Family processes in the development of youth depression: Translating the evidence to treatment

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ABSTRACT

There is strong evidence that family factors play a role in the development, maintenance and course of youth depression. However, to date few clinical trials of psychotherapy for youth depression employ family therapy interventions or target the known family risk factors. This is surprising given recent meta-analytic findings showing only modest effect sizes for psychotherapy for youth depression, and that cognitive therapies do not outperform non-cognitive therapies. The aim of this review is to 1) use a developmental systems approach to review empirical evidence on family risk factors for youth depression to identify potential targets for treatment, 2) examine the extent to which these family risk factors have been targeted in clinical trials for youth depression, and 3) provide a road map for the development of empirically validated family-based interventions for youth depression.

Strong evidence was found supporting a relationship between family factors at multiple system levels and depressive symptoms or disorders. Support for several different hypothesized causal mechanisms as well as bidirectional effects was found. A comparison of the identified risk factors and psychotherapy trials for youth depression indicated that few RCT’s target family factors; among those that do, only a few of the family risk factors are targeted. Recommendations for translation of empirical knowledge of family risk factors and mechanisms to develop empirically valid family-based interventions to enhance existing treatments for youth depression are provided.

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1. Introduction

An extensive body of evidence supports the role of family processes in the development, course and maintenance of depression in children and adolescents (Sander & McCarty, 2005). Family environment, marital and family relationships (Cummings, Keller, & Davies, 2005), parenting behavior (Alloy, Abramson, Smith, Gibb, & Neeren, 2006) and attachment (Sexson, Glanville, & Kaslow, 2001) have been related to the development and maintenance of childhood and adolescent depressive symptoms and disorders, as well as treatment response and relapse among depressed adolescents (Birmaher et al., 2000). This evidence comes from diverse research traditions and populations, employing diverse methodologies, and examining varying aspects of family relationships (Goodman & Gotlib, 1999; Sander & McCarty, 2005; Sheeber, Hops, & Davis, 2001).

Surprisingly, the direct translation of these research findings into specific techniques for treatment of depressive disorders has been limited (Davies & Cummings, 2006). Few treatment studies of youth depression include family-based interventions; those that do target only a few of the specific family risk factors identified in the research literature (Sander & McCarty, 2005; Weisz, McCarty, & Valeri, 2006).

Furthermore, the evidence for efficacy of current treatments for youth depression may not be as strong as it once appeared (Weersing & Brent, 2006). In a recent meta-analysis of treatments for youth depression, Weisz et al. (2006) reported a modest overall effect size for current treatments for youth depression, which was considerably lower than effect sizes found for treatments of other child and adolescent disorders. Cognitively based treatments, which make up the majority of empirically tested treatments, were not significantly better than non-cognitively based treatments. Taken together, these findings suggest not only that there is considerable room for improvement in the treatment of youth depression, but also the possibility that treatment effects could be increased by also targeting non-cognitive risk factors. Family factors would seem to be an obvious candidate, given the strong evidence for family factors in depression. Despite this, few treatment studies of youth depression have included family interventions which target the wide variety of family risk factors for youth depression (Sander & McCarty, 2005; Weisz et al., 2006).

This is in contrast to clinical trials research on externalizing disorders, substance abuse and eating disorders, in which family interventions have been demonstrated (Diamond & Josephson, 2005).

The aim of this review is to address the gap in the translation of research findings on family processes in depression and treatment of youth depression. The first goal is to examine the empirical evidence on family risk factors and mechanisms in the development of youth depression, in order to identify potential targets for treatment. The second goal is to examine the extent to which these family risk factors have been targeted in randomized controlled psychotherapy trials for youth depression. The third goal is to provide a road map for the development of family-based treatments for youth depression to target the broad array of family risk factors identified in the empirical literature.

Several considerations guide our approach. First, there is a need to move beyond identifying separate risk factors, to developing models of causal risk mechanisms (Garber, 2006). Research on family and parental factors related to depression have tended to view parental psychopathology, rearing styles, marital functioning and family structure as separate domains, and often research is conducted on only one of these variables at a time. Researchers are increasingly using developmental and family systems theory to integrate diverse findings into models, which can describe the complex interrelationships between risk factors and specify possible causal pathways from risk to outcome (Cowan & Cowan, 2002; Davies & Cummings, 2006; Goodman & Gotlib, 1999; Goodman & Gotlib, 1999; Sheeber et al., 2001). While developmental and interpersonal models have been available for decades in the developmental literature (Cicchetti & Toth, 1998), the adult literature on depression (Gotlib, 1990), and within family therapy research (Steinglass, 1987) these models have less frequently been applied to the treatment of child and adolescent depression, where cognitive models have been predominant.

1.1. A systems approach to the evidence

While the terms “family systems” or “systemic” have been used in various ways in the literature, in this review, “systemic” refers to an approach which views the individual in the context of his environment. Individual characteristics such as genes, temperament and personality are assumed to unfold within, and in interaction with, a particular environment. Environmental influences can be conceived as operating at varying degrees of proximity to the child, from the immediate family (parents and siblings), to extended family, to the broader social context outside the family (e.g., school, work, social-cultural factors) (Bronfenbrenner, 1986). It is important to note that this approach makes no further assumptions about etiology or treatment of depression. Rather, the only assumption is that examining multiple factors at various systemic levels will give a more complete...
picture of the mechanisms involved in the development of youth depression. Within this approach, the term subsystem refers to any part of a larger system: for example, the individual child subsystem refers to the child, the marital subsystem refers to the spousal relationship, the whole family subsystem refers to the mother, father and child(ren) unit.

1.2. Why use a systemic model for youth depression?

Systemic models allow for the integration of multiple types of data and theories by viewing each as occurring at different levels of analysis: individual, family, community and culture (Bronfenbrenner, 1986). The literature on family factors in depression has drawn from several populations (depressed parents, depressed youth, and community samples), and includes diverse factors such as parental rearing style, family environment, marital conflict, attachment, support and conflict. Rather than viewing these as separate factors, organizing these factors according to the different system levels facilitates model building and clinical application. Moreover, this approach lends itself to developing hypotheses about how individual risk factors interact to produce risk mechanisms, and is consistent with current multivariate approaches to studying risk processes in youth. Furthermore, placing risk factor evidence at each level of the system generates hypotheses about potential targets for treatment, including the level at which targets should be addressed, which can be tested in clinical trials.

Fig. 1 illustrates a systemic model of risk for the development of youth depression. In contrast to models which describe hypothesized causal pathways from family factors to youth depression (Cummings & Davies, 1994; Downey & Coyne, 1990; Goodman & Gotlib, 1999), the purpose of this model is to describe the multiple factors which can influence or maintain the development of depression, the level at which each factor operates, and the ways in which each factor may interact with other factors at the same or different level of the system. The model does not specify the direction of causality, but allows for the possibility that relationships may be reciprocal and transactional rather than unidirectional. The purpose of this model is twofold: to integrate family risk factor evidence into hypothesized mechanisms of transmission which can be tested in subsequent research, and to promote the translation of this evidence into clinical interventions targeting the hypothesized mechanisms at the appropriate system level.

While several reviews of family factors in youth depression have appeared recently, this is the first to use a systemic framework to integrate the empirical findings in order to facilitate clinical application. Furthermore, we include a broader range of studies, including some studies of normal populations which have bearing on theoretical models but which have largely been ignored in the youth depression treatment literature. We also address the question of specificity of these family factors to depression, by including studies which examine other psychopathological outcomes in addition to depression.
2. Part I. Risk factors and mechanisms in the development of youth depression

In Table 1, family risk factors for depression are organized according to system levels, from the individual level to the broader social level. Level 1, the individual subsystem, focuses on risk factors related to characteristics of each individual family member (mother, father, or child). This includes genetic, temperamental, and cognitive or personality factors of parents or child. Level 2, the parent–child subsystem, includes aspects of the parent–child relationship such as attachment, rearing style, support and conflict. Level 3, the marital subsystem, includes aspects of the spousal relationship such as marital conflict. Level 4, the whole family system, includes triadic relationships (i.e. mother, father and children in interaction together), as well as subsystems interacting with each other and global family environment. Finally, Level 5, the extra-familial level, includes factors beyond the family which impact on the family and its members, such as social–economic factors and stress.

While it is beyond the scope of this review to cover all family risk factors, we have selected the risk factors at each level which have received the most attention in youth depression research. Therefore, at Level 1, we briefly review child factors such as genes, temperament, and cognitive style of the child; and the parent factor of depression; at Level 2, the parent–child relationship including attachment, rearing, support and conflict, autonomy and relatedness; at Level 3, the marital relationship; at Level 4, global family environment, triadic relations and relationship between subsystems within the family; and at Level 5, stress.

2.1. Methodological note: Levels of evidence required for causal models

In this review, we highlight the differences between cross-sectional vs. prospective longitudinal designs, and between self-report vs. observational assessment methods, because these designs produce evidence of varying strength for causal hypotheses linking specific family factors to youth depression.

2.1.1. Cross-sectional vs. longitudinal design

Most studies of family factors and depression use cross-sectional designs, which makes it impossible to prove etiological hypotheses, since child factors (e.g. depression, negative affect, helplessness) may influence parenting, or a third factor such as marital quality or shared genes could explain both. Prospective longitudinal designs provide stronger evidence for causal hypotheses; however, longitudinal designs cannot completely eliminate the possibility of a third variable causing the association, or of subtle child factors influencing parent behaviors (Cowen & Cowan, 2002). Experimental and intervention studies provide additional support for causal hypotheses, but processes involved in etiology and treatment may be different, so it cannot be assumed that mechanisms of change due to an intervention are the same as those which led to the symptom in the first place.

2.1.2. Self-report vs. observational assessment

The majority of studies of family processes in depression assess family functioning via self-report questionnaires, which have several limitations. Many studies rely on youth report of family factors and depressive symptoms, which can lead to over-inflation of the association due to shared-method variance (i.e. same informant for both the predictor and the outcome variable) (Ge, Conger, Lorenz, & Simons, 1994). Furthermore, adolescents and parents often perceive their relationship quite differently (Sheeber et al., 2001) and this difference is difficult to interpret. Depressed adolescents' negative cognitive bias may influence their views of themselves as well as their parents as excessively negative, leading to a spurious correlation between perceived parenting and depression (Alloy et al., 2006). On the other hand, social desirability may lead to an inflation of parents' report of their own positive rearing behaviors. In addition, adolescents and parents bring to bear different sets of “data” when reporting on their relationship, both of which may be valuable even if discrepant, as they reflect the different perspectives, history of interactions and the emotional meanings attached to them (Sheeber et al., 2001).

Observational studies help fill this gap by objectively measuring parent–child interactions, and by allowing the sequential analysis of behaviors within an interaction, to provide a clearer, real-time view of how adolescents and parents reciprocally affect each other. Data from observational studies can help elucidate what “actually” occurs (i.e. behaviorally), and to compare to parent and youth perceptions. Furthermore, observational data lend themselves to the development of intervention strategies, by suggesting specific points in the interaction sequence to target for change. In contrast, global perceptions of relationships such as warmth and control may be more difficult to target (Sheeber et al., 2001). While multi-method, multi-informant, longitudinal designs are clearly the gold standard; in practice we seek an accumulation of converging evidence from studies of varying methodological design and quality.

2.2. Level 1: Individual factors

2.2.1. Child factors

Characteristics of the individual child such as genetic and biological factors, temperament, cognitive vulnerability, self-image and personality fall at level 1 and are clearly important in the development of depression (Alloy et al., 2001). However, since the focus of this review is family factors in youth depression, and since several excellent reviews have examined individual child factors in depth (Alloy et al., 2001; Garber & Robinson, 1997; Goodman & Gotlib, 1999) we will limit our discussion to studies which examine the interaction between child and family factors in depression.

Depressive disorders in children and adolescents have been associated with the cognitive triad described by Beck (1967) of negative
self-image, negative view of the world, and negative expectations of the future (Beck, 1967; Stark, Schmidt, & Joiner, 1996). Beck (1967) hypothesized that early parent–child interactions play an etiological role in the development of maladaptive cognitive processes. The mechanisms underlying the link between parent–child relationship and maladaptive cognitions are increasingly receiving attention (Blatt & Homann, 1992). Dysfunctional cognitive styles in children have been associated with negative parenting practices in both cross-sectional and prospective designs, and there is evidence that dysfunctional cognitions may mediate the relationship between negative parenting and childhood depression (Alloy et al., 2006). In their review, Alloy et al. (2006) concluded that one mechanism through which negative parenting leads to depression is through the development of a negative cognitive style which increases vulnerability for depression (the cognitive vulnerability hypothesis). In contrast, there has been less support for the hypothesis that children’s negative cognitive style is related to the parents’ negative cognitive style via modeling (Stark et al., 1996). Children with a negative cognitive style have been found to interpret family interactions more negatively; this may in turn lead to more (actual) negative family interactions, in a negative feedback loop which maintains depressive symptoms (Sheeber et al., 2001).

