thoughts, and impulses to exist without avoidance or suppression. Paradoxically, efforts to suppress anger often increase its intensity by heightening cognitive and physiological arousal. Acceptance reduces this struggle and fosters a more regulated emotional

environment. Neurobiologically, acceptance practices engage prefrontal regulatory regions while reducing limbic activation, suggesting overlapping mechanisms with mindfulness but with a distinct emphasis on values-driven behavior (Lindsay et al., 2023).

ACT also incorporates values clarification, an essential process for clients whose anger disrupts interpersonal relationships, work performance, or personal well-being. By identifying valued directions, such as patience, kindness, fairness, or assertiveness, clients can evaluate whether anger-driven behaviors align with long-term goals. This process encourages behavioral choices guided by values rather than emotional impulses, strengthening prefrontal executive functioning. For individuals with chronic anger, values-based action provides a framework for choosing constructive responses during provocation, such as pausing, articulating needs calmly, or removing oneself from escalating situations.

A subtly different but highly effective intervention within the mindfulness family is affect labeling, the simple act of naming one's emotion during moments of distress. Affect labeling reduces amygdala activation and increases right ventrolateral prefrontal cortex (rVLPFC) engagement, demonstrating a direct neural pathway through which putting feelings into words regulates emotional intensity (Lieberman et al., 2007). In anger treatment, affect labeling allows clients to name nuances within their emotional experience, such as irritation, frustration, resentment, indignation, or moral outrage, rather than collapsing them into undifferentiated anger. Increased granularity supports better differentiation, which predicts improved emotion-regulation outcomes. Recent studies indicate that frequent practice of affect labeling enhances attentional control and reduces reactivity during anger-provocation tasks (Krämer et al., 2023).

The integration of mindfulness, acceptance, and affect labeling into anger treatment has strong empirical support. Randomized controlled trials of mindfulness-based CBT for anger demonstrate greater reductions in aggression, anger rumination, and self-reported anger intensity compared to traditional CBT or mindfulness alone (Richard et al., 2023). Similarly, ACT-based interventions show significant improvements in anger regulation among individuals with trauma histories, chronic irritability, or personality disorders, suggesting that acceptance and values-based approaches are particularly beneficial for clients with longstanding relational or identity vulnerabilities (Lindsay et al., 2023). These interventions may be especially useful for individuals whose anger arises from secondary emotions such as shame, fear, or perceived invalidation, allowing them to acknowledge underlying vulnerabilities without resorting to defensive aggression.

From a clinical perspective, mindfulness and acceptance strategies are essential for treating anger because they address domains that cognitive restructuring alone cannot

fully reach. While CBT effectively targets distorted appraisals, mindfulness and acceptance cultivate meta-awareness, emotional tolerance, interoceptive sensitivity, and nonreactivity. These capacities are essential for preventing rapid anger escalation, particularly among clients with heightened physiological arousal or neurobiological vulnerabilities involving limbic hypersensitivity or impaired prefrontal control.

Importantly, these approaches can be integrated flexibly within broader psychotherapeutic frameworks. For example, clients may begin with mindfulness practices to increase awareness of physiological cues, use affect labeling to modulate acute arousal, and implement acceptance and values work to address chronic or relational patterns. Combining these modalities with CBT enhances overall treatment outcomes, providing a comprehensive toolkit to manage complex anger presentations.

DBT-Informed Interventions and Emotion Regulation Training

Behavior Therapy (DBT) and its emotion-regulation components offer a robust, empirically supported framework for addressing anger dysregulation, particularly in individuals who exhibit high emotional sensitivity, rapid escalation, and chronic difficulties returning to baseline affective states. Originally developed for borderline personality disorder (BPD), DBT has been widely adapted for anger management across diverse populations, including those with trauma histories, impulsive aggression, and neurodevelopmental vulnerabilities. DBT's multimodal structure, integrating mindfulness, skills training, behavioral shaping, and motivational enhancement, aligns closely with contemporary neurobiological models of emotion regulation, which emphasize deficits in prefrontal inhibitory control, heightened limbic reactivity, and impaired autonomic recovery. By targeting these mechanisms through structured skills and experiential practice, DBT enhances clients' capacity for self-regulation and reduces the frequency and intensity of anger episodes (Linehan, 2015).

A key DBT construct relevant to anger treatment is emotion vulnerability, the tendency for emotional systems to be more easily activated and less efficiently modulated. Neurobiologically, individuals with high anger vulnerability often exhibit hyperresponsive amygdala activation to perceived threat, injustice, or interpersonal invalidation, coupled with underactivation in the dorsolateral prefrontal cortex (dlPFC) and anterior cingulate cortex (ACC), which are essential for inhibitory control and cognitive reappraisal (Kolla et al., 2023). DBT's emotion-regulation module directly targets this imbalance by teaching clients to identify emotional cues early, differentiate between primary and secondary emotions, and apply targeted strategies to reduce vulnerability and mitigate escalation. Increasingly, neuroimaging evidence suggests that DBT skills enhance functional

connectivity between prefrontal and limbic regions, contributing to improved impulse control and more adaptive emotional responses over time (Schaefer et al., 2023).

One of DBT's most important contributions to anger treatment is distress tolerance, a collection of skills designed to help individuals withstand acute emotional surges without resorting to impulsive or aggressive behavior. Techniques such as cold-water immersion, paced breathing, and temperature-modulation strategies (e.g., the "TIPP" skills: Temperature, Intense exercise, Paced breathing, Paired muscle relaxation) reduce sympathetic arousal and help regulate the "fight-or-flight" response. These physiological interventions directly target autonomic dysregulation, which is particularly salient in anger episodes characterized by rapid heart rate, increased blood pressure, and muscle tension. Studies show that such techniques improve heart rate variability (HRV), an index of parasympathetic functioning strongly associated with emotion regulation and reduced anger reactivity (Lehrer & Gevirtz, 2023). For clients with impulsive anger bursts, distress tolerance creates crucial space between impulse and action, allowing regulatory strategies to become effective.

DBT's mindfulness skills also play a foundational role in modulating anger. These practices cultivate nonjudgmental observation of internal experiences, enabling clients to recognize rising anger cues before they reach full intensity. Mindfulness facilitates "urge surfing," a technique in which individuals observe urges, such as the impulse to yell or retaliate, without acting on them. This process supports top-down inhibitory control by fostering awareness of the transient nature of emotional states. Neurobiological studies demonstrate that mindfulness practices increase activation in prefrontal regions, reduce amygdala responsivity, and strengthen integration between cognitive and affective networks (Farb et al., 2023). Mindfulness also interrupts anger rumination, a process that prolongs and intensifies anger through repeated cognitive rehearsal of perceived injustices or slights. By redirecting attention to the present moment, mindfulness diminishes rumination-related activation in default-mode network (DMN) regions, reducing the persistence of anger over time.

Central to DBT's emotion-regulation module is the principle of opposite action, a behavioral intervention in which clients engage in actions that counteract the action tendencies of anger. Whereas anger urges individuals to confront, blame, or assert dominance, opposite action involves approaching the situation with gentleness, requesting clarification rather than assuming hostility, or disengaging from conflict when appropriate. By repeatedly engaging in opposite action, clients weaken the learned associations between anger triggers and maladaptive behaviors. Behavioral activation of this sort aligns with principles of neural plasticity: engaging in alternative responses strengthens pathways

associated with regulatory behavior while reducing reliance on habitual reactive patterns. Empirical studies show that consistent practice of opposite action improves anger outcomes and decreases aggression in community and clinical populations (Neacsiu et al., 2022).

Interpersonal effectiveness is another cornerstone of DBT particularly relevant for anger, as many anger episodes occur in relational contexts involving perceived invalidation, unmet needs, or conflict escalation. DBT teaches strategies such as DEAR MAN (Describe, Express, Assert, Reinforce, Mindful, Appear confident, Negotiate) to improve communication and problem-solving while reducing reliance on aggressive or passive-aggressive behaviors. Interpersonal effectiveness skills help clients articulate feelings and needs directly, reducing misinterpretations and reactive anger cycles. Given that anger often functions as a protective defense against vulnerability, interpersonal skills increase clients' willingness to express primary emotions, such as hurt or fear, instead of escalating into secondary anger. Neurobiologically, improved interpersonal communication may reduce activation of threat-detection circuits and increase regulatory engagement within prefrontal networks (Uddin et al., 2023).

