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Parental Expressed Emotion and Youth Psychopathology: New Directions for an Old Construct

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Abstract

Levels of parental expressed emotion (EE) are prospectively associated with the symptomatic course of a range of childhood psychiatric disorders. This article reviews the literature linking parental EE to youth psychopathology and proposes a novel framework for understanding its mechanisms of action. We find that, despite noteworthy methodological limitations, parental EE is linked consistently to a more deleterious course of mood, anxiety, and psychotic disorders in youth. Its mechanism of action is unknown. Models of "toxic family stress" (referring to frequent, sustained, and uncontrollable stress without protective influences) provide one framework for understanding how high EE environments interact with individual biological vulnerabilities to promote illness onset and recurrence. Research aimed at understanding biological responses (e.g., stress reactivity, arousal) to familial EE is needed. Such work may inform efforts to understand how EE affects the course of psychiatric disorders and may guide the development of novel interventions emphasizing emotion regulation strategies.

Keywords

Expressed emotion; Family dynamics; Child and adolescent psychopathology

For nearly five decades, expressed emotion (EE) has been studied as an aspect of family functioning that portends poor outcome for adults with mental illness [1–3]. EE is defined by attitudes of high criticism, hostility, and/or emotional over-involvement in the caregivers of a patient with a psychiatric disorder, as measured during a semi-structured interview or speech sample. Across the full spectrum of psychiatric disorders, high levels of EE have been linked to poorer clinical course, diminished treatment response, and higher rates of relapse. Indeed, it is one of the most robust predictors of long-term outcome in the adult psychopathology literature [4, 5]. High parental EE is associated with a wide range of mental and physical health conditions in pediatric populations, [6–8] and it has been found to predict clinical course and treatment response for depressive, anxiety, and bipolar disorders [9–12]. In addition, it has been associated with early temperamental abnormalities [13], self-injurious behavior, [14] and higher levels of externalizing comorbidity among

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youth with intellectual disabilities [15–17]. Thus, it is a risk factor of interest in several forms of youth psychopathology.

Despite its potential role in the onset and course of psychiatric disorders, our understanding of how parental high-EE attitudes emerge in the context of youth mental illness is limited. Further, mechanistic models of how EE attitudes interact with the unfolding of childhood psycho-pathology are lacking. These gaps in understanding are problematic in that they undermine efforts to intervene effectively with maladaptive family dynamics. Although EE is thought to complicate several forms of psychopathology, few interventions directly targeting the emotional dynamics and associated interaction patterns of high-EE families have been articulated. Of those that have, success has been relatively limited. In this review, we discuss the literature linking EE to child and adolescent psychopathology, highlighting emerging findings that may advance our understanding of family processes and mental illness. We propose a novel framework for studying the effects of parental EE on youth psychopathology and discuss how this perspective may guide new family-based interventions.

The framework for this review is guided by two assumptions: first, that parental EE predicts the course of child psychiatric illness, even if it is not a direct causal agent in psychopathology. Second, that greater emphasis on understanding its mechanisms of action is needed. To that end, we suggest that efforts to understand EE's prognostic power borrow from innovative models of "toxic family stress". These models draw on findings from multiple disciplines to explain how dysfunctional family climates influence youth well-being at biological, neurological, and psychological levels, with lasting adverse consequences. They suggest one potential mechanism—heightened stress reactivity—by which EE may be linked to poor outcomes among youth with psychiatric illness. The effects of emotionally toxic environments, we suggest, are not limited to increased risk of illness occurrence, but to poorer course and treatment response once onset has occurred.

The Effects of Stressful Home Environments

All children experience some type of stress in the home environment at one time or another, and for most youth, these experiences serve the important function of helping them learn to regulate emotions and manage their behavioral responses effectively. Exposure to stress prompts a number of physiological responses including increases in heart rate, respiration, and cortisol production. In healthy family systems, when youngsters experience stress, the presence of supportive caregivers cushions the impact of biological responses and facilitates the child's return to baseline [18]. When supportive relationships are lacking, however, stress response systems are weakened, children become hyper-vigilant for potential threat, and brain functioning is compromised [19, 20].

