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Psychosocial Mechanisms of Outcome in Pediatric Psychiatry

by

Alessandro S. De Nadai

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy Department of Psychology College of Arts and Sciences University of South Florida

Co-Major Professor: Eric A. Storch, Ph.D. Co-Major Professor: Marc S. Karver, Ph.D. Vicky Phares, Ph.D. Michael T. Brannick, Ph.D. Brent J. Small, Ph.D.

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Keywords: Psychiatry, pediatric, alliance, adherence, motivation, expectancies

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Abstract

Nearly half of all youths experience a mental health disorder at some point during childhood (Merikangas et al., 2010). Pediatric psychopathology is associated with a substantial amount of impairment in the school, social, and home domains, and such symptoms can have adverse impacts on subsequent development (Beauchaine & Hinshaw, 2013; Patel, Flisher, Hetrick, & McGorry, 2007). Fortunately, a number of medications have demonstrated efficacy in treating a number of mental health conditions (Martin, Scahill, & Kratochvil, 2010). Despite these demonstrated effects, treatment response is often incomplete, and the mechanisms by which pharmacotherapy lead to behavior change are not well understood. However, research in pediatric psychopharmacology has often not considered the role of psychosocial variables, despite their promise to explain much variance in psychiatric outcomes and the robust influence they have demonstrated in psychotherapy-based behavior change (e.g., Shirk & Karver, 2011). This study investigated the role of four psychosocial variables in treatment outcome in pediatric psychiatric practice: medication adherence, therapeutic alliance, motivation for behavior change, and expectancies for positive treatment outcome. Surprising patterns of effects were found, with psychosocial variables being associated with both decreases and increases in symptomology depending on the circumstance (e.g., externalizing behavior), and many inconsistencies were observed among these patterns. While psychosocial variables are often portrayed as having uniformly positive impacts on treatment, their role in pediatric psychiatry may not be as straightforward as is commonly depicted in other diseases and therapeutic approaches. In

particular, the nature of their effects on outcome may vary across symptom presentations and intervention approaches. Based on these findings, recommendations for clinical practice and future research are discussed which affect all patients, researchers, and medical providers who participate in pediatric psychiatric treatment.

Introduction

Psychiatric disorders in children are associated with devastating individual consequences and present an enormous public health burden. During childhood, 49.5% of youth experience a mental disorder at some point, and 22.2% of children experience symptoms that are characterized by severe impairment and/or distress (Merikangas et al., 2010). By adolescence, nearly two million American children perceive more than half of their days as "mentally unhealthy" (Perou et al., 2013). Pediatric psychopathology is also associated with impaired performance in the school, social, and home environments, and can lead to family disruption and even suicide (Beauchaine & Hinshaw, 2013). Such impairment is compounded longitudinally, as a majority of adults with mental disorders experience onset of symptoms during youth that continue to persist into adulthood (Kessler et al., 2005). This adversely affects the achievement of developmental milestones, including academic, vocational, and social goals (Patel, Flisher, Hetrick, & McGorry, 2007). These behavioral difficulties can also affect other health functioning; for instance, youth with mental disorders are at a higher risk of contracting HIV relative to peers without any mental disorders (Donenberg, 2005; Donenberg, Emerson, Bryant, Wilson, & Weber-Shifrin, 2001). On a broader level, the associated costs of mental illness in young people are staggering, as annual costs associated with pediatric mental illness are estimated at \$247 billion dollars (Perou et al., 2013), and mental disorders are the costliest health condition to treat in children (Soni, 2009). As a whole, mental health problems in children are widespread, debilitating, and have detrimental effects on families, communities, and society.

