Explorations of Parenting Environments in the Evolution of Psychiatric Problems in Children

Understanding the genetic basis for behavior has fascinated the field of psychiatry for the past two decades. However, in this decade, it is again becoming apparent that understanding salient social environments, particularly early adverse caregiver/offspring relationships, is also necessary to the understanding of the etiology of psychiatric symptoms. Biological parents with psychiatric disorders who are rearing their own children may heighten risk for their children both by transmitting genes and by their role in shaping an adverse rearing environment. This issue of the *Journal* contains four articles on parental risk factors—in particular, parental depression and/or antisocial behaviors, including abuse—and their consequences for the offspring.

"Parents with mental disorders...want to do better for their children."

Teicher and colleagues, in this issue, report that young adults who endorsed exposure to both domestic violence and parental verbal aggression (e.g., blaming, yelling, belittling) as children but did not experience physical or sexual abuse were more likely to report dissociative, anxiety, and depressive symptoms than those reporting physical or sexual abuse alone or in combination. One limitation of this study is that it employs retrospective self-report only. Although retrospective reports of past psychosocial variables have their place, they do not correlate well with evidence obtained at the time of exposure (1) and may not accurately reflect the role of maltreatment in symptom development. Suggestive as these findings are, they need replication in a longitudinal prospective research study in which careful measurement of parent/child relationships are conducted throughout childhood. Several already-collected samples are available for such analyses (2).

Also in this issue, Weissman and colleagues followed a cohort of offspring of parents with moderate to severe depression and compared them to offspring of parents with no illness for 20 years. The parental sample was well-characterized, although the parent/child relationships were not. Children with at least one depressed parent had higher rates of mood and anxiety disorders, substance dependence, and self-reported physical illness in mid-adulthood than children of non-ill parents. Treatment rates for the offspring of depressed parents were dismal; only 38% of this high-risk cohort had any form of treatment, whether psychological or pharmacological. These findings add to recent data from the National Comorbidity Survey Replication concerning the high rates of long delays and failures among individuals with serious mental disorders to seek or receive treatment (3). The study by Weissman and colleagues was not designed to distinguish between the genetic and relationship risk factors in this population, but it does highlight one method of identifying a group of children at high risk for both psychiatric and medical problems.

In contrast to other studies, Young and colleagues, in this issue, included genetic data in their research in an effort to identify the relative role of a specific allele as well as the social quality of the rearing environment. They sought to determine whether a putative high-risk allele of the monoamine oxidase (MAO-A) gene interacted with childhood maltreatment to increase rates of conduct disorder symptoms in teens. An interaction of this allele with an adverse rearing environment was first reported in a community sample examined prospectively from childhood to adulthood (4). In their effort at replication, Young and colleagues examined a clinical sample of adolescents currently in an intensive treatment setting for youth with conduct disorder and substance abuse. They were genotyped for the allele of the MAO-A gene, and parental maltreatment was assessed

with an in-depth semistructured clinical interview. The study found a clear main effect of the various maltreatments on conduct disorder symptoms but no effect of the allele and no gene-environment interaction. The impact of the maltreatment environment seems clear, but the effect of the high-risk MAO-A allele, alone or in interaction with an adverse environment, is either small or uncertain. Thus, on a community level, it would not yet make sense to use this particular allele as part of screening for high-risk children.

However, several studies have estimated the effects of the entire genome on child and adolescent psychopathology using twin and adoption designs. These studies have provided clear evidence of an interaction between genetic factors and adverse rearing environments (5). In this issue of the *Journal*, Kim-Cohen and colleagues discuss a particularly strong twin design using a prospective community sample of twins. This design was enriched for high-risk families, and assessment was performed with self-report and multi-informant observational measures. More than 1,000 families participated in a home-visit assessment when the twins were 5 years of age and again 18–24 months later. The group was divided into four subsets: 1) mothers with major depression during their child's lifetime (15%), 2) mothers with one or more antisocial symptoms but no depression (20%), 3) mothers with both major depression and antisocial symptoms (14%, comorbid group), and 4) the remaining 51% with neither major depression nor antisocial symptoms. The study found that children of mothers in the comorbid group were more likely to live under financial hardship, be exposed to domestic violence, and have a mother with drinking problems and elevated risk for suicide. These children also received the lowest levels of maternal warmth and highest levels of maternal hostility and were at highest risk for physical maltreatment. Not surprisingly, 20% of these children met criteria for conduct disorder at age 7. However, they showed similar levels of internalizing symptoms as children of depressed-only mothers. In addition, it appears that the increased environmental and relationship difficulties primarily led to externalizing symptoms at this young age and that children of the mothers in the comorbid group are at extremely high risk for early-onset psychiatric difficulties.