There is increasing evidence that environments may affect children in different ways, due to temperament differences, differential susceptibility for environmental influences (Belsky, 2005) or through gene–environment interactions (Eley, Sugden, Corsico, Gregory, Sham, & McGuffin et al., 2004). Children with negative temperaments may be more sensitive to the effects of parental criticism, parental depression, and family conflict, and may need more parental support than children with an optimistic or positive temperament (Belsky, 2005). Youth with difficult or negative temperaments were found to be more sensitive to the effects of interparental discord on adolescent delinquency and depressive symptoms (Davies & Windle, 2001). Child temperament has also been found to interact with environmental and genetic factors to create increased risk for depressive and other disorders (Mufson, Nomura, & Warner, 2002). Furthermore, there is accumulating evidence that genetic predisposition may interact with adverse environmental events to increase risk for the development of depression in adults (Caspì, Sugden, Moffitt, Taylor, Craig, & Harrington et al., 2003) and children (Eley et al., 2004). Thus individual genetic vulnerability appears to require some kind of environmental stress to be fully potentiated into a depressive disorder.

Finally, individual and family factors may interact in different ways, depending on the developmental phase of the child. For example, Nolen-Hoeksema, Girgsd and Seligman (1992) found that negative family events were more predictive of depressive outcomes in early childhood, whereas pessimistic cognitive style combined with negative events to predict depressive symptoms in later childhood.

2.2.2. Parent factors

The individual characteristics of parents, such as psychopathology, personality, and cognitive style, may influence the development of child depression (Beardslee, Versage, & Gladstone, 1998; Goodman & Gotlib, 1999). In this section we will examine the impact of parental depression on the development of youth depression, as that has formed the basis of many of the explanatory models of family factors in child depression.

Children of depressed parents are at increased risk for a variety of poor behavioral and psychological outcomes relative to children of non-psychiatrically impaired parents (Beardslee et al., 1998; Downey & Coyne, 1990; Goodman & Gotlib, 1999; Weissman & Jensen, 2002). This increased risk can be observed throughout the developmental course, from infancy to toddlerhood, childhood, adolescence and adulthood (Goodman & Gotlib, 1999). For example, based on data from their 10 year longitudinal follow-up study of children of depressed parents, Weissman, Warner, Wickramaratne, Moreau, and Olfson (1997) concluded that children of depressed parents are at increased risk for anxiety disorder and major depression in childhood, major depression in adolescence, alcohol dependence in adolescence and early adulthood, and social impairment. Risk for major depression and phobia’s was three times greater compared to children of nondepressed parents, while risk for panic disorder and alcohol dependence was five times higher. Hammen, Burge, and Stansbury (1990) likewise found increased rates of psychiatric disorder in children of unipolar depressed mothers compared to children of bipolar depressed, chronically medically ill or healthy mothers. Many of these children had multiple diagnoses, more than half met criteria for an affective disorder, and 45% met criteria for major depression, compared to 11% of the children of healthy mothers (Hammen et al., 1990). Effects of parental depression have even been documented across three generations: children in families with two generations of depression (parents and grandparents) were found to be at highest risk for psychopathology compared to children of depressed parents only. Subclinical impairment across the developmental spectrum has also been observed in the children of depressed parents, including in infancy increased fussiness, more difficult temperaments, less secure attachments to mothers, and lower functioning on motor and mental development tests; in toddlerhood, increased stress reactivity and delayed self-regulation skills; and in childhood and adolescence, more school problems, lower social competence, lower self-esteem and more behavior problems (Goodman & Gotlib, 1999).

2.2.3. Hypothesized risk mechanisms

While genetic factors partly explain the transmission of depression from parent to child, genetic factors do not account for all of the variance (Bice, Harold, & Thapar, 2002), suggesting that environmental mechanisms also play a role (Goodman & Gotlib, 1999). Adverse family environments may be necessary to potentiate a genetic risk factor into the development of adolescent depression (Eley et al., 2004), or adult depression (Caspì et al., 2003).

One hypothesized environmental mechanism is that the symptoms of depression directly interfere with the tasks of parenting, such as responding appropriately to children’s needs and maintaining emotional involvement (Goodman & Gotlib, 1999). For example, depressed mothers displayed more negative/hostile and disengaged behavior, and less positive social behavior compared to nondepressed mothers, in a meta-analysis of 46 observational studies examining parenting behavior among depressed mothers (Lovejoy, Graczyk, O’Hare, & Neuman, 2000). Effect sizes (averaged across studies) were moderate for negative and disengaged behaviors, and small for positive behaviors, suggesting that the presence of negative behaviors rather than the absence of positive social interactions best differentiated depressed from nondepressed mothers.

Other investigators hypothesize that the impaired social and interpersonal skills of depressed parents, rather than specific symptoms, may lead to difficulties in parenting (Berg-Nielsen, Vikan, & Dahl, 2002; Hammen, Shih, Altman, & Brennan, 2003). Additionally, the depressed parent’s negative cognitive style may explain the transmission of depression from parent to child (Alloy et al., 2001). Finally, other aspects of family environment such as marital difficulties (Cummings et al., 2005) and stress (Goodman & Gotlib, 1999) may partly or fully account for the relationship between parental depression and child outcomes (Downey & Coyne, 1990; Emery, Weintraub, & Neale, 1982; Rutter & Quinton, 1984).

2.3. Level 2: Parent–child subsystem

The parent–child subsystem level of the family has received the most theoretical and empirical attention in relation to the development of child depression (Sander & McCarty, 2005). Attachment theory (Bowlby, 1969, 1980), cognitive theory (Beck, 1967), and psychodynamic theory (Blatt & Homann, 1992) all postulate disturbances in the early parent–child relationship as an underlying
cause of depression. While systemic theory also assumes that parent-child relationships impact on the development of depression, parent-child relationships are viewed as one of several subsystem within the family, which may interact with other subsystems.

In this section we consider several different constructs which have been used to describe the parent–child relationship and which have been linked to youth depression: attachment, parental warmth and control, parental support and parent-adolescent conflict, autonomy and relatedness, and expressed emotion.

2.3.1. Attachment

Bowlby (1980) made the first theoretical argument for the relationship between attachment and depression. According to his theory, the infant’s repeated experience of the primary caretaker’s response to his needs leads to a mental representation of the self in relation to others, the Internalized Working Model. The internalized working model forms the basis for later expectations about relationships with others, and shapes the child’s behavior toward significant others, and his capacity to regulate his emotional states. According to this theory, securely attached children will develop a self-concept as being worthy of being loved and cared for, of others as being reliable, caring and available, and an expectation of their needs being met in interpersonal relationships in the future. In contrast, insecurely attached individuals will develop a self-concept characterized by feeling unworthy of being loved, and of significant others as being unavailable to meet emotional needs in the future. Bowlby (1973) conceptualized attachment processes as extending throughout development, and attachment can be assessed at various stages of development, from early infancy (Ainsworth, Blehar, Waters, & Wall, 1978) to early and middle childhood, adolescence and adulthood (Ungerer & McMahon, 2005).

Two types of designs have been used to test the relationship between attachment and depression: prospective longitudinal studies which examine the relationship between infant attachment and later psychopathology, and studies assessing attachment in children and adolescents with depression.

2.3.1.1. Infant attachment and later psychopathology: prospective longitudinal studies. Several prospective longitudinal studies have examined the relationship between infant attachment classification and subsequent internalizing and externalizing psychopathology. While the focus of this review is on depression, prospective studies often assess both internalizing and externalizing symptoms as outcomes. Furthermore, these data can help determine the specificity of the relationship between attachment and depression, compared to other disorders. Using the Strange Situation paradigm (Ainsworth et al., 1978) infant attachment can be classified as either secure, avoidant, ambivalent/resistant, or disorganized (Sroufe, Carlson, Levy, & Egeland, 1999). In general, stronger relationships between infant attachment and later psychopathology have been found in high risk compared to low risk samples. For example, in a high-risk sample of teenage mothers and their infants, avoidant attachment was associated with aggression in middle childhood, and aggression and antisocial behavior in adolescence, whereas ambivalent attachment was related to anxiety and depressive disorders (Sroufe, Egeland, Carlson, & Collins, 2005; Warren, Huston, Egeland, & Sroufe, 1997). In contrast, studies using low risk samples have failed to demonstrate a clear relationship between infant attachment status and later psychopathology (for review, see Ungerer & McMahon, 2005).

Disorganized attachment has been found to predict later internalizing as well as externalizing behavior problems (Lyons-Ruth & Jacobvitz, 1999). Thus the evidence does not support a specific, one to one relationship between attachment and depression. Rather, attachment relationships appear to confer risk or protective effects in a cumulative, incremental fashion, in combination with individual risk factors and other environmental factors. These data also suggest the hypothesis that the negative impact of insecure attachment is more likely to be potentiated in environments with high degree of adversity or stress.

2.3.1.2. Attachment and depression in childhood and adolescence. The majority of studies of attachment in childhood and adolescence assess attachment as measured by the youth’s report of attachment. Two measures have been frequently used: the Inventory of Parent and Peer Attachment (Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990), a self-report measure which assesses affective and cognitive dimensions of adolescents’ relationships with parents and close friends, particularly in terms of psychological security, and the Adult Attachment Interview (AAI) (George, Kaplan, & Main, 1985), which assesses the adolescent’s memories of childhood experiences with parents, and the impact of these experiences on current relationships. Thus, the operationalization of attachment in youth is based on the youth’s self-reported affective and cognitive schemas about attachment, in contrast to the behavioral and interactive assessment used in the Strange Situation paradigm for assessing infant attachment. While this is consistent with Bowlby’s theory that the actual attachment relationship will be internalized as the internal working model, the types of data yielded by these two designs are quite different.

Essau (2004) found that perceived attachment to parents but not peers on the IPPA was significantly worse in depressed adolescents compared to adolescents with no psychiatric disorder in a community sample. Armsden et al. (1990) compared self-reported attachment security in early adolescents who were either currently depressed, depressed remitted, nondepressed psychiatric controls or maternal illness controls. Depressed adolescents reported less secure parent attachment compared to nondepressed psychiatric controls, nonpsychiatric controls, and less secure peer attachment compared to nonpsychiatric controls. However, the attachment security of the adolescents with remitted depression was not significantly different from the nonpsychiatric control group. This finding suggests that the negative bias during the depressive episode may account for the lower self-reported attachment of the depressed children.

While the above studies suggest a relationship between self-reported youth attachment and depression, they do not address the actual relationship between parent and child, making it impossible to rule out negative attributional bias as the cause of the correlation. Self-reported measures of attachment strategies presumably reflect the accumulation over time of interactions with attachment figures as perceived by the child, consolidated into a relatively stable, cognitive-affective perceptual schema which then shapes the interpretation of new interactions with significant others (Kobak, Sudler, & Gamble, 1991). However, the actual quality of the current relationship with the parent may also affect the individual’s experience, and may serve to either confirm or disconfirm the attachment schema. For example, Sroufe et al. (1999) found that individuals whose attachment patterns switched from insecure in infancy to secure in adolescence had a better prognosis than those who were insecure at both time points, or secure as infants but insecure as adolescents.

Studies which examine both perceived attachment strategy and observational measures of parent–child interaction can help disentangle these issues, and elucidate possible mechanisms through which attachment in childhood and adolescence is related to actual interactions with parents and peers. For example, Kobak, Cole, Ferenz-Gillies, Fleming, and Gamble (1993) tested the hypothesis that secure adolescents would be better able to negotiate age-appropriate autonomy from parents, while maintaining a cooperative relationship with parents. Consistent with their hypothesis, in a disagreement task with parents, adolescents with secure attachment were able to assert their own point of view while still listening to their mothers (“balanced assertiveness”) and showed less avoidance and dysfunctional anger than insecure adolescents (Kobak et al., 1993). In a related study, both insecure and preoccupied attachment as measured by the Adult
Attachment Interview (AAI) were significantly related to depressive symptoms, and the interactions of depressed teens and their mothers were characterized by maternal dominance and dysfunctional anger (Kobak et al., 1991).

Abela, Hankin, Haigh, Adams, Vinokuroff, and Trayhern (2005) hypothesized that excessive reassurance-seeking behavior may interact with insecure attachment, leading to child depression. That is, insecurely attached children may be prone to ask for excessive reassurance from their parents; parents in turn may feel irritated and respond in a rejecting manner, thereby reinforcing the child’s insecurity and low self-esteem, and increasing depressive symptoms. They examined children’s self-reported attachment to parents, reassurance-seeking behavior, and depression in 6–14 year old children of depressed parents. Children who reported both insecure attachment and high excessive reassurance seeking had higher levels of current depressive symptoms, were more likely to have a past history of depression, and had greater severity of depressive episodes, compared to those that had just one (insecure attachment or excessive reassurance seeking).

