Trauma as a Contributor to Violence in Autism Spectrum Disorder

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In examining contributors to violence among individuals with autism spectrum disorder (ASD), one factor that has received little attention is a history of psychological trauma. This study's purpose was to explore the possible mechanisms for an association between trauma and violence in persons with ASD. The literature regarding the neurobiology and theoretical underpinnings of ASD is reviewed and compared with the literature on the neurobiology and theoretical underpinnings of trauma as a risk factor for violence in individuals without ASD. Information from this comparison is then used to formulate possible mechanisms for a trauma-violence association in ASD. Individuals with ASD may possess sensitized prefrontal-cortical-limbic networks that are overloaded in the face of trauma, leading to unchecked limbic output that produces violent behavior, and/or cognitive dysfunction (including deficits in theory of mind, central coherence, and executive function) that impacts trauma processing in ways that portend violence. While these mechanisms for a trauma-violence association in ASD may have case-based support, more research is needed to confirm these mechanisms and clarify whether in fact trauma increases violence risk in ASD. To facilitate the investigation, it would be helpful for clinical and forensic evaluators to obtain a careful trauma history when evaluating all individuals, including those with ASD.

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For more than two decades there has been a growing interest in the relationship between autism spectrum disorder (ASD) and violence. A review of the literature, including descriptive case reports,[1–29] prevalence studies,[30–46] and previous reviews,[47–54] indicates no conclusive evidence that individuals with ASD are more violent than individuals in the general population. However, among individuals with ASD, specific generative (e.g., comorbid psychiatric disorders,[8,38,40,47] social cognitive deficits,[48–50] and emotion regulation problems[41,50,51]) and associational (e.g., younger age,[39] repetitive behavior,[29,39,45] and a diagnosis of Asperger's syndrome[38]) factors have been shown or posited to increase the risk of violent behavior.

One factor that has received little attention as a possible contributor to ASD-related violence is a history of psychological trauma. This factor was not examined in the above-cited studies, and research on the effects of trauma in individuals with ASD is remarkably limited. Only a few published reports have examined this possibility.[55–57] For example, Mandell *et al.*[55] found that, among 156 children with ASD treated in a community mental health setting, 18.5 percent had been physically abused and 16.6 percent had been sexually abused, and that both physically and sexually abused children were more likely to engage in sexual acting out and abusive behavior than were nonabused children. Mehtar and Mukaddes[56] noted that, in a sample of 69 children and adolescents with ASD consecutively referred to a university autism clinic, 26 percent had a history of trauma (most commonly, witnessing or being a victim of accidents, disasters, or violence), and of those, 94.4 percent were noted to show increased aggression in the six months following the trauma. However, it is unclear how aggression was defined in this study, and the sample (university clinic-referred) may have been selected for disturbance. Kawakami *et al.*[57] studied 175 youths and adults with ASD and found higher rates of past physical abuse and neglect (36.1% and 25%, respectively) in those with a criminal history

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than in those without such a history (7.2% and 2.2%, respectively). They concluded that physical abuse and neglect were significant risk factors for criminal behavior (a third of which was violent) in persons with ASD.

Although these studies may provide preliminary support for a trauma–violence association in ASD, one relied on caregiver reports,[55] and another may have recruited subjects selected for disturbance.[56] More research is needed to confirm a trauma–violence association in ASD. In the meantime, given the limited literature that addresses trauma as a potential contributor to violence in ASD and the lack of reports that examine how such a relationship, if established, could be explained, the aim of this review was to explore possible mechanisms for an association between trauma and violence in individuals with ASD.

Method

The literature regarding the neurobiology of (and theories to explain) ASD was reviewed and compared with the literature on the neurobiology and theoretical underpinnings of trauma as a risk factor for violence in individuals without ASD. Information from this comparison was then used to formulate possible mechanisms for an association between trauma and violence in individuals with ASD. Case reports providing potential support for these hypothesized mechanisms are also cited.

Results

To explore the possible mechanisms of an association between trauma and violence in individuals with ASD, I first considered the neurobiology of ASD and compared it to the neurobiology of individuals at risk for violence who have been traumatized.

