

A Translational Neuroscience Framework for Personalizing and Optimizing Preventive Intervention Models

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Numerous interventions have been tested and found “effective” in preventing onset of behavioral problems that antedate substance misuse. However, effects sizes are invariably small to modest due to substantial heterogeneity in outcomes; thus, measurable benefits do not accrue to a significant number of recipients. Very few prevention trials have employed highly sensitive and specific measures of neurodevelopmental processes (e.g., functional magnetic resonance imaging) that may explain differential program effects. Given substantial advances in understanding how the brain is impacted by environmental experiences and are related to child/adolescent development and later risk for substance use disorders (SUDs), prevention science is now primed to explore more explanatory underlying conditions that interfere with intervention effects.

Design of a new generation of effective interventions to address the conditions that increase liability for SUD requires a comprehensive and transdisciplinary understanding of its etiology and the translation of that knowledge to prevention science, practice and policy. This “neuro-prevention” framework employs cross-cutting designs that are integrative in terms of theoretical perspectives and empirical methods, permitting a greater understanding, for example, of how neurobiological mechanisms correlate and interact with environmental conditions to influence behavior. Accordingly, teams comprised of neuroscientists and intervention researchers afford better opportunities to parse this complex web of factors affecting those behaviors and more precisely identify underlying processes that may respond favorably to well-targeted interventions.

To maximize program effectiveness for a greater number of recipients prior to entrenchment of problems, it is important that we determine which personal characteristics, in interaction with prevailing conditions (e.g., economic instability, parental SUD, deviant peer networks), are most amenable to which program components. The etiological literature is instructive in that regard. For example, deficits in specific neurocognitive functions have been implicated in traits associated with risk for behavioral problems (e.g., disinhibition, heightened reward sensitivity, dysregulated emotion) and SUD vulnerability. Also, psychophysiological markers of these traits reflective of emotion regulation (measures of sympathetic and parasympathetic system reactivity), involved in the establishment of neurocognitive-emotion linkages during development, also have been related to behavioral problems. These developmentally relevant neurocognitive and physiological mechanisms are highly sensitive to environmental exposures and experiences; factors commonly identified in traditional prevention trials as influential, such as economic instability, deviant peer networks, caregiver mental illness, discrimination, and family dysfunction. Determining program components on the basis of a wider complement of interactive neurobiological and social-environmental factors implicated in behavioral problems is likely to significantly increase effect sizes.

Ultimately, an understanding of how the interrelationship between brain and environment can impact critical points in the developmental trajectory to alter risk status has potential to: (a) increase accuracy of our predictive analytics; (b) guide refinement or design of targeted interventions; (c) determine when adjunctive or preparatory treatments are needed prior to participation in preventive intervention; and (d) identify social conditions that undermine healthy development to guide policy reforms. In sum, a neuro-prevention framework applies neuroscience technologies and findings to enhance understanding of heterogeneity in program responses as a foundation to further inform preventive intervention models.

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