The results of the studies mentioned in this editorial support a similar yet unsurprising theme: both genetic risk factors (i.e., parental depression and/or antisocial behaviors) and relationship risk factors (i.e., verbal abuse, witnessing of violence, physical or sexual maltreatment) play a role in the development of conduct disorder symptoms in younger children, dissociative and mood symptoms in young adults, and overt mood and anxiety disorders, as well as possible increased risk of physical disorders, by mid-adulthood. There is opportunity for psychiatrists to initiate preventive interventions for children identified with these genetic and relationship risk factors. For children of depressed parents, there are several evidence-based approaches to prevention of depression that are effective in reducing onset of depression during adolescence (6, 7). However, it is disconcerting that there are so little data on whether successful treatment of a parents' mental disorder improves the prospects for their children.

Cumulatively, the studies discussed in this editorial make one powerful and urgent point: clinicians treating parents with mental disorders must also consider the development of their children. In our clinical experience, we find that parents with mental disorders, including those with severe difficulties, want to do better for their children. This desire can motivate them to participate earnestly in treatment programs aimed at protecting the health of their children. A study conducted by Beardslee and colleagues (6) indicates that even small changes in parents' disclosure of their illness and interaction with their families may have a notable impact on their children. However, for many parents, their illness and its consequences may preclude major changes in their parenting and relationships with their family. Even in clinical situations, it appears that including the children in a discussion about their parents' illness is helpful. This discussion often involves acknowledging and learning about that illness and suggesting constructive ways for the child to cope with its consequences. Beardslee and colleagues suggest that

preventive interventions of this kind can be effectively administered by individual practitioners, not necessarily child psychiatrists, although specific field tests to demonstrate this point have not been completed. Child psychiatrists could focus on developing systems of care in which formal prevention services for children are available.

As psychiatrists, we might learn from the harm reduction strategies currently used in smoking cessation assessment with parents (i.e., if parents cannot stop smoking, how can they minimize exposure of their children to secondhand smoke?). Future research could explore what harm reduction may mean in the context of psychiatric disorders. Keeping the pathology out of key family rituals and routines is a powerful family-systems intervention that, perhaps, should be investigated. Practitioners can help parents maintain at least some positive family rituals without the influence of the symptomatic behavior. Data collected some years ago suggest, quite specifically, the value of maintaining rituals when a parent has severe alcohol problems (8). While these interventions may seem simplistic, they are practical on a large public health scale and may decrease or delay the onset of psychiatric illness in youth, for whom every year of development without illness allows time to develop ego skills to deal with their inherited risk factors.

The following vignette describes a ritual for harm reduction.

> "Ms. A" was a bright single mother who had dropped out of medical school because of the development of severe bipolar disorder. She had four children from three different fathers and first came to treatment when the third child, 5 years old, was suspended from kindergarten for aggressive and out-of-control behavior. In meeting her, it was apparent that her barely managed mania kept the household in chaos. In addition to working with her on a better pharmacologic strategy for her own illness, the authors also suggested that she obtain a kitchen table so that she could start having family dinners with everyone sitting at a table rather than haphazardly eating throughout the apartment. Over the course of the next months, the authors focused on how the family could talk at the table without people leaving their seats and without yelling or bickering. The authors coached Ms. A to use this time to connect with each of her children about their activities and schoolwork and to maintain positive communication for the 20- to 30-minute period of dinner, even if she later could not control her own activity level and frustration.

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