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Neurobiochemistry and Criminal Responsibility: The Future of the "Biochemical Defence" in Indian Criminal Justice System

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ABSTRACT

Neurobiochemical research, encompassing genetic markers like the low-activity Monoamine Oxidase A (MAOA-L) variant and neurotransmitter dysregulation, provides compelling biological evidence for aggressive behaviour stemming from volitional impairment—a diminished capacity to control impulses. This scientific reality directly clashes with the Indian Criminal Justice System's legal standard for the insanity defence under BNS Section 22. Indian jurisprudence, established through landmark precedents, rigidly adheres to the M'Naghten Rule (the cognitive test), requiring the accused to prove complete mental incapacity to know the nature or wrongfulness of the act. This paper critically examines the incongruity between modern neuro-criminology and Indian criminal jurisprudence. The analysis focuses on the severe evidential barriers facing biochemical data due to the law's omission of volitional deficits. Furthermore, we assess the risks posed by the judiciary's inconsistent assessment of novel scientific evidence in the absence of a standardized admissibility framework like Daubert. The findings demonstrate that the existing framework fails to justly assess neurobiologically compromised offenders, creating a potential constitutional challenge under Article 21. Legislative reform is

essential to introduce the doctrine of Diminished Responsibility (DR) into the BNS, providing an intermediate category of culpability (e.g., reducing murder to culpable homicide) that aligns with evidence of partial impairment.

The Foundational Principles of Criminal Liability in India

a) The Enduring Doctrine of *Mens Rea* and Culpable Intent

The Indian criminal justice system, codified under the newly introduced Bharatiya Nyaya Sanhita (BNS), maintains the fundamental principle that criminal liability requires both the *actus reus* (the guilty act) and the *mens rea* (the guilty mind). This doctrine of *mens rea* emphasizes that culpability is not determined solely by the commission of a wrongful act, but rather by the perpetrator's corresponding mental state, necessitating a conscious, voluntary, and intentional action. The concept ensures that punishment aligns with the offender's mental condition at the time of the offense, fostering justice and fairness.

General exceptions embedded within the BNS function to negate this required culpable intent. Provisions such as BNS Section 22 establish a legal defence by demonstrating that, due to specific circumstances, the necessary *mens rea* could not have existed. Crucially, Indian jurisprudence starts from the fundamental assumption that every individual is prudent, sane, and fully responsible for their actions. Consequently, the substantial burden of rebutting this presumption falls entirely upon the accused. The introduction of sophisticated neurobiochemical evidence must, therefore, be framed as an effort to negate the core elements of intent required for conviction. If neurochemistry only suggests an increased *risk* or predisposition to aggressive behaviour, it inherently fails to prove the complete absence of the necessary knowledge or intent required for a successful defence. This establishes an immense evidential barrier for the biochemical defence.

b) BNS Section 22: Unsoundness of Mind and the Strict Cognitive Test

BNS Section 22, titled 'Act of a Person of Unsound Mind,' directly replaces the former Section 84 of the Indian Penal Code (IPC). This provision stipulates that nothing constitutes an offense if it is done by a person who, "at the time of doing it, by reason of mental illness, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law".

The structure and interpretation of BNS Section 22 adhere rigidly to the nineteenth-century English M'Naghten Rule. This approach focuses exclusively on the **cognitive capacity** of the accused-their ability to understand the physical character of their conduct and distinguish between moral right and legal wrong. Judicial precedent requires that the claimed unsoundness of mind must be of such a degree that it completely extinguishes or grossly impairs the accused's rational mental processes and reasoning power. It is consistently held that mere mental illness, partial delusion, or a temporary behavioural disturbance is insufficient for exemption from criminal liability.

The definitive legal interpretation of the insanity defence was provided by the Supreme Court of India in the landmark case of ***Dahyabhai Chhaganbhai Thakkar v. State of Gujarat***. The Court laid down pivotal principles that strictly govern the use of this defence, which are directly applicable to BNS Section 22:

1. **Burden of Proof:** While the prosecution must prove the act and *mens rea* beyond a reasonable doubt, the accused bears the onus of proving the defence of legal insanity by the lower standard of a **preponderance of probabilities**.

2. **Critical Time:** The unsoundness of mind must be established specifically at the **time of the commission of the act**.

3. **Nature of Incapacity:** Mere medical evidence of prior mental illness or partial delusion is insufficient; the accused must prove the complete **incapacity of knowing the nature of the act or that it was wrong or contrary to law**.