There is reasonable evidence that specific brain regions and the interconnections between them are critical in producing difficulties in social cognition, emotion recognition, emotion regulation, and repetitive behaviors in individuals with ASD. For example, neuropathologic studies in ASD have shown smaller, more densely packed neurons in the amygdala, hippocampus, entorhinal cortex, mammillary body, anterior cingulate gyrus, and nuclei of the septum[58,59]; a decreased number of Purkinje cells in the posterolateral and inferior cerebellum[59,60]; and cortical abnormalities.[60] Morphometric studies have noted volumetric reductions in the amygdala and hippocampus relative to total brain volume in patients with ASD compared with control patients[61]; according to Tuchman,[62] these findings (along with the histopathologic observations noted earlier) suggest dendritic tree and neuropil underdevelopment, reflecting incompletely developed connections between limbic system structures and the cerebral cortex.

Structural MRI studies in high-functioning individuals with ASD compared with age- and IQ-matched controls, have shown decreases in gray matter in the right paracingulate gyrus and left inferior frontal gyrus and increases in gray matter in the amygdala/periamygdaloid cortex, middle temporal gyrus, and inferior temporal gyrus and in the cerebellar regions.[63] Functional (f)MRI studies in individuals with ASD have demonstrated activation of frontotemporal regions, but not the amygdala, when making inferences about others' mental states (theory-of-mind tasks) from the eyes, in contrast to control subjects who showed activation of the superior temporal gyrus and amygdala during such tasks.[64] Positron emission tomographic (PET) studies similarly using theory-of-mind tasks in subjects with ASD, and control subjects have revealed a lack of normal activation of the left prefrontal area in those with ASD.[65]

Quantitative electroencephalogram (EEG) studies have found significant differences between individuals with ASD (Asperger's syndrome) and control subjects in the amplitude of slow and fast waves in the frontal, temporal, and temporal-parietal cortices (so-called mirror neuron areas, i.e., areas containing visuomotor neurons that may have a role in imitation, empathy, understanding, and predicting others' behavior, and language). Such studies have also revealed abnormalities in the anterior cingulate, amygdala, uncus, insula, hippocampal gyrus, parahippocampal gyrus, fusiform gyrus, and the orbitofrontal and ventromedial areas of the prefrontal cortex.[66]

Neurochemical studies have shown increased platelet serotonin levels[67] and elevated plasma norepinephrine levels[68] in individuals with ASD.

In addition to findings from neurobiological studies of individuals with ASD, it is helpful to consider existing theories regarding the basis for various symptoms seen in ASD. For example, one theory involves

the mirror neuron system (MNS), as noted above. According to this theory, mirror neurons are involved in imitation of movements, copying appropriate social interactions, understanding others' intentions, and language function.[69] Such neurons are located primarily in the frontal, parietal, and temporal cortices. It has been shown that MNS activity correlates with empathic concern and social competence[70] and that children with ASD show decreased activity in MNS regions during tasks that require them to mirror facial expressions of different emotions.[71] Moreover, mirror neurons have strong connections to the limbic system including the anterior cingulate and the amygdala, and delayed conductivity in the MNS for imitation has been found in individuals with ASD.[72] Quantitative EEG studies have shown abnormal findings in the amygdala and parietal regions in individuals with ASD.[66]

According to another theory, called salience landscape theory,[73] sensory information is normally conveyed to the amygdala, where it is compared with stored information, resulting in selection of an appropriate emotional response; the salience of the information is compared with an existing environmental landscape in the person's mind. In individuals with ASD, however, the pathways from the sensory areas of the brain to the amygdala may be altered, leading to extreme emotional responses to minimal stimuli and triggering of the autonomic nervous system with associated heart racing and distress. Self-stimulation and repetitive behaviors may therefore serve a self-soothing function for these persons.

Other theories focus on the concept of theory of mind: the ability to identify and understand the thoughts and feelings of others to make sense of their behavior. Such a capacity has long been conceptualized as a core deficit of individuals with ASD.[74] Hill and Frith[75] posited possible malfunction in the medial prefrontal cortex, temporal-parietal junction, and temporal poles as contributory to this deficit.