Toxic family stress is a term used to describe a particular type of stress exposure that is frequent, sustained, and uncontrollable, and which occurs in the absence of buffering protective factors. Converging findings from the fields of neuroscience, behavioral genetics, and pediatrics suggest that when youth are exposed to this type of stress—frequently associated with abuse, neglect, and parental psychopathology—they exhibit heightened

stress responses (e.g., hypercortisolemia) that interfere with functioning at physiological, neural, emotional, and behavioral levels. In particular, sustained activation of stress response systems is thought to disrupt the development of neural circuitry, and, in extreme cases, may cause neural death or interfere with neurogenesis [21]. Even in less acute situations, frequent exposure to familial stress may be harmful inasmuch as it lowers the threshold for triggering stress responses among high-risk children [22].

In the context of child and adolescent mental health research, models of toxic family stress have important implications for understanding how family environmental factors interact with genetic predispositions to elicit and maintain mental illness. First, viewed through the lens of diathesis/stress models, environments that foster a lower threshold for stress responding may leave youth vulnerable to the onset of psychiatric symptoms. Second, the emergence of mental illness—especially if it is recurrent and significantly impairing—is itself a significant stressor for youth, and family responses may either buffer the effects of this challenge or amplify them. Third, the family climate may become a risk factor for illness recurrence if parents' responses to the early signs of mental illness are mal-adaptive or counterproductive. Finally, as we discuss below, sustained exposure to stressful home environments may affect the child's response to treatment.

EE as a Form of Toxic Family Stress

EE is a measure of how families respond to an episode of psychiatric disorder, and it has enjoyed a long and august history since it first emerged in the 1960s. At that time, researchers sought to identify family dynamics associated with relapse among adult patients with schizophrenia who were discharged from the hospital to the homes of their parents. They found markedly higher rates of relapse among patients who returned to homes that were high in EE (i.e., highly critical, hostile, or overprotective) as determined during a clinical interview with parents called the *Camberwell Family Interview* [1–3]. This interview and other measurement protocols ask relatives to describe the development of the patient's illness and its impact on the family, especially in the prior 3 months. Responses are then coded for tone and content including descriptions of the relationship, critical remarks, emotional displays, and evidence of extremely self-sacrificing or overprotective behavior. These features form the basis for the two primary dimensions of the EE measure: criticism and emotional overinvolvement. Criticism captures blame, dislike or resentment that parents may feel toward an ill offspring. Emotional overinvolvement reflects attitudes of over-protectiveness, marked over concern, inordinately self-sacrificing behaviors, or exaggerated emotional responses regarding the child. A third EE dimension, hostility, has been largely dropped from EE research because of its high correlation with levels of criticism.

There are several features of the EE construct that have contributed to its impressive longevity in a field that abounds with other parent and family measures. First, EE is distinct from other measures of family functioning in that it is not focused on beliefs about parenting, global levels of stress, or parenting practices. Rather, EE examines the attitudes and emotions expressed by caregivers about the patient, so that its emphasis is on dyadic relationships. This focus is meant to provide a glimpse into day-to-day family life, including aversive patterns of interaction between parent and child that may be highly stressful to

both. Despite claims to the contrary [23] this approach does not place blame on parents or ascribe them a causal role in the child's mental illness. Although such causal perspectives once dominated thinking about the role of the family in psychiatric disorder (for review, see Strachan et al. [24]), current conceptualizations of EE emphasize the bi-directional nature of negative affective exchanges between family members. They view the emergence of illness as a salient developmental event for parent and child alike, and one that alters relationships and patterns of family interaction. Thus, EE focuses on how families respond to and reorganize around episodes of mental illness with the idea that these patterns have implications for recovery and subsequent recurrence [25]. With this in mind, parental EE may be an outcome of child mental illness but also a stressor that interacts with the child's biological vulnerability.

