Psychiatric Fallout From Toxic Exposure

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An Update on Psychiatric Effects of Toxic Exposures
Psychiatric effects of environmental and chemical toxins were described in the medical literature as early as 1850. In the 21st century there has been, sadly, no shortage of victims of environmental disasters to assess and treat according to guidelines presented in this Special Report on environmental and chemical toxins, with an introduction by Chair James S. Brown, Jr, MD, MPH, MS.
Neurobiology and Clinical Manifestations of Methamphetamine Neurotoxicity

The short- and long-term behavioral and physiological effects of methamphetamine neurotoxicity are devastating. Illicit drugs such as “ecstasy” are often adulterated with methamphetamine. Solvent abuse that results in altered mental status and potentially lethal physical abnormalities remains a frequent presentation in the emergency setting. Occupational exposure to various solvents also results in neuropsychiatric injury, often referred to as “chronic solvent encephalopathy.” Contaminants including thallium or infectious agents such as anthrax can be consumed with illicit drugs. Methamphetamines are also considered an “emerging contaminant” of water, a term that describes the growing numbers of toxins that enter the human environment—especially surface water. For a mobile-friendly view of the Figure, click here.
Synergistic neurotoxicity of methamphetamine
A significant percentage of methamphetamine users are HIV-positive and are being treated with antiretroviral agents, which may increase methamphetamine concentration in the plasma. Betulinic acid and its derivatives might potentiate neurotoxicity in high-dose methamphetamine users. The Figure represents synergistic neurotoxicity of methamphetamine (METH) and betulinic acid (BA) to monoaminergic terminals in the rat striatum of male late adolescent rats. For a mobile-friendly view of the Figure, click here.
Significance for the practicing psychiatrist
Understanding the relationship between the molecular mechanisms underlying the neurotoxicity of methamphetamine and related clinical manifestations is imperative to provide more effective treatments. This article highlights neurobiology of methamphetamine toxicity, clinical manifestations of methamphetamine neurotoxicity, and implications for treatment of chronic methamphetamine users. For a mobile-friendly view of the Monarch notes, click here.
Chemicals that receive the most attention are those we are routinely exposed to in our daily lives. The sources of these exposures are as varied as the chemicals themselves: pesticides on produce, flame-retardant compounds on furniture, metals in drinking water, and various chemicals used to manufacture consumer products that have simplified our lives. Research on the impact of these chemicals has highlighted several neurological targets that are disturbed. These findings are further supported through population-based studies that have established some chemicals as significant risk factors for neurological deficits.
Significance for the practicing psychiatrist
The etiopathogenesis of neuropsychiatric disorders is complex. The application and integration of a multidimensional biomarker assessment can significantly improve diagnostic accuracy and assist in formulating a suitable therapeutic intervention strategy. Exposure to neurotoxic chemicals is a significant contributor to a variety of neuropsychiatric disorders. Such exposures and the subsequent pathogenesis that arises must be delineated from other exogenous or endogenous etiologies to effectively diagnose and treat the disorder. Application of a systems toxicology approach can leverage biomarkers of neurotoxicity and facilitate these processes. For a mobile-friendly view of the Monarch notes, click here.
The Influence of Diet on ADHD

Emerging evidence reveals that aspects of diet can indeed affect ADHD. For example, research has consistently shown that restriction/elimination diets may be effective in reducing ADHD symptoms. In addition, for some youths, a diet free of processed foods containing additives, particularly colorings and preservatives, may improve symptoms. Further, the best established evidence to date indicates that the severity of ADHD symptoms may be reduced by a combination of supplementation with omega-3 fatty acids combined with reducing or removing processed foods, especially those high in food colors and preservatives.
Environmental Toxicants and Autism Spectrum Disorder
A growing body of scientific literature associates symptoms of autism spectrum disorder (ASD) with environmental toxin exposure, including exposures from contaminants in herbal remedies, heavy metals (especially during fetal growth), polychlorinated biphenyls, bisphenol A, and organophosphate pesticides. The case vignette in this article describes ASD symptoms from the use of Ayurvedic herbs, which emphasizes the importance of asking patients about herbal use.
Understanding the Link Between Lead Toxicity and ADHD

A report on the current status of lead as a contributor to pediatric psychiatric problems with recommendations on how to counsel families anxious about lead exposure. The author points out that levels above 5 µg/dL require clinical interventions and that levels lower than 1 µg/dL have been associated with behavioral problems. The importance of this finding is reflected by the recent Flint, Michigan, drinking water crisis in which 99,000 residents were potentially exposed to lead-contaminated water between 2014 and 2015.
Significance for the practicing psychiatrist
When ADHD is present, a survey of possible lead exposure should be considered. Lead’s association with ADHD is well-established even at the historically reduced levels currently typical in the US population. Recent evidence supports a causal, not merely correlational, association even at supposedly safe levels of lead exposure. Clinicians can offer many practical steps to parents to mitigate potential harmful effects and risks of low-level lead exposure on brain development. For a mobile-friendly view of the Monarch notes, click here.