Emotion regulation in DBT also emphasizes reducing vulnerability to emotion dysregulation, particularly through lifestyle modifications that stabilize physiological and psychological functioning. Skills such as PLEASE (Physical health, balanced Eating, Avoid mood-altering substances, adequate Sleep, and Exercise) target chronic conditions that increase anger susceptibility. Sleep deprivation, for example, reduces prefrontal-amygdala connectivity and increases emotional reactivity, lowering thresholds for anger (Palmer & Alfano, 2023). Similarly, substance use exacerbates impulsive aggression by impairing inhibitory control and increasing limbic activation. Addressing these vulnerabilities enhances overall emotional stability and improves responsiveness to higher-order regulation strategies.

DBT's multimodal structure also supports anger treatment through behavioral reinforcement and coaching. The inclusion of phone coaching and skills generalization helps clients apply regulation strategies during real-world anger episodes, reducing reliance on impulsive responses and strengthening adaptive habits. Additionally, the dialectical framework, balancing acceptance and change, allows clinicians to validate clients' anger as understandable while simultaneously challenging unhelpful behaviors. This balance enhances engagement and reduces defensiveness in clients who may feel shame or guilt about their anger.

Evidence for DBT's effectiveness in reducing anger is robust. Meta-analytic findings demonstrate significant reductions in anger intensity, anger rumination, and aggressive

behaviors among individuals receiving DBT, particularly those with BPD and trauma-related disorders (Schaefer et al., 2023). Adaptations of DBT for forensic populations and adolescents with behavioral dysregulation have similarly shown reductions in violent incidents, improved emotional control, and enhanced interpersonal functioning (Miller et al., 2023). Neuroimaging studies suggest that DBT strengthens activation in regulatory brain regions and improves integration between cognitive and affective neural networks, providing biological support for clinical improvements.

In summary, DBT-informed anger interventions provide a comprehensive, neuroscience-aligned framework for treating anger dysregulation. By combining mindfulness, distress tolerance, emotion regulation, interpersonal effectiveness, and lifestyle stabilization strategies, DBT addresses both the biological and psychological dimensions of anger. Its emphasis on skill acquisition, experiential practice, and real-world generalization makes it uniquely suited for individuals with chronic, reactive, or trauma-related anger difficulties.

Cognitive Bias Modification and Experimental Approaches

Cognitive Bias Modification (CBM) represents an emerging class of interventions aimed at altering implicit cognitive processes that contribute to anger and aggression. While traditional psychotherapies rely on conscious reflection and deliberate skill use, CBM targets automatic patterns of interpretation, attention, and appraisal that occur rapidly, often outside of awareness, during anger-provoking situations. These automatic cognitive processes are increasingly understood as central contributors to anger dysregulation, particularly in individuals who exhibit hostile attribution biases, rapid threat detection, or rigid negative interpretations of ambiguous social cues. Contemporary cognitive and affective neuroscience research demonstrates that aggressive individuals display accelerated limbic activation coupled with biased interpretation of ambiguous social stimuli, reflecting strongly ingrained cognitive-emotional templates (Vitiello et al., 2023). CBM interventions aim to recalibrate these patterns, reducing vulnerability to rapid anger escalation.

One of the most studied forms of CBM in anger treatment is Interpretation Bias Modification (IBM), an intervention that trains individuals to adopt benign or neutral interpretations of ambiguous interpersonal cues. Hostile attribution bias, interpreting ambiguous actions as intentionally harmful, is a robust predictor of aggression and anger intensity across clinical and developmental populations (Ciesinski et al., 2023). For example, an individual may perceive a colleague's curt email as intentionally disrespectful, or interpret a partner's delayed response as rejection or betrayal. IBM seeks to disrupt these tendencies by repeatedly exposing clients to ambiguous scenarios and prompting them to generate non-hostile interpretations. Over time, this repeated practice strengthens

cognitive pathways associated with benign interpretation, leading to measurable reductions in anger reactivity. Empirical studies support the efficacy of IBM, with meta-analytic data showing small-to-moderate reductions in aggression and anger across both laboratory-based and digitally delivered interventions (Ciesinski et al., 2023).

At the neurobiological level, IBM appears to influence functional connectivity between limbic and prefrontal networks. Preliminary imaging studies show that individuals with strong hostile attribution tendencies exhibit heightened amygdala activation in response to ambiguous facial expressions and reduced engagement of regulatory regions such as the dorsolateral prefrontal cortex (dlPFC). Training that induces repeated benign interpretations increases dlPFC activation during subsequent emotion-processing tasks, suggesting enhanced top-down modulation of threat responses (Krämer et al., 2023). These findings indicate that CBM may strengthen neural circuits involved in cognitive reappraisal, improving regulatory capacity in individuals prone to anger dysregulation.

Another promising intervention involves Attention Bias Modification (ABM), which targets attentional patterns that predispose individuals to notice threat-related cues more readily than neutral cues. Anger-prone individuals often display vigilance for signs of disrespect, dominance threat, or interpersonal injustice, patterns supported by increased activity in the amygdala, insula, and salience network (Krämer et al., 2023). ABM retrains attention by encouraging clients to shift focus away from threatening or provocative stimuli toward neutral or positive alternatives. For example, individuals may complete computerized tasks where their attention is consistently guided away from angry faces and toward neutral expressions. Over time, this training reduces attentional capture by anger-relevant cues and dampens physiological arousal during provocation. Although ABM research in anger is less extensive than IBM, early randomized trials show reductions in aggressive behavior and improved emotional control in at-risk populations (Zvielli et al., 2023).

Beyond modifying cognitive biases, several experimental approaches seek to enhance anger regulation by targeting underlying neural mechanisms directly. One such approach is cognitive control training (CCT), which focuses on strengthening executive functions such as working memory, inhibitory control, and cognitive flexibility. Individuals with chronic or impulsive anger frequently demonstrate deficits in these cognitive domains, correlating with reduced dlPFC recruitment and impaired regulation during emotional tasks (Panasiti et al., 2023). CCT uses repetitive, adaptive tasks, such as n-back working memory training, response inhibition tasks, or task-switching paradigms, to enhance cognitive capacity. Improved executive functioning translates into better emotion regulation and reduced anger reactivity. For example, studies show that working memory training reduces aggressive behavior by improving individuals' ability to hold multiple interpretations in

mind, delaying impulsive reactions (Denson et al., 2019). CCT may also increase prefrontal-limbic connectivity, supporting neural resilience under stress.

Another innovative experimental modality involves neurofeedback, which trains individuals to modify brain activity patterns associated with anger. Using real-time feedback from EEG or fMRI, clients learn to increase activation in regulatory prefrontal regions or decrease activation in limbic regions associated with emotional reactivity. Preliminary studies indicate that neurofeedback targeting frontal midline theta or alpha asymmetry may reduce anger, improve inhibitory control, and enhance emotional stability (Sitaram et al., 2023). While neurofeedback for anger remains in early developmental stages, findings suggest that targeted modulation of neural oscillations could integrate effectively with psychotherapeutic interventions.

Additional experimental strategies include virtual reality (VR) interventions that simulate anger-provoking scenarios in a controlled environment. VR allows clients to practice regulatory strategies, such as mindful attention, cognitive reframing, or opposite action, while experiencing physiological arousal similar to real-world anger triggers. VR-based exposure may reduce anger reactivity by weakening conditioned responses and enhancing regulatory pathways. Early studies show VR can reduce aggression in forensic populations and improve regulation in adolescents with behavioral dysregulation (Fisher et al., 2023).

CBM and experimental interventions offer several clinical advantages. First, they directly target automatic processes that are resistant to traditional talk therapy. Second, they can be delivered digitally, increasing accessibility and reducing barriers to care. Third, they complement CBT, DBT, and mindfulness-based approaches by creating cognitive and neural conditions that facilitate skill acquisition. For example, clients who complete CBM may find it easier to engage in cognitive restructuring because their baseline interpretive tendencies are less hostile. Similarly, improving attentional control enhances mindfulness skills by reducing habitual threat scanning.

However, these approaches also present limitations. Effect sizes remain modest, and interventions may require repetitive, sustained practice to produce clinically meaningful change. CBM effects may not generalize fully from computerized tasks to complex interpersonal contexts without additional therapeutic support. Moreover, individual differences, such as cognitive capacity, trauma history, or neurodevelopmental conditions, may modulate responsiveness to CBM interventions.

Despite these challenges, the integration of CBM and experimental approaches within comprehensive anger treatment is promising. These interventions reflect a shift toward precision-based psychotherapy that leverages cognitive science and neuroscience to

modify underlying mechanisms of anger dysregulation. When combined with established therapies such as CBT and DBT, CBM enhances treatment efficacy by addressing automatic processes that contribute to rapid anger escalation.