From the perspective of toxic family stress, the EE construct is of interest as it may represent a maladaptive pattern of responding to psychiatric illness that is physiologically arousing for children and occurs in the absence of buffering factors (e.g., parental warmth). That is, when facing the stress of emerging illness, parents who respond with criticism, blame, or excessive control (i.e., overprotectiveness) may create emotionally charged environments that exacerbate symptoms, which in turn may fuel negative parent/child interactions and interfere with healthy behavior management strategies.

EE as a Prognostic Indicator

Over the past 20 years, EE has been examined as a correlate of psychological adjustment among diverse groups of youth, including those from community, clinic, and inpatient settings, and across a broad range of psychiatric and medical conditions (see Table 1). It has been examined across the full developmental spectrum, with studies documenting its correlates among toddlers [26], preschoolers [27], school age children, [28, 29] and adolescents [14, 30]. The most rigorous studies are those that have examined EE as a potential prognostic indicator. These studies suggest that EE is stable over periods of up to 2 years [27] and predicts poor clinical outcomes over time. For example, in a community sample of youth, maternal EE ratings taken at a preschool baseline predicted subsequent diagnoses of ADHD in 3rd grade [27]. A study of identical twin pairs found that maternal EE predicted subsequent antisocial behavior after controlling for shared environmental effects [32]. In mood disorders, maternal levels of EE predicted the onset of depression in high risk and clinical samples [33]. EE is also linked to the persistence of mood symptoms over time in major depressive disorder [9] and bipolar disorder [30].

Levels of EE may influence treatment outcomes. Among adolescents with eating disorders, maternal criticism accounted for 28–34 % of the variance in treatment outcome [34] and was a more robust correlate of outcome than diagnosis, length of illness, or body weight. Parental EE also predicted treatment response [10] and functional outcomes [11] among youth receiving exposure-based treatment for OCD. Among adolescents with bipolar disorder, EE moderated the effects of psychosocial treatment, such that youth whose parents were high-EE at baseline showed greater improvement in depression and mania scores in family-focused treatment compared to youth in a brief treatment control. These gains lasted up to 2 years following the conclusion of treatment [35]. In a sample of high-risk youth with

depression or sub-threshold bipolar disorder, early family intervention produced the largest symptomatic improvements in youth with high-EE parents [36].

Notably, although high levels of EE are found more often among youth with psychiatric and behavioral difficulties, they are not always associated with the acuity of these problems. Several studies with carefully characterized samples of youth meeting criteria for an Axis I disorder do not find links between parental EE and the concurrent severity of the child's mental illness [10, 37]. Reviews of the adult literature find no cross-sectional associations between EE and symptom severity [38]. Indeed, the association between EE and prospective outcomes—but not concurrent symptomatic states—suggests that EE may moderate the course of childhood psychiatric illnesses. In turn, this makes family criticism and emotional overinvolvement promising targets for interventions aimed at altering relationship patterns that interfere with treatment.

Limitations of EE Research

Although EE has a strong record as a prognostic indicator, several limitations of existing studies merit consideration. First, there is substantial variability in rates of high EE across studies (Table 1). Thus, it is difficult to know whether EE is a proxy for normative family distress when dealing with an illness or a specific maladaptive pattern of responding to particular forms of psychopathology in youth. Second, there has been little consideration of how current measures of parental EE should be modified for younger age groups. Although the broad concepts of criticism and emotional overinvolvement have relevance and intuitive appeal for child and adolescent patients, complications arise when using measurement tools developed for adults with children. Finally, although parental EE is linked to elevated rates of various forms of youth psychopathology, research aimed at understanding the mechanisms by which these attitudes predict poor outcomes across disorders is scant. In the sections that follow, we elaborate on these issues and their implications for research and clinical practice.