4. **Conduct Assessment:** The court must examine the accused's conduct and behaviour immediately *before*, *during*, and *after* the offense to determine their mental state and whether the plea is a strategic manoeuvre to evade responsibility.

Further reinforcing the stringent evidentiary requirements, the Supreme Court in subsequent rulings has emphasized the necessity of examining the depth of incapacity. While some earlier interpretations held that unsoundness of mind which 'materially impairs' the cognitive faculties could suffice, the current prevalent judicial view requires a greater degree, often leaning towards the **complete extinguishment** of the ability to know the nature or consequences of the act. Furthermore, in cases like ***State of Madhya Pradesh***, the Apex Court underlined the procedural requirement that where evidence suggests prior mental illness, the authorities have a duty to ensure the accused is subjected to medical examination by a

competent psychiatric expert. Failure to do so can seriously damage the prosecution's case. Cases like ***Siddhapal Kamala Yadav v. State of Maharashtra*** exemplify the courts' strict interpretation, where the defence was rejected because the accused's plea of epilepsy and subsequent calm and quiet behaviour following the act failed to demonstrate the requisite loss of reasoning power at the critical time.

This adherence to a purely cognitive threshold presents a profound disconnect when attempting to incorporate modern neurobiological defences. The law requires the accused to prove they did not *know* the wrongfulness of the act. However, the emerging field of neuro-biochemistry primarily focuses on demonstrating **volitional impairment**—the inability to control aggressive impulses, even when the individual intellectually understands the difference between right and wrong. This fundamental conflict renders BNS Section 22 an inadequate legal mechanism for scientific evidence that points toward compromised impulse control rather than total cognitive breakdown.

The burden of proof rests with the accused, who must establish legal insanity not beyond a reasonable doubt, but by the lower civil standard of a **preponderance of probabilities**. If the defence under BNS Section 22 is successfully established, the accused is acquitted of the offense. However, the legal process anticipates the issue of public safety: the court retains the authority under the Criminal Procedure Code to order the detention of the acquitted person in a mental health institution for treatment and safety, thereby justifying incapacitation based on inherent risk, irrespective of culpability.

The Neurobiological Correlates of Aggression and Impulsivity

a) The MAOA Gene, Epigenetics, and the Gene-by-Environment ($G \times E$) Interaction

Neuro-criminology has increasingly focused on genetic factors that predispose individuals to violent and aggressive behaviour. The Monoamine Oxidase A (MAOA) gene is among the most studied entities in this field. MAOA encodes an enzyme responsible for catabolizing monoamine neurotransmitters, including dopamine, serotonin, and noradrenaline, thereby regulating their concentration in the synapse.

Genetic variation in the MAOA gene, specifically allelic variations in its Variable Number of Tandem Repeats (VNTR), determines its activity level. The low-activity variant, often referred to as MAOA-L, results in less efficient breakdown of these key monoamines, potentially leading to neurochemical dysregulation. While numerous studies suggest a correlation between MAOA-L and higher levels of aggression, the influence of this gene alone is insufficient to predict criminal behaviour.

The most compelling scientific evidence rests upon the **Gene-by-Environment (G\times E) interaction**. Studies have demonstrated that individuals possessing the MAOA-L genotype, when combined with severe early environmental adversity, such as childhood maltreatment, display significantly elevated levels of aggression and are correctly predicted to commit crime. The gene provides the underlying biological predisposition, while the traumatic environment acts as the requisite developmental trigger.

Epigenetic mechanisms provide a molecular explanation for this complex interaction. Epigenetics refers to heritable modifications in gene expression that occur without altering the underlying DNA sequence. Traumatic early-life experiences, such as abuse, can induce epigenetic changes (e.g., DNA methylation) in regulatory genes like MAOA, NR3C1, and OXTR. These persistent marks significantly impact neurodevelopment, leading to impaired stress response and poor self-regulation in adulthood, which are recognized precursors to aggressive behaviour. Arguing that the accused's neurobiology was not merely inherited but actively and physically *damaged* by maltreatment shifts the narrative from pure biological determinism to one of environmentally induced impairment, which may prove more mitigating in a legal context concerned with social determinants of crime.

Table 1: Illustrative Model of Gene-by-Environment Interaction on Aggression

This table is a conceptual model based on behavioural genetics research demonstrating the interaction between the low-activity MAOA gene and adverse environmental exposure in predicting aggression.

