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Introduction and Rationale for Individualized Substance Abuse Prevention from an Ontogenetic Perspective

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Abstract

Background—Progress in substance abuse prevention science could be accelerated by more closely aligning studies, methodologies, and intervention program testing with the ontogenetic orientations of its underlying theories and etiology research. This article introduces the Ontogenetic Prevention approach, describes some aspects of what this orientation offers for substance abuse prevention, and provides an overview of this full special issue.

Methods—Review of the substance abuse prevention literature illustrates specific aspects of Ontogenetic Prevention.

Conclusions—The Ontogenetic Prevention perspective and each of the manuscripts in this special issue provide channels whereby substance abuse prevention can evolve from a science that relies largely on universal intervention to diversification for meeting the needs of individuals under specific developmental circumstances.

Keywords

Development; etiology; ontogeny; prevention; substance abuse; theory

INTRODUCTION

This special issue describes and promotes the ontogenetic perspective of prevention, which emphasizes orienting prevention to meet the needs of individuals. It is widely agreed that individuals vary considerably in substance abuse (SA) risk factors as well as salencies and trajectories of such factors (1,2). Yet, these risk factor variances are rarely considered in the implementation or testing of prevention programs, especially in universal contexts.

Ontogenetic Prevention (OP) emphasizes capitalizing on interpersonal and ontogenetic heterogeneity to improve intervention impact (or outcomes for individuals), does not assume an intervention can generate positive outcomes in all persons, and is consistent with adaptive intervention, which targets prevention efforts for those who need them while adjusting dosage and program components accordingly (3). In “Neurocognition as a Moderator and Mediator in Adolescent Substance Misuse Prevention,” Riggs and Greenberg (4) illustrate this point,

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Declaration of Interest

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reviewing how executive cognitive functioning may be a risk factor, intervention mediator, and/or outcome moderator of prevention. In fact, OP assumes any intervention may generate iatrogenic outcomes in some persons; evidence about *who* benefits, is not impacted, or experiences iatrogenic effects from a program is more instrumental than overall efficacy.

Although aspects of OP resemble the public health notion of selective or indicated prevention, greater exigency is placed on grounding in etiology and developmental research, prudent alignment of intervention with recipient needs, and detailed understanding of the range of impacts an intervention has on various types of recipients. The value of identifying null and iatrogenic outcomes and how such discoveries advance prevention as well as etiology is illustrated by Dishion et al.'s (5) report of iatrogenic outcomes of a group intervention for boys with problem behaviors. The intervention resulted in deviancy training and increased problem behavior among recipients; their report was cited over 600 times as of October 2008 and replicated by other researchers (6).

WHY IS THE ONTOGENETIC PARADIGM NEEDED?

SA is important to prevent partly because of the frequently consequent illnesses associated with it (7). SA and its secondary outcomes impair functioning of individuals and societies by way of criminal and psychiatric antisocial behavior, other psychiatric disorders, STDs, and risk of harm to persons and victims (8–10). In spite of the dangers of SA, certain forms of substance use are normative, can be health enhancing (11), and a large proportion of persons with SA recover without professional intervention (12). So, whereas potentially great benefits could result from SA prevention, complexities of SA development and outcomes require caution to avoid unintended outcomes (akin to the Hippocratic oath).

A common goal among prevention programs is altering one to a few risk factors to bias development away from SA. However, evidence supports the widely-accepted ecological, multifactorial, and liability-threshold theories that manifold factors contribute to SA ontogeny (13,14). Identification of the appropriate risk factors to target for each intervention has been among the greatest challenges of SA prevention research. Accordingly, prevention programs that are implemented as universal interventions may be expected to generate small efficacies because in all persons they attempt to alter the same one to few factors using the same “dosage” of an often manualized program. In fact, null to small efficacies have consistently resulted from over two decades of clinical trials on dozens of universal interventions, with few exceptions (15). One upshot of the incongruity between how most interventions are (a) conceived (targeting few risk factors) vs. (b) implemented and researched (as universal programs) may be an underestimation of the impact they could have upon the persons most likely to benefit, namely, those who experience the targeted risk factor(s). A lack of research to disassemble universal interventions and identify specific ingredients of change has also hindered progress in this area.

HOW ONTOGENETIC AND UNIVERSAL PREVENTION ALIGN

OP is not incongruous with universal prevention but rather is a paradigm that may improve the impact of prevention programs by way of tailoring them (which should improve efficacy and effectiveness). One of the more efficacious universal SA prevention programs, the *Good Behavior Game*, is designed to reduce SA risk specifically in disruptive students in poorly managed classes using mechanisms of behavior change identified through decades of experimental research (e.g., reinforcement, peer pressure) (16). The expectation of non-iatrogenic or beneficial impacts for the other students also is grounded in such research.