Measurement of High-EE Attitudes

The rate of high EE in families of healthy youth (as measured with Five-Minute Speech Samples) is between 20 and 40 % [6, 8, 39–42]. Still greater variability is observed across different Axis I disorders with reports ranging from 29 % in ADHD samples [43] to 51 % in studies of depressed youth [6].This spread is also evident across different investigations of the same disorder. For example, Hibbs et al. [28] found that 82 % of youth with OCD had a parent who was high in EE whereas Peris et al. [10] reported rates of 55 % in a treatment-seeking sample of youngsters with OCD. Although due, in part, to sampling differences (e.g., inpatient versus outpatient samples), this level of variability within the same disorder is striking.

The variability may be due to differences in how EE is measured, scored, and reported across studies. Newer studies tend to rely on the Five Minute Speech Sample (FMSS [44]), a protocol that has been found to identify high-EE attitudes less frequently compared to the original CFI [45]. In addition, some studies have used EE scoring procedures for which reliability and validity have not been established [33, 46–48]. Other research groups have

dissected the FMSS measure such that they report on individual coding elements (e.g., relationship dissatisfaction) in relation to key outcomes without reporting on high/ low EE classifications at all, or adjust the scoring procedures to include “borderline criticisms” in the high-EE category. Overall rates of high-EE are presented in only a fraction of studies. Thus, an important step for advancing research on EE and youth psychopathology resides with establishing a clear set of reporting conventions including adherence to validated scoring procedures and consistent reporting of rates of high EE.

Developmental Considerations

A second shortcoming of much of the current pediatric EE research is the limited recognition of developmental issues that may arise when applying the construct to younger populations. This is important given that EE was developed as a way to capture dysfunctional dynamics in the context of adult relationships. The tendency to attribute blame for undesirable behaviors or to describe self-sacrificing or overprotective behaviors is likely to differ when parents speak about child and adolescent patients. Some evidence suggests that the criteria used to determine EE status—namely subgroup ratings of criticism and emotional over-involvement—may not fit neatly for children [25, 49, 50].

Although the adult literature shows linkages between both the criticism and emotional overinvolvement dimensions of EE and the patient’s functioning [51], the emotional overinvolvement dimension proves much more problematic for pediatric populations. Indeed, parental criticism and overinvolvement scores are frequently unrelated to each other in pediatric populations [7] and the majority of studies find that high EE classifications are mainly due to parents’ scores on the criticism dimension [8, 14, 27–30]. Although early research suggested that criticism was linked to externalizing disorders and emotional overinvolvement to internalizing disorders, the bulk of the evidence suggests that overinvolvement does not relate to child and adolescent mental health outcomes at all [26, 49, 50]. Overinvolved behaviors often described in the adult schizophrenia literature—such as parents who become consumed with whether their adult offspring has showered and is eating properly—may not reflect inappropriate behaviors among parents of children or teens, especially those whose level of functioning is compromised by psychiatric illness. In particular, behaviors that may undermine the autonomy of adult patients–such as assisting with daily living skills or chores or intervening in social disputes–may not reflect the same level of enmeshment for young children, and, in some cases may be adaptive and necessary.

A related question pertains to how far downward the EE construct can be extended. EE has been studied reliably in toddlers and preschoolers with evidence for its stability over time and links to developmentally relevant variables such as attachment and temperament [26, 27, 46, 52]. However, some of the criteria used to make high-EE designations appear to have little to no utility in younger age groups, and they are particularly worrisome for research attempting to link EE to infant wellbeing [53]. For example, one aspect of EE coding on the FMSS involves indicating whether the parent provided excessive detail about the past during the 5-min narrative, a feature thought to reflect an inappropriate level of involvement with the patient. Within the context of the original scoring system (developed with adult patients in mind), this distinction is based on the respondent’s detailed accounts of the patient’s early

childhood or infancy. Yet, by definition, parents of small children have less “past” to talk about, and many innocuous statements or adaptive behaviors are likely to be coded as evidence for high EE.