Similarly, Allen, Moore, Kuperminc, and Bell (1998) have hypothesized that insecurely attached adolescents will have more difficulty establishing autonomy from parents while maintaining a sense of relatedness, and that difficulty establishing autonomy will increase risk for internalizing and externalizing symptoms. Consistent with the first part of their model, adolescents with secure attachment strategies on the AAI had current adolescent–mother relationships characterized by autonomy and relatedness: they could disagree amiably with their mothers, had a realistic rather than idealizing view of their mothers, felt supported by their mothers, and had mothers who were attuned to their self-perceptions (Allen et al., 2003). A strength of this study was that the adolescent–mother relationship was assessed with a combination of observational, adolescent-reported and mother-reported measures. In a related study, securely attached adolescents reported higher competence with peers, lower self-reported internalizing symptoms, and lower peer- and mother-reported deviant behaviors, supporting the hypothesized link between attachment security and adolescent psychosocial functioning (Allen et al., 1998). Turning to the role of the father in attachment and autonomy processes in adolescents, Allen, Porter, McFarland, McElhaney, and Marsh (2007) found that adolescents’ relationships with their fathers and peers significantly contributed to attachment security, even after controlling for maternal relationship factors. Furthermore, attachment insecurity was linked to higher levels of depressive symptoms across adolescence, primarily in females. In contrast, Pavlidi and McCauley (2001) found that while depressed adolescents reported poor attachment to parents, observed parent–adolescent attachment quality was not found to be significantly different from nonclinical adolescents.

Finally, attachment representations may be changeable, for example through new positive relationships in therapy (van Ijzendoorn, 1995), or through improvements in the attachment relationship with parents.

2.3.1.3. Attachment and youth depression: Summary. In summary, although a direct causal link between infant attachment and depressive symptoms is not supported, the available evidence suggests that early attachment may operate as a risk or protective factor for the development of both internalizing disorders and externalizing disorders, with the strongest relationships being found in high-risk samples, and with infants with a disorganized attachment pattern. It may be that severe disruptions of attachment, or disruptions of attachment within the context of highly stressful, chaotic or aversive environments, are necessary to potentiate the development of disorder, although further evidence is required. In adolescence, perceived insecure attachment to parents has been related to cognitive processes such as maladaptive attributional style, observed parent–child interactions such as excessive reassurance seeking and autonomous behavior, and symptoms of psychopathology such as depression, internalizing and externalizing symptoms. Two models have been suggested in the literature: the mediation model, in which parent–child relationship deficits lead to insecure attachment strategies which in turn leads to internalizing or externalizing behavior, and the moderation model, in which adolescent attachment style interacts with actual parent–child relationship to produce depressive or other symptoms. While the data are not inconsistent with either hypothesis, they do not definitively confirm one over the other. What is clear, however, is that in adolescence, self-reported attachment and observed parent–child relationships, while correlated, are constructs which can be separately measured, and should be differentiated in studies examining adolescent attachment.

Finally, the available evidence cannot rule out the hypothesis that the attributional bias of depressed adolescents accounts for the lower self-reported attachment security. While these findings are consistent with Bowlby’s original model linking deficient early caretaking with insecure attachment and a negative sense of self in relation to others, further research is required to establish causal links between attachment processes as they unfold over time, actual parent–child relationships, cognitive-affective schemas, and the development of depressive symptoms.

2.3.2. Level 2: Parental rearing, conflict and support, autonomy and relatedness

Many different aspects of the parent–child relationship have been examined in relation to youth depression, but most fall under one of two broad dimensions: warmth/support and control/autonomy. A third dimension, conflict, has also been examined, most often in relation to parental support, and usually in adolescent as opposed to child samples. While overlapping, each of these terms has been defined and operationalized in somewhat different ways, which will briefly be described before discussing specific findings.

Warmth and control. Broadly defined, parental warmth or care refers to acceptance and responsiveness, emotional closeness, and availability on the positive end, and criticism, rejection, lack of warmth or acceptance on the negative end (Gladstone & Parker, 2005). Two forms of parental control have been differentiated: psychological control, which refers to over protectiveness, guilt-induction, shaming, threats of withdrawal of love, or intrusiveness, and behavioral control, which refers to consistency in limit-setting, supervision and monitoring of children (Barber, Olsen, & Shagle, 1994). Whereas studies of parenting in community samples have found associations between behavioral control and positive outcomes, studies of psychopathology have more often focused on the negative consequences of high psychological control combined with low warmth, or the so-called “affectionless control” style (Rapee, 1997).

Support and conflict. Parental support is a variable frequently examined in studies of adolescents, usually in combination with conflict. While similar to warmth, it is typically operationalized with different measures. Several aspects of conflict have been examined: severity, tactics and resolution.

Autonomy and relatedness. While the construct of autonomy is similar to the positive side of psychological control, and the concept of relatedness to warmth and support, studies employing these variables are grounded in developmental theory which views establishment of autonomy as the central psychological task of adolescence (Allen, Hauser, Bell, & O’Connor, 1994). Furthermore, autonomy and relatedness are often operationalized using observational measures of the parent–child interaction.

Expressed emotion. Expressed emotion (EE) refers to the degree of criticism and emotional involvement expressed in the verbal descriptions of patients with schizophrenia and depression by their family members (Asarnow, Thompson, Hamilton, Goldstein, & Guthrie, 1994), and has been shown to correlate strongly with relapse. These
constructs are similar to the constructs of rejection (warmth) and psychological control, but are assessed differently.

In the following section the empirical evidence on the association between the parent–child relationship and youth depression will be reviewed, beginning with non-observational (self-report) studies, followed by observational studies.

2.3.3. Parental care and control in children and adolescents: Non-observational studies

Cross-sectional studies. Depressed adults consistently have reported greater perceived parental rejection and psychological control compared to nondepressed adults in retrospective studies of the parent–child relationship (Gladstone & Parker, 2005; Parker, 1983). However, since retrospective designs cannot rule out the possibility that this is due to the known negative cognitive and attentional bias of depressed individuals, only studies of depressed youth will be included in this section.

In one of the first studies directly assessing current interpersonal relationships of depressed children, Puig-Antich, Lukens, Davies, Goetz, Brennan-Quattrocioc, and Todak (1985a) interviewed the mothers of prepubertal depressed children. The psychosocial functioning of the depressed children was significantly worse than both psychiatric and normal control children (including poorer communication, less warmth and greater rejection in the mother–child relationship). Father–child relationships were also significantly poorer in the depressed and psychiatric children compared to normal controls. Even after the children had fully recovered, including normal school functioning, the parent–child relationships, though improved, did not reach the level of the control group, suggesting some enduring family relationship difficulties even after remission of symptoms (Puig-Antich et al., 1985b).

Two comprehensive reviews of parental rearing and youth depression have found consistent evidence for a relationship between low care and high psychological control parenting style (“affectionless control”) and depression; however, the evidence is stronger for care than control (Alloy et al., 2006; Rapee, 1997). Reviewing cross-sectional studies, Alloy, Abramson, Smith, Gibb, and Neener (2006) found consistent support for an association between low parental care and depressive symptoms in children and adolescents, whereas findings for control/overprotection were mixed: two out of three studies reported a significant relationship between high control and depressive symptoms (Garber, Robinson, & Valentiner, 1997) and one failed to find an association (Greaven, Santor, Thompson, & Zuroff, 2000). In studies examining rearing and depressive disorders, five out of six studies found a significant association between “affectionless control” parenting and youth diagnosis of depression, with one study (Burbach, Kashani, & Rosenberg, 1989) finding that “affectionless control” was not associated with depression, but rather with other psychiatric disorders (Alloy et al., 2006). In a study examining psychological as well as behavioral control, Barber et al. (1994) found parental psychological control to be related to internalizing symptoms, whereas low behavioral control was related to externalizing symptoms.

Prospective studies. Three longitudinal studies have examined parental support and parent–adolescent conflict in relationship to onset, course, and remission of depressive symptoms or disorder, in order to test the causal hypothesis that increases in parent–adolescent disagreement are related to increases in depressive symptoms over time. Rueter, Scaramella, Wallace, and Conger (1999) found that increases in parent-reported parent–adolescent disagreements from age 12/13 to age 14/15, predicted increase in adolescent-reported internalizing symptoms in the same time period, which in turn predicted first onset of diagnosis of depressive or anxiety disorder at age 19. The authors statistically tested and rejected several alternative hypotheses, including the bidirectional hypothesis that emotionally distressed adolescents induce more conflict in family members. In addition, by using parent reports of parent–adolescent conflict to predict youth reports of symptoms, they avoided shared-method variance problems. Similarly, Stice, Ragan, and Randall (2004) found that deficits in perceived parental but not peer support in 11–15 year old girls were related to increases in depressive symptoms and first onset of major depression two years later. They also found that youth depressive behavior led to decreases in perceived peer social support, but not perceived parent social support, suggesting “support erosion” due to depression occurs more in peer social relations than with parents. Sandford et al. (1995) looked at factors which predicted persistence of major depression one year later in a sample of clinically depressed adolescents, and found that adolescents who continued to be depressed were less involved with their fathers, and less responsive to maternal discipline compared to remitters. In a related finding from a treatment study, parent–child conflict and low affective involvement at baseline and follow-up predicted lack of recovery, chronicity and recurrence of depression at two year follow-up in a randomized controlled trial comparing cognitive–behavior therapy to family therapy for depressed adolescents (Birmaher et al., 2000).

Several studies have examined prospective relationships between parental care and control and child depression. In a ten year follow-up study of offspring of depressed and nondepressed parents, Nomura, Wickramaratne, Warner, Mufson, and Weissman (2002) found affectionless control parenting to be associated with a six-fold increased risk of major depressive disorder (MDD) and parent–child discord with a four-fold increased risk of MDD, but only among offspring of nondepressed parents. In contrast, among offspring of depressed parents, parental affectionless control was not associated with offspring diagnosis of MDD, anxiety or substance abuse. Affectionless control also predicted a 12 fold risk of substance abuse in children of nondepressed parents, suggesting a lack of specificity of affectionless control for depression. Multivariate analyses indicated that parental depression was more important than family discord for predicting offspring MDD or anxiety disorders, whereas family discord was more important for predicting substance abuse disorders. Reporting on the same sample, Miller, Warner, Wickramaratne, and Weissman (1999) found that maternal affectionless control was associated with daughter low self-esteem, which in turn was related to daughter depression at 10 year follow-up, but only among daughters of depressed mothers.

Similarly, Liu (2003) found that perceived parental care and indifference of 6th grade Taiwanese schoolchildren predicted depression scores one year later, with children’s self-esteem fully mediating the relationship between low care and depression and partially mediating that between indifference and depression. Furthermore, girls and boys may respond differentially to parenting from mothers vs. fathers: In a prospective study of parental care and control, mothers’ reports of their own rejecting and controlling behavior toward their 5 year old children predicted self-criticism in the daughters at age 12, whereas mother-reported paternal control and rejection predicted self-criticism in the sons at age 12 (Koestner, Zuroff, & Powers, 1991).

Finally, Hale, van der Valk, Akse, and Meeus (2008) tested a bidirectional model of perceived parental rejection and adolescent depressive and aggressive symptoms over four yearly assessments in young adolescents. In the first two assessments, perceived parental rejection predicted adolescents’ depressive symptoms, and adolescent depressive symptoms predicted perceived parental rejection, supporting bidirectional effects between parental rejection and adolescent depressive symptoms. However, in the last two assessments, adolescent depression predicted parental rejection, but parental rejection did not predict adolescent depression, suggesting that by later adolescence, the depressive behavior of the adolescent was influencing the parental rejection, not vice-versa. Interestingly, parental rejection predicted adolescent aggression at all four time periods. The authors hypothesize that in early adolescence, parental
rejection and adolescent depression are reciprocally related, but by later adolescence, a transactional process has been established whereby parents react negatively to their adolescents’ depressive behavior, and adolescents who feel rejected react with aggression.

In summary, the findings from prospective longitudinal studies show that key aspects of the parent–child relationship such as care, control, support and conflict predict increases in depressive symptoms, first onset of depressive disorder, increased risk for major depression, and persistence, lack of recovery, chronicity or relapse of major depression. The prospective design provides stronger evidence compared to cross-sectional studies for a causal relationship between parent–child relationship factors and risk for depression. While some evidence supports unidirectional parent–child effects, there is also evidence for bidirectional effects of child depressive symptoms and parenting. Finally, strong support is lacking for a specific relationship between parent–youth variables and depression. Rather, these parent–youth variables are also related to anxiety, substance use, and externalizing symptoms and disorders.

2.3.3.1. Observational studies of parent–child relations. In this section studies using observational methods are reviewed.