Three OP-oriented programs are described in this issue. In “A Person-Centered Approach to Individualizing a School-Based Universal Preventive Intervention,” Caldwell, Bradley, and

Coffman discuss *TimeWise*, a unique SA preventive intervention designed to help youths to intentionally use their free time in enjoyable, beneficial, and constructive activities rather than substance use; some sessions are tailored for individual needs (e.g., overcoming one's barriers to leisure activities) (17). Examining the impact of parenting practices in reducing SA risk, "Parenting Practices as Mediators of Treatment Effects in an Early-Intervention Trial of Multidimensional Family Therapy" by Henderson et al. explores the use of *Multidimensional Family Therapy* in treating at-risk youth and their families. In their study, the program is demonstrated to improve parental monitoring as a specific mechanism toward reducing SA risk (18). Grounded in development theory, "A School-Based, Family-Centered Intervention to Prevent Substance Use: The Family Check-Up" by Stormshak and Dishion reviews the capabilities of this specialized modality that utilizes screening, needs assessments, collaboration with recipients, and complete tailoring of intervention for at-risk youth (19).

WAYS ONTOGENETIC PREVENTION CAN CONTRIBUTE TO PREVENTION SCIENCE

Certain hindrances to progress in prevention science stem from wide reliance on universal testing of prevention programs. The ubiquitous null to small efficacies of programs has led to a potentially incorrect presumption (perhaps even expectation) that outcomes of any SA prevention effort will be small. Methodologically, huge samples are used to boost statistical power to detect often inconsequential effects, which restrains progress by requiring substantial resources and effort for each study, which in turn limits the ability to troubleshoot, refine, replicate, and improve programs. How this restrains progress in prevention is illustrated by the fact that it remains unknown whether universal prevention outcomes reflect small impacts on many persons or large impacts on few persons, partly because such programs are not tested in indicated groups.

The universal approach also has engendered competition between programs vying for universal implementation rather than a collective determination of the types of persons who could benefit most from each program. Reliance on large sample designs also precludes explication of the ontogenetic processes within individuals that lead to prevention outcomes, such as what might be elucidated using idiographic designs (20). Finally, and potentially the most stifling hindrance to dissemination of prevention programs, is skepticism that the field of prevention cannot sizably reduce SA risk, especially in persons most at risk for SA.

Three means whereby OP can utilize individual differences to improve the impact of specific programs on individuals are: grounding prevention programs in etiology (1,2,4,5,16–19); intervention decisions guided by screening and needs assessments; and small-sample, practical randomized clinical trials. Two innovative assessments are investigated in this special issue. Arria, Vincent, and Caldeira (21) in "Measuring Liability for Substance Use Disorder among College Students: Implications for Screening and Early Intervention," study the potential and limitations of the *Transmissible Liability Index* (a measure of individual's transmissible risk for SA) as a screener for college freshmen. Next, test-retest reliabilities and latent factor structures are reported in Ridenour, Clark, and Cottler's "The Illustration-based Assessment of Liability and EXposure to Substance use and Antisocial behavior® for Children," a child report instrument that measures many SA risk factors, does not require reading, appears to enhance children's attention to the task, and is enjoyable (22).

Use of a range of methodologies is needed to explicate ontogenetic factors related to the impacts of a prevention program. For example, Kraemer et al. (23) demonstrated how different traditional statistical techniques erroneously detect interaction effects that are actually type I errors under different conditions. Specifically, methods are needed to elucidate idiographic processes that facilitate prevention outcomes in applied settings (7,20,23).

Two techniques are highlighted in this issue for small sample clinical trials in applied settings. Connell, in “Employing Complier Average Causal Effect Analytic Methods to Examine Effects of Randomized Encouragement Trials,” demonstrates how average causal effect analysis can refine efficacy estimates of randomized clinical trials by accounting for level of study participant compliance with an intervention (24). The final article of this issue, “A Small Sample Randomized Clinical Trial Methodology Using N-of-1 Designs and Mixed Model Analysis”, by Ridenour, Hall, and Bost merges mixed model techniques with N-of-1 (case study) experimental designs for statistically powerful randomized clinical trials that can use sample sizes several times smaller than traditional universal SA prevention studies (7). Supporting points that were raised earlier, this technique is demonstrated using re-analyses of an existing prevention dataset to clarify differential impacts experienced by distinct types of persons.

It is hoped that this special issue can increase momentum toward diversity and sophistication in idiographic SA prevention research. OP offers the prospect of greater beneficial impacts for individuals, clearer elucidation of prevention mechanisms, amalgamation of (rather than competition between) programs, and ultimately, more effective reduction in SA risk. This issue provides examples and methods toward accomplishing these goals of OP.

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