The Origins of EE Attitudes

The current evidence suggests that high EE attitudes are (1) a common (albeit variable) correlate of youth psychiatric disorders, (2) relatively stable over time [15, 27, 31], and (3) linked in some cases to poor clinical outcomes. An important question then—particularly for those interested in intervention and prevention—is how high EE attitudes emerge in response to childhood psychiatric illness. Research with adult patients indicates that length of illness plays a role in high EE attitudes, such that parents express more criticism toward patients with longer histories of illness [54]. Given that many forms of mental illness have their origins in childhood and that the experience of illness is relatively new to pediatric patients and their families, studies of EE in pediatric samples may shed light on how unfolding mental illness elicits maladaptive response patterns in families. Pediatric EE studies may also guide treatment development as they offer specific avenues of intervention for families who respond to psychiatric illness with excessive levels of negative affectivity or other mal-adaptive behaviors.

In the adult literature, models of EE posit that the dysfunctional family dynamics stem from bi-directional interactions in which patient characteristics (i.e., challenging features of illness) elicit hostile, blaming responses from caregivers who are predisposed to controlling behavior and difficulties with affect regulation [55]. Efforts to extend these theoretical accounts to child and adolescent patients have emphasized the role of child temperament in eliciting high EE attitudes [25]. From this perspective, EE attitudes may begin in early childhood with a youngster whose behavioral, self-regulatory, or cognitive limitations (e.g., behavioral inhibition, irritability, mood lability) pose significant challenges for parents. These features may reflect the child’s underlying genetic vulnerability to psychiatric illness, a vulnerability that may be shared by one or more parents. The result may be that a genetically predisposed child is matched with a parent who is vulnerable to maladaptive patterns of responding with hostile and critical or anxious and overprotective behaviors.

High EE attitudes have been linked to elevated rates of psychopathology in parents [26, 28, 50, 57–60]. It is also associated with lower levels of perceived parental support [17, 58], poorer family communication [61] and higher levels of marital problems and family conflict [39]. High EE dynamics are observed in actual parent–child interaction [40, 50, 62], and are associated with disrupted attachment patterns [52]. These findings suggest that EE is related to numerous aspects of family functioning that might undermine the ability of parents to cope effectively with child psychopathology. For children and adolescents who are already challenged by the early vestiges of mental illness, parents’ negative affective responses—possibly reflecting the parents’ own vulnerability to psychiatric disorder—may create a family atmosphere in which children experience multiple stressors as sustained and uncontrollable From the perspective of family intervention, these findings underscore the importance of carefully assessing broader family functioning–including parental coping skills and strategies for managing mental illness—at the outset of treatment.

Attributional Models

Models of EE in the adult literature suggest that critical comments stem from attributions that caregivers make about the causes of undesirable patient behaviors [63–65]. Within this framework, attributions of personal control and responsibility lead to high levels of criticism (and thus, high EE) whereas beliefs that external factors (i.e., illness) are responsible for unwanted behavior lead to more neutral or low EE responses. Considerable empirical work [5] supports this model for understanding EE in the context of adult psychopathology and a more limited body of work suggests its relevance for younger age groups. For instance, Bolton et al. [56] reported that, among mothers of clinic-referred youth with behavior problems, those who made attributions of blame and personal responsibility about their children's behavior were more likely to be high in EE. A similar association has been found in pediatric OCD [10]. Notably, the attribution model explains the "critical comments" component of the EE construct better than the emotional overinvolvement component.

Although parental psychopathology and parental attributions may be two factors that predispose parents to poor patterns of responding to mental illness, they are unlikely to account fully for the variance in high EE attitudes. Certainly they do not account for child-level variables that may be correlated with high EE attitudes, such as the child's temperament, willingness to participate in treatment, emotion regulation skills, and empathy for others. They also do not consider "resiliency" variables that may influence the emergence of EE attitudes, such as a parent's emotion regulation skills, problem-solving abilities, or positive atttributional biases. Understanding these variables is essential to developing effective interventions for high EE families.