X-Axis: Childhood Maltreatment Exposure (Severity)	**Y-Axis: Aggression/Antisocial Score**	**Line A (MAOA-H Genotype)**	**Line B (MAOA-L Genotype)**
Low Maltreatment	Low/Baseline Score	Low Aggression	Low Aggression
High Maltreatment	Moderate Score	Moderate Aggression	Significantly Elevated Aggression

Source: https://pmc.ncbi.nlm.nih.gov/articles/PMC4306065/

b) Neurochemical Dysregulation and Impaired Volitional Control

The biochemical defence focuses on specific neurotransmitter imbalances that impair the brain's regulatory circuits, particularly those located in the Prefrontal Cortex (PFC), amygdala, and striatum.

1. **Serotonergic Hypofunction:** Research strongly associates impulsive and aggressive behaviour with deficient serotonergic function, characterized by low cerebrospinal fluid levels of serotonin metabolites (like 5-HIAA). Serotonin typically exerts an inhibitory action in the brain, regulating emotion and behaviour. Low serotonin levels are linked to poor function in the orbitofrontal cortex (OFC), a region vital for impulse control and observed as impaired in individuals exhibiting antisocial behaviour.

2. **Dopaminergic Disinhibition:** The serotonergic system modulates the activity of the dopaminergic system. When serotonin function is deficient (hypofunction), it can lead to the disinhibition and subsequent hyperactivity of the dopamine system. Since the dopamine system is critically involved in reward processing, motivated behaviour, and behavioural activation, this resulting hyperactivity reinforces impulsive and potentially aggressive actions.

This combined neurochemical profile—low inhibitory serotonin and heightened excitatory dopamine—disrupts the executive function required for impulse control, directly corresponding to a deficit in **volitional capacity**. The impairment is primarily related to the ability to resist an urge or impulse (irresistible impulse), rather than the cognitive knowledge that the act is wrong. This critical distinction confirms that neurobiological evidence is inherently aligned with an argument for partial responsibility, as opposed to the complete exoneration required by BNS section 22.

Table II: Conceptual Model of Serotonin-Dopamine Dysregulation in Impulsive Aggression

This table illustrates the pathway where deficient inhibitory neurotransmission leads to disinhibited behavioural activation in brain regulatory circuits, drawing from research on neurochemical mechanisms of impulsive aggression.

Neurotransmitter	**Normal Function**	**Dysfunction (Biochemical Defence Basis)**	**Behavioural Consequence**
Serotonin (5-HT)	Inhibitory control, mood stabilization	Hypofunction/Low Levels	Loss of inhibitory control
Dopamine (DA)	Reward, motivation, behavioural activation	Hyperactivity/Disinhibition	Heightened impulsivity, aggression
Key Brain Region	**Prefrontal Cortex (PFC) Regulation**	**Impaired Serotonin Modulation of DA**	**Irresistible Impulse/Poor Self-**

			Regulation

Source: https://pmc.ncbi.nlm.nih.gov/articles/PMC2612120/

c) The Scientific Limitations of Determinism

Despite the intriguing correlations found in neuroscience, researchers consistently caution against interpreting these findings as simple, deterministic causes of crime. The aetiology of violent behaviour is exceedingly complex and determined by a combination of countless biological, social, and environmental variables.

The attempt to find single biomarkers reliably connected to aggression has historically been fraught with difficulty, yielding conflicting results even for seemingly robust candidates like testosterone and cortisol. Crucially, experts acknowledge a "wide gap" between merely possessing a genetic variant, such as MAOA-L, and manifesting violent behaviour. This gap is mediated by a multitude of interacting factors, making the claim that genetics alone *caused* the offense scientifically tenuous and statistically unreliable for individual prediction. This inherent scientific complexity creates an immediate hurdle in judicial proceedings, as complicated, multi-factorial scientific testimony risks confusing the court (the trier of fact), thereby failing the basic relevance and reliability tests required for admissibility.

Admissibility and Judicial Gatekeeping of Biochemical Evidence in India

a) Legal Framework for Expert Evidence

The admissibility of neurobiological evidence in India is governed primarily by the Indian Evidence Act, 1872. Section 45 of the IEA, now section 39 of BSA, allows for the opinion of persons especially skilled in "science or art", now extend to "or any other field" to be considered relevant facts when the court must form an opinion on such a point. Under Sections 40 and 45, the facts and grounds upon which the expert's opinion is based-including scientific theory, hypothesis, and documented empirical methodology-are also relevant.