Cross-sectional studies: support and conflict. Several studies have combined observational and self-report assessments to investigate the relationship between perceived and observed parent–youth relationships. For example, Sheeber and Sorensen (1998) found that depressed adolescents and their mothers both described their families as being less supportive and more conflictual than non-distressed adolescents and their mothers. In observed problem-solving interactions, the same depressed adolescent–mother pairs demonstrated less facilitative and more depressive interactions. However, neither adolescent nor mother exhibited higher rates of aggressive behavior. In a second study combining both self-report and observational measures, Sheeber, Davis, Leve, Hops, and Tildesley (2007) found that both clinically and sub-clinically depressed adolescents had less supportive and more conflictual relationships with their mothers as well as fathers compared to healthy adolescents. Furthermore, a supportive relationship with either father or mother did not moderate the relationship between conflict and depression. Self-report and observational data were combined to create factors of support and conflict; interestingly, observational data did not load as highly as self-report data on the two constructs.

McCarty, Lau, Valeri, and Weisz (2004) examined whether the two dimensions of expressed emotion (EE), criticism and emotional involvement, were correlated with observed parent–child interactions. Parents rated high on the critical dimension of EE were observed to be higher on antagonism, negativity, disgust, harshness and to be less responsive in their interaction with their children compared to low or borderline critical parents. In contrast, none of the observed parent–child interaction variables corresponded to the parental emotional over-involvement dimension of EE. Similarly, parents of depressed children have been found to score higher on the criticism dimension of EE compared to parents of nonclinical (Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990) or schizophrenia spectrum disordered children (Asarnow et al., 1994) whereas the over-involvement dimension did not differentiate depressed from nonclinical children (Asarnow et al., 1994).

Parental support and conflict: Prospective observational studies. Finally, several studies have combined observational assessment of parent–child interactions with prospective longitudinal design, which provides the strongest test for causal hypotheses. Burge and Hammen (1991) found observed critical interaction quality and low task involvement during a mother–child disagreement task to be associated with children’s depressive symptoms and maladaptive school performance six months later in children aged 8 to 16. Positive mother–child interactions were associated with good school performance six months later.

Ge, Lorenz, and Conger (1994) examined whether maternal warmth and support moderated the relationship between stress and depressive symptoms in girls and boys, in a longitudinal study employing home observations of youth aged 9–17. Observed maternal warmth and support moderated the relationship between stressful life events and depressive symptoms in girls, not boys. Furthermore, they found differences in the trajectories of girls and boys’ depressive symptoms and stress: Girls’ depressive symptoms increased after age 13, whereas boys’ depressive symptoms remained relatively stable after age 13. Boys reported more stressful life events than girls before age 12, but after 13, the pattern reversed, with girls’ reporting a greater increase in stressful life events, particularly between 13 and 15. The authors conclude that girls not only experience more stressful life events during adolescence, but are also more sensitive to the effects of these stressful events than boys, for whom the increases in negative events were not associated with increased depressive symptoms. This gender difference in experienced stressful life events and vulnerability to these events may explain why the moderating effect of maternal support was found for girls but not boys.

Prospective studies employing multi-method, multi-informant measurement illustrate how parents and children perceive the parent–child relationship differently. For example, Sagrestano, Paltok, Holmbeck, and Fendrich (2003) found that child reports of increases in parent–child conflict and decreases in parental monitoring were associated with increases in depressive symptoms in low income, African American youth aged 9–15. On the other hand, parent-reported increases in positive parenting were associated with decreases in child depression two years later. Increases in observed “peer relationship” between parent and child (that is, a relationship characterized by the child acting like a parent, and the parent acting like a child), were related to increases in depressive symptoms at follow-up. However, 12 other dimensions of observed parenting or family atmosphere were not associated with increases in depression scores.

Prospective observational studies: Autonomy and relatedness. Several developmental researchers have hypothesized that difficulty in negotiating the central adolescent task of establishing autonomy from parents can increase vulnerability to depression and other disorders (Allen et al., 1994; Powers & Welsh, 1999). According to Powers and Welsh’s model, the transition into adolescence leads to increases in the child’s expression of autonomy, which in turn leads to increases in conflict with parents and decreases in the adolescent’s submissive behavior. Families which cannot tolerate conflict or in which conflict is extreme, may not provide the necessary support for the adolescent’s developmental need to negotiate conflict and a more autonomous relationship with the parents (Powers & Welsh, 1999). Furthermore, adolescent girls and their mothers may have particularly difficult time with this phase of development, given girls’ socialization to be more submissive and conflict-avoidant, and mothers’ potential loss of power as adolescent autonomy increases (Powers & Welsh, 1999).

To test this model, Powers and Welsh (1999) examined longitu-dinally the relationship between mother–daughter interactions reflecting autonomy, and daughters’ internalizing symptoms. They tested two directional models: a causal model in which mother–daughter interpersonal behaviors predicted increases in daughter’s internalizing symptoms one year later, and a reactive model in which daughters’ internalizing behaviors predicted difficulty negotiating autonomy. To combine subjective perception with objective interaction, mothers and daughters rated their own videotaped interaction. Daughters high in internalizing symptoms rated themselves as having high levels of conflict and submission with mothers, whereas their mothers, rating the same interaction, viewed their daughters’ behaviors as humorous rather than reflecting conflict or submission. The daughters’ perceived submissive behavior to mother was associated with increased risk of internalizing symptoms one year
later, supporting the causal/maintaining model. Furthermore, both too high and too low levels of mother’s conflict, humor and sarcasm were related to daughter’s internalizing symptoms one year later, supporting their hypothesis that a moderate degree of conflict is protective (Powers & Welsh, 1999).

Allen et al. (1994) have conducted a series of longitudinal studies examining the relationship between adolescent autonomy development and internalizing and externalizing symptoms. Observed difficulty establishing autonomy and relatedness with parents at age 14 and 16 was linked to observed depressed affect and self-reported externalizing behaviors at age 17. Difficulties with autonomy were more strongly linked to depressed affect, and difficulties maintaining relatedness more strongly linked to externalizing behaviors. Lack of hostile, relatedness-inhibiting behaviors in the parent–adolescent dyad was associated with adolescent depressive affect, similar to Power and Welsh’s (1999) finding of submissiveness in internalizing girls.

In a related longitudinal study, autonomy and relatedness with both mother and peers contributed to increases in depressive symptoms in adolescents (Allen et al., 2006). Specifically, adolescent–mother interactions in which the adolescent undermined autonomy and relatedness in a disagreement task, and adolescent peer interactions in which the adolescent was withdrawn, angry or dependent, each independently predicted increases in depressive symptoms one year later (Allen et al., 2006). In contrast, Pavlidis and McCauley (2001) found no differences in observed autonomy and relatedness between clinically depressed adolescent–mother dyads compared to nonclinical pairs, but lower self-reported attachment to parents among the depressed adolescents.

In summary, findings from observational studies of parent–youth interactions are generally consistent with findings from self-report studies on care, support, conflict, expressed emotion and autonomy. Specifically, there is evidence for the critical dimension of EE corresponding to observed antagonistic and harsh parenting, for observed critical and uninvolved parenting to be prospectively related to depressive symptoms, and for observed difficulty with autonomy and relatedness between adolescents and parents to be prospectively related to observed depressed affect. However, some interesting discrepancies between self-report and observational data were also found. In Sheeber and Sorensen’s (1998) study, depressed adolescents and their mothers experienced themselves as being more conflictual and less supportive; however, their interactions were not characterized by higher aggressive behavior, but rather by lower facilitative behavior, and higher depressive behavior. The impact of parental support is also unclear. Sheeber et al. (2007) found no evidence in their cross-sectional study that father or mother support moderated the relationship between conflict and depressive symptoms, and Powers and Welsh (1999) found that maternal support was not prospectively related to adolescent girls’ internalizing symptoms. In contrast, Ge et al. (1994) found that observed maternal support moderated the relationship between stress and later depressive symptoms in girls, but not boys. Finally, observational measures were found to be less related to outcome variables than self-report (Sagrestano et al., 2003) and loaded less strongly than self-report measures on combined variables (Sheeber et al., 2007). Powers and Welsh’s study (1999) provides some hypotheses for these discrepancies. Mothers and daughters perceived the same interaction quite differently: the high internalizing girls felt submissive and in conflict; their mothers found them humorous. Mothers may interpret submissive behavior in a positive light, as cooperative, helpful, or close. Such an interpretation may be appropriate for younger children, but may be less appropriate for adolescents, who may desire more autonomy but be afraid to express it directly, leading to feelings of resentment. Similarly, an explanation for the conflicting results with regard to maternal support is that too much maternal support in adolescence may be related to a lack of autonomy and avoidance of healthy levels of conflict in the mother–adolescent relationship.

Finally, these studies illustrate how the framework of adolescent autonomy development helps integrate the findings reviewed above on parental rearing, conflict and support, and leads to testable hypotheses about the relationship between these variables.

2.4. Level 3: Marital relationship

In this section, the impact of the marital relationship on child functioning will be reviewed. The majority of this evidence comes from prospective longitudinal studies of community samples which examine a broad range of outcomes, from adjustment problems to internalizing and externalizing symptoms. These studies provide the strongest evidence for causal models linking marital relationship to child outcomes and allow investigators to determine the specificity of the relationship between marital factors and depression (compared to other symptoms such as aggression). While these studies are broader in scope than this review’s specific focus on depression, they are included in this section as they provide data to test the hypothesis that marital relations impact on child psychopathology.

There is clear evidence that marital discord and divorce are associated with child adjustment problems and psychopathology (Cummings, 1994; Emery, 1982). Studies examining marital discord and divorce show that much of the deleterious effect of divorce on child functioning can be explained by the high degree of marital discord in divorcing families rather than the fact of the divorce or separation per se (Emery, 1982). Among intact couples, marital conflict may have the most negative impact on children (Cummings, 1994; Hudson, 2005). In response to interparental conflict, children show behavioral distress, report feeling anger, sadness, fear, guilt, shame or worry, and show physiological reactions (Cummings & Wilson, 1999). Interparental conflict has been associated particularly with aggression, conduct problems and other externalizing disorders, but also with internalizing problems, impaired social skills, and child–parent and peer relationships (Cummings, 1994; Hudson, 2005).

Marital conflict appears to play a role in the transmission of psychological problems from depressed parent to child (Downey & Coyne, 1990). For example, Emery et al. (1982) found little association between parental unipolar or bipolar depression and children’s disturbed school behavior, after controlling for the effect of marital discord. In their study of the children of psychiatrically ill parents, Rutter and Quinton (1984) concluded that the main risk in most cases stemmed from the associated psychosocial dysfunction in these families. Thus, marital conflict may explain part of the increased risk for problems among children of depressed parents. In contrast, Nomura et al. (2002) found parental depression to be a stronger predictor of offspring depression compared to marital discord and other family environment risk factors, suggesting a direct effect of parental depression on offspring depression.

While the negative effects of marital conflict on children have been well documented, some amount of conflict may be inevitable in family relationships, and conflict may serve a number of useful functions within a family, such as resolving disagreements, developing problem-solving skills, and modeling effective interpersonal conflict solving (Cummings, 1994; Cummings & Wilson, 1999). Determining constructive as opposed to destructive interparental conflict tactics becomes an important question (Cummings & Wilson, 1999). Indeed, the type of conflict tactics used by parents appears to have an effect on children. In general, interparental hostility and withdrawal have been more strongly related to externalizing than internalizing problems in children (Katz & Woodin, 2002). For example, interparental verbal and nonverbal hostility, threat of physical aggression, and withdrawal increased the likelihood of children’s aggressive responding, whereas interparental calm discussion, humor, support, affection and problem solving decreased likelihood of children’s aggressive responding in children aged 8–16 (Cummings, Goake-Morey, & Papp, 2004). However, interparental conflict was found to predict internalizing
problems when the husband was angry and withdrawn (Katz & Gottman, 1993). Avoidant conflict tactics have been found to be related to higher rates of internalizing, but not externalizing problems in children (Marchand & Hock, 2003).

Finally, conflict resolution has been shown to ameliorate children's distress in the face of interparental conflict in experimental analogue studies, in which children are exposed to videotapes depicting “parents” (i.e. actors) fighting, with varying degrees of conflict resolution (Davies & Cummings, 1994). Complete resolution of conflict has been shown to bring children's distress levels back to baseline levels, but even partial resolution of conflict reduces children's distress (Cummings, 1994). Children's distress is also reduced when “parents” resolve their conflict off screen, or when they tell children that the conflict has been resolved (Cummings, Simpson, & Wilson, 1993).

In summary, marital discord, particularly marital conflict, has been related to both internalizing and externalizing problems in children in cross-sectional, longitudinal and experimental studies, has been found to be elevated in families of depressed children and depressed parents, and may partially account for the relationship between parental depression and child psychopathology, including depression. How parents fight seems to matter, with actual or threatened physical aggression, anger combined with withdrawal, and avoidant tactics having particularly deleterious effects on children, and calm discussion, humor and problem solving decreasing negative effects. Resolution of the conflict, whether directly observed or only explained by parents, has been found to ameliorate the negative impact of interparental conflict in analogue experiments. The bulk of this research has been conducted in nonclinical populations, and stronger relationships have been found between interparental conflict and externalizing symptoms than internalizing symptoms.