New Directions for the EE Construct: A Toxic Stress Perspective

Models of toxic family stress may clarify how parent-, child- and family-level variables interact to create and maintain high EE attitudes among parents. To date, research on toxic family stress has examined how children respond to environments characterized by extreme adversity and instability (e.g., poverty, abuse, parental psycho-pathology), emphasizing the heightened stress responding and hypervigilance that emerge when youngsters confront sustained stressors in the absence of adequate family support [20]. There are several indications that, for youth with psychiatric illness, high EE dynamics may function in a similar manner. Indeed, for youngsters experiencing the first onset of psychiatric illness, high EE may reflect an absence of appropriate support in the face of a significant stressor. The toxic stress framework is illustrated in recent models of mood disorder which postulate that excessive activation of the hypothalamic–pituitary–adrenal (HPA) axis may reflect inadequate neuroendocrine control over the immune system. This in turn may lead to increased levels of inflammatory markers and a more deleterious course of illness [66].

It is also possible that, in some cases, the family interactional behaviors associated with EE (for example, problem discussions that do not resolve) are the stressors in this stress/vulnerability equation. As noted, high EE attitudes are often stable over time suggesting that they may pose ongoing challenge for a biologically vulnerable child. Moreover, these attitudes—while likely emerging in response to child symptoms–have the potential to form a vicious and self-perpetuating cycle. That is, affectively charged family dynamics are likely

to exacerbate psychiatric illness and complicate recovery, in turn begetting further criticism and over-involvement from parents struggling to respond effectively. These reciprocal and escalating patterns of influence are documented in the adult EE literature [40] and are likely operative among pediatric patient populations as well. What is less clear, however, is how EE affects children and families at a biological level.

Does EE affect physiological indices of the stress response in the same manner as other forms of toxic stress? In an innovative series of studies of college students, Hooley et al. [67, 68] found that formerly depressed students who heard audiotapes of their mothers criticizing them were less likely to activate areas of the dorsolateral prefrontal cortex (DLPFC) during fMRI than students without a history of depression. These differences were not apparent in response to maternal praise. In a second study, formerly depressed students responded to maternal criticism with greater amygdalar activation and less activation in the DLPFC and anterior cingulate cortex than healthy students [68]. These studies suggest that the criticism dimension of EE affects vulnerable individuals at the neural level, even while these individuals are in remission. Although the cause/effect relationship between changes in neural activation and the frequency of maternal criticism cannot be ascertained from these studies, they provide some indication for links between EE and stress responding.

Among youth with both internalizing and externalizing disorders, poor family functioning is consistently associated with heightened stress responding across a range of experimental tasks. With respect to EE dynamics in particular, Christiansen et al. [43] reported that, among youth with ADHD, parental high EE status was associated with elevated levels of salivary cortisol. Moreover, this heightened stress reactivity moderated the link between EE and comorbid oppositional defiant disorder. In an earlier report, Hibbs et al. [69] found that youth with OCD from high EE homes had elevated patterns of baseline stress responding as measured via skin conductance on laboratory tasks. In addition, they were slower to habituate following exposure to an experimental stressor, a finding that is in line with patterns of hyper-vigilance observed among youth exposed to chronic family stress.

These laboratory findings may offer a plausible explanation for why EE is related to poor treatment outcome in pediatric OCD [10, 11]. Cognitive behavior therapy (CBT) is considered the current treatment of choice for pediatric OCD [70]; it relies heavily on exposure-based exercises in which youth are gradually exposed to anxiety provoking stimuli. Traditional theories of learning and behavior suggest that the process of activating fears and habituating to them is central to clinical improvement, with faster and more consistent returns to baseline associated with better clinical outcomes [71]. Thus, if habituation during exposure tasks is disrupted or delayed, or if some youth begin these tasks from a state of elevated activation, the learning process may be undermined and treatment gains may be limited. As a result, high levels of EE should predict slower gains in CBT. These possibilities—while speculative at this point—provide one testable model of how EE influences treatment outcomes.