However, the path to admissibility is not automatic. The evidence must first meet the basic principle of relevancy, meaning it must make the fact in issue (the accused's mental capacity at the time of the act) more probable. A judge also retains significant discretion to exclude evidence if its probative value is deemed to be outweighed by the potential for harm, confusion, or undue prejudice.

b) Status of Scientific Reliability Standards

The Indian legal landscape currently lacks a mandatory, codified framework analogous to the U.S. *Daubert* standard for rigorously assessing the reliability and methodology of novel scientific evidence. Although some Indian courts have been influenced by *Daubert* principles, adopting a more flexible approach that looks at reliability, professional qualifications, and impartiality, the assessment of expert opinion remains non-uniform and highly dependent on judicial discretion.

This lack of a stringent, uniform standard poses a critical risk. Without robust guidelines, judges may give undue weight to complex, but ultimately unreliable, testimony, or conversely, dismiss credible scientific opinion. To uphold constitutional guarantees of justice and the right to life (Article 21), the judiciary must solidify its "gatekeeping function" to ensure that scientific evidence presented is empirically documented and scientifically sound, not merely speculative.

c) International Precedents and Admissibility Challenges

International experience demonstrates significant judicial apprehension toward incorporating behavioural genetic evidence into the culpability phase of criminal trials. In the U.S., courts have considered, and often excluded, low-activity MAOA gene evidence offered by the defence. For instance, the New Mexico Supreme Court upheld the exclusion of MAOA evidence intended to negate the premeditation required for first-degree murder, highlighting the complexity of variables and the lack of clear predictive correlation between genetics and individual violent behaviour.

The rationale for international exclusion applies strongly to the Indian context: since BNS Section 22 requires proof of complete cognitive incapacity (a very high legal bar), and international courts have struggled to admit MAOA evidence even to prove *diminished* intent (a lower bar), an Indian judge is highly likely to exclude the evidence as irrelevant or overly prejudicial for negating *mens rea*. Furthermore, Supreme Court rulings, such as in ***State of U.P. v. Krishna Gopal***, place importance on the "orality of the trial process," emphasizing the primacy of credible eyewitness testimony over medical opinions that merely point to alternative possibilities. This judicial tendency creates an inherent bias against complex scientific claims that seek to challenge established factual narratives of intent. Where genetic evidence has been successfully introduced abroad, its utility has been largely confined to the **sentencing phase** to argue for mitigation, rather than the initial culpability phase for seeking acquittal.

The Need for Legislative and Judicial Reform

a) The Inadequacy of BNS Section 22 for Neuroscientific Findings

The BNS, in retaining the language of "unsoundness of mind" from the IPC, failed to incorporate modern scientific understandings of the complex biological, psychological, and social causes of criminal tendencies. The law's restrictive focus on the cognitive capacity to *know* right from wrong makes it incapable of addressing neurological impairments that primarily affect **volitional capacity**.

The neurobiochemical findings regarding serotonergic-dopaminergic dysregulation fundamentally argue for a state of **irresistible impulse**—a condition where the accused, due to neurological dysfunction (often traceable through MAOA or epigenetic markers), knows the act is wrong but lacks the ability to restrain the impulse. Since Indian law does not formally recognize the irresistible impulse doctrine, evidence demonstrating substantial impairment of impulse control cannot achieve the full acquittal afforded by BNS section 22.

b) Advocating for the Doctrine of Diminished Responsibility (DR)

The scientific evidence clearly demonstrates *partial impairment* rather than *total incapacity*, necessitating the introduction of a doctrine of intermediate culpability. The doctrine of Diminished Responsibility (DR) offers the most scientifically and legally appropriate framework. DR acknowledges that a mental abnormality can substantially impair a defendant's capacity, thus lessening culpability without complete exoneration.

In jurisdictions that recognize DR, the defence typically reduces a charge of murder to manslaughter (culpable homicide in the Indian context), recognizing that while the accused committed the act, their capacity to take full responsibility was compromised. This intermediate outcome is perfectly suited for neurobiological evidence, which suggests *predisposition* and *partial impairment* of control, rather than complete psychological blackout.

India previously considered adopting DR following the 42nd Law Commission Report in 1971, but no legislative reform was enacted. The BNS revision represented a missed opportunity to align Indian criminal law with global scientific and legal trends. A specific legislative amendment to the BNS is required to introduce DR, preferably by replacing the term "unsoundness of mind" with a broader "mental impairment" that explicitly includes impairment of volitional capacity as grounds for mitigation.