In addition, weak central coherence theory[76] argues that individuals with ASD are unable to synthesize information and make sense of it in a normal fashion. They cannot form a coherent understanding of what is occurring, because they fail to take note of how context affects the meaning of what is said or done. This effect may manifest in the ability to respond to only part of what is said, the part that relates to the person's special interest area. According to Thompson *et al.*,[66] weak central coherence likely involves a lack of appropriate connectivity between the posterior sensory processing areas of the brain and the frontal areas that modulate responses to the sensory input ("top-down" modulation). This lack of connectivity may result in piecemeal recall, causing the person to focus on trivial detail while missing the big picture.

Still another theory to explain features of ASD is that of executive dysfunction. This theory is based on the observation that executive functioning (including attention, planning, inhibition, and mental flexibility) seems to be impaired in individuals with ASD, functions controlled largely by the prefrontal cortex.[66] However, executive dysfunction is not unique to ASD and can be seen in other conditions that involve frontal lobe dysfunction (e.g., head trauma, attention deficit hyperactivity disorder, obsessive compulsive disorder, and Tourette's syndrome).

A final theory to consider is the polyvagal theory,[77] which proposes that the social engagement difficulties, attachment problems, tactile sensitivity, and poor listening skills in individuals with ASD are due to dysfunction in three circuits that regulate reactivity: the unmyelinated vagus, which induces immobilization (such as when animals feign death); a sympathetic–adrenal component, which fosters mobilization (fight or flight); and the myelinated vagus, which helps regulate social communication, self-soothing, and calming. According to this theory, to engage in social behavior, a person must feel safe, and attaining a feeling of security involves evaluating the environment. The neural structures involved in this process include the fusiform gyrus and the superior temporal sulcus, which are not activated in individuals with ASD. As a result, the limbic defense system involving the amygdala is not properly inhibited and the individual experiences vigilance and anxiety. This effect is coupled with difficulty regulating visceral states (e.g., vagal regulation of the heart to slow it down). Porges[78] also commented on neurophysiological interactions between what he called the social engagement system, the hypothalamic-pituitary-adrenal (HPA) axis, oxytocin and vasopressin, and the immune system.

In summary, neurobiological studies in individuals with ASD suggest structural and functional abnormalities involving the prefrontal cortex, frontal and temporal cortices, limbic system (particularly the amygdala and anterior cingulate), and the intercon-

nections between these areas as contributory to many of the symptoms of this disorder. In addition, elevated platelet serotonin levels and plasma norepinephrine levels have consistently been observed in individuals with ASD. Most explanatory theories of ASD symptoms (e.g., impaired theory of mind ability, weak central coherence, executive dysfunction) imply abnormal functioning in these same regions in the pathogenesis of such symptoms, and the polyvagal theory further suggests vagal and sympathetic–adrenal involvement.

I turn now to individuals (without ASD) who have been traumatized. Research has shown that a history of psychological trauma may increase the risk of aggression.[79–85] For example, Heide and Solomon[79] noted that childhood trauma (e.g., from abuse and neglect) causes disorganized or insecure attachment, which compromises right brain development, leading to impairment in the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), amygdala, and connecting circuits and may result in a diminished sense of self and disconnection from other people (OFC impairment), impaired ability to regulate affect (OFC and ACC impairment), impaired development of empathy (ACC impairment), and aggression (from decreased inhibition by the OFC of hypothalamic regions associated with aggression). They also noted that childhood trauma causes dysregulation of the HPA axis, leading, in the long-term, to decreased cortisol secretion and increased vulnerability to stressors later in life, including maladaptive coping strategies that may manifest in aggressive behavior toward self or others.

Dutton[80] noted that insecure attachment during the "rapprochement subphase" of early development (1.5–2 years) represented a form of trauma in immature men, with resulting difficulties in ego development and tendencies to use control and violence as safeguards against abandonment by intimate others. He noted that the prefrontal cortex (PFC) and orbitofrontal cortex (OFC) and the interconnections among them were central in the generation of impulsive aggression. Citing studies by Raine *et al.*[81] and Schore,[82] he theorized that the PFC/OFC acts as a brake and that subcortical areas (e.g., the limbic system) serve as an accelerator for violence. He also noted that aggression is associated with low 5-HT (5-hydroxytryptamine) levels, as well as elevated NE (noradrenergic) function, changes that can be induced by separation from an attachment object in particular.