To address pediatric mental illness, two major approaches have been psychotherapy and medication treatment (Olfson, He, & Merikangas, 2013; Olfson & Marcus, 2010). While both modalities have displayed efficacy for a number of conditions (Kendall, 2011; Martin, Scahill, & Kratochvil, 2010), psychopharmacological interventions are used over five times more frequently than psychotherapy for youths (Olfson, Blanco, Wang, Laje, & Correll, 2014), and an estimated 14.2% of adolescents report taking a psychotropic medication in the preceding 12 months (Merikangas, He, Rapoport, Vitiello, & Olfson, 2013). Medication use is also expanding over time, as the number of physician visits resulting in psychotropic medication prescriptions has more than doubled during the 15 year period prior to 2010 (Olfson, et al., 2014). However, the potential of side effects and adverse developmental impacts of medication use has led to some concern, especially given the extensive usage of pharmacotherapy in children. Also, while pharmacotherapy is frequently efficacious, real world treatment response is often incomplete (e.g., Franklin et al., 2011; The TADS Team, 2007; Walkup et al., 2008). Given that the development of new psychotropic agents has slowed greatly relative to the rest of medicine (Cowen, 2011), new "miracle drugs" to improve psychiatric outcomes are not on the developmental horizon.

Given this context, what can be done to improve the real world effectiveness and safety of contemporary pharmacotherapy? Many efforts to address these concerns have manifested through the development of new pharmacological agents, which rely on an assumption that the mechanism of change in pharmacotherapy is largely biological. Unfortunately, this approach to pediatric psychopharmacology has led to limited improvements in patient outcomes in the past several decades. While some progress has been made in reducing side effects, newer pharmacological agents have not produced drastic improvements in patient outcomes (e.g.,

Lieberman et al., 2005). As it currently stands, purely biological approaches to psychiatry have not produced fully optimal outcomes, and millions of children are not obtaining optimal symptom relief as a result. In addition, these suboptimal individual-level outcomes can aggregate into a large scale attenuation of therapeutic effects, leading to additional burden on the overall healthcare system when patients remain in active treatment for excessively long periods.

However, a different approach is to directly incorporate psychosocial variables in pharmacotherapy approaches, which have demonstrated substantial impact on outcomes in psychotherapy and conventional medicine (e.g., Hall, Ferreira, Maher, Latimer, & Ferreira, 2010; Osborn & Egede; Shirk, Karver, & Brown, 2011). These variables are known as "common factors" in the psychotherapy literature, as they have been highlighted for their importance across all methods of psychotherapy for over 70 years (Rosenzweig, 1936), but in the context of pharmacological interventions they have been characterized more frequently in terms of nuisance confounds and "placebo effects" (Miller, Colloca, & Kaptchuk, 2009). However, while psychosocial variables have often been considered to be research confounds, perhaps instead they can be construed as novel mechanisms to capitalize upon in order to provide new avenues for outcome improvement. At present, quantitative data to justify the roles of such variables are limited in pediatric psychiatry, but common factors are active in some capacity during all mental health treatment, including psychiatry (Patterson, 1985; Verhulst, Kramer, Swann, Hale-Richlen, & Beahrs, 2013). These psychosocial variables are also likely to work in tandem with biological treatments, as placebo effects have a variety of neuroendocrine consequences (e.g., modulation of neurotransmitter and hormonal functioning, changes in brain-based metabolism; Finniss, Kaptchuk, Miller, & Benedetti, 2010). To artificially dichotomize such effects as separate from biology results in imprecision in modeling the effects of pharmacotherapy for youth.

Among psychosocial variables, one factor that has received relatively little attention in pediatric psychiatry has been medication adherence, despite adherence rates being remarkably poor for a number of widely used medications (Brown & Bussell, 2011; Fischer et al., 2010). Another series of variables that have received limited empirical examination in pediatric psychiatry include the therapeutic alliance, motivation for behavior change, and expectancies for positive treatment outcome. These variables are readily available for modification and do not depend on hypothetical scientific developments in order to bring about improved patient outcomes. At present, patient adherence, therapeutic alliance, and patient expectancies and motivation remain understudied as active ingredients in pediatric psychiatry, as biological interventions are often construed as the integral active components. However, increasing the understanding of psychosocial variables in pediatric psychiatry could provide a foundation for innovative approaches that aim to improve care for a large number of patients. A review of relevant psychosocial variables follows.