Table III: Comparison of Legal Standards and Neurobiological Reality

Parameter	**BNS Section 22 (Cognitive Test)**	**Proposed Diminished Responsibility (DR)**	**Neurobiochemical Evidence (MAOA-L/Serotonin)**
Capacity Focus	Cognitive: Incapacity to **Know** (M'Naghten Rule)	Volitional/Cognitive: Substantial impairment to **Control** or **Understand**	Volitional: Impaired impulse control/Self-regulation
Legal Outcome	Complete Acquittal (with possible detention)	Reduction of Charge (e.g., Murder to Culpable Homicide)	Supports DR, fails to meet BNS section 22 standard
Scientific Fit	Extremely Low (Requires total psychosis)	High (Acknowledges partial impairment)	Suggests need for intermediate sentencing options

Source: https://www.cambridge.org/core/journals/international-annals-of-criminology/article/unsoundness-of-mind-and-neurological-impairments-a-reappraisal-of-section-22-of-the-bharatiya-nyaya-sanhita-2023/56973B608EEE5CC7F99A6F6F01742FAE

c) Ethical, Constitutional, and Dispositional Implications (Neurolaw in India)

The increasing use of neurobiological data in legal proceedings is recognized globally as a "double-edged sword." While it facilitates the pathways to a better understanding of human behaviour and can mitigate culpability for past acts, it simultaneously risks supporting the perception that the offender is inherently prone to future violence.

This raises serious constitutional and policy concerns in the Indian context. If neurobiological evidence is used to demonstrate a permanent, high-risk biological structure (e.g., a "gene for violence"), there is a substantial danger that judges, prioritizing incapacitation, might favour indefinite commitment or institutionalization over mandated rehabilitation. This would undermine the progressive, rights-based philosophy of the Mental Healthcare Act, 2017 (MHCA 2017), which aims to ensure persons with mental illness live life with dignity and non-discrimination.

The failure of BNS Section 22 to accommodate volitional impairment creates a potential constitutional vulnerability. If a defendant can scientifically prove substantial biological impairment but is denied any

form of defence (full acquittal or mitigation) because the law only recognizes cognitive failure, forcing a full conviction and potentially the maximum penalty, the provision could be challenged as arbitrary and violative of the Right to Life and Liberty (Article 21) by failing to accurately assess true culpability.

Therefore, any legislative reform must be holistic. The disposition of neurobiologically impaired offenders must align with the MHCA 2017, prioritizing rehabilitation and community-based treatment plans, such as cognitive-behavioural therapy (CBT), education, and vocational training, over purely punitive or incapacitate incarceration. Given the MHCA's current shortcomings regarding continuous community care, legislative efforts must ensure robust, state-supported mental health services are available to facilitate the successful reintegration of these offenders and mitigate recidivism risks.

Conclusion and Recommendations

The future of the "Biochemical Defence" in the Indian Criminal Justice System faces formidable challenges rooted in the colonial-era structure of culpability preserved within the Bharatiya Nyaya Sanhita (BNS). Neuro-biochemistry, exemplified by the MAOA gene and serotonin/dopamine dysregulation, establishes a compelling case for *diminished* volitional capacity, yet BNS Section 22 only grants exemption for *total cognitive* incapacity. Consequently, scientific evidence intended to mitigate aggressive behaviour falls into the legal void between full responsibility and complete exoneration.

To ensure that the Indian legal system adheres to its constitutional mandate of justice and aligns with modern scientific understanding, the following recommendations are imperative:

1. **Legislative Reform of the BNS:** The BNS must be amended to formally introduce the doctrine of **Diminished Responsibility (DR)**. This reform should broaden the general exceptions to include substantial impairment of **volitional capacity** due to mental or neurobiological abnormality, allowing scientific evidence to reduce charges based on partial culpability.

2. **Judicial Gatekeeping Standardization:** The Supreme Court must establish clear, uniform guidelines for the admissibility and reliability of novel scientific evidence, particularly neurogenetic and neuroimaging data, under Section 45 of the Indian Evidence Act. A modified *Daubert* standard focusing on scientific methodology, peer review, and demonstrated empirical reliability is necessary to prevent the admission of speculative biomarkers and ensure rigorous judicial gatekeeping.

3. **Rights-Based Dispositional Policy:** Legal outcomes for neurobiologically impaired offenders must strictly follow the rehabilitative, rights-based framework of the Mental Healthcare Act, 2017. Disposition should prioritize targeted pharmacological or behavioural therapies and community-based reintegration measures, rather than resorting to prolonged preventative incapacitation based on inherent genetic risk factors.

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