2.5. Level 4: Whole family system

In this section, we turn to studies which have examined interconnections between the above subsystems within the context of the broader family system. Thus we move from consideration of the individual, dyadic relationships (spouse-spouse or parent–child) to triadic relationships, that is mother, father and child(ren). The shift from dyadic to triadic relationships allows interrelationships between various subsystems to be examined, which may impact on dyadic and individual processes (Bronfenbrenner, 1986; Minuchin, 1974). By examining a broader level of the system, transactional processes may be revealed which might otherwise go unnoticed. For example, if a depressed mother is observed to be withdrawn from and critical of her children, a causal relationship may be hypothesized:

Maternal depression—Critical withdrawn parenting—Child depression.

However, if the scope is broadened to include the father, we may notice a high degree of marital conflict, with father alternating between hostile and withdrawn behavior, and mother exhibiting hostile, dependent, and withdrawn behavior. With this additional data, several different levels of the system can be modeled: individual (mother, father and child), marital relationship separate from child, and marital relationship including child. The causal relationship between these factors is not immediately evident, and can be modeled in a series of competing hypotheses, or modeled as transactional, circular causal models.

The shift from dyadic to triadic family processes also has important implications for research design. In the above example, marital conflict can be included in a statistical model, as a third variable which accounts for the relationship between maternal depression and child depression, or as a moderator or mediator variable. For example, marital conflict may moderate the relationship between maternal depression and child depression, such that the interaction between maternal depression and marital conflict leads to child depression.

Alternately, marital conflict may mediate the relationship between maternal depression and child depression, such that maternal depression leads to increased marital conflict, which leads to child depression. Conversely, maternal depression may mediate the relationship between marital conflict and child depression, such that marital conflict leads to maternal depression, which leads to child depression. Furthermore, reciprocal causal pathways can be tested. Thus including more levels of the family system can lead to research designs in which competing hypotheses are tested against each other. Finally, there is greater ecological validity in studying triadic systems: most families, even divorced or single-parent families, include some variation of father, mother and children and how these parents interact clearly affects child functioning (Emery, 1982).

2.5.1. Global family environment

Several studies have examined the perceived family environment of depressed youth. Stark, Humphrey, Crook, and Lewis (1990) compared the perceived family environment of depressed only, anxious only, comorbid depressed/anxious, or normal 4th to 7th grade children and found that all disordered children perceived their families as less supportive, more conflictual, less involved with outside recreational activities, more enmeshed, and less involved in decision making processes, compared to the normal controls. Children with comorbid anxiety/depression and their mothers reported the greatest degree of family disturbance compared to anxious only and control children. Moreover, in a study of adolescent school refusers comorbid for anxiety and depression, adolescents and their families described their family environment as low in cohesion (disengaged) and in adaptability (rigid) ( Bernstein, Warren, Massie, & Thuras, 1999). Adolescents in extremely disengaged families were significantly more depressed than adolescents in connected families, and adolescents in extremely rigid families had higher somatic complaints. Finally, Kashani, Suarez, Jones, and Reid (1999) compared perceived family environment of clinically depressed or anxious youth, and found that depressed youth reported less pride in their families; less trust, respect and loyalty between family members; viewed their families as less adaptable when under stress; and felt less supported by family members.

Studies of perceived family environment are limited by the potential bias on the part of child or parent; however, observational studies can address this limitation. In an observational longitudinal study, Jacobvitz, Hazen, Curran and Hitchens (2004) examined whether boundary disturbances in the family interactions of 24 month old children predicted depressive, anxiety and ADHD symptoms at age 7. Both disengaged family patterns (low warmth, affection, eye contact or emotional responsiveness between family members), as well as by enmeshed family patterns, (one parent turns to a child for inappropriate intimacy or guidance, or undermines the authority of the spouse) predicted depressive symptoms at age 7, even after controlling for maternal depression. In contrast, controlling interactions predicted anxious symptoms, and hostility predicted ADHD symptoms. Girls and boys showed different reactions to family patterns: girls who experienced the enmeshed pattern were more vulnerable to developing depressive symptoms, whereas boys were more vulnerable to developing ADHD symptoms. The fact that observed disengagement or enmeshment at age 2 predicts depressive symptomatology at age 7 provides stronger evidence for a causal role of actual, not only perceived, family processes in the development of depressive symptoms. (However, the possibility of a third variable such as genetic factors explaining both cannot be ruled out.) Furthermore, it broadens the issue of warmth and control to the larger family context: what has sometimes been treated as a measure of parent–child relationship may in fact reflect a broader family style, which in turn may impact on parent–child relationships.

Similarly, Rueter and Conger (1995) hypothesized that an overall family climate characterized by warmth and supportiveness in early
adolescence would buffer against the increased parent–child conflict in middle adolescence, by supporting conflict resolution, and keeping conflict at low to moderate levels. Conversely, a hostile or coercive environment would make it more difficult to resolve conflicts, leading to escalation of conflict. Using prospective longitudinal data of adolescents from age 12/13 to 15/16, they found that families which displayed critical, coercive, and angry behavior towards each other became more hostile one year later, which in turn predicted more defensive and disruptive behavior during problem-solving tasks, and which further predicted exacerbated parent–child disagreements. In contrast, families strong in communication, attentive listening, and warmth remained warm one year later, and these emotionally supportive families’ problem-solving abilities improved. Furthermore, warmth and supportiveness directly and more strongly related to supportive families’ problem-solving abilities, including family system (individual, marital dyad, parent–child dyad, entire family), making it difficult to interpret findings from bivariate analyses, or determine causal mechanisms. Multivariate designs address these limitations by simultaneously measuring several risk factors at different levels, and testing competing causal models. For example, Cummings et al. (2005) examined interrelationships between maternal and paternal depressive symptoms, family functioning, and child adjustment in a community sample of families with kindergarten children. Increased parental depressive symptoms were associated with greater marital conflict, insecure marital attachment, less parental warmth, more psychological control in parenting, and with children’s internalizing and externalizing problems, peer exclusion, and lower prosocial behavior. Marital conflict mediated the relationship between maternal depressive symptoms and children’s internalizing problems, whereas maternal attachment mediated between maternal depressive symptoms and peer exclusion. In fathers, marital conflict and marital attachment mediated the relationship between paternal depressive symptoms and children’s internalizing problems. While parental depressive symptoms were related to less effective parenting, parenting did not mediate the relationship between parental depression and child outcomes. Once again, girls and boys responded differentially to mothers and fathers: mother’s depressive symptoms were more strongly associated with girls’ peer exclusion than boys, and father’s depressive symptoms were more strongly and negatively associated to boys’ prosocial behavior than girls.

Similarly, Cox, Paley, Payne, and Burchinal (1999) examined the effects of the marital relationship on parenting by measuring marital interactions in couples expecting their first baby. Marital withdrawal rather than conflict (assessed prior to the infant’s birth) predicted less sensitive parenting for both husbands and wives when their infants were 3 and 12 months. For fathers, the interaction of marital conflict and withdrawal predicted less sensitive parenting at 12 months. Interestingly, relationships between parental depressive symptoms and parenting were relatively weaker.

Finally, Low and Stocker (2005) found that mother’s and father’s marital hostility was linked to parent–child hostility, which was linked to children’s internalizing problems. There were different patterns found for mothers and fathers: Mothers’ marital hostility was directly associated with child externalizing problems, whereas fathers’ marital hostility was indirectly associated with externalizing problems via father–child hostility, and father’s depressed mood was related to child internalizing problems indirectly via father–child hostility. Similarly, Hops, Biglan, Sherman, Arthur, Friedman, and Osteen (1987) compared the children of depressed mothers with marital distress to children of depressed mothers without marital distress, and found that children of depressed mothers with marital distress displayed significantly higher irritable affect than children of normal mothers, whereas children of depressed mothers without marital distress did not. Taken together, these findings suggest that marital distress may be an important factor in the relationship between maternal depression and youth depression, with interpersonal hostility directed at the child being one hypothesized mechanism of transmission.

In contrast, Nomura et al. (2002) examined the impact of several measures of family discord (poor marital adjustment, parent–child discord, low family cohesion, affectionless control, divorce) on risk for psychiatric disorders among offspring of depressed and nondepressed parents, in a prospective longitudinal study. While all family discord measures were associated with a diagnosis of either major depression, anxiety disorder or substance abuse among the offspring of nondepressed parents, among the offspring of depressed parents, family discord was not related to child diagnosis (with the exception of low family cohesion increasing the risk of substance abuse). Parental depression was more important than family discord for the development of offspring major depression, whereas family discord was more important than parental depression for the development of substance use disorder in offspring.

2.5.3. Interaction patterns in families with a depressed member

Micro-analytic studies of interaction patterns in families with a depressed member allow for the observation of reciprocal processes as they unfold between several subsystems of the family, and shed light on hypothesized interactional mechanisms which may promote or maintain the development of depressive behavior. Two different models have been hypothesized: 1) Reinforcement of depressive behavior in the family, and 2) Effect of child’s response to interpersonal conflict on the development of depressive symptoms.

2.5.3.1. Reinforcement models of depressive behavior. Social learning theory models of depression hypothesize that depressive behavior may be reinforced within the family (Davis, Sheeber, & Hops, 2000). For example, mothers of depressed adolescents have been found to increase facilitative behavior in response to the adolescent’s depressive behaviors during a problem-solving task, compared to mothers of nondepressed adolescents (Sheeber, Hops, Andrews, Alpert, & Davis, 1998). Further evidence comes from studies showing that depressed affect in one family member can reduce or “suppress” subsequent expression of aggression in other family members. For example, high levels of depression in children have been found to be associated with subsequent low levels of conflict and aggression in family members of both depressed and comorbid depressed/conduct disordered children (Dadds, Sanders, Morrison, & Rebgut, 1992). Dysphoric affect in mothers has been found to suppress the expression of aggressive affect from children or husbands, while aggressive affect from children and fathers suppressed mother’s dysphoric affect (Hops et al., 1987). Thus, aversive behavior (both depressive and aggressive) may be functional in that it decreases the aversive behavior of other family members, and therefore may be reinforced within the family (Biglan, Rothling, Hops, & Sherman, 1989; Hops et al., 1987). In addition, depressed children may receive less reinforcement for positive, nondepressive behavior. For example, mothers of depressed clinic children rewarded their children at much lower rates than mothers of nondepressed clinic children or nonclinical children during a challenging task (Cole & Rehm, 1986). Thus, not only do depressive disorders appear to be reinforced in the family via positive and negative reinforcement processes, depressed children appear to receive lower levels of reinforcement for positive, problem-solving behaviors.
In contrast, some evidence suggests that depressed youth may receive less attention due to their aversive behavior, consistent with Coyne's (1976) interactional theory of depression, which states that depressive behavior is aversive to others and elicits negative interpersonal reactions. For example, Pineda, Cole, and Bruce (2007) found that parents of depressed adolescents were less attentive and reacted more negatively to their adolescents compared to parents of nondepressed adolescents, consistent with Coyne's interactional model rather than the social learning model of depression. However, these two theories are not necessarily incompatible. For example, Biglan et al. (1989) found that while distressed behavior in couples induced negative feelings in the long-term, in the short run, it deterred hostile reactions. In their model, a husband who initially suppresses his aggression towards his depressed wife becomes resentful over time; however, the reinforcing effect of his suppressed anger has already occurred. Furthermore, it may be precisely because depressive behaviors are so aversive that they are so (negatively) reinforcing. If family members reduce their aggressive behavior in reaction to depressive behaviors as a way to escape from them or reduce them in the short run, they may in the long run reinforce them.

2.5.3.2. Response to interparental fighting and aggression and development of depression. The way a child responds to interparental conflict and distress may mediate the relationship between marital conflict and the child’s depressive symptoms (Davis, Hops, Alpert, & Sheeber, 1998). Children are more likely to intervene in interparental conflict in families with high levels of marital conflict, and this has detrimental effects (Cummings, 1994). For example, Davis et al. (1998) found that adolescents became aggressive after involvement in interparental conflict, with differential patterns emerging for boys vs. girls. Boys who responded to their mother’s attack on the father by becoming aggressive to the mother (defense of or alliance with father) were more likely to show increased aggressive behavior one year later (Davis et al., 1998). On the other hand, girls who responded to their father's initiating conflict with their mother by becoming aggressive to their mother (attack of mother; alliance with father) were more likely to display higher levels of aggressive functioning one year later. The relation between response to interparental conflict and later depressive symptoms was weaker than for aggressive symptoms. However, suppression of sadness toward the mother when mother initiated conflict with father was related to an increase in depressive symptoms in a subset of girls. Intervening in reaction to interparental depressive behavior has been found to predict depressive symptoms in adolescents: females who responded to interparental depressive behavior with either facilitative or depressive behavior, or with the suppression of aggression showed increases in depressive symptomatology one year later, whereas for males, both depressive and aggressive responses to interparental depressive behaviors related to increases in depressive symptoms (Davis, Sheeber, Hops, & Tildesley, 2000).