Considering the Role of Adherence in Pediatric Psychiatry

Adherence refers to the extent that patients follow a prescribed medication regimen, and is critical to ensure the effectiveness of interventions (Osterberg & Blaschke, 2005). Indeed, billions of dollars in drug development are rendered ineffectual if patients do not take their medications as prescribed. In child psychiatry, adherence has often been observed to be poor, and adherence rates have been reported to be under 50% for commonly used medications such as serotonin reuptake inhibitors (frequently used for depression and anxiety) and stimulants (frequently used for attention-deficit/hyperactivity disorder, i.e., ADHD; Gau et al., 2006; Murray, de Vries, & Wong, 2004; Richardson, DiGiuseppe, Christakis, McCauley, & Katon, 2004). While data on the adherence-outcome relationship are limited in pediatric

psychopharmacology, nonadherence to sertraline has been associated with a nearly 30% reduction in response rates in adult depression (von Knorring, Åkerblad, Bengtsson, Carlsson, & Ekselius, 2006). Psychiatric medication nonadherence is also a major contributor to drug-related emergency hospital admissions (Procyshyn, Barr, Brickell, & Honer, 2010), and poor medication adherence has been associated with increased rates of relapse and hospital readmission in patients with schizophrenia and bipolar disorder (Velligan et al., 2009). Conversely, improvements in adherence predict improved outcomes in treatment for pediatric ADHD and depression (Pappadopulos et al., 2009; Woldu et al., 2011). These adherence-outcome relationships are also seen in other fields of medicine, as meta-analytic estimates indicate that the difference between low and high adherence makes for a 26% difference in desired medical outcomes, and variability in adherence can compromise medical outcomes as much as 71% (DiMatteo, Giordani, Lepper, & Croghan, 2002). Poor adherence is not restricted to psychiatry and remains a common problem in a variety of chronic health conditions in youth, including diabetes, renal disease, and AIDS (DiMatteo, 2004). Inadequate adherence is also costly in terms of financial expenditures. Increases in costs ranging from \$750 to \$2,000 have been observed for each nonadhering patient in adult antidepressant treatment (Revicki, Simon, Chan, Katon, & Heiligenstein, 1998; Thompson, Peveler, Stephenson, & McKendrick, 2000), and costs associated with vocational absenteeism due to antidepressant noncompliance are over \$1,000 annually per nonadherent patient (Birnbaum et al., 2010). In contrast, adequate adherence to antidepressants has been associated with a reduction in overall medical costs (Cantrell, Eaddy, Shah, Regan, & Sokol, 2006).

Adherence stands to be an important mechanism of outcome in pediatric psychiatry, especially given the frequent usage of psychiatric medications in youth along with low observed

adherence rates. As it stands already, unsuccessful treatment as a result of nonadherence burdens an already overtaxed mental health care system. Thus, increasing understanding of how children adhere to psychiatric medications can help individual patients, reduce financial costs to the medical system, and allow the mental health system to treat the millions of youths in need more effectively. Despite these wide ranging consequences, medication adherence in pediatric psychiatry remains an understudied variable (McGuinness & Worley, 2010).

In considering variables that have traditionally affected adherence, a number of factors have emerged. With regard to physical factors, side effects of medication as well as convenience of administration predict adherence (Julius, Novitsky, & Dubin, 2009; Mitchell, 2006). Exemplars of these phenomena include patients who may find it easier to adhere to once-a-day medication dosing relative to multiple intradaily doses, and patients who experience greater side effects can sometimes reduce the amount of medication they are taking when they experience these undesired effects. Demographic factors also have relationships with adherence. Overall adherence is worse for children relative to adults (Costello, Wong, & Nunn, 2004) and in particular adolescents can show increased difficulties with adherence (Matsui, 2007).