Implications for Community Care

The uptake of the EE construct in community mental health settings has been disappointing. Few practicing clinicians assess EE despite its significant record in predicting the course of

psychiatric disorders and possibly, moderating the effects of psychosocial interventions. This gap between academic research and community practice–well known in the evidence-based treatment literature–derives in part from the expensive and cumbersome nature of the EE assessment and coding systems. The gap probably also derives from the lack of useful treatment information gleaned from knowing that a family is high or low in EE. A high EE family may benefit from family therapy, but does one really need an extensive family assessment to make that recommendation? Moreover, if the family is likely to benefit, is it from neutralizing heated family dynamics, teaching more adaptive skills for coping with or managing a particular illness, or by offering therapeutic support that alleviates family stress? These remain open questions, and underscore the need for further research on EE and psychosocial intervention.

Mechanistic models of EE, including those focused on attributions of causality, interaction patterns, or bidirectional-interactional models of toxic family stress are useful to the extent that they inform treatment. For example, attributional models imply that parents would have lower levels of EE if they understood that much of the child's behavior is the product of a biologically-based illness, or at least factors beyond the child's control; this position emphasizes the role of psychoeducation in intervening with high-EE behaviors in families. Psychoeducation is a mainstay of most family interventions, and provides a platform for changing key views about mental illness. It may help not only in changing attributions of causality, but also in informing parents of circumstances where a child could exert more self-control, where more parental guidance is needed, and how developmental considerations should affect expectations of the child.

Nonetheless, psychoeducation alone is unlikely to produce changes in affectively charged family dynamics, and further skills training is likely to be needed. Studies of family interaction suggest the importance of training in communication and problem-solving skills to reduce the frequency and intensity of negative exchanges or to increase the frequency of positive interactions. One study that focused on improving family communication [72] found that family-focused treatment was effective in reducing symptoms in patients with bipolar disorder to the extent that it increased the frequency of *positive* communication within families.

Models of developmental psychopathology [73] point to integrated treatments that address child, parent, and family problems at biological, cognitive, and affective levels of analysis. Skill-based treatments that teach parents to mix criticism with praise, to deliver critical feedback in specific and constructive ways, and to engage more effectively with treatment personnel may reduce strain within the household. Critically, they may provide parents with strategies for expressing their concerns in ways that are tolerable and less stressful to the child, and offer all family members tools for negotiating conflicts or disagreements. Skills training may also lower levels of emotional arousal among family members when they become "triggered" by other family members' behavior, by giving them a set of tools for responding appropriately.

Finally, models of toxic stress are likely to lead to emphasis on improving emotion regulation for parents and children. Interventions to enhance emotion regulation may include

affect labeling and monitoring, relaxation training, or mindfulness meditation or other self-soothing strategies. They may decrease the impact of toxic interchanges on individuals within a system. For example, a parent faced with a highly oppositional child may be less likely to respond with criticism if he or she is able to tolerate the negative affect caused by such exchanges and respond with neutrality. A parent attempting to help a child with severe OCD to resist urges to ritualize may be better equipped to do so if she can tolerate the distress that arises in response to watching her child struggle. Emerging evidence in the pediatric OCD literature suggests that teaching parents skills for managing and tolerating their own affective arousal may lead to better treatment outcomes [74]. Although identifying of the mechanisms of action underlying links between high EE and poor youth outcomes is not a requirement for family treatment, these examples illustrate the potential for more targeted, empirically-guided approaches to intervening with families in need. Given that the child and adolescent psychopathology literature documents relatively little success with changing the high EE family dynamic thus far, such efforts are sorely needed.

