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Letter to the Editor

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Naltrexone as a possible treatment for the violent patient in forensic and non-forensic settings

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The article in February 2021 of *CNS Spectrums* titled "Pharmacological interventions to reduce violence in patients with schizophrenia in forensic psychiatry" had thoroughly elucidated the lack of reliable studies that could draw firm conclusion regarding the pharmacological intervention in the reduction of violence in schizophrenia spectrum disorders (SSDs) in forensic settings due to substantial methodological limitations.[1] The article used the WHO definition of violence which stated "A violent act is an aggressive, domineering, forceful, or assaultive verbal or physical action, most often directed against self or others."[1] The article also claimed that there are links between a range of mental disorders and violence as evidenced by a systematic review and meta-analysis of 20 original studies that illustrated a clear association between schizophrenia, substance use disorders (SUDs), and violence, while risk rose markedly when SUD was present.[1] Despite this supposed link, the authors also briefly acknowledged that the overall contribution of violent offenses committed by patients with schizophrenia to society's general level of violence is very small. However, the authors did not consider that this supposed link of SSD–SUD might also be a false positive triggered by these methodological flaws. Additionally, the article's emphasis on SSD–SUD connection to violence may give the readers the false notion that violence is prevalent in SSD–SUD patients. In reality, this banal connection between substance users and increased aggression is well documented as more common in the general population.[2]

Unfortunately, these flawed studies in forensic and non-forensic settings have further obfuscated the alleged link between mental illness and aggression/violence, thus creating uncertainty around any treatment approaches. Therefore, we should step back and reassess this hypothesis of SSD–SUD-aggression nexus, the hypotheses behind the etiology of violence/aggression, as well as the various pharmacological tools used as interventions.

Since we do not fully grasp at this time the true mechanism of action that promotes aggressive/violent behavior, then let us propose that violence/aggression is a heterogeneous disorder with multiple etiologies. Hence, practitioners could become more focused on practicing proactive rather than reactive interventions, thus developing an effective bio-psycho-social-cultural anticipatory management model for aggressive behavior in inpatient psychiatric settings (forensic and non-forensic).

Moreover, let us also propose that violent behavior is a habitual form of reactive/impulsive or deliberate self-preservation mechanism precipitated by a perceived distress or a threat (real or imagined) to the autonomous self, or by an overwhelming need to have power over others. In addition, if we accept the notion that behaviors are habits with addictive proclivity that utilize a self-reinforcing mechanism through the mesolimbic pathway and the Nucleus Accumbens, then reducing and/or modulating the connectivity in the subcortical region of the brain (specifically in the NAc-ML-DA) and strengthening the firing-wiring network in the prefrontal cortex through pharmacological and behavioral interventions might diminish the frequency of violent acts. Medications such as Naltrexone or other drugs with a similar mechanism of action (nonselective opioid receptor antagonist that may attenuate extracellular dopamine in the NAc) should be further investigated as tools that reduce the activation triggered by positive or negative stimuli which the NAc craves.

Despite the brain's neuroplasticity that welcomes new wiring through new experiences, changing the deeply ingrained habits can be a daunting task. Consequently, the violent individual might often resist change, and would automatically revert back to his or her comfortable old wiring state whenever a perceived threat arises. Cognitive behavioral interventions combined with Naltrexone and a self-correcting (cybernetic) model could play an important role in revamping the neural structure of violent behavior. The repeated process of self-correction can lead to a new wiring leading to a fortification of the new connectivity in the prefrontal cortex, which will eventually supersede the old connections. Furthermore, retraining our psychiatric staff in forensic and non-forensic settings in behavioral methods that refine their interactions with patients is critical to make the restrictive/containing and involuntary environment less hostile and more palatable to the patient thus increasing compliance with treatment. However, a cookie-cutter approach may not work for every aggressive patient, therefore, we ought to tailor the

violent-reduction management plan to fit the intersectionality model of genetics and environment, thus considering the patient's background, which could include exposure to chronic violence and trauma, history of violent behavior, drug and alcohol use, impulsivity, as well as sociopathic/narcissistic tendencies. Genetics might also play role in the future when new associations among certain codons and violence are identified, although having a link does not automatically translate into violent action. Keeping in mind the base rate bias, all of the components mentioned above could become amplified when laced with psychosis, which might occasionally lead to aggressive conduct, although does not mean that SSD patients are more prone to violence than other individuals in the general population.

In regards to conventional treatment of aggression, the current medical interventions are limited to an armamentarium of psychotropic medications that act as chemical straitjackets by momentarily deterring the violent act. For example, neuroleptics such as Clozapine might be temporarily effective in slowing down the person's motor ability and dampening some of the implicated neurotransmitters, but does not alter or repair the defective inner brake system prompted by the strong connectivity to the subcortical region of the brain from years of coalescent conditioned behavior and presumed genetic tendencies. In addition, psychotropic over-sedation, and poor titration can also be problematic resulting in akathisia, agitation, and/or a rebound effect leading to aggression.

Finally, practitioners should probably be asking themselves whether to accept this old problem of human aggression/violence as an innate human characteristic, or reframe it as a medical condition caused by a dysfunction in the neurochemical interaction matrix. If the human violent behavior turns out to be solely a product of a biological shortcoming, then it would be extremely disheartening to accept that centuries of wars and violence that led to millions of deaths had been nothing more than a manifestation of a dysregulated chemical system that had gone awry.

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