In addition to these factors, one unique aspect of adherence in pediatric psychiatry is that the pathology itself can reduce adherence (Smith & Shuchman, 2005). This is particularly impactful for child psychiatry, as a recursive process can exist where adherence to psychiatric intervention is reduced by psychopathology, and then this low adherence precludes improvements in psychopathology, which subsequently continues to impact adherence. For example, a depressed child may be less likely to be motivated to take medication, adhere more poorly, and remain more depressed as a product of this nonadherence, which subsequently continues to attenuate adherence as part of a vicious cycle.

Although physical, demographic, and psychopathology-related factors can affect adherence, they are not fully predictive of behavior, and at times provide only limited avenues for adherence improvement. However, other psychosocial variables provide a potential opportunity to improve adherence and merit further consideration, especially as significant variability has been observed among physicians with regard to their approaches to adherence (Drotar, 2009). One traditional approach has been defined as the health beliefs model, which focuses on the perceived benefits of medications in contrast to perceived harms (Rosenstock, 1966). A related framework that has also been applied to adherence behavior is social cognitive theory, which focuses on expectations for positive outcomes and expectations for ability to complete adherence behavior (Bandura, 1998). Such approaches have been successful for increasing adherence and health-promoting behavior in a number of medical conditions, including asthma and heart disease (Bandura, 2004). Yet another approach to psychosocial variables in adherence has been the theory of planned behavior (TPB), which focuses on three components: patient attitudes towards adherence, patients' perceived subjective norms towards adherence, and patient expectations about their ability to engage in adherence (Ajzen, 2011). The TPB model predicts engagement for a number of health behaviors, ranging from increases in exercise to reductions in risk-taking behavior (McEachan, Conner, Taylor, & Lawton, 2011). However, these traditional models suffer from some shortcomings. They were originally conceptualized in adults, and the complexity of these models is compounded in pediatric care as these variables can apply to both parents and children (Matsui, 2007). Also, while these traditional approaches have made some inroads into predicting adherence, much variance remains to be explained (Riekert, Ockene, & Pbert, 2013). They are further limited by their focus on patient-level behavior, neglecting patient-provider and family-based interactions (Clark &

Janevic, 2013). To address this limitation, models that incorporate the patient-provider interaction (such as therapeutic alliance) merit addressing in the context of adherence and outcome (Diamond, 2012).

Considering the Role of Therapeutic Alliance in Pediatric Psychiatry

The therapeutic alliance focuses on the interaction between patient and clinician, and in adults has been conceptualized in terms of three components: the bond between clinician and patient, agreement on the tasks to be completed in therapy, and agreement on the therapeutic goals to be achieved (Bordin, 1979). In children, these individual components have been identified as important, but at times have not emerged as separate factors, and instead a one-factor model of alliance has been primarily found (Shirk, et al., 2011). Differences between adult and pediatric alliance models have arisen for a number of reasons, including distinct perspectives provided by children and the presence of multiple parties in therapy (e.g., parents and children; Zack, Castonguay, & Boswell, 2007). Children may also not have the cognitive capacity to differentiate task and goals (which are more cognitively based) from the bond (which is more emotionally based; Shirk & Karver, 2011). With regard to the process of alliance in child therapy, alliance formation involves the clinician simultaneously serving in a position of active listening while also providing a directive framework for treatment (Shirk, et al., 2011).

The alliance has been highlighted most extensively in the psychotherapy literature, where it has shown a robust effect on treatment outcome across psychotherapies (Horvath, Del Re, Flückiger, & Symonds, 2011). Additionally, alliance can predict other important child therapy processes such as patient engagement and retention (Castro-Blanco & Karver, 2010; Garcia & Weisz, 2002). However, while the necessity of the physician-patient relationship has been identified as critical in adherence for overall pediatric practice (Winnick, Lucas, Hartman, &

Toll, 2005), it has received very little attention in child psychiatry despite the patient-provider relationship being central to establishing a diagnosis and to making a treatment prescription.

