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Multimodal neuroimaging in adverse childhood experiences and related PTSD

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Background: Adverse Childhood Experiences (ACE) are strongly associated with various mental health disorders, including Post-Traumatic Stress Disorder (PTSD). Although neuroimaging studies have investigated the neurobiological correlates of ACE and PTSD, there is no consensus on the specific neurobiological mechanisms underlying ACE and related PTSD. Many studies use single-modality imaging techniques, often examining structural and functional data separately, potentially overlooking crucial interactions and shared information across modalities. This study addresses this gap by using multimodal neuroimaging (MN) approaches to explore the neurobiological impact of ACE and related PTSD.
Methods: Two studies were conducted using complementary and joint multimodal neuroimaging analyses (CoMNA and JoMNA). The CoMNA study performed complementary analyses of T1-weighted MRI and diffusion MRI (dMRI) in a total of 78 adults to assess cortical morphometry and white matter integrity related to ACE. JoMNA study used a total of 119 participants with ACE (70 with ACE-related PTSD and 49 ACE-exposed controls). T1-weighted MRI, dMRI, and resting-state functional MRI (rs-fMRI) data were acquired, and joint connectivity matrix independent component analysis was employed to examine shared information across modalities and identify structural and functional connectivity differences between groups.
Results: The CoMNA study identified reduced cortical volume in the right superior parietal lobe (rSPL) associated with childhood abuse. Reduced rSPL volume also mediated the relationship between childhood abuse and PTSD avoidance symptoms, with corresponding changes in connected white matter integrity. The JoMNA study revealed functional hyperconnectivity in the medial prefrontal cortex and inferior temporal regions, hypoconnectivity in the lateral prefrontal cortex, and structural hypoconnectivity in white matter pathways, including the right orbitofrontal region, in the ACE-related PTSD group. These connectivity abnormalities were significantly correlated with the severity and number of ACEs and PTSD symptoms.
Conclusion: Both studies identified significant neurobiological alterations associated with ACE and PTSD, including structural and functional alterations in brain regions involved in cognitive control, self-referential processing, and social behavior. These findings underscore the potential of MN to elucidate the complex neurobiological basis of ACE-related psychopathology. Future, larger MN studies could contribute to developing predictive models to identify individuals at risk for psychopathology and enable personalized treatment approaches.