Trauma-Focused and Schema-Focused Approaches

Trauma-focused and schema-focused psychotherapies offer essential pathways for treating anger that stems not from discrete cognitive distortions or learned behavioral patterns, but from deeply ingrained emotional, relational, and neurobiological adaptations formed in the context of overwhelming stress or chronic relational injury. For many clients, anger functions as a secondary, defensive response that emerges when vulnerable emotions, fear, shame, abandonment pain, become intolerable. Trauma-related anger often reflects hyperactive threat detection systems, impaired contextual processing, and difficulties integrating memory and emotion within a coherent sense of self (Heberlein et al., 2023). Schema-related anger, by contrast, reflects longstanding core beliefs about self, others, and the world, most often involving themes of mistrust, defectiveness, entitlement, or emotional deprivation, that shape how individuals interpret interpersonal events. Trauma-focused approaches work by processing dysregulated memories and recalibrating threat circuits, whereas schema-focused approaches modify maladaptive emotional and cognitive templates. Together, they target anger at the level of its origin.

In individuals with posttraumatic stress disorder (PTSD) or complex trauma, anger often arises from hypervigilance, perceived threat, betrayal trauma, moral injury, or chronic invalidation. Neurobiological models highlight that trauma alters several key systems: the amygdala becomes hyperreactive, prefrontal regulatory regions show reduced activation, and the hippocampus demonstrates impaired contextual modulation, making ambiguous interpersonal cues appear threatening or unjust (Palmier-Claus et al., 2023). Trauma-focused therapies, such as trauma-focused cognitive behavioral therapy, prolonged exposure (PE), and eye movement desensitization and reprocessing (EMDR), aim to reprocess traumatic memories that continue to drive physiological and emotional hyperreactivity. These interventions reduce anger by desensitizing threat cues, altering associative learning, and improving integration of traumatic memories into semantic networks. Functional neuroimaging studies show that trauma therapy increases activation in the ventromedial prefrontal cortex (vmPFC) and decreases amygdala reactivity during threat processing, indicating recalibration of regulatory pathways (Marusak et al., 2023). These neural changes align with clinical observations that clients become less reactive and more reflective when faced with anger-eliciting situations.

Trauma-focused therapies are particularly effective for anger that is episodic, trauma-triggered, or associated with flashbacks, dissociation, or moral injury. In moral injury, a

form of trauma involving violations of one's core values, anger is often entwined with guilt, shame, or profound disillusionment. Therapies that integrate meaning-making, such as Adaptive Disclosure or specialized EMDR protocols, help clients recontextualize painful events without collapsing into shame or rage. For clients whose anger masks underlying fear or helplessness, trauma-focused work reveals the primary emotions driving dysregulation and fosters compassion for the self that endured trauma.

Dialectical Behavior Therapy–Prolonged Exposure (DBT-PE) is another trauma-informed model relevant to anger dysregulation. DBT-PE combines the emotion-regulation and stability-building components of DBT with exposure-based trauma processing. The rationale is that trauma survivors who exhibit intense anger often lack the regulatory capacity to tolerate trauma work without destabilization. DBT-PE addresses this by strengthening inhibitory control and distress tolerance before exposure. This staged-care model is supported by research showing that DBT increases prefrontal control capacity, allowing clients to approach trauma memories with less reactive anger and greater cognitive integration (Schaefer et al., 2023). DBT-PE is particularly beneficial for trauma survivors with BPD features, chronic anger, and emotional lability.

Whereas trauma-focused treatments address dysregulated threat circuits, schema therapy is uniquely suited to anger that originates from enduring cognitive-emotional patterns developed in childhood. Schema therapy conceptualizes anger as emerging from unmet core needs, such as safety, connection, or fairness, and the activation of maladaptive schemas, such as Mistrust/Abuse, Defectiveness/Shame, Entitlement, or Subjugation (Young et al., 2003). These schemas shape interpretations of interpersonal situations in ways that amplify anger. For example, a client with a Mistrust/Abuse schema may interpret neutral feedback as a personal attack, whereas someone with an Entitlement schema may respond with intense anger when limits are set. Schema modes such as the Angry Protector, Bully/Attack, or Detached Self-Soother further illustrate how anger becomes a defensive adaptation.

Schema therapy uses experiential, cognitive, and relational interventions to modify these deeply embedded patterns. Imagery rescripting is one of the most powerful schema-focused interventions for anger, particularly when anger reflects childhood experiences of fear, humiliation, or neglect. In imagery rescripting, clients revisit early memories while the therapist provides corrective emotional experiences, protection, validation, nurturance, thereby altering the emotional meaning of the memory. Neurobiologically, imagery rescripting may recalibrate activation in limbic structures by linking previously stored traumatic or painful memories with new, soothing emotional associations (Fischer-Kern et

al., 2023). For anger, the process helps clients recognize that their reactions originate from earlier unmet needs and allows them to respond less defensively in current relationships.

Schema therapy's limited reparenting provides another essential mechanism for treating anger rooted in early deprivation or invalidation. Through consistent empathy, boundary setting, and emotional attunement, therapists model the relational patterns that were absent during critical developmental periods. Clients internalize these relational experiences, which supports development of healthier schemas and emotion-regulation capacities. This relational corrective experience has strong neurobiological plausibility: attachment research shows that consistent attunement enhances prefrontal development and reduces limbic reactivity, exactly the systems implicated in chronic anger (De Winter et al., 2023).

For clients whose anger expresses unacknowledged vulnerability, schema therapy facilitates identification of primary emotions beneath defensive anger. Many individuals use anger as a protective response to shame, abandonment, or emotional pain. Through mode work, clients learn to access underlying vulnerable states, such as the Abandoned Child or Lonely Child modes, with compassion rather than hostility. This process reduces reliance on maladaptive anger modes and fosters emotional integration. As clients connect with underlying pain, anger often becomes less explosive and more proportional.

Trauma-focused and schema-focused approaches can be integrated for clients with complex presentations. For example, individuals with chronic anger related to childhood abuse may benefit from both trauma processing and schema modification. Trauma-focused techniques reduce physiological reactivity, while schema work reshapes longstanding cognitive-emotional patterns that perpetuate anger in relationships.

Trauma-focused and schema-focused therapies provide essential tools for addressing anger that originates from deep relational wounds, maladaptive cognitive-emotional patterns, or chronic activation of threat circuits. By targeting the root causes of anger, rather than just its manifestations, these approaches foster long-term emotional integration, relational stability, and psychological resilience.

Interpersonal, Systems-Based, and Couple/Family Approaches

Interpersonal and systems-based approaches conceptualize anger not solely as an individual pathology but as an emergent property of relational patterns, communication deficits, family-of-origin dynamics, and contextual stressors. From this perspective, anger is both shaped by and expressed within interpersonal systems, romantic partnerships, family constellations, cultural environments, and organizational structures. Systems-based interventions address anger by modifying these relational patterns, enhancing

communication, strengthening attachment security, and changing interactional cycles that perpetuate emotional dysregulation. This section integrates systemic theory with contemporary neurobiological research, highlighting how relational contexts modulate threat systems, prefrontal regulation, and autonomic functioning.

A central premise of interpersonal models is that anger is inherently relational. It emerges in response to perceived violations of expectations, threats to belonging, or ruptures in attachment bonds. Attachment theory has been particularly influential in shaping interpersonal anger treatment. Individuals with insecure attachment, especially those with anxious or disorganized patterns, are more likely to exhibit anger dysregulation, misinterpret neutral cues as rejection, or escalate rapidly in conflict (De Winter et al., 2023). Neurobiologically, insecure attachment is associated with heightened amygdala activation and reduced recruitment of prefrontal regions during interpersonal stress, creating vulnerability to reactive anger in relational contexts. Emotionally focused therapy (EFT), one of the most empirically supported couple therapies, directly targets these attachment vulnerabilities. EFT helps partners identify primary emotions (e.g., fear, shame, abandonment pain) beneath secondary anger, transforming conflictual cycles into supportive, regulated interactions. By fostering secure attachment, EFT reduces hypervigilance and decreases activation of neural threat circuits implicated in anger (Johnson et al., 2023).

Interpersonal approaches also emphasize circular causality, the notion that anger escalates through repetitive interaction patterns rather than linear cause-and-effect sequences. For example, one partner's withdrawal may trigger the other's anger, which in turn increases withdrawal, reinforcing a negative cycle. These recursive patterns are maintained by emotional reactivity, cognitive biases, and physiological arousal. Systemic therapy helps couples identify and de-escalate these cycles by shifting focus from blame to pattern awareness. Integrating neuroscience, clinicians can highlight how physiological arousal, elevated heart rate, reduced heart rate variability (HRV), and narrowed attentional focus, creates conditions under which escalation becomes almost automatic (Lehrer & Gevirtz, 2023). Teaching partners to pause, regulate, and re-engage reduces the likelihood of triggering limbic cascades that drive conflict.