2.5.4. Summary of whole family level

Family-level processes such as cohesion, enmeshment and warmth have been associated with depressive symptoms in both self-report as well as observational studies. Prospectively assessed family environment of very young children has been found to predict internalizing and externalizing symptoms in middle childhood, and supportive family environment in early adolescence predicted lower parent–child conflict during middle adolescence. Several studies have implicated marital conflict as one potential mechanism in the transmission of depression from parent to child; however, one well-designed longitudinal study found stronger evidence for a direct effect of parental depression on child depression. Micro-analytic studies suggest that reinforcement processes may help maintain depressive behavior in children and adolescents. Finally, intervening in interparental aggressive or depressive behavior appears to have negative consequences for adolescents, with intervention in interparental conflict associated with later aggressive behavior, and intervention in interparental depressive behavior associated with later depressive behavior.

2.6. Level 5: Extra-familial context: stress

Bronfenbrenner (1986) argued that developmental research should examine how extra-familial factors may have direct or indirect influences on child and family functioning. Goodman and Gotlib (1999) in their influential review of the effect of maternal depression on child functioning, highlight the overall stressful context of depressed women’s lives as an important level to investigate to elucidate the underlying mechanism through which maternal depression leads to child psychopathology. We therefore briefly discuss this level, as it has implications for research as well as treatment.

Stress has broadly been defined as “environmental events or chronic conditions that objectively threaten the physical and/or psychological health or well-being of individuals of a particular age in a particular society” (Grant et al., 2003). While stress can theoretically occur at any level of a system, it is particularly useful to consider stressful environmental events which impinge upon family functioning. In a comprehensive review of prospective studies, stressful events were found to predict increases in both internalizing and externalizing symptoms in youth in 53 out of 60 studies, with stronger associations found with internalizing symptoms (Grant, Compas, Thurm, McManion, & Gipson, 2004). Effect sizes ranged from 1% to 21%, with an average of 4% for studies testing unique variance accounted for (Grant et al., 2004). Families with depressed mothers are more likely to face stresses such as poverty, single parenthood, or job or financial difficulties (Goodman & Gotlib, 1999). Furthermore, families with a depressed parent tend to be more isolated and less likely to encourage their children to participate in social groups and activities outside the immediate family, which may limit children’s ability to develop social relationships or receive extra-familial support, and which may negatively impact on the separation-individuation process (Cummings & Davies, 1994).

While it is beyond the scope of this article to extensively review the vast literature linking stress to youth depression, a study by Ge et al. (1994) illustrates how stress can impact on family functioning and youth depression, and how models linking stress to particular family subsystem functioning and youth depression can be developed and tested. In cross-sectional data from a community sample of 7th grade students, stressful life events experienced by parents were related to parents’ depressed mood which then was found to disrupt parenting practices (via harsh or inconsistent parenting), which in turn placed adolescents at increased risk for developing depressive symptoms. These results were found with all parent–child combinations: fathers and sons, fathers and daughters, mothers and sons, and mothers and daughters. The relevant point for researchers interested in the development of youth depression is that the larger social context may interact with aspects of the family environment to lead to increased or decreased risk for depression. There are also policy implications of such research: if extra-familial factors such as poverty, social or ethnic identity, language, socio-economic status, school or neighborhood have an impact on child depression, then interventions at this level may be important for preventing or treating depression. Psychologists tend to focus on the individual or family level of intervention, but in some cases, interventions at a broader level may prove to be more effective, and intervention trials should include tests at this level as well.

2.7. Summary of empirically supported family risk factors for youth depression

Family risk factors for youth depression were reviewed at five system levels. At Level 1 (individual parent), parental depression has been associated with increased risk of psychiatric disorders among
<table>
<thead>
<tr>
<th>Level of family system</th>
<th>Family risk factor targeted</th>
<th>Authors</th>
<th>Sample</th>
<th>Target of treatment</th>
<th>Study design</th>
<th>Results (child outcomes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individual parent</td>
<td>Maternal depression³</td>
<td>Billings and Moos (1986); Timko et al. (2002)</td>
<td>249 &lt; 18 year old children of depressed parents already receiving treatment and children of nondepressed parents</td>
<td>Depressed parents</td>
<td>Not randomized; compared children of fully remitted, partially remitted, or no improvement, and children of never depressed parents</td>
<td>At 1 year follow-up, children of remitted depressed parents whose depression remitted functioning better than children of parents who remained depressed but worse than children of never depressed parents; at 4-year follow-up, children of stably remitted comparable to children of never depressed; children of non-remitters functioning most poorly; at 10 year follow-up, offspring of remitted depressed parents comparable to offspring of nonremitted depressed parents, and sig, worse than offspring of never depressed parents</td>
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<td></td>
<td></td>
<td>Byrne et al. (2006)</td>
<td>260 4–16 year old children of dysthymic mothers</td>
<td>Dysthymic mothers</td>
<td>Medication vs. IPT vs. medication + IPT</td>
<td>Children of mothers with ≥ 40% reduction in dysthymic symptoms had fewer internalizing symptoms compared to children of mothers with &lt; 40% symptom reduction 2 years post maternal treatment</td>
</tr>
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<td></td>
<td></td>
<td>Clark et al. (2003)</td>
<td>19 1–24 mo old infants of depressed mothers</td>
<td>Depressed mothers</td>
<td>IPT vs. M-ITG vs. waitlist</td>
<td>Greater child adaptability in the two active conditions compared to waitlist; no treatment effects for children's cognitive and motor development</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Forman et al. (2007)</td>
<td>176 6 mo infants of postpartum depressed mothers</td>
<td>Depressed mothers</td>
<td>IPT vs. waitlist</td>
<td>No association between reductions in maternal postpartum depression and child behavior problems, attachment, emotionality, temperament</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lee and Gotlib (1989, 1991)</td>
<td>75 7–13 year old children of depressed, psychiatric or medically ill mothers (mothers)</td>
<td>Depressed, psychiatric or medically ill mothers</td>
<td>No randomization; child outcomes examined after mothers began treatment</td>
<td>No differences in child internalizing or externalizing symptoms between treatment groups</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Murray et al. (2003)</td>
<td>193 newborns of mothers with postpartum depression</td>
<td>Depressed mothers</td>
<td>Nondirective counseling vs. CBT vs. psychodynamic therapy vs. routine primary care</td>
<td>Some short-term (4.5 and 18 mo) benefit to mother–child relationship and infant behavior problems. No group differences for attachment, or for cognitive or emotional development at 5 years.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Modell et al. (2001)</td>
<td>24 4–15 year old children of depressed mothers</td>
<td>Depressed mothers</td>
<td>Open trial medication</td>
<td>Reduction in maternal depression associated with few child internalizing, externalizing and learning problems</td>
</tr>
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<td>Verdelli et al. (2004)</td>
<td>9 6–18 year old depressed children referred for outpatient treatment with depressed mothers</td>
<td>Depressed mothers</td>
<td>Open trial IPT</td>
<td>Reduction in maternal depression associated with improved child global functioning but not child depressive symptoms</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Verduyn et al. (2003)</td>
<td>119 2–4 year old children of depressed mothers from the community</td>
<td>Depressed mothers</td>
<td>Parent CBT + parenting skills vs. support group vs. no intervention</td>
<td>No group differences on child behavior or maternal depression; children of mothers in CBT group showed pre–post improvement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Weissman et al. (2006)</td>
<td>151 7–17 year old children of depressed mothers</td>
<td>Depressed mothers</td>
<td>Medication treatments</td>
<td>Maternal response to trt correlated with reduction in child symptoms and diagnosis</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Level of family system</th>
<th>Family risk factor targeted</th>
<th>Authors</th>
<th>Sample</th>
<th>Target of treatment</th>
<th>Study design</th>
<th>Results (child outcomes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 — Parent-youth</td>
<td>Support</td>
<td>Mufson et al. (1999)</td>
<td>33 12–18 year old clinic-referred depressed adolescents</td>
<td>Youth</td>
<td>IPT vs. clinical monitoring; 12 weekly sessions</td>
<td>Overall ES: .61 IPT Depression ES: .54 IPT</td>
</tr>
<tr>
<td></td>
<td>Conflict</td>
<td>Mufson et al. (2004)</td>
<td>63 12–18 year old high school students with depression, dysthymia, depression NOS, adjustment disorder w/depressed mood</td>
<td>Youth</td>
<td>IPTA vs. TAU (16 sessions)</td>
<td>.45 IPT .44 IPT</td>
</tr>
<tr>
<td></td>
<td>Autonomy</td>
<td>Rosello and Bernal (1999)</td>
<td>71 13–18 year old clinic clinic-referred depressed adolescents</td>
<td>Youth</td>
<td>IPT vs. CBT vs. WL</td>
<td>.11 CBT .47 IPT .72 IPT</td>
</tr>
<tr>
<td></td>
<td>Communication</td>
<td>Diamond et al. (2002)</td>
<td>32 12–17 year old clinic-referred depressed adolescents</td>
<td>Parents + youth</td>
<td>ABFT (12 weeks) vs. WL (6 weeks)</td>
<td>.68 ABFT .72 ABFT</td>
</tr>
<tr>
<td>Parenting style</td>
<td>Conflict</td>
<td>Brent et al. (1997)</td>
<td>107 13–18 year old depressed adolescents (clinic referred and via advertisement)</td>
<td>Family (SBFT) youth (CBT)</td>
<td>CBT vs. SBFT vs. NST (12 weeks)</td>
<td>.32 CBT .15 SBFT .36 CBT .18 SBFT</td>
</tr>
<tr>
<td></td>
<td>Communication</td>
<td>Treatment of Adolescent Depression Study (TADS; March et al., 2004)</td>
<td>439 12–17 year old depressed adolescents (outpatients)</td>
<td>Youth (CBT, med) parent (psychoed) youth + parent (optional conjoint sessions)</td>
<td>CBT vs. medication vs. CBT + Med vs. placebo (psychoed and parent–youth sessions not separately tested)</td>
<td>.09 CBT −.07 CBT</td>
</tr>
<tr>
<td></td>
<td>Problem solving</td>
<td>Clarke et al. (1999)</td>
<td>123 14–18 year old depressed or dysthymic adolescents</td>
<td>Youth (CWD-A) parent (parent group)</td>
<td>CWD-A (16 sessions) vs. CWD-A (16 sessions) + parent group (8 group sessions) vs. WL</td>
<td>.32 CWD-A .15 CWD-A + parent group −.14 CWD-A + parent group</td>
</tr>
<tr>
<td></td>
<td>High expectations</td>
<td>Lewinsohn et al. (1990)</td>
<td>59 14–18 year old depressed (major or minor) adolescents</td>
<td>Youth (CWD-A) parent (parent group)</td>
<td>CWD-A (14 sessions) vs. CWD-A (14 sessions) + parent group (7 group sessions) vs. WL</td>
<td>.53 CWD-A .73 CWD-A + parent group .68 CWD-A 1.31 CWD-A + parent Group</td>
</tr>
<tr>
<td>Attachment</td>
<td>Pos. reinforcement</td>
<td>Asarnow et al. (2002)</td>
<td>23 4th–6th graders with depressive symptoms</td>
<td>Youth (CBT) youth + parents (MFT)</td>
<td>CBT + MFT vs. WL</td>
<td>.41 CBT + MFT .25 CBT + MFT</td>
</tr>
<tr>
<td>Marital subsystem</td>
<td>Dysfunc. interactions</td>
<td>Cohesion, disengagement</td>
<td>Extr familial</td>
<td>Stressful life events</td>
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<tr>
<td>Sanford et al. (2006)</td>
<td>41 13–18 year old depressed adolescents</td>
<td>Family</td>
<td>TAU vs. TAU + family psychoeducation</td>
<td>.64 (TAU + FP)</td>
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<tr>
<td>Parental warmth/control</td>
<td>Sanford et al. (2006)</td>
<td>107 13–18 year old depressed adolescents</td>
<td>Family (SBFT) youth (CBT)</td>
<td>CBT vs. SBFT vs. NST (12 weeks)</td>
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<tr>
<td>Sanford et al. (2006)</td>
<td>41 13–18 year old depressed adolescents</td>
<td>Family</td>
<td>TAU vs. TAU + family psychoeducation</td>
<td>.505 (TAU + FP)</td>
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<tr>
<td>Sanford et al. (2006)</td>
<td>41 13–18 year old depressed adolescents</td>
<td>Family</td>
<td>TAU vs. TAU + family psychoeducation</td>
<td>.64 (TAU + FP)</td>
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</table>

IPT = Interpersonal Therapy; IPTA = Interpersonal Therapy — Adolescent; M-ITG = Mother–Infant Therapy Group; CBT = Cognitive–Behavioral Therapy; TAU = Treatment As Usual; SBFT = Systematic Behavior Family Therapy; CWD-A = Coping With Depression — Adolescent; MFT = Multiple Family Therapy.  
1Studies can be listed more than once if family factors at multiple system levels are targeted.  
2Unless noted, all studies are RCT’s.  
3Reported in Gunlicks & Weissman, 2008.  
4All effect sizes (ES) for overall outcomes and for depression outcomes from Weisz et al. (2006) unless otherwise noted.  
5Effect sizes for overall and depression outcomes from Sanford et al. (2006), not Weisz et al.
offspring, including depression, anxiety, substance abuse, as well as social, emotional and behavioral problems. Several mechanisms have been proposed to explain this increased risk, including impaired parenting skills, poor social skills, cognitive style of the parent, stressful family environment, and genetic transmission. While support for a causal relationship is strengthened by evidence from several prospective studies, the evidence does not definitively support one causal mechanism over another, and the consensus is that several mechanisms of transmission are likely. The impact of parental depression is not specific for child depression: while it increases risk for offspring depression, it is also linked to other forms of psychopathology, such as anxiety and substance use disorders. Individual characteristics of the child, such as cognitive style, temperament and genetic vulnerability, can increase risk for depression, either as a direct effect or in interaction with adverse environmental factors.

