Transmission and Treatment of Depression

Three articles in this issue make a substantial contribution to our understanding both of how depression is transmitted from parents to children and of approaches to treating depression. Tully et al. (1) present one of a series of reports from an etiological study: the Sibling Interaction and Behavior Study (SIBS) is a finely crafted adoption study designed to weigh environmental versus genetic theories on the transmission of psychopathology from parents to children. The results provide important support for environmental theories of transmission of mothers', but not fathers', psychopathology to children. These results have immediate treatment implications. Two treatment studies are a fortunate complement to the Tully et al. etiological study: a report from the interpersonal psychotherapy for depressed mothers treatment project by Swartz et al. (2) and a report from the STAR*D-Child project by Pilowsky et al. (3). Results of the STAR*D study, a nonrandomized effectiveness study, suggest that if a mother's depression is successfully treated, her children will also show clinical improvement. Results of the study of interpersonal psychotherapy for depressed mothers adds additional heft: mothers were randomized to a treatment and a control condition, and the children of the treated mothers showed clinical improvement. Both studies reinforce the SIBS findings: environmental mechanisms are important in the relationship between maternal depression and child psychopathology.

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We have accepted for some time that genetic factors account, in part, for the transmission of major depressive disorders from parents to children. Although authors of recent studies have guessed that genetic factors account for about 40% of the variance in adult depression (4), the role of genes in accounting for parent-to-child transmission is probably less. Genes that influence depressive symptoms in childhood, at least in girls, are different from those that influence the same symptoms in midadolescence and, presumably, later in development (5). Clinical researchers have also proposed environmental mechanisms for transmission. The most crucial data favoring this hypothesis are from studies showing that parental depression decreases the warmth and increases the negativity of parents' relationships with their children, and these parenting difficulties have then been linked to psychopathology in their children (e.g., reference 6). However, recent data from genetically informed studies raise questions about the interpretation of these findings: genetic factors in the child evoke parental warmth and negativity, and to a large extent, these same genetic factors increase the child's liability to depression (7). Thus, the association between parental depression, parenting qualities, and children's depressive disorders could be accounted for by genes common to all three. To complicate matters further, a third route of transmission is strongly suggested by human and animal studies: fetal exposure. One proposed mechanism is that the fetus of a depressed or anxious mother is exposed to high levels of maternal cortisol, which then "programs" the fetus's hypothalamic-pituitary-adrenal axis. Data suggest, for example, that adolescents whose mothers were anxious during pregnancy have an increased liability to depression, a liability that is mediated by the adolescents' abnormal cortisol patterns: elevated cortisol levels without normal diurnal variation (e.g., reference 8).

Thus, given a growing number of plausible explanations for mother-child transmission of depression, we need research designs to weigh these alternatives, as illustrated by the three articles in this issue. The adoption design examines the effect of environ-

mental mechanisms by controlling the effects of genetics and prenatal exposure, which cannot account for similarities between adoptive mothers and their children. The randomized controlled clinical trial can be considered another powerful tool because it functions as a true experiment. The study of interpersonal psychotherapy for depressed mothers controls the "environmental exposure" of the child by reducing depressive symptoms in the experimental group but not in the control group. The STAR*D study, although not a true experiment, used statistical analysis to suggest that reductions in mothers' symptoms preceded changes in their children, a finding that supports the experimental evidence from the study of interpersonal psychotherapy for depressed mothers. Thus, these studies add up. The treatment researchers arrived on the scene, necessarily, after many children of depressed mothers had become ill themselves. The studies thus provide powerful evidence of the role of maternal depression in the *maintenance* of child psychopathology by environmental mechanisms. The adoption study provides strong and novel evidence of a substantial role of maternal depression in *initiating* child depression and other psychopathology, again by environmental mechanisms. Future studies will now have to take a closer look at specific environmental mechanisms that account for the findings from these studies. Likely mediators are the effects of maternal depression on mothers' parenting and marital difficulties, the mother's role in shaping her children's ties to peers and school, and the effects on children of the stigma and misunderstanding that are still associated with severe psychiatric illness.

All three research projects, however, share a limitation. Each leaves open the question of the child's effects on parents: Can a child's psychopathology evoke psychopathology in a parent or affect a parent's response to treatment? There is ample evidence that heritable child characteristics have a major impact on the behavior of their parents toward them and even on how much their parents argue about them (7), and one adoption study suggests that these effects might extend to influencing parents' psychopathology (9). In addition, recent longitudinal studies suggest, for example, that a boy's antisocial behavior may have sizable impact on his mother's subsequent depressive symptoms, particularly at the boy's transition to first grade and, later in development, during his transition into adolescence (10). Moreover, recent preliminary evidence suggests that successful treatment of the child may also reduce parental depressive symptoms (11). All these studies should lead to some caution about one-way effects of parental symptoms on their children, as suggested by the three studies here.

The clinical reader can draw four lessons from these studies. First, they provide strong evidence that depression is a family matter. The division of our field into adult and child psychiatry often works against best treatment practices. Clinicians treating depressed adult parents should routinely consider the effects of parental depression on their patients' children. Interpersonal psychotherapy for depressed mothers is an important advance and might, after further research, become a standard part of mental health training programs focusing on adults. Clinicians must extend their focus on family to marriage. For example, the STAR*D-Child team has reported evidence suggesting that the benefits a child receives from pharmacological treatment of their mothers was greater in two-parent households than in single-mother households (12). This extends the literature on the role of marital problems in reducing favorable responses to both psychotherapy and pharmacotherapy of depression. Likewise, clinicians caring for children need to take the family perspective—as a large number already do—by extending treatment to parental psychopathology and marital difficulties when needed.

Second, within this family perspective, the clinician has exceptional opportunities not only for more precise clinical assessment and improved treatment but also for prevention. These studies, and many others, clarify the fact that maternal depression is a potent but malleable risk factor for child psychopathology, and there is reason to be-

lieve that early detection of depression in mothers, along with short-term support for their children, may prevent the development of disorders before they begin (13).

Third, clinical trials of antidepressants or of psychotherapy with patients who are parents should include, when possible, assessments of both the marriage and the children. It is astonishing that our field has gone for almost half a century, after the first randomized controlled trials of the pharmacotherapy of major depression, without inquiring about the effects of these treatments on the children. We are still waiting. (As noted, the STAR*D effectiveness study did not randomize its subjects.)

Fourth, these studies encourage us to teach ourselves and our trainees about psychopathology from a developmental and family perspective. Many will want to be part of the unfolding story of the many family routes to depression: through genetic influences, through fetal exposure, and through the multiple social impacts of parental depression. The fundamental excitement of this research, both for understanding and for comprehensive prevention and treatment, lies in the interplay among these factors as children grow into adulthood.

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DAVID REISS, M.D.

Address correspondence and reprint requests to Dr. Reiss, Yale Child Study Center, PO Box 207900, New Haven, CT 06520-7900; dxreiss@earthlink.net (e-mail). *Editorial accepted for publication June 2008 (doi: 10.1176/appi.ajp.2008.08060916).*

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