Communication training is another cornerstone of systemic anger treatment. Ineffective communication, characterized by criticism, defensiveness, contempt, or stonewalling, activates neural threat networks in both partners, reducing cognitive flexibility and impairing problem-solving. Evidence from interpersonal neurobiology shows that negative communication patterns escalate physiological synchrony in couples, meaning both partners' stress responses rise simultaneously, reinforcing mutual dysregulation (Timmons

et al., 2023). Interventions such as the Gottman Method, integrative behavioral couple therapy (IBCT), and communication-focused systemic therapies teach partners to express complaints as needs, validate emotions, and repair ruptures promptly. These skills reduce interpersonal threat perception, enhance prefrontal engagement, and increase relationship satisfaction.

In addition to couple-level processes, anger often reflects family-of-origin patterns shaped by generational emotional regulation norms, trauma histories, or rigid family roles. Multigenerational family therapy models, including Bowenian therapy and structural family therapy, address anger by examining triangulation, emotional cutoffs, parentification, and cross-generational transmission of emotion regulation patterns. For instance, individuals raised in dismissive or emotionally volatile families may develop maladaptive anger scripts that persist into adulthood. Research in developmental neuroscience suggests that chronic exposure to inconsistent caregiving or emotional invalidation disrupts maturation of the vmPFC and ACC, regions critical for emotion regulation, leading to adult anger vulnerability (Heberlein et al., 2023). Working with the family system allows clinicians to modify interaction patterns that reinforce anger, foster healthier emotional expression, and strengthen supportive relationships.

Family-based interventions are particularly effective in treating adolescent anger and aggression. Programs such as functional family therapy (FFT), multisystemic therapy (MST), and parent–child interaction therapy (PCIT) have shown significant reductions in aggression, oppositional behavior, and anger dysregulation. These approaches work by modifying parenting practices, reducing coercive cycles, enhancing parental attunement, and restructuring environmental stressors. Neurobiological evidence suggests that improved parenting behaviors increase prefrontal regulatory capacity in adolescents and reduce baseline cortisol levels, supporting long-term reductions in anger reactivity (Marusak et al., 2023). Family-based models also address contextual factors, peer networks, school environments, and community stressors, that can exacerbate anger dysregulation.

Interpersonal and systemic models increasingly incorporate mentalization-based approaches, which emphasize understanding one's own and others' internal states. Deficits in mentalization contribute to anger escalation by fostering misinterpretation of others' intentions and reducing reflective functioning. Individuals who struggle with mentalization are more likely to attribute malevolence to neutral behaviors, intensifying anger and interpersonal conflict. Mentalization-based therapy (MBT) fosters curiosity about internal experiences, enhances perspective-taking, and slows reactive responding. Neurobiological findings indicate that mentalization training increases activation in

prefrontal regions associated with cognitive empathy and reduces amygdala reactivity during social interaction (Bateman & Fonagy, 2023). MBT is especially useful in populations with relational trauma, BPD features, or complex anger patterns.

Cultural and systemic contexts also shape anger expression and treatment. Cultural norms influence whether anger is expressed directly, suppressed, or reframed through nonverbal cues. Systemic oppression, discrimination, and chronic stress exposure can heighten anger in marginalized communities. Trauma-informed systemic approaches acknowledge that anger may reflect adaptive responses to external threats, not internal pathology. Interventions grounded in liberation psychology, community-based healing, and culturally responsive family therapy validate these contextual realities while fostering regulated and empowered expression.

Across all models, systemic interventions excel at identifying patterns, enhancing regulation within relationships, and restoring connection. They shift therapy from focusing solely on individual dysregulation to understanding anger as embedded in relational dynamics, attachment history, and social context. When partners, parents, or family members learn to co-regulate, communicate effectively, and break maladaptive cycles, clients experience improved emotional security, reduced physiological reactivity, and decreased anger intensity.

Integrative and Multimodal Approaches

Integrative and multimodal approaches to anger treatment reflect a contemporary shift toward combining complementary evidence-based strategies to address the full neurobiological, cognitive, emotional, and interpersonal complexity of anger dysregulation. While individual modalities such as CBT, DBT, mindfulness-based interventions, trauma-focused therapies, and schema therapy offer powerful mechanisms of change, many clients present with multifactorial anger patterns shaped by childhood adversity, chronic stress, relational trauma, neurodevelopmental differences, and entrenched cognitive-emotional schemas. Multimodal approaches allow clinicians to tailor interventions to the specific neurobiological drivers of anger, such as limbic hyperreactivity, prefrontal under-regulation, autonomic dysregulation, memory intrusion, hostile attribution bias, or attachment insecurity, while addressing systemic, interpersonal, and contextual factors. Integrative care aligns with current precision-oriented frameworks in clinical psychology and psychiatry, emphasizing personalized treatment matching based on identifiable mechanisms.

A central premise of integrative anger treatment is that different mechanisms of anger may require different therapeutic "entry points." Clients whose anger stems from rapid

limbic activation and poor inhibitory control may benefit most from DBT distress-tolerance skills, HRV biofeedback, and mindfulness practices. Those whose anger reflects distorted interpretation patterns or rigid cognitive schemas may respond best to cognitive restructuring, cognitive bias modification (CBM), or schema therapy. Trauma-related anger, in contrast, requires reprocessing of traumatic memories, recalibration of threat circuits, and strengthening of emotion regulation capacities before trauma exposure. Integrative models help clinicians determine which mechanisms are primary and which are secondary, enabling a staged and targeted approach.

One widely used multimodal model is CBT + mindfulness integration, in which cognitive restructuring and behavioral activation are paired with mindfulness training to reduce rumination and increase present-moment awareness. This combination targets both top-down cognitive mechanisms and bottom-up attentional and physiological mechanisms. For example, cognitive restructuring reduces the hostile attribution bias that activates anger, while mindfulness reduces attentional fixation on perceived injustice, allowing prefrontal regulatory circuits to maintain greater control (Krämer et al., 2023). Studies demonstrate that integrated CBT-mindfulness protocols yield larger reductions in anger intensity and anger rumination than CBT or mindfulness alone (Bjureberg et al., 2023). Neurobiologically, this combination enhances dlPFC–amygdala connectivity and reduces default mode network (DMN) hyperactivity, supporting greater emotional resilience.

Another common integrative strategy involves DBT + trauma-focused therapy, particularly for clients with complex trauma who present with explosive anger, emotional instability, and interpersonal sensitivity. DBT provides the regulatory foundation, distress tolerance, emotion regulation, interpersonal effectiveness, necessary for clients to tolerate trauma processing without becoming overwhelmed. Once regulatory skills are established, trauma-focused interventions such as EMDR, prolonged exposure, or trauma-focused CBT can be introduced to address the traumatic memories that drive chronic anger. Research on DBT-Prolonged Exposure (DBT-PE) demonstrates substantial reductions in anger, aggression, and emotional reactivity among trauma survivors, with neuroimaging studies showing increased prefrontal control and decreased limbic hyperreactivity (Schaefer et al., 2023). This staged approach ensures that trauma work proceeds within a safer physiological window of tolerance.

More recently, clinicians have begun integrating schema therapy with mindfulness, DBT, and interpersonal approaches to address longstanding relational and identity-based anger. Schema therapy conceptualizes anger as emerging from unmet core emotional needs and maladaptive schemas (e.g., mistrust/abuse, entitlement, subjugation), and interventions such as imagery rescripting and mode work help clients access vulnerable emotions

beneath anger. Integrating mindfulness allows clients to observe activating schemas without fusing with them, while DBT skills help stabilize emotional arousal during schema activation. Interpersonal and systems-based interventions further target relational patterns that reinforce schemas, such as cycles of criticism and defensiveness, withdrawal and pursuit, or invalidation and escalation. This combined approach supports transformation at both the internal schema level and the interpersonal behavioral level.

Beyond traditional psychological interventions, multimodal approaches increasingly integrate biophysiological strategies that target autonomic dysregulation and stress reactivity. HRV biofeedback, paced breathing, and vagal-tone enhancement strategies address low parasympathetic functioning, a major vulnerability for anger escalation (Lehrer & Gevirtz, 2023). Neurofeedback interventions may complement psychotherapy by enhancing prefrontal regulation or reducing limbic overactivation during emotional tasks. Exercise, sleep regulation, and anti-inflammatory lifestyle modifications support neurobiological stability and reduce the physiological burden associated with chronic anger (Palmer & Alfano, 2023). When integrated thoughtfully into psychotherapy, these modalities help clients regulate physiological arousal, creating fertile ground for cognitive, emotional, and relational change.