The parent–child subsystem, level 2, has been most investigated in the context of child depression. Evidence from cross-sectional and prospective studies using self-report and observational methods has supported an association between increased risk of depressive symptoms and disorders in youth, and parental rearing style, low parental support, attachment difficulties, difficulty establishing autonomy and parent–youth conflict. Evidence for the role of psychological control and maternal support in predicting depressive symptoms is mixed. Evidence for both unidirectional and bidirectional effects has been found. While some parent–child relationship factors have been specifically linked to depressive or internalizing disorders, studies which examine a wider range of outcomes do not tend to support a specific relationship between these factors and depression.

At Level 3, the marital relationship, cross-sectional, prospective longitudinal and experimental studies have supported a relationship between marital conflict and immediate distress in children and adolescents, as well as later development of internalizing and externalizing disorders. The type of conflict strategy used by couples appears to be important, with hostility and withdrawal associated with more negative child outcomes including internalizing symptoms, and calm discussion, humor, and physical contact associated with more positive outcomes. Experimental evidence suggests that resolution of parental conflict can help ameliorate the harmful effects on children. While marital conflict has been associated with both internalizing and externalizing symptoms, stronger associations have been found with externalizing symptoms.

At Level 4, the whole family system, disengaged as well as enmeshed family patterns have been linked prospectively with increased risk for symptoms of depression, anxiety and ADHD, with disengaged patterns more strongly associated with internalizing symptoms, and enmeshed patterns with ADHD symptoms. Supportive family environment has been prospectively associated with lower parent–adolescent conflict. Several multivariate studies found that when adverse family environment is controlled for, the association between parental depression and youth depression is reduced or eliminated, suggesting that these other family factors may account for the association. Similarly, marital conflict has been found to be more predictive of negative child outcomes than parental depression. However, one longitudinal study found parental depression to be more important in predicting youth depression than family discord. A longitudinal observational study demonstrated negative consequences for adolescents who are involved in interparental conflictual or depressive behaviors, with involvement in interparental conflict more strongly linked to later adolescent aggressive behavior, and involvement in interparental depressive behavior more strongly linked to later depressive behavior.

Finally, at Level 5, the extra-family subsystem, there is evidence from prospective longitudinal studies that stressful events increase risk for youth internalizing or externalizing symptoms, either through main effects or in interaction with other levels of the family system, with stronger effects for internalizing symptoms.

3. Part II. Targeting family risk factors in psychotherapy of youth depression: Evidence from clinical trials

In this section, we turn to the clinical trials research on psychotherapy for youth depression to examine which of the identified family risk factors and mechanisms have been targeted in youth depression trials. We first examine who is the target of treatment (i.e. youth, parent or family), and which risk factors at which system level are targeted. We then briefly describe the main types of psychotherapies tested for youth depression. Finally, we examine the evidence for efficacy of youth treatments for depression at the five system levels.

3.1. Who is the target of treatment?

The majority of treatments for youth depression target the depressed child or adolescent. In a recent meta-analysis of the effects of psychotherapy for youth depression (Weisz et al., 2006), the youth alone was the focus of treatment in 30 out of 35 studies. Among the five studies which treated family members, two studies treated the youth and parents, one study treated the youth primarily but also included one family education group session, and two studies treated the entire family (i.e. youth and family together). An additional study not included in this meta-analysis targeted the entire family (Sanford et al., 2006). Finally, 10 studies targeted depressed parents in order to reduce symptoms of child depression or other psychopathology (Gunlicks & Weissman, 2008).

3.2. Which risk factors are targeted in psychotherapy for youth depression?

The majority of RCT’s for psychotherapy for youth depression target cognitive factors. 31 out of 35 studies in Weisz et al.’s (2006) meta-analysis involved a cognitive change element compared to 12 non-cognitively based treatments. Only 8 out of 35 studies targeted family processes. Of those eight, only five interventions made family or interpersonal relationships the central focus, while three tested CBT interventions plus additional parent or family sessions. Of the five emphasizing relationships, only two actually treated the entire family (Brent et al., 1997; Diamond, Reis, Diamond, Siqueland, & Isaacs, 2002), while the remaining three treated the youth but made interpersonal issues central (Mufson et al., 2004; Mufson, Weissman, Moreau, & Garfinkel, 1999; Rossello & Bernal, 1999). One major CBT trial included an optional family component that was not separately tested (March et al., 2004).

3.3. Which family risk factors are targeted?

Among the few studies which address family risk factors, which of the empirically supported risk factors have been targeted? Table 2 compares risk factors identified in Part I with the risk factors targeted among the few psychotherapy trials which have addressed family factors. The majority of studies have targeted risk factors of the individual parent (Level 1), or of the parent–child relationship (Level 2). At Level 1, ten studies tested the effect of treating maternal depression on child depression and psychopathology (Gunlicks & Weissman, 2008). Ten RCT’s of psychotherapy for youth depression targeted parent–child relationship factors (Level 2), such as conflict resolution, improving communication, and parental support. No studies targeted marital conflict (Level 3) to reduce youth depression. Three studies targeted the entire family (Level 4). Finally, four studies targeted stressful life events (Level 5).

3.4. What kinds of treatments have been tested in RCT’s for youth depression?

CBT. By far the most commonly used psychotherapy for youth depression is cognitive–behavioral therapy (CBT). A recent meta-
analysis of psychotherapy trials for youth depression classified 31 out of 35 studies as involving a cognitive change emphasis (Weisz et al., 2006). Although many different variations of CBT for youth depression have been developed, most include two main components: cognitive restructuring and behavioral activation. Two studies also evaluated the benefit of adding a parent group to the youth CBT (Coping with Depression) compared to youth CBT alone (Clarke, Rohde, Lewinsohn, Hops, & Seeley, 1999; Lewinsohn, Clarke, Hops, & Andrews, 1990). The parent group targets the interpersonal factors related to depression, by increasing parental support of youth, and reducing negative interactions and conflict between parent and teens. Parents are taught communication and conflict resolution skills which they role play with each other; in the last two sessions, they are joined with their adolescents to practice these skills together. A CBT treatment for prepubertal children, Stress Busters (Asarnow, Scott, & Mintz, 2002) included one multiple family group session at the end of the treatment which focused on increasing parental support for child competency; however, this was not separately tested.

**Interpersonal Psychotherapy (IPT):** Three studies tested Interpersonal Therapy for Depression, adolescent version (Mufson et al., 2004; Mufson et al., 1999; Rossello & Bernal, 1999), an individually-based psychotherapy which targets the interpersonal difficulties of adolescents identified in epidemiological and clinical research. Depressive symptoms are assumed to be related to interpersonal conflicts; thus resolution of interpersonal conflict and development of positive interpersonal relationships is the presumed mechanism of recovery. The treatment focuses on developmental issues common to adolescents with depression, such as separation and authority issues with parents, interpersonal disputes, role transitions, loss, peer pressure, single-parent families, and stressful life events.

**Family therapy for depression.** Three RCT’s tested some form of family therapy, and one included an optional family therapy component which was not separately tested for efficacy.

**Systemic Behavior Family Therapy.** Systemic Behavior Family Therapy (SBFT) was tested against CBT and Supportive Psychotherapy in one study (Brent et al., 1997). SBFT combines techniques from functional family therapy (Alexander & Parsons, 1982) and from problem-solving therapy (Robin & Foster, 1989), and includes techniques such as reframing problematic behaviors, changing dysfunctional family interactional patterns, improving communication and problem-solving skills, improving parenting skills and sensitivity to developmental issues.

**Attachment Based Family Therapy (ABFT).** Attachment Based Family Therapy (Diamond et al., 2002) stresses the role of attachment problems in the development of adolescent depression, and addresses several family risk factors which have been empirically linked to youth depression: parental criticism, hostility and disengagement, parental stress, ineffective parenting, parent–adolescent conflict and support, and autonomy development. The goal of ABFT is to strengthen the attachment relationship between parent and adolescent, by helping the adolescent openly address longstanding conflicts with parents which have strained the attachment relationship, and by developing appropriate autonomy from parents.

**Family psychoeducation.** One study compared family psychoeducation plus treatment as usual to treatment as usual for adolescent depression (Sanford et al., 2006). Twelve 90 minute sessions were conducted with all family members in the home, with one booster session at 3 month follow-up. The goals of the sessions were to increase knowledge about depression and its effect on the family, strengthen family communication and reduce expressed emotion, and to enhance coping, problem solving and crisis-management.

**Treatment of Adolescent Depression Study (TADS) parent component.** The Treatment of Adolescent Depression Study (TADS) (March et al., 2004) which compared the efficacy of medication to CBT alone and CBT combined with medication, included a parent component which was not separately tested but was considered essential to treatment retention (Wells & Albano, 2005). The parent component included two mandatory sessions of parent psychoeducation, as well as optional parent–youth sessions which could be drawn from five family therapy modules which addressed 1) family problem-solving skills; warmth and hostility 2) family communication skills, 3) behavioral contingency schedules 4) positive reinforcement, and 5) parent–adolescent attachment.

3.5. **Efficacy of interventions targeting family factors.**

How effective are interventions that primarily target family and interpersonal relationships? Table 2 displays results from treatment studies targeting family factors in order to improve youth depression, including ten studies which treated maternal depression to reduce child psychopathology. Effect sizes from Weisz et al.’s (2006) meta-analysis are included. Unfortunately, the small number of studies makes it difficult to draw firm conclusions. Weisz et al.’s (2006) meta-analysis of psychotherapy of youth depression found an average effect size of .34 for all treatments, and cognitively based interventions fared no better than non-cognitive interventions. Effect sizes for family or parent-based treatments of youth depression varied widely, from .14 to 1.31 for child CBT + parent group, from .18 to .72 for family therapy, and from .44 to .72 for Interpersonal Therapy (IPT). Since many factors independent of type of treatment can influence effect size, it is difficult to directly compare effect sizes. When family or parenting interventions have been compared to individual, cognitively based interventions, the findings have been mixed. In the only randomized controlled trial which directly compared CBT to family therapy, Brent’s group found cognitive–behavioral therapy (CBT) to be more effective in the short term than systematic behavioral family therapy (SBFT), however, at two year follow-up the treatment effects were the same. Furthermore, SBFT was found to reduce parent–child conflict, which was a predictor of relapse. Rossello and Bernal (1999) found larger effect sizes for IPT compared to CBT; however these differences were not significant. Among the cognitive–behavioral trials, Lewinsohn’s group found evidence for a marginally significant increase in effect by adding a parent group to a youth CBT group (Lewinsohn et al., 1990); however a later study failed to replicate this finding (Clarke et al., 1999).

How effective are treatments that target a depressed parent (Level 1) at reducing symptoms of child depression or other psychopathology? In their review, Gunlicks and Weissman (2008) found evidence that reduction or remission of parental depressive symptoms was associated with reduction in child symptoms. Specifically, five out of nine studies found that treatment of parental depression was associated with improvements in child psychopathology, and five out of the six studies examining psychosocial outcomes reported that mothers’ treatment was associated with child improvement, including better academic functioning, better global functioning, better mother–child relationship. In contrast, they found little evidence that treating postpartum depression has significant positive effects on infant attachment, temperament, and cognitive development.