Nevertheless, some empirical data has addressed the alliance in psychiatry. In adult depression, the average alliance throughout treatment accounts for 19% to 56% of variance in pharmacological treatment outcome (Krupnick et al., 1996; Weiss, Gaston, Propst, Wisebord, & Zicherman, 1997), and alliance can predict outcomes even when already accounting for technique effects from cognitive behavioral therapy (CBT) and/or antidepressant medication (Klein et al., 2003). Early alliance in treatment may be particularly predictive of antidepressant outcomes (Blatt & Zuroff, 2005). Alliance may also have a specific effect on medication outcome, as it has displayed differential effects between active compounds and placebo medication in SSRI treatment for depression (Strunk et al., 2010). Alliance and expectancies have also predicted adherence and outcome in pharmacotherapy for bipolar disorder (Gaudiano & Miller, 2006; Sylvia et al., 2013; Zeber et al., 2008) and in the usage of antipsychotic medication (Frank & Gunderson, 1990; McCabe et al., 2012). Alliance may affect other therapy process variables as well, as the odds of medical treatment adherence are 2.16 times greater overall if a physician is a good communicator (Zolnierek & DiMatteo, 2009). Thus, these limited data indicate that alliance is not only an accessory to psychiatric treatment, but may in fact drive a significant proportion the treatment process and subsequent outcome in pharmacotherapy.

A number of reasons have been posited regarding why alliance may affect outcomes. One theory focuses on the sufficiency of strong alliance formation, which allows the patient to enact changes that might not otherwise be made alone (Norcross, 2010). In this context, the alliance is the principal stimulus that leads patients to identify and enact positive change as a result of therapy. Another aspect is that alliance helps patients engage in other therapy elements (De

Nadai, King, Karver, & Storch, 2014). Under this conceptualization, a strong alliance provides a foundation for communication in therapy that enhances patient engagement in specific techniques and interventions provided by the clinician, which then result in therapeutic change. Alliance has been found to be consistently related to adherence in a number of mental health treatments, and may work partially through improving expectancies for treatment (Thompson & McCabe, 2012). In psychiatry, it has been suggested that alliance can directly improve patient outcomes, and may also indirectly improve outcomes through its positive effects on adherence (Priebe & McCabe, 2008). However, this hypothesis remains understudied. No comparative data exist for children, though doctor-patient communication (an analogue of alliance) has been shown to predict adherence to a variety of pediatric medical treatments (DiMatteo, 2004). Child psychiatry also differs from traditional therapy with adults due to the dual importance of both child-clinician as well as parent-clinician alliance (Joshi, 2006), as child and parent alliance may have some orthogonal contributions to mental health outcomes (Bickman et al., 2012; Hawley & Weisz, 2005). While understudied, alliance affects both process and outcome variables in psychological and medical treatments.

Considering the Role of Motivation for Behavior Change in Pediatric Psychiatry

While patient motivation has been identified as a key principle in routine psychiatric practice (Chanut, Brown, & Dongier, 2005), its effects have been rarely quantified in pharmacotherapy for pediatric psychopathology. Motivation for behavior change in the context of psychopathology has most often been conceptualized in terms of the transtheoretical model for change, which posits that patients are often at different stages of readiness for change. This succession of stages include precontemplation (has not considered change), contemplation (has some desire to change, but also some desire to maintain the status quo and has not initiated

change), preparation (has started to take steps that lead to change), action (has initiated the change process), and maintenance (working to retain changes that have been made; Prochaska & DiClemente, 2005). Interventions tailored to specific stages of changes have displayed efficacy in changing behaviors ranging from smoking cessation to physical activity promotion (Cahill, Lancaster, & Green, 2010; Marshall et al., 2003), and while it was originally derived in the context of substance use disorders, the relationship between readiness for change and outcome in a number of psychotherapy approaches has been identified through meta-analysis (Norcross, Krebs, & Prochaska, 2011). The transtheoretical model has proven quite flexible, permitting for application to a wide range of behaviors (including exercise, domestic violence, and organ donation) and in a broad array of treatment settings, ranging from primary care to college campuses (Lundahl et al., 2013; Prochaska, Redding, & Evers, 2008).