Another emerging direction is the use of digital and technological adjuncts, including mobile apps delivering mindfulness practices, emotion-regulation exercises, CBM tasks, or real-time physiological feedback. Digital tools enhance accessibility and support daily practice, reinforcing synaptic changes associated with emotional regulation. For example, smartphone apps that prompt affect labeling or provide breathing exercises during early signs of anger can help clients intervene in-the-moment before escalation occurs. Some platforms incorporate personalized algorithms that detect physiological changes (via wearables) and deliver tailored interventions. Early research shows that digital adjuncts improve treatment adherence, reduce anger rumination, and extend therapeutic gains outside the therapy room (Bjureberg et al., 2023).

Integrative approaches also emphasize contextual and systemic factors. For clients whose anger is rooted in relational conflict, systemic family therapy and couples-based interventions may be essential. For clients affected by systemic oppression, discrimination, or sociocultural stressors, culturally responsive and liberation-focused frameworks are crucial. Trauma-informed community interventions, group programs, and multigenerational approaches acknowledge that anger is shaped not solely by individual factors but by broader social conditions. Integrative care thus adapts to the intersecting realities of biology, psychology, relationships, and environment.

The most advanced integrative models draw on principles of precision psychotherapy, which aim to match treatment components to client-specific neurobiological and psychological profiles. Precision models consider factors such as amygdala reactivity, prefrontal executive functioning, autonomic flexibility, trauma history, cognitive style, and attachment patterns to determine the ideal combination of interventions. In anger dysregulation, for example, a client with high threat sensitivity and insecure attachment may benefit from EFT or schema therapy before cognitive restructuring becomes effective. Conversely, a client with strong cognitive control but high rumination may respond well to mindfulness-based interventions combined with reappraisal strategies. Precision approaches hold promise for optimizing outcomes by tailoring interventions to the mechanisms most responsible for the client's anger.

Ultimately, integrative and multimodal approaches recognize that anger dysregulation is multidimensional, involving interactions among neural systems, cognitive processes, emotional learning, interpersonal relationships, and environmental stressors. No single modality sufficiently addresses all these layers. By synthesizing techniques from CBT, DBT, mindfulness, trauma therapy, schema therapy, interpersonal approaches, and biophysiological regulation, integrative models provide a flexible and comprehensive framework for long-term healing. These approaches are well suited to real-world clinical practice, where clients present with complex, layered, and chronic anger patterns that require both depth and breadth of intervention.

Case Applications and Clinical Vignettes

Case applications provide an essential bridge between theory and practice, illustrating how neurobiological, psychological, and systemic factors converge in real-world anger presentations. The following vignettes, drawn from composite clinical profiles, demonstrate how assessment, formulation, and intervention are informed by the mechanisms described throughout this course. Each vignette highlights a different anger phenotype and illustrates targeted psychotherapeutic strategies aligned with the client's neurobiological profile.

Case Vignette 1: Impulsive Reactive Anger (IED Phenotype)

"Marcus," a 32-year-old man, was referred after multiple workplace outbursts involving yelling, slamming objects, and sudden anger episodes that he described as "coming out of nowhere." Episodes were brief but intense, followed by remorse and exhaustion. Marcus denied premeditated aggression, describing instead a pattern of rapid emotional escalation. He reported chronic irritability, sleep disturbance, and difficulty "cooling down" after conflicts.

Assessment and Formulation:

Assessment revealed elevated state and trait anger on the STAXI-2 and significant anger rumination. Psychophysiological symptoms included racing heart, facial warmth, and muscle tension, with minimal awareness of early warning cues. His history included inconsistent parenting and exposure to familial volatility, but no significant trauma. Screening for ADHD revealed subthreshold symptoms of impulsivity.

Neurobiological conceptualization included hyperreactive amygdala response, low serotonergic inhibition, and low HRV, consistent with research on IED and impulsive aggression (Coccaro & Lee, 2023; Lee et al., 2024). Marcus demonstrated limited prefrontal inhibitory control during high arousal and a very narrow window between trigger and behavioral response.

Intervention:
Treatment integrated anger-focused CBT, DBT distress tolerance, and HRV biofeedback.

1. Physiological Regulation First:
   Early sessions prioritized bottom-up regulation, including paced breathing (6 breaths/min) and temperature-modulation strategies. After six sessions, HRV improved modestly, and Marcus reported noticing physiological cues earlier.

2. Cognitive Restructuring:
   Once physiological stabilization improved, cognitive restructuring targeted Marcus's rapid hostile interpretations (e.g., "They're disrespecting me"). Thought logs revealed automatic assumptions of intentional harm—a mild form of hostile attribution bias (Vitiello et al., 2023).

3. Chain Analysis and Trigger Mapping:
   Detailed functional analysis clarified micro-triggers that Marcus previously overlooked, such as tone-of-voice shifts, ambiguous facial expressions, and perceived criticism.

4. Opposite Action and Urge Surfing:
   During provoking moments, Marcus practiced pausing, labeling his internal state ("anger rising"), and either using brief time-outs or responding with a pre-scripted assertive statement.

Outcome:
Marcus's episode frequency decreased from several times weekly to once every 3–4 weeks. He reported feeling "more in control" and less physically overwhelmed. His workplace supervisor noted measurable improvements in emotional stability. Treatment

demonstrated how integrating physiological stabilization with cognitive and behavioral work can effectively regulate neurobiologically driven impulsive anger.

Case Vignette 2: Trauma-Related Anger and Moral Injury

"Elena," a 41-year-old former ICU nurse, sought therapy for chronic anger, interpersonal withdrawal, and emotional explosions toward family members. She described carrying "rage that won't go away" after COVID-19–related moral injury experiences in which she was forced to withhold care during resource scarcity. She reported nightmares, guilt, hypervigilance, and intense anger toward supervisors and systemic failures.

Assessment and Formulation:
   Elena met criteria for PTSD, with prominent hyperarousal and moral injury components. Anger emerged when she felt dismissed or misunderstood, often triggered by reminders of ethical conflicts. Screening revealed elevated limbic hyperreactivity, intrusive trauma memories, and emotional numbing alternating with explosive anger. Neurobiologically, her presentation aligned with amygdala hyperactivation, reduced vmPFC engagement, and altered HPA-axis functioning, common in trauma-related anger (Marusak et al., 2023).

   Elena did not respond well initially to CBT-style cognitive restructuring, events felt morally absolute and cognitively "unreframeable."

Intervention:
   Treatment followed a DBT-informed stabilization + trauma-focused processing pathway.

1. Stabilization Phase:
   DBT emotion-regulation and distress-tolerance strategies were introduced to reduce dangerous anger episodes and increase window of tolerance. Mindfulness of anger sensations helped Elena differentiate anger from underlying grief and betrayal.

2. Affect Labeling and Values Clarification:
   Sessions focused on differentiating moral injury–based anger ("anger at violations") from reactive anger in family settings. Affect labeling reduced arousal, consistent with findings that labeling lowers amygdala activation (Lieberman et al., 2007).

3. Trauma Processing Phase (EMDR):
   EMDR targeted morally injurious memories using modifications recommended for complex trauma. Reprocessing reduced intrusive imagery and physiological reactivity. Elena reported a shift from "boiling anger" to "grief I can sit with."

4. Meaning-Making Work:
   Using Adaptive Disclosure principles, Elena explored themes of identity, ethics, and systemic betrayal, eventually reconnecting with values of compassion and integrity.

Outcome:

After 20 sessions, Elena reported a 60–70% reduction in anger intensity and improved connections with family. She noted that the anger no longer felt "explosive" but "contextual." Her case illustrates how trauma-focused approaches, supported by adequate stabilization, reshape anger driven by moral injury and threat dysregulation.

Case Vignette 3: Schema-Driven Anger in Interpersonal Conflict

"Devon," a 28-year-old graphic designer, sought therapy for chronic anger in romantic relationships. He described reacting harshly to perceived criticism, becoming cold and contemptuous during conflict, and later feeling ashamed. His partner described him as "always on edge," triggered by minor slights. Devon acknowledged a pattern of pushing others away when feeling vulnerable.

Assessment and Formulation:

Devon endorsed traits consistent with avoidant and narcissistic relational patterns, including emotional inhibition, fear of dependency, and intense sensitivity to shame. His anger frequently surfaced in relational contexts involving feedback, perceived rejection, or partner requests for emotional closeness.

Schema assessment revealed activation of Defectiveness/Shame, Mistrust/Abuse, and Emotional Inhibition schemas. His primary anger mode resembled the Angry Protector, which activated whenever vulnerability surfaced. Neurobiologically, Devon likely experienced heightened insula activation during social evaluation combined with reduced prefrontal modulation—patterns consistent with insecure attachment and shame-based reactivity (De Winter et al., 2023).

Intervention:
Treatment followed a schema therapy + interpersonal emotion-regulation format.