Dent and Jowitt[83] reported that multiple, severe traumatic events in childhood can significantly contribute to aggressive, antisocial, and violent behavior in children and adolescents and that repeated physical and sexual victimization in particular is a risk factor for adolescent homicide. Beauregard *et al.*[84] similarly noted childhood sexual abuse to be a risk factor for sexual homicide of children.

As a final example, Losey[85] reported that a history of being bullied can increase an individual's risk for homicidal behavior, depending on various cognitive (e.g., feeling unable to escape bullying), psychiatric (e.g., depression, impulsivity, substance use), social (e.g., feeling alienated, bystander behavior), and family-related (e.g., lack of emotional support, poor parenting techniques) contextual factors.

Given that trauma is associated with impaired development and functioning of the prefrontal (including orbitofrontal) cortex, the anterior cingulate cortex, the amygdala, and the HPA axis (changes that may increase a traumatized individual's risk for aggression) and given that dysfunction in many of these same areas is implicated in the pathogenesis of ASD symptoms, it is reasonable to posit that individuals with ASD would be particularly vulnerable to the effects of trauma in increasing their risk for violent behavior. Moreover, similar neurochemical abnormalities (e.g., elevated plasma norepinephrine levels) have been demonstrated in individuals with ASD and in traumatized individuals, including those who have become aggressive. In addition, cognitive explanations for ASD symptoms including impaired theory-of-mind abilities, weak central coherence, and executive dysfunction have implications in terms of how individuals with ASD may process traumatic experiences (e.g., overfixation on peripheral aspects, misattribution of sources of negative emotions, and inflexibility in considering alternatives to aggression as a means of coping or seeking misdirected revenge), possibly increasing the risk of violence. Finally, it could be inferred from polyvagal theory (which hypothesizes dysregulation of the unmyelinated vagal and sympathetic–adrenal) and myelinated vagal systems, causing hypervigilance and anxiety in individuals with ASD, that such individuals are especially susceptible to adverse effects of traumatic experiences that produce similar neurophysiologic effects.

Thus, a possible mechanism for an association between trauma and violence in ASD is that individuals with ASD possess sensitized and dysfunctional prefrontal-cortical-limbic networks that, under normal circumstances, impose on them difficulties in social cognition, emotion recognition, emotion regulation, and repetitive behaviors. Primed to function like the brains of traumatized individuals, the brains of nontraumatized individuals with ASD may fall short of triggering violent behavior because of adequate social supports, predictable daily routines, consistently safe environments, and other factors. Once exposed to a traumatic event (or repeated traumatic events), however, the individual with ASD may experience a "network overload" in which already impaired prefrontal and cortical abilities to modulate limbic and hypothalamic agitated/defensive/hypervigilant output is further compromised, to a degree unmanageable by internal (e.g., withdrawing or engaging in repetitive behaviors) or external (e.g., reassurance or redirection by others) interventions. The result may be violent behavior.

From a cognitive standpoint, another possible mechanism for a trauma–violence association in ASD is that individuals with ASD, by virtue of their difficulty in identifying and understanding the mental states of others, their poor ability to consider context in processing and appropriately responding to sensory information, and their executive dysfunction, may misattribute the source of anger, rage, fear, and other intense emotions caused by a previous trauma to persons not associated with the traumatic event, because of some irrelevant similarity of that person (or group) to the perpetrator or some other aspect of the event. Thus, for example, an individual with ASD who has endured repeated physical and sexual abuse by a perpetrator with a gold necklace and odor of cigarette smoke on his person may become fixated later on exacting revenge on an individual (or group of individuals) observed to have these same characteristics (weak central coherence); he may choose to confront such an individual and, while doing so, fail to recognize the confused, surprised, and fearful reactions of that person (impaired theory of mind abilities), instead interpreting these reactions as further evidence of guilt; and he may be unable to consider alternatives to violence as a means of dealing with his anger, rage, and other negative emotions resulting from the trauma (mental inflexibility associated with executive dysfunction).