In considering pediatric psychiatry, parental motivation for change has been identified to predict adherence to psychiatric medication (De Nadai, 2013), and better outcomes have been found in pharmacotherapy for depressed adolescents who are in the action stage of change at baseline (Lewis et al., 2009). While data are limited with youth, higher levels of precontemplation have been associated with less change during pharmacotherapy for adults with obsessive compulsive disorder (OCD; Pinto, Pinto, Neziroglu, & Yaryura-Tobias, 2007). Unfortunately, stages of change research has received relatively little attention in psychiatry, despite the robustness of its supporting literature and its likely relevance for psychiatric research and practice (Cole, Bogenschutz, & Hungerford, 2011). Notably, psychiatric disorders often present barriers to motivation for change that prevent successful intervention for change in the disorder itself (Dilallo & Weiss, 2009), creating a self-sustaining barrier to symptom change. While patient motivation affects both distal patient symptom outcomes as well as proximal

therapy process outcomes, the relative magnitude of its direct and indirect effects have rarely been quantified for any health condition, and it remains an understudied variable in pediatric psychiatric practice.

Considering the Role of Expectancies in Pediatric Psychiatry

Expectancies for psychiatric care can be distilled into two major aspects: what the patient expects his/her role to be in treatment (role expectancies) and what the patient expects for treatment outcome (outcome expectancies; Dew & Bickman, 2005; Glass, Arnkoff, & Shapiro, 2001). Outcome expectancies have received particular focus in mental health treatments (Delsignore & Schnyder, 2007), and they have been identified to predict treatment outcomes in psychotherapy through meta-analysis (Constantino, Arnkoff, Glass, Ametrano, & Smith, 2011), including CBT for social phobia, fear of flying, and pediatric obsessive compulsive disorder (Lewin, Peris, Lindsey Bergman, McCracken, & Piacentini, 2011; Price, Anderson, Henrich, & Rothbaum, 2008; Safren, Heimberg, & Juster, 1997). Outcome expectancies do not exist in a therapeutic vacuum but rather work jointly with other common factors, as patients with positive treatment expectancies have been found to have stronger alliances (Connolly Gibbons et al., 2003; Constantino, Arnow, Blasey, & Agras, 2005; Greenberg, Constantino, & Bruce, 2006; Hersoug, Hoglend, Havik, & Monsen, 2010) and adhere better to psychological treatments (Constantino, Ametrano, & Greenberg, 2012). This mechanistic relationship with alliance may be particularly strong, as Joyce et al. (2003) found that alliance could account for approximately one-third of the relationship between expectancies on outcome. Motivation for change has also been associated with outcome expectations (McKee et al., 2007), though expectancies are distinct from motivation, as patients may be motivated for change yet still not expect noteworthy positive changes from therapy (Arnkoff, Glass, & Shapiro, 2002). This construct distinction has

also been observed in pediatric psychotherapy, where parent outcome expectancies have been found to predict adherence above and beyond parent motivation for treatment (Nock, Ferriter, & Holmberg, 2006).