1. Mode Model and Psychoeducation:
   Devon learned to identify when the Angry Protector mode was activated and distinguish it from underlying vulnerable child states, which he typically suppressed.

2. Imagery Rescripting:
   Early memories involving harsh criticism and emotional neglect were revisited and rescripted, providing corrective emotional experiences that softened shame-based

anger. This shifted Devon's internal narrative from "I'm defective" to "I never learned safe closeness."

3. Limited Reparenting and Emotional Attunement:
The therapeutic relationship modeled calm curiosity and acceptance, providing an alternative attachment template. Over time, this co-regulation reduced Devon's amygdala-driven relational hyperreactivity.

4. Interpersonal Effectiveness Training:
Borrowing from DBT and EFT, Devon practiced expressing needs using softened start-ups, validating partner concerns, and tolerating discomfort during conflict conversations.

5. Cognitive Reframing of Schema-Driven Interpretations:
When Devon interpreted neutral partner behaviors as criticism, therapy focused on generating more balanced appraisal options.

Outcome:
   By session 30, Devon's romantic conflicts decreased significantly. He reported "still feeling anger, but not controlled by it" and demonstrated improved vulnerability and relational openness. The case highlights the need for schema-level and attachment-oriented interventions when anger emerges from deeply ingrained interpersonal learning.

<div align="center">Ethical, Cultural, and Professional Considerations in Treating Anger</div>

   Treating anger requires a sophisticated understanding of ethical, cultural, and professional responsibilities that extend far beyond symptom reduction. Because anger dysregulation may involve aggression, trauma histories, safety concerns, and complex relational dynamics, clinicians must balance therapeutic effectiveness with rigorous adherence to professional standards. The APA (2017), ACA (2014), AAMFT (2015), and NASW (2021) Codes of Ethics provide essential guidance for navigating issues of assessment, risk, boundaries, telehealth, cultural responsiveness, trauma-informed care, and interdisciplinary collaboration. Integrating these ethical frameworks with contemporary neurobiological models ensures that treatment is competent, culturally informed, and grounded in client safety and autonomy.

   A central ethical responsibility involves conducting thorough, ongoing assessment of risk related to anger, violence, and impulsive aggression. Professional codes consistently emphasize the duty to protect clients and others from foreseeable harm. APA Standard 3.04 (Avoiding Harm), ACA B.2.a (Serious and Foreseeable Harm), NASW 1.07.c, and AAMFT 1.11 all indicate that confidentiality may be breached when there is imminent risk of danger. When clinicians evaluate anger, they must consider individual risk factors such

as history of violence, substance use, neurocognitive impairments, interpersonal contexts, access to weapons, or emotional states linked to trauma. Because anger often escalates rapidly, ethical assessment requires attention not only to overt threats but also to more subtle cues such as physiological dysregulation, dissociation, or increasing irritability. At the same time, clinicians must avoid misdiagnosis or biased interpretation of anger presentations, which could violate ethical standards regarding accuracy in assessment (APA 9.01) and cultural sensitivity (ACA E.5.b; NASW 1.05). Ethical anger assessment therefore requires a combination of neurobiological understanding, clinical sensitivity, and adherence to evidence-based protocols.

Informed consent plays an especially important role in anger treatment because many interventions, such as emotion-focused trauma processing, exposure-based work, or mindfulness strategies, may evoke strong emotional reactions. Ethical codes emphasize transparent communication about the nature and risks of treatment (APA 3.10, ACA A.2.a, AAMFT 1.2, NASW 1.03). Clients should understand potential difficulties, such as emotional flooding during trauma work or temporary increases in arousal as they learn new regulation skills. In modalities like DBT or trauma-focused CBT, where treatment involves structured skill practice and between-session assignments, clinicians must clarify expectations and boundaries. When technology-assisted tools are used, such as HRV biofeedback, VR-based exposure, or mobile emotion-regulation apps, clients must also be informed about data privacy, limitations of technology, and issues of digital confidentiality. Respect for autonomy, emphasized strongly in the NASW and ACA codes, requires that clients participate collaboratively in decisions about pacing, treatment components, and therapeutic goals.

Cultural competence is another ethical cornerstone of anger treatment. The APA Multicultural Guidelines (2017), ACA Section C.5, AAMFT 1.1, and NASW 1.05 require clinicians to understand how cultural, racial, spiritual, gendered, and community-based factors shape anger expression and interpretation. Anger norms vary widely across cultures; in some contexts, direct expression of anger is discouraged, while in others it may be viewed as a legitimate form of boundary-setting or protest. For minoritized clients, anger may reflect adaptive responses to systemic oppression or chronic discrimination rather than intrapsychic dysfunction. Ethical care therefore requires clinicians to explore the cultural meaning of anger, family narratives about emotional expression, and social contexts that influence anger triggers. Without this grounding, therapists risk pathologizing culturally normative behaviors or overlooking legitimate anger rooted in injustices. Professional codes warn explicitly against stereotyping or imposing dominant-culture values on clients (APA 3.01; ACA C.5), underscoring the importance of cultural humility and client-centered formulation.

Trauma-informed ethics are especially critical because many clients experience anger not as a primary emotion but as a protective response to overwhelming fear, shame, or loss. Ethical trauma treatment requires attending to safety, stabilization, and pacing—principles aligned with APA 3.04, NASW 1.01, and AAMFT 1.10. Premature exposure to traumatic memories may retraumatize the client or destabilize their affective system, particularly when anger has historically served as a buffer against vulnerability. Clinicians must therefore ensure sufficient emotion regulation skills before trauma processing, consistent with staged-model approaches (e.g., DBT-PE). Ethical trauma-informed care involves monitoring dissociation, respecting windows of tolerance, and validating anger as a survival-based adaptation rather than a moral failing. Because trauma-related anger often arises from experiences of betrayal, coercion, or injustice, therapists must avoid blame, pathologization, or invalidating interpretations. Compassionate attunement, central to trauma ethics, is mandated implicitly across all major professional codes as part of the duty to avoid harm and promote wellbeing.

Work with couples and families introduces additional ethical complexity. The AAMFT Code of Ethics places particular emphasis on managing power differentials and ensuring safety in family systems, warning clinicians against initiating conjoint sessions when coercive control or intimate partner violence is present. Anger in families may involve cycles of criticism, withdrawal, or triangulation, and engaging these systems requires careful ethical attention to confidentiality, alliance balance, and informed consent. The ACA (B.4.b) and NASW (1.07.e) codes highlight that confidentiality extends differently across family members; clinicians must clarify how information will be used and what cannot be kept confidential when safety is at risk. Adolescents present a further ethical challenge because clinicians must balance parental rights with adolescent privacy while adhering to reporting laws. When anger contributes to family conflict, therapists must prevent scapegoating of one member and avoid reinforcing harmful dynamics, remaining consistent with ethical mandates for nonmaleficence and systemic sensitivity.

The use of telehealth and digital tools for anger management raises additional ethical considerations. Remote therapy may introduce confidentiality challenges, particularly when clients lack private space or when high-arousal anger episodes occur outside the clinician's physical presence. APA Telepsychology Guidelines, ACA Section H, and NASW Technology Standards emphasize therapist competence in digital modalities, awareness of technological risks, and procedures for emergency management. Ethical telehealth requires establishing clear crisis protocols, verifying client locations at each session, and preparing strategies for managing in-session escalation. When apps or wearable devices are used to support anger regulation (e.g., HRV feedback, mindfulness reminders), clinicians must ensure these tools are evidence-based and compliant with privacy

regulations. Transparency regarding data collection, storage, and potential vulnerabilities is essential.

Professional scope of competence is another major ethical consideration. Anger dysregulation often co-occurs with complex trauma, personality disorders, brain injuries, substance use disorders, or neurodevelopmental conditions. The APA (2.01), ACA C.2, AAMFT 3.1, and NASW 1.04(a) codes require clinicians to recognize the limits of their expertise and seek consultation or referral when needed. Collaboration with psychiatrists, primary care providers, neurologists, or social service agencies may be necessary when anger is influenced by medical, neurological, or social determinants. Ethical collaboration involves obtaining appropriate client consent (ACA B.3), protecting confidentiality (APA 4.06), and coordinating care while avoiding fragmentation. Interdisciplinary approaches are particularly important when anger poses safety risks or reflects underlying physiological dysregulation beyond the scope of psychotherapy alone.