3.6. **Summary of family risk factors targeted in youth depression treatment.**

In summary, the majority of tested treatments for youth depression do not target family factors or involve family members in the treatment. Among those that do, the parent–child relationship (Level 2), is the main focus, particularly conflict resolution, problem-solving skills, communication and support, attachment, increasing warmth and decreasing hostility, and increasing positive reinforcement of the adolescent. Marital conflict (Level 3) has not been targeted in RCT’s for youth depression, and whole family relations (Level 4) and stressful life events (Level 5) are targeted in only a few studies. In a promising trend, investigators have begun to target parental depression (Level 1)
as a means to reduce or limit child psychopathology, with positive results. Family-based treatments as well as individual treatments targeting family issues show clear promise in terms of efficacy compared to waitlist, with effect sizes ranging from small to large. However, the data are inconclusive with regard to relative efficacy of targeting family factors vs. cognitive or behavioral factors, or the relative efficacy of targeting the individual adolescent vs. including other family members. Comparing the existing treatments which target family factors, IPTA targets a broad range of family factors, but involves only the youth, which may limit efficacy. ABFT demonstrates a strong effect size and targets the largest number of family factors, but its primary focus on parent–youth attachment may not be appropriate for all families. Lewinsohn and Clarke's Coping With Depression group targets parent–youth conflict, but provides minimal direct practice between parent and teen, which may limit efficacy. Finally, the TADS study demonstrates the feasibility of targeting multiple family risk factors using flexible modules, integrated with standard individual CBT; however, the lack of efficacy data is a limitation.

4. Part III. Road map for the development of empirically supported family treatments for depression

In this section, we discuss recommendations for applying the empirical findings on family factors in depression to family treatment for depression. We will address two questions: 1) Which family risk factors should be targeted? 2) What are the steps needed to empirically validate family-based treatments?

4.1. Which family factors should be targeted?

The evidence reviewed demonstrates that family factors at all levels of the family system play a role in youth depression. These family risk factors are highly intercorrelated, and although several different models have been tested to identify causal risk processes, there is insufficient evidence to support one model over another, or to determine where in the process from family risk to disorder to intervene. Furthermore, different family factors may be differentially related to depressive symptoms. A meta-analysis examining which of these factors has the strongest relationship to depressive symptoms and changes in depressive symptoms would help identify potential targets. There is also evidence for bidirectional pathways between risk factors at the different system level (e.g. between parent factors and child factors), making it difficult to determine causal priority, or where to intervene. RCT's can help identify potential targets of treatment by systematically varying the target (e.g. parent, child family) as well as by testing hypothesized mechanisms related to outcome. For example, since ABFT is presumed to operate by improving parent–youth attachment, demonstrating that changes in attachment mediate treatment outcome would provide empirical evidence for targeting the parent–youth relationship.

Component profile analysis, an approach used to identify specific techniques employed in effective treatments, can also be used to identify potential targets for treatment (McCarty & Weisz, 2007). Applying this method to youth depression treatments which target family factors, four studies demonstrated the effect size criteria of .5 or greater: ABFT (ES = .72; Diamond et al., 2002), IPTA (ES = .54; Mufson et al), CBT plus parenting training (ES = 1.31; Lewinsohn et al., 1990); and psychoeducation plus treatment as usual (ES = .64; Sanford et al., 2006). Communication and problem solving were targeted in all four studies; parent–youth conflict and parental support in three out of four studies, parental warmth and control, autonomy and expressed emotion in two out of four studies, and attachment and cohesion/disengagement in one out of four studies. Thus communication, problem solving, parent–youth conflict and parental support may be key elements to target in family-based treatments.

4.2. Steps in the development and validation of family-based interventions for youth depression

Validation of new psychotherapeutic treatments proceeds in a series of standard phases including 1) model development, 2) manual development, 3) efficacy testing, 4) effectiveness and implementation, and 5) testing moderation and mediation hypotheses. We first summarize recommendations based on this review for model and manual development. We then examine to what extent family-based interventions have been validated at each of these phases.

4.2.1. Developing a model

We have argued that a systemic framework can help facilitate the translation of empirically supported family risk factors to interventions for youth depression, by integrating risk factor evidence at multiple system levels into hypothesized risk mechanisms which can be tested in intervention research. We propose an integrative rather than competitive model: each of the “competing” theories of depression can be seen as operating at different levels of analysis. Determining the most effective level for intervention becomes an empirical rather than theoretical question. Furthermore, a systemic approach allows for the assessment of multiple risk factors at multiple levels, from the individual, to the parent–youth dyad, to the family, to the broader school and social environment. Not every family with a depressed youth will present with the same constellation of risk factors, and a systemic approach allows for flexibility in tailoring interventions to the needs of specific families.

Several excellent models of development of depression in the family context have already been proposed by developmental and family researchers (Cummings et al., 2005; Davis et al., 2000; Sheeber et al., 2001) and by adult depression researchers (Gotlib, 1990). These models, while differing in specifics, each propose that depressive behavior develops in the context of family, interpersonal, and social contingencies in interaction with genetic and cognitive propensities of the child. While we do not dispute the importance of individual processes in the development of depression, we focus on family-level processes for several reasons. First, since both genetic and environmental vulnerability may be required for the development of depression (Eley et al., 2004), addressing adverse environmental factors may be one way to prevent the development of depression. Second, cognitive and interpersonal theories of depression place strong emphasis on the early parent–child relationship in the development of vulnerability to depression (Beck, 1967). Intervening directly in this interpersonal process could potentially prevent the development of this vulnerability, or could help change maladaptive relationship patterns which maintain depressive symptoms. Finally, we emphasize family-level interventions because the application of the empirical evidence on family risk factors for depression has lagged far behind application of individual cognitive and interpersonal models of depression (Sander & McCarty, 2005). It should be noted however that individual, cognitive or interpersonal approaches can be integrated in this model at the individual level of analysis.

From a clinical perspective, the family level may be the most efficient point of entry into the family system as well. Depressed adolescents are living within a family context, and the evidence strongly supports the likelihood of problematic family relationships. Depressed adolescents are usually referred to professionals by parents who want to help, but may not know how. Beginning treatment with the entire family may improve retention rates, by providing parents with much needed support (e.g. Wells & Albano, 2005). Furthermore, by beginning at the level of the whole family, the clinician can assess the adolescent's individual vulnerability in the specific context of his or her family, which may help identify features of his or her environment that help maintain the depression. With this broader view of the adolescent in his family context, the therapist can easily
move down to the level of individual cognitive vulnerability (e.g. by recommending CBT), or up to the level of school environment (by intervening with school staff) to address problems occurring at multiple levels.

4.2.2. Developing a manual

Several manualized family therapy protocols are available for youth depression: Diamond’s Attachment Based Family Therapy (Diamond et al., 2002), the TADS family modules (Wells & Albano, 2005), and Brent and colleagues’ Systemic Behavioral Family Therapy (Brent et al., 1997). Lewinsohn and Clarke’s parenting intervention, while targeting the parental subsystem rather than the entire family, provides a good example of a highly structured and detailed program targeting depressed youth and their parents.

4.2.3. Evidence for validation of family-based interventions for youth depression

Table 3 shows studies which have been conducted to validate family therapy for depression at each phase. With the exception of Phase I, treatment development, there has been little empirical research at the other phases. At Phase II, randomized controlled trials, there have been only two trials: one trial comparing ABFT to waitlist control (Diamond et al., 2002), and one randomized controlled clinical trial against a known effective treatment, CBT (Brent et al., 1997).

There have been no studies comparing family therapy to treatment as usual, or to medication. In Phase III, assessing the additive benefit of family therapy, three RCT’s have been conducted (Lewinsohn et al., 1990; Clarke et al., 1999; Sanford et al., 2006). Further questions such as whether family therapy can improve efficacy in combination with IPT or with medication have not been conducted. Only one study has examined moderation effects (Phase IV) — that is, whether there are differential treatment effects for specific subpopulations (e.g. youth with comorbid conditions, treatment-resistant depression, or adverse family environments). Brent’s group examined relative efficacy for adolescents with comorbid anxiety compared to those with depression only, and for clinic referred compared to non-clinic referred (Brent et al., 1998). Finally, only one study (Kolko, Brent, Baugher, Bridge, & Birmaher, 2000) examined mediation effects (Phase V); that is, whether the hypothesized therapy mechanisms mediate change after psychotherapy. Finally, dismantling studies (Phase VI), or studies which test the relative efficacy of specific components of family treatments, have not yet been conducted.

4.2.4. Further research directions

Further evidence is needed to build an empirical basis for determining which family risk factors or processes to target in treatment of youth depression. One relatively simple approach would be to conduct a meta-analysis to determine the relative strength of the relationship between various family factors and depressive symptoms and disorders. Since this may vary with the age and gender of the child, moderation effects need to be included. Component profiling analysis is also recommended to identify the components of effective family treatments; however, we first need to have a larger evidence base of family treatment studies (McCarty & Weisz, 2007). Most importantly, we need more family intervention studies for youth depression to test efficacy of family therapy, and to begin to fill in the blank spaces in Table 3 to go beyond the basic question of whether family therapy works, to examine the relative efficacy of family treatments compared to other bona fide treatments, to examine what combination of treatments is most effective for which subgroups of patients, to evaluate whether the hypothesized mechanisms of change actually mediate therapeutic change, and to identify the essential components of treatment. These studies also have the potential to answer questions about the mechanisms of change after psychotherapy, and to provide evidence for causal risk models for the development of depression (Cowan & Cowan, 2002). For example, the hypothesis that family therapy decreases youth depression by reducing dysfunctional parent–adolescent conflict can be tested in an RCT by examining whether parent–adolescent conflict mediates improvement in depressive symptoms. If confirmed, this finding would lend support to the hypothesis that parent–adolescent conflict leads to or maintains depression, which in turn can be tested in basic developmental research into depression. Thus, family therapy intervention research has the potential to improve evidence-based treatment of youth depression, and to further our basic knowledge of the development of depression within the family.

5. Discussion

The results of this review comparing family risk factors for youth depression to current empirically evaluated treatments for youth depression suggest indeed that there is a significant gap in the translation of basic research findings to treatment studies. Put simply, we know more than enough about family risk factors and possible underlying mechanisms to begin to apply this information to treatment. Review after review article has documented this evidence, and has recommended the application of these findings to treatment. To date, this has occurred to only a limited degree. This is all the more striking given the lack of evidence that CBT is more effective than other forms of treatment for youth depression, and lack of evidence that cognitive processes mediate changes over the course of psychotherapy for youth depression (Kolko et al., 2000; Spielmans, Pasek, & McFall, 2007; Weersing & Weisz, 2002). Furthermore, the relatively modest effect size for cognitive-based therapies for youth depression compared to treatment of other youth disorders suggests the need to enhance existing treatments by targeting other risk factors.
5.1. Why have the findings from family process research gone largely unheeded in clinical trials for youth depression?

If the preference for individual, cognitive-based approaches is not supported by the empirical evidence, on the one hand, and if there is strong evidence that targeting family factors could improve treatment of youth depression on the other, why has this been largely neglected in the treatment literature? One explanation is that clinical applications always lag behind basic research findings. Intervention researchers may be less aware of the evidence from developmental and family process research. Furthermore, whereas cognitive therapy researchers have highlighted the importance of empirically validating, family therapy proponents have been less concerned with empirically demonstrating treatment efficacy or confirming hypothesized models. However, the very success of the cognitive paradigm may also be a factor. Scientific paradigms serve to define the concepts and data for inclusion in a particular field, as well as defining what lies outside the field (Kuhn, 1962). The cognitive model has been tremendously influential in the last forty years in organizing the selection and interpretation of data about depression. During roughly the same time period, evidence supporting family factors in depression has been accumulating. However, this evidence has not been as systematically applied to treatment of youth depression. More than a dozen recent reviews of family factors in depression have called for the application of these findings to treatment of youth depression (e.g., Alloy et al., 2006; Goodman & Gotlib, 1999; Sander & McCarty, 2005; Sheeber et al., 2001). At the same time, recent articles from experts within the field of youth depression have questioned the evidence for the superiority of cognitive therapy for youth depression compared to other therapies (Weersing & Brent, 2006; Weisz, Iyengar, Kolko, Birmaher, & Brent, 2006; Weisz et al., 2006). Thus the field appears to be poised to integrate and apply these findings which have been available for several decades. There are promising signs that this integration is beginning. There are manualized treatments available to address the interpersonal and family relationships associated with youth depression; however they need to be integrated into a developmental and family framework, and they need to be tested systematically. The fact that TADS included a comprehensive family component as part of the first study comparing CBT to medication indicates the degree to which CBT researchers have incorporated concerns about family processes into their study designs. Interventions targeting parental depression as a means of reducing child psychopathology represent another promising research trend. And finally, the fact that leading CBT researchers are taking a critical look at the accumulated data on CBT for youth depression is a welcome sign, which will improve the family evidence within a systemic framework, and they need to be tested systematically. We hope that this review, by organizing treatments for youth depression. We hope that this review, by organizing future research on psychotherapy of adolescent depression should focus on which combination of interventions is most helpful for which individuals and their families.

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References