Potential support for these mechanisms for an association between trauma and violence in individuals with ASD is provided by numerous case reports describing individuals with ASD who engaged in violent behavior. For example, Baron-Cohen[3] described a 21-year-old man with ASD who was repeatedly violent toward his 71-year-old "girlfriend," as well as toward others. It was noted that this man's mother committed suicide when he was 11 years of age, and that he saw his mother dead. He was also noted to have a stepmother who "hated him." In another case, Simblett and Wilson[21] reported on a 22-year-old woman with ASD and a history of violent and destructive behavior in various settings; this woman's mother died when she was 19, leaving her in the care of her father who drank heavily and whose marriage to her mother had been stormy. In a third case, Bankier *et al.*[7] described a 26-year-old man with a history of repeated violent attacks on his mother whose father repeatedly physically abused him as a child and whose parents subsequently divorced. A fourth case[15] involved a sexual serial killer whose parents divorced when he was a child and whose mother departed without making arrangements for his care when he was 18. In a fifth case, Barry-Walsh and Mullen[20] described a man who experienced "considerable abuse" in a secure hospital during his adolescence, leading to fear and mistrust of health professionals; he eventually threatened and stalked some of the professionals involved in his care. In a sixth example, Silva *et al.*[18] described a man who committed serial homicides; his father had committed suicide when the man was 28 years of age.

Other case reports have described individuals with ASD committing violent acts who had a history of being bullied[2,9,12,14,16,25] or who had experienced separation from caregivers at a critically early age because of perinatal or other events (e.g., maternal psychiatric hospitalization,[21] neonatal hypoxia,[6] neonatal allergic reaction,[17] neonatal dehydration,[6] and maternal bleeding[6]). Although most of these case reports focus on the role of social cognitive (e.g., theory of mind) and other ASD-related deficits in explaining the violent behavior referenced, it is interesting to consider the potential role of trauma in contributing to the violence, in light of the above discussion. For example, Murrie *et al.*[14] described a 31-year-old man with ASD charged with 11 counts of arson; in the year before the offense, he became increasingly preoccupied with those who had wronged him during

his childhood and of the need to avenge himself. After hearing an arson report on the news, he concluded that fire-setting was the best way to address his concerns. Over a two-month period he broke into various summer homes in his neighborhood, setting them on fire. When questioned by police, he explained that the fires were a means of exacting revenge on schoolmates who had harassed him during his youth. It was determined that there was no actual relationship between the summer homes and the schoolmates, but this man described small details of these homes that reminded him of peers who had harassed him. Thus, his inability to consider alternatives to arson as a means of addressing his concerns suggests mental inflexibility associated with executive dysfunction, and his choosing to set fire to homes that reminded him of peers who had harassed him suggests weak central coherence; taken together, these deficits may have significantly affected this man's processing of earlier traumatic bullying/harassment by peers in a way that fueled his subsequent violent behavior.

Discussion

In this article, I have attempted to explore the possible mechanisms for an association between trauma and violence among individuals with ASD, given little attention in the current literature to the role of psychological trauma in affecting violence risk in individuals with ASD, either alone or in combination with better studied generative or associational risk factors (e.g., comorbid psychiatric disorders,[8,38,40,47] social cognitive deficits,[48–50] emotion regulation problems,[41,50,51] diagnosis of Asperger's syndrome,[38] younger age,[39] and repetitive behaviors[29,39,45]). Studies in individuals without ASD intuitively suggest such an association, given that a history of trauma has been reported to increase their risk of aggression.[79–85] To date, this is the first attempt to explore the possible mechanisms for an association between trauma and violence in ASD.

Neurobiological studies in individuals with ASD implicate prefrontal, frontal, temporal, and limbic brain regions and their interconnections as central in the genesis of ASD symptoms; these systems are also thought to be dysfunctional in traumatized individuals without ASD who are at risk for violent behavior. Thus, a sensitized prefrontal-cortical-limbic network in individuals with ASD may be the mechanism underlying an association between trauma and violence in this group; such a sensitized network at baseline may produce the difficulties in social cognition, emotion recognition, emotion regulation, and repetitive behaviors characteristic of ASD, whereas exposure to trauma may "overload" the network, leading to unchecked limbic (e.g., amygdalar) output that portends violent behavior.

In addition, from a cognitive standpoint, individuals with ASD, by virtue of their difficulty in identifying and understanding the mental states of others, their poor ability to consider context in processing and appropriately responding to sensory information, and their executive dysfunction, may process traumatic experiences in ways that include overfixation on peripheral aspects, misattribution of sources of negative emotions, and inflexibility in considering alternatives to aggression as a means of coping or seeking revenge, increasing their risk of engaging in violence.