Summary

Despite decades of research linking high EE to poor outcomes for youth with mental illness, parallel research on efficacious interventions for EE is lacking. To date, most family therapy trials have focused on specific child/adolescent disorders, with symptom reduction emphasized as a primary goal. Enhanced family functioning has received relatively less attention. Yet the role of EE in predicting the course of multiple forms of child and adolescent psycho-pathology and in some cases, predicting treatment response argues for the value of addressing negative parental attitudes or family interaction patterns in treatment. Effective intervention with high EE families will require a clearer understanding of the determinants of critical attitudes in parents, the effects of these attitudes on developing children, and the recursive effects of these bidirectional interaction patterns on other family members. Moreover, it will require an appreciation of how poor family functioning influences child functioning on physiological, neural, emotional, and behavioral levels. Research that examines the impact of critical or overinvolved home environments at the neural or immunological level may clarify the mec hanisms by which EE leads to poorer outcomes of psychiatric disorder and help to identify novel strategies for intervention.

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Table 1

Rates of High EE Attitudes across Child and Adolescent EE Studies[a]

	Study	N	Mean age[b] (SD)	Rate of high EE (%)
Axis I disorders				
Depression				
	Asarnow et al. [6]+	35	10.99 (1.53)	51
	Asarnow et al. [75]+	83	12.62 (2.76)	61
	McCleary and Sanford [79]	57	15.1 (1.5)	40
	Silk et al. [33]	29	12.9 (2.8)	66
	Tompson et al. [59]	22	10.77 (.87)	36.4
ADHD				
	Asarnow et al. [75]+	73	11.09 (2.88)	52
	Christiansen et al. [43]	59	10.6 (2.8)	29
	Daley et al. [49]	80	3.08 (−)	43
	Peris and Hinshaw [29]+	81	9.75 (1.79)	62
OCD				
	Hibbs et al. [28]+	49	14 (2.8)	82
	Przeworski et al. [11]	62	11.7 (2.64)	16
	Peris et al. [74]	58	12.33 (2.56)	55
DBDs/externalizing				
	Baker et al. [76]	18	4.63 (.53)	41
	Hibbs et al. [28]+	34	11.7 (3.4)	88
	Peris and Baker [27]	60	6.8 (.4)	73
	Owen-Anderson et al. [42]	20	7.34 (1.06)	47
Bipolar disorder				
	Miklowitz et al. [35]+	52	14.5 (1.6)	46
Schizophrenia				
	Asarnow et al. [6]	30	10.20 (1.93)	23
Eating disorder				
	van Furth et al. [34]	46	17.1 (2.4)	21
Gender identity disorder	Owen-Anderson et al. [42]	20	6.59 (1.78)	45
High risk samples				
Children at risk for Bipolar disorder	Miklowitz et al. [36]	35	12.3 (2.8)	49
Youth at risk for depression				
	Brennan et al. [82][c]	522	15.17 (.27)	12
	Silk et al. [33]	21	13.2 (2.3)	29
	Tompson et al. [59][d]	52	10.11 (1.37)	36.5
Disadvantaged minority youth				
	McGuire and Earls [31]	39	8.5 (−)	31
Children at elevated risk for Behavioral/emotional Problems	Boger et al. [60]	276	6.0 (−)	27

	Study	N	Mean age[b] (SD)	Rate of high EE (%)
Adolescent self injury	Wedig and Nock [14]	36	15.26 (1.48)	25
Other				
Diabetes mellitus				
	Liakopoulou et al. [78]	55	12.9 (1.99)	71
	Worrall-Davies et al. [81]	45	9.8 years (−)	46
Childhood asthma	Wamboldt et al. [80]	30	7.5 (−)	43
Adolescent asthma	Wamboldt et al. [80]	84	14.7 (−)	48
Intellectual disability				
	Beck et al. [16]	33	9.02 (3.54)	60
	Dossetor et al. [57]	92	-	35
	Hastings et al. [15]	75	9.75 (4.04)	52
	Kubicek et al. [77]	38	1.72 (.60)	40
Learning disability				
	Lam et al. [17]	27	10.8 (2.3)	40

[+] Results reflect families where at least one parent is high in EE

[a] Studies were included based on their reporting of rates of overall high EE within published findings and on their adherence to originally validated scoring procedures

[b] Age is reported in years

[c] Paternal EE

[d] Including only those cases with a history of maternal depression and no past or current child depression