Despite the wide ranging effects observed for psychological treatments, expectancies have received comparatively little attention in psychopharmacological interventions. With regard to extant data, adolescents' outcome expectations for depression treatment have predicted outcomes across psychological and psychopharmacological modalities (Curry et al., 2006), and similar findings have been found in adult depression (Sotsky et al., 1991). Distinguishing patients with high and low expectations may provide a particularly stark contrast, as 90% of patients with strong expectancies showed treatment response in a single-blind trial of reboxetine for depression, in comparison to 33% of patients with low expectancies (Krell, Leuchter, Morgan, Cook, & Abrams, 2004). It has been argued that common factors account for a majority of variance in adult antidepressant therapy for depression, and in particular expectancies may be a central mechanism for this effect (Kirsch, 2013). Alliance has also been demonstrated as a mediating mechanism whereby expectancies exert outcome effects in pharmacotherapy for both unipolar and bipolar depression (Gaudiano & Miller, 2006; Meyer et al., 2002). In addition to these findings in depressive disorders, outcome expectancies along with perceived quality of clinicians' explanations of medications have been related to adherence in ADHD treatment (e.g., Berger, Dor, Nevo, & Goldzweig, 2008; McNicholas, 2012).

Troublingly, there may be lower expectations for primarily pharmacological approaches. Rapaport et al. (1996) found that when surveying depressed patients about possible sources of successful relief, medication alone was perceived as the least likely to help (8%), relative to talking therapy alone (25%) and combined medication and talk therapy (62%). Lax et al. (1992)

also found that patients with OCD had stronger treatment expectations for psychological treatment relative to pharmacotherapy, though expectations were strong for both treatment modalities. Expectancies may also impact trials of clinical compounds, as response rates are higher in antidepressant trials when the medication under evaluation is compared to another active medication as opposed to placebo (Rutherford, Sneed, & Roose, 2009). There are some data that indicate that this finding may be due to higher expectancies for symptom reduction, given that when patients are certain they are receiving active treatment they expect better outcomes, as opposed to placebo-controlled trials where patients are uncertain if they are receiving treatment that will be helpful (Rutherford, Sneed, Devanand, Eisenstadt, & Roose, 2010).

The powerful influence of outcome expectancies is well known in other areas of medicine. Indeed, many physicians attempt to capitalize on their effects - over half of surveyed physicians have reported that in the prior year, they have prescribed medications to raise patient expectations as opposed to using them primarily for their original intended therapeutic effects (Tilburt, Emanuel, Kaptchuk, Curlin, & Miller, 2008). There is some evidence that expectancy itself works through neural mechanisms that regulate the experience of emotion (Enck, Benedetti, & Schedlowski, 2008; Rutherford et al., 2010), and thus expectancy enhancing interventions (Constantino et al., 2012) are not completely independent from biological interventions. However, the role of outcome expectancies for medication therapy in pediatric psychiatry remains poorly understood, with regard to both direct treatment effects as well as indirect effects through variables such as adherence and alliance.

Considering the Influence of Alliance, Motivation, and Expectancies on Adherence in the Context of Outcome in Pediatric Psychiatry

A conceptual model of how common factors can affect psychiatric outcomes can be found in Figure 1. This figure illustrates several notable facets. First, direct effects on outcome are expressed by separate constructs including alliance, motivation, and expectancies, which frequently have been amalgamated into a generic unitary placebo construct. Second, these common factors are addressed for both parents and children through multi-informant reporting, permitting comparisons among them. Third, mechanisms of effects are incorporated, with alliance and adherence both functioning as mediators of improved psychiatric outcomes. At present, while placebo effects have been identified to account for a substantial amount of outcomes in pharmacotherapy (Leucht, Hierl, Kissling, Dold, & Davis, 2012), few attempts have been made in the psychiatric literature to quantitatively break down the nature of these placebo effects. This limits treatment targeting, as the proportion of outcomes attributable to different common factors and different treatment participants has not been identified. This model also readily includes potential moderators. For example, adherence rates may well differ for children of different ages, given that parents often take more responsibility for medication administration for younger youth relative their older counterparts (Hsin, La Greca, Valenzuela, Moine, & Delamater, 2010).

Implications of the Present Study

While it is logical that psychosocial variables affect pediatric psychiatric treatment, the research base is