Clinician countertransference poses unique ethical challenges when working with anger. Clients who display hostility, volatility, defensiveness, or intimidation can evoke strong emotional reactions in therapists, including fear, frustration, avoidance, or overaccommodation. All major professional codes emphasize the importance of monitoring personal reactions to avoid impaired objectivity (APA 2.06; ACA F.6.b; AAMFT 3.10; NASW 4.05). Countertransference unaddressed may lead to boundary violations, misattunement, premature termination, or avoidance of essential clinical exploration. Clinicians must reflect on their own histories with anger, cultural beliefs about conflict, and physiological responses in-session. Ethical care may involve seeking supervision, engaging in personal therapy, or establishing clear in-session safety strategies. Because anger can be highly activating for both client and therapist, maintaining therapeutic presence while upholding firm, compassionate boundaries is essential.

Overall, ethical anger treatment is a complex, multidimensional process that requires clinicians to integrate rigorous professional standards with cultural awareness, trauma-informed sensitivity, and scientific understanding of neurobiological regulation. Professional ethics are not peripheral to anger treatment, they are central to ensuring client safety, promoting autonomy, protecting therapeutic integrity, and delivering competent, culturally grounded care that addresses the full complexity of anger within personal, relational, and systemic contexts.

Advances in affective neuroscience, clinical psychology, and trauma research have transformed our understanding of anger from a simple behavioral issue into a multidimensional construct grounded in neurobiological, cognitive, emotional, interpersonal, and sociocultural systems. Anger dysregulation is not a unitary

phenomenon but a diverse clinical presentation shaped by individual histories, neurocircuitry, environmental stressors, and relational patterns. A modern, evidence-based approach to anger requires clinicians to move beyond reductionist frameworks and adopt integrative models that consider the interplay of limbic hyperreactivity, prefrontal control deficits, threat-detection biases, traumatic conditioning, attachment insecurity, and systemic influences. As neuroscience continues to illuminate the mechanisms underlying anger, mental health providers are increasingly equipped to tailor interventions that address the specific drivers of dysregulation in each client.

One of the most significant clinical implications of this work is the reframing of anger as fundamentally an emotion-regulation problem. Historically, anger interventions emphasized behavioral suppression or cognitive reframing without adequately addressing the neurobiological substrates of arousal. Contemporary research demonstrates that many clients struggle with anger not because they "choose" to be angry but because their neurobiological systems react too rapidly, too intensely, or too automatically. Heightened amygdala reactivity, reduced dorsolateral prefrontal functioning, impaired interoceptive awareness, and chronic autonomic dysregulation reduce the capacity for reflective responding in moments of perceived threat (Krämer et al., 2023; Palmier-Claus et al., 2023). Recognizing these mechanisms allows clinicians to integrate bottom-up interventions, such as HRV biofeedback, paced breathing, and mindfulness, with cognitive and relational strategies to create a more comprehensive pathway to regulation.

Another critical implication involves the role of trauma and early adversity in shaping anger responses. Trauma survivors frequently experience anger as a secondary or protective reaction that shields them from overwhelming primary emotions such as fear, shame, or grief. Moral injury, betrayal trauma, and chronic invalidation often generate enduring anger states that cannot be resolved through cognitive means alone. Trauma-focused modalities such as EMDR, prolonged exposure, DBT-PE, and imagery rescripting are necessary to recalibrate threat circuitry and integrate fragmented emotional memories (Fischer-Kern et al., 2023; Marusak et al., 2023). Without resolving these underlying trauma processes, symptom-level anger treatments are likely to produce only partial or temporary change. Thus, a trauma-informed conceptualization is essential for any comprehensive anger treatment plan.

Anger is also deeply embedded in **interpersonal and systemic contexts**. Attachment patterns shape the ways individuals interpret conflict, perceive threat, and engage in relational regulation. Insecure attachment, notably anxious, avoidant, and disorganized patterns, is consistently associated with heightened reactivity and poor recovery following interpersonal stress (De Winter et al., 2023). Couple and family therapies, including EFT,

IBCT, systemic family therapy, and mentalization-based interventions, play crucial roles in addressing relational anger cycles. These models bring into focus the bidirectional nature of anger, emphasizing that emotional escalation is rarely an individual phenomenon but part of a larger system of communication, expectations, and interpersonal histories. Importantly, systemic and culturally responsive approaches help clinicians recognize when anger reflects reactions to chronic stressors such as racism, socioeconomic marginalization, or unresolved intergenerational trauma.

Cultural and ethical considerations remain foundational to competent anger treatment. Anger has different meanings across cultures, genders, and communities, and pathologizing normative expressions—or failing to recognize anger as a response to systemic oppression—can cause harm and erode trust. Ethical practice requires cultural humility, awareness of implicit biases, and sensitivity to the sociopolitical conditions surrounding anger expression (APA, 2017; NASW, 2021). Additionally, given the safety implications of anger dysregulation, clinicians must remain vigilant in assessing risk, maintaining clear boundaries, and adhering to professional ethics related to confidentiality, informed consent, and scope of competence.

Looking toward the future, several promising directions are emerging in the neuroscience and clinical treatment of anger. One exciting area involves precision psychotherapy, where assessment tools, such as neurocognitive profiling, physiological monitoring, and fine-grained behavioral analysis, help match clients to interventions most likely to address their specific dysregulation mechanisms. For example, individuals with high threat sensitivity may benefit most from mindfulness-based and physiological regulation techniques, while those with prominent cognitive biases may respond best to cognitive bias modification or schema work. Precision models hold the potential to significantly improve treatment outcomes by reducing trial-and-error approaches and streamlining intervention selection.

Another evolving trend is the integration of digital and wearable technologies into anger regulation. Mobile applications, virtual reality platforms, and wearable HRV monitors can capture real-time physiological data, deliver in-the-moment interventions, and extend treatment beyond the therapy room (Bjureberg et al., 2023). Advances in artificial intelligence may eventually enable personalized emotion-regulation prompts based on early indicators of escalation, offering immediate support during high-risk moments. Such tools can enhance accessibility, particularly for individuals living in underserved areas or those hesitant to engage in traditional therapy.

Innovations in neuromodulation and neurofeedback also show promise. Early evidence suggests that targeted brain-based interventions may strengthen prefrontal regulation networks or reduce limbic hyperreactivity, complementing traditional psychotherapy

approaches (Sitaram et al., 2023). While still emerging, these methods represent a frontier of integrative care for clients refractory to behavioral and cognitive interventions.

The neurobiology of anger offers a powerful lens through which clinicians can better understand, assess, and treat one of the most challenging yet fundamentally human emotional responses. Anger is not merely a behavior to be suppressed or a cognitive distortion to be corrected; it is a dynamic interplay of neural activation, emotional learning, attachment experiences, relational contexts, and sociocultural forces. Effective treatment demands an integrated, ethical, and culturally informed approach that honors both biological realities and human complexity. By drawing on the full range of contemporary evidence, from neuroscience to trauma frameworks to systemic therapies, mental health professionals are well-positioned to facilitate meaningful and lasting change for clients struggling with anger dysregulation. As the field evolves, the integration of emerging technologies, precision-based interventions, and culturally responsive practices promises to expand the possibilities of treatment and enhance the capacity of clinicians to promote safety, connection, and emotional wellbeing.

References

American Association for Marriage and Family Therapy. (2015). *AAMFT Code of Ethics*.

American Counseling Association. (2014). *ACA Code of Ethics.*

American Psychological Association. (2017). *Ethical principles of psychologists and code of conduct.*

American Psychological Association. (2017). *Multicultural guidelines: An ecological approach to context, identity, and intersectionality.*

Ballesteros, J., et al. (2023). Cognitive-behavioral therapy plus standard care for violence in psychotic disorders: A systematic review. *Schizophrenia Research, 256*, 89–101.

Bateman, A. W., & Fonagy, P. (2023). Mentalization-based treatment: Advances in theory and practice. *American Journal of Psychotherapy, 77*(1), 23–38.

Bertsch, K., Hill, L., & Herpertz, S. C. (2023). Social threat sensitivity and anger: Insights from affective neuroscience. *Neuroscience & Biobehavioral Reviews, 155*, 105399.

Bjureberg, J., et al. (2023). Internet-delivered emotion regulation treatment for maladaptive anger: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 91*(2), 153–167.

Chester, D. S. (2017). The role of the ventral striatum in retaliatory aggression. *Social Cognitive and Affective Neuroscience, 12*(4), 650–660.

Ciesinski, N. K., et al. (2023). Cognitive bias modification for aggression and anger: A systematic review and meta-analysis. *Aggression and Violent Behavior, 70*, 101937.

Coccaro, E. F., & Lee, R. (2023). Serotonergic function in impulsive aggression. *Neuroscience & Biobehavioral Reviews, 146*, 105056.

Coccaro, E. F., et al. (2023). Neural mechanisms of impulsive aggression in intermittent explosive disorder. *Neuropsychopharmacology, 48*(2), 321–334.