Support for these mechanisms for a trauma–violence association in ASD may be suggested by case reports of individuals with ASD who have been violent, in whom a history of potential trauma can be identified.

Although interesting, these mechanisms and case reports do not establish or prove that trauma increases the risk of violence in individuals with ASD. More research is needed to answer that question (building on previous studies examining populations of individuals with ASD who have and have not been violent and comparing the two groups for a history of trauma). To facilitate further study, it would be helpful for clinical and forensic evaluators to obtain a careful, detailed trauma history when evaluating all individuals, including those with ASD, recognizing that the latter may have difficulty conveying a history of traumatic experiences due to such factors as cognitive limitations, alexithymia, and poor emotional recall.[86]

Limitations

The limitations of this article are as follows: first, although the proposed mechanisms for a trauma–violence association in ASD are plausible and grounded in a review of the relevant literature, such mechanisms lack empirical confirmation by way of neurobiological or other scientific studies in traumatized individuals with ASD who have been violent. Second, although the case reports cited offer potential support for the proposed mechanisms for a

trauma–violence association in ASD, they do not specifically confirm prefrontal-cortical-limbic dysfunction as responsible for the violent acts described, nor do they prove that trauma actually increases risk of violence in individuals with ASD. Third, in the referenced case reports, other potentially confounding factors may have accounted for violent behavior in the individuals reviewed (e.g., undiagnosed or unreported psychopathic features or psychiatric disorders or socioeconomic status). This possibility reflects the broader question of the complexity of research on violence and its determinants, an area in which all studies face limitations in accuracy of diagnosis (of violence and of the variables under study), reliability of reporting of violence and other behavior, and the multifactorial etiology of violence. Finally, brain studies, although interesting and potentially enlightening in proposing neuroanatomical and neurophysiological bases for behavior, do not necessarily correlate with behavior, and the current state of neuroscience is that the meaning of brain differences between various groups remains unclear.

Future Research

This article has identified possible mechanisms for an association between trauma and violence in individuals with ASD. However, more research is needed to answer two important questions: whether there is an association between trauma and violence in ASD in the first place; and what mechanisms underlie such an association, if confirmed.

Initial studies, building on the limited research conducted to date,[55–57] should focus on the first question—whether there in fact is a trauma–violence association in ASD—and would ideally employ a prospective design; assess a large, unbiased sample of individuals with ASD for a history of psychological trauma using effective and well-validated screening tools/criteria; control for the effects of comorbid psychiatric disorders and other potential risk factors mitigating violence risk, either by use of stringent inclusion/exclusion criteria or (perhaps more practically) by conducting appropriate analyses that control for the effects of such variables; use clear definitions for "ASD," "violence," and "traumatic event"; measure violence by a combination of individual/family self-report and legal record corroboration; and compare individuals with ASD who engaged in violence with those who did not in terms of any history of trauma.

If an association between trauma and violence risk in ASD is confirmed, studies could then target the second question, starting with clarifying the neurophysiologic and psychological responses to trauma in individuals with ASD. As previously noted, the neurobiology of trauma has been studied in individuals without ASD, with evidence to suggest that such individuals may be at increased risk for aggression based on abnormalities in prefrontal, orbitofrontal, limbic, and HPA axis functioning.[78–84] If similar (and perhaps more pronounced) changes are found in traumatized individuals with ASD, it would lend support to the mechanisms for a trauma–violence association in individuals with ASD proposed in this article.

Conclusion

In this article, I have attempted to explore the possible mechanisms for an association between trauma and violence in individuals with ASD. A review of the literature suggests that such mechanisms involve sensitized prefrontal-cortical-limbic networks that are overloaded in the face of trauma, or cognitive dysfunction in the realms of theory of mind, central coherence, and executive function that affects trauma processing in ways that portend violence.

More research is needed to confirm an association between trauma and violence risk in ASD, and if confirmed, to ascertain the validity of the above-proposed mechanisms for such an association. To facilitate further study, it would be helpful for clinical and forensic evaluators to obtain a careful, detailed trauma history when evaluating all individuals, including those with ASD.

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