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## Depression in At-Risk Adolescents and Their Parents

**To the Editor:** In their randomized controlled trial, Dr Garber and colleagues<sup>1</sup> provided important information on the successful prevention of adolescent-onset depression using an evidence-based psychotherapy in high-risk adolescents. In their study, if a parent was currently depressed the adolescent did not benefit from treatment. This observation is consistent with other data suggesting that if a currently depressed mother is successfully treated, her children's symptoms are more likely to improve.

An ancillary study of children of parents receiving treatment as part of the Sequenced Treatment Alternatives to Relieve Depression (STAR\*D) found that remission of maternal depression after 3 months of medication was significantly associated with reductions in the children's diagnoses and symptoms.<sup>2</sup> Conversely, a failure to remit was associated with worsening in child outcomes, paralleling observations by Garber et al that cognitive behavioral therapy did not confer a protective effect among the offspring of currently depressed parents. The STAR\*D findings were sustained in offspring of mothers who remitted later after the first 3 months of treatment<sup>3</sup> and were supported by consistent findings in an independent randomized controlled trial covering 9 months and using another evidence-based psychotherapy for the depressed mothers.<sup>4</sup>

The combined findings of these studies suggest that a range of efficacious treatments can be recommended to prevent adolescent-onset major depression, targeting both high-risk adolescents and currently depressed parents.

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**In Reply:** Drs Weissman and Talati present converging evidence that treating parental depression may be important for children's adjustment and recommended that interventions should target currently depressed parents and their high-risk offspring. Several empirical findings support this

perspective. First, offspring of depressed parents are at increased risk for depression and other psychiatric disorders. Second, interventions aimed at treating currently depressed children<sup>1,2</sup> and preventing depression in at-risk youth (in our study) have been found to be less effective when a parent is currently depressed. Third, treatment of depression in parents of children with current psychopathology resulted in lower levels of children's self-reported depressive symptoms.<sup>3</sup> Fourth, a significant association has been found between remission in mothers' depression and decreases in their children's symptoms and disorders.<sup>4</sup> Thus, it is fair to conclude that reducing depression in parents likely will yield more positive outcomes in their children.

Important questions remain, however, particularly regarding mechanisms. Parental depression may be a marker of a variety of non-mutually exclusive processes underlying the intergenerational transmission of depression, including genetic vulnerability, neurobiological dysregulation, neuroticism, a chronic and severe course, exposure to stressful life events, and dysfunctional parenting behaviors. It is not clear which of these factors accounts for the poor outcomes found in some offspring of depressed parents and which factors change with remission of parents' depression. The study by Weissman et al<sup>4</sup> acknowledged that the observed association between maternal remission and child improvement could have been due to some shared third variable (eg, reduced family stress, less genetic vulnerability) or to reverse causation (changes in children's symptoms leading to reductions in maternal depressive symptoms).

Experimental studies allow for a more direct test of the effects on children of treating their parents' depression. Swartz et al<sup>3</sup> found that treatment of depressed mothers resulted in lower levels of depressive symptoms in both the mothers and children, and the authors speculated that changes in maternal depression mediated the effect of treatment condition on children's depressive symptoms over time. Further research is needed that explicitly tests this mediation hypothesis.

Moreover, studies need to examine whether reducing depression in parents is sufficient to improve their children's functioning or if it is necessary to more directly target the hypothesized underlying mechanisms (eg, parenting behaviors, coping with stress). Thus, the current state of knowledge indicates that programs aimed at reducing or preventing depression in youth need to attend to the extent of depression in the parents. Whether treatment of parental depression alone is necessary or sufficient, or needs to be augmented with programs that specifically target putative mechanisms, remains an important question for future investigation.

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### Substance Abuse and Violent Crime in Patients With Schizophrenia

**To the Editor:** The study by Dr Fazel and colleagues<sup>1</sup> linked treatment for schizophrenia, conviction for violent offenses, and a history of substance misuse. The authors found that 27.6% of a sample of patients with schizophrenia and a coexisting substance use disorder committed a violent offense, compared with 8.5% of a control group of patients with schizophrenia without a substance use disorder and 5.1% of a general population control group. They concluded that schizophrenia alone has a modest association with violent offending on the basis of the small difference in the rate of conviction for violent offenses between the population control group and the non-substance-using schizophrenia group.

We are concerned that methods used in this study have 2 important limitations that might have led to a significant underestimation of the risk of violent crimes committed by people with schizophrenia. First, the population control group included people with substance use disorders. Given an association between substance use and violent crime,<sup>2</sup> it is probable that a population control group including people with substance use disorders would have a substantially higher rate of violent offenses than a non-substance-using group. Therefore, the comparison between this group and the non-substance-using schizophrenia group would underestimate the difference in the rates of violent crime.

Second, the study excluded patients with schizophrenia who committed a violent offense during their first episode of psychotic illness and were treated for the first time in prison. In a meta-analysis, we estimated that 38.5% of homicides during psychotic illness were committed by patients who had not received prior treatment.<sup>3</sup> It is therefore likely that this exclusion led to a significant underestimate of rates of violent crime in both substance-using and non-substance-using schizophrenia groups.

Fazel et al<sup>1</sup> concluded that their findings should reduce stigma about the relation between schizophrenia and violence. We are concerned that a diminution of their important findings by underestimation of the true violence risk in schizophrenia might actually increase stigma by drawing attention away from the need to develop policies to prevent and treat schizophrenia, particularly combined with substance misuse.

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**In Reply:** Dr Smith and colleagues raise concerns that our longitudinal analyses may have underestimated the risk of violent crime in schizophrenia. Specifically, they suggest that the rate of violent crime in patients without comorbid substance abuse should be compared with individuals in the general population who are not substance abusers. We believe that this is a different question. Because substance abuse is highly prevalent<sup>1</sup> and possibly treatable in schizophrenia,<sup>2</sup> our approach has potentially important implications for psychiatric services and public health.

Nevertheless, when analyzing the data as suggested by Smith et al, the adjusted odds ratio is 1.6 (95% confidence interval, 1.4-1.8) for patients with schizophrenia who are not substance abusers compared with population controls who are not substance abusers. However, the sensitivity of register-based data on substance abuse diagnoses in the general population is likely to be worse than for schizophrenia since inpatient admissions (which are very common among individuals with schizophrenia) allow for the assessment of substance abuse. Despite this caveat, the new analysis does not change our interpretation. We note that the MacArthur Violence Risk Assessment Study (which included 160 patients with schizophrenia), another of the very few longitudinal studies in this field, found comparable risk estimates for schizophrenia and other serious mental disorders among persons who were not substance abusers.<sup>3,4</sup>

Smith et al also raise the possibility that using 2 separate discharge diagnoses as a case criterion may have underes-