Dabek, M., et al. (2023). Prefrontal damage and aggression after traumatic brain injury: A neuroimaging review. *Brain Injury, 37*(2), 195–208.

Dahlgren, M. K., et al. (2023). Serotonin and impulsive aggression: An updated neurobiological review. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 8*(5), 442–455.

De Winter, S., et al. (2023). Attachment insecurity and neural responses to social threat: A meta-analytic review. *Social Cognitive and Affective Neuroscience, 18*(1), 15–28.

Denson, T. F., et al. (2019). Working memory training and anger regulation: A randomized controlled trial. *Journal of Experimental Psychology: General, 148*(2), 279–295.

Dougherty, D. D., et al. (2023). Inflammatory markers and affective dysregulation: A systematic review. *Journal of Affective Disorders, 331*, 65–78.

Farb, N. A. S., et al. (2023). Mindfulness training and prefrontal-limbic regulation: A systematic review of neuroimaging findings. *Neuroscience & Biobehavioral Reviews, 147*, 105044.

Fett, A.-K. J., et al. (2023). Threat misperception in psychosis: A neuroimaging meta-analysis. *Psychological Medicine, 53*(4), 1178–1190.

Fischer-Kern, M., et al. (2023). Neural correlates of imagery rescripting: A systematic review. *Neuroscience & Biobehavioral Reviews, 149*, 105182.

Fisher, E. L., et al. (2023). Virtual reality interventions for aggression reduction: A systematic review. *Clinical Psychology Review, 101*, 102291.

Geniole, S. N., et al. (2019). Neural correlates of anger and aggression: Insights from neuroendocrine research. *Hormones and Behavior, 111*, 50–59.

Geniole, S. N., et al. (2020). Hormonal mechanisms of aggression: The role of testosterone and cortisol. *Current Opinion in Psychology, 33*, 123–130.

Gerlicher, A., et al. (2023). Sympathetic arousal and emotional reactivity in anger. *Psychophysiology, 60*(4), e14201.

Gröndal, M., Ask, K., & Winblad, S. (2023). The distinction between irritability and anger and their associations with impulsivity and subjective wellbeing. *Scientific Reports, 13*, 10398.

Heberlein, A., et al. (2023). Neurobiological consequences of early trauma: A systematic review. *Neuroscience & Biobehavioral Reviews, 147*, 105026.

Johnson, S. M., et al. (2023). Attachment science and couple therapy: Updated empirical foundations for EFT. *Journal of Marital and Family Therapy, 49*(3), 567–584.

Keay, K. A., & Bandler, R. (2017). Defensive behaviors and fear: The role of the periaqueductal gray. *Progress in Neurobiology, 155*, 89–112.

Kolla, N. J., Tully, J., & Bertsch, K. (2023). Neural correlates of aggression in personality disorders: A systematic review. *Translational Psychiatry, 13*(1), 330.

Krämer, U. M., et al. (2023). Neural mechanisms of approach-related anger: A functional neuroimaging review. *Neuroscience & Biobehavioral Reviews, 153*, 105375.

Lazarus, R. S. (1991). *Emotion and adaptation.* Oxford University Press.

Lee, R., et al. (2024). Autonomic dysregulation in intermittent explosive disorder: A psychophysiological review. *Biological Psychology, 180*, 108576.

Lehrer, P., & Gevirtz, R. (2023). Heart rate variability biofeedback for emotional regulation: A transdiagnostic review. *Applied Psychophysiology and Biofeedback, 48*(1), 1–17.

Lieberman, M. D., et al. (2007). Putting feelings into words: Affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science, 18*(5), 421–428.

Linehan, M. M. (2015). *DBT skills training manual* (2nd ed.). Guilford Press.

Lindsay, J. A., et al. (2023). Acceptance-based therapies and emotion regulation: A meta-analytic review of neural mechanisms. *Clinical Psychology Review, 102*, 102244.

Marusak, H. A., et al. (2023). Childhood adversity and altered emotional brain networks: A meta-analytic review. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 8*(3), 301–312.

McCloskey, M. S., et al. (2023). Cognitive-behavioral therapy for impulsive aggression: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 91*(1), 24–38.

Miller, A. L., et al. (2023). Dialectical behavior therapy for adolescent aggression: A systematic review. *Journal of Clinical Child & Adolescent Psychology, 52*(4), 655–670.

Mohammadi, S., et al. (2023). Sex differences in neural responses to emotional stimuli: A systematic review. *NeuroImage, 275*, 120204.

Montoya, E. R., et al. (2023). The dual-hormone hypothesis revisited: Testosterone, cortisol, and aggression. *Psychoneuroendocrinology, 147*, 105988.

Neacsiu, A. D., et al. (2022). Opposite action and behavioral activation in emotion regulation: A meta-analytic review. *Clinical Psychology Review, 99*, 102103.

Okuda, M., et al. (2015). Prevalence and correlates of anger in the community: Results from a national survey. *The Journal of Nervous and Mental Disease, 203*(10), 756–762.

Osman, S., et al. (2023). Hormonal modulation of irritability and anger in premenstrual dysphoric disorder. *Journal of Psychiatric Research, 158*, 23–33.

Palmer, C. A., & Alfano, C. A. (2023). Sleep, emotion regulation, and psychopathology: A transdiagnostic perspective. *Sleep Medicine Reviews, 67*, 101744.

Palmier-Claus, J. E., et al. (2023). Neurobiological correlates of irritability across mood disorders: A systematic review. *Journal of Affective Disorders, 331*, 169–182.

Palop Larrea, V., et al. (2024). Anger and physical and psychological health: A narrative review. *Revue d'Études – Société & Santé.*

Panasiti, M. S., et al. (2023). Executive functioning and reactivity in aggression: A neurocognitive review. *Cognitive, Affective, & Behavioral Neuroscience, 23*, 67–82.

Porsch, R. M., et al. (2023). Genetic influences on irritability and aggression: A twin study. *Behavior Genetics, 53*(1), 45–57.

Reyna, C., et al. (2023). Anger as a bargaining emotion in social and organizational contexts. *Current Opinion in Psychology, 50*, 101647.

Richard, Y., et al. (2023). A systematic review of neural, cognitive, and clinical correlates of anger and aggression. *Current Psychology.* https://doi.org/10.1007/s12144-022-03143-6

Schaefer, J. D., et al. (2023). Neurobiological changes after DBT in borderline personality disorder: A meta-analysis. *Frontiers in Psychiatry, 14*, 1123456.

Sell, A., et al. (2009). Human anger: Evolutionary causes and consequences. *Philosophical Transactions of the Royal Society B, 364*, 3665–3674.

Shafti, M., et al. (2023). A systematic review of the co-occurrence of self-harm and aggression. *Clinical Psychology & Psychotherapy.* https://doi.org/10.1002/cpp.2885

Shamay-Tsoory, S. G., & Abu-Akel, A. (2016). The social salience hypothesis of oxytocin. *Biological Psychiatry, 79*(3), 194–202.

Shaw, P., et al. (2023). Emotional dysregulation in ADHD: Neural substrates and developmental pathways. *The Lancet Psychiatry, 10*(5), 389–402.

Sitaram, R., et al. (2023). Advances in neurofeedback for emotion regulation: Implications for anger and aggression. *NeuroImage: Clinical, 38*, 103379.

Timmons, A. C., et al. (2023). Physiological synchrony and coregulation in couples: A meta-analytic review. *Psychological Bulletin, 149*(6), 823–851.

Tonnaer, F., et al. (2023). Amygdala–prefrontal connectivity and trait anger: A meta-analytic review. *Brain Imaging and Behavior, 17*, 2149–2163.

Uddin, L. Q., et al. (2023). Neural substrates of self-referential processing in borderline personality disorder. *Current Psychiatry Reports, 25*(2), 71–82.

Uljarević, M., et al. (2023). Neural correlates of emotion regulation in autism: A systematic review. *Autism Research, 16*(7), 1331–1348.

van der Veen, D. C., et al. (2023). Trauma-focused therapy and sexual functioning in PTSD: Findings from an intensive treatment trial. *Journal of Traumatic Stress, 36*(1), 105–118.

Vitiello, B., et al. (2023). Hostile attribution bias and aggression: A developmental and clinical neuroscience perspective. *Development and Psychopathology, 35*(3), 1005–1021.

Whelan, Y. M., et al. (2023). MAOA genotype, childhood maltreatment, and aggression: An updated systematic review. *Neuroscience & Biobehavioral Reviews, 147*, 105035.

Young, J. E., Klosko, J. S., & Weishaar, M. (2003). *Schema therapy: A practitioner's guide.* Guilford Press.

Zvielli, A., et al. (2023). Attention bias modification for anger and aggression: A randomized controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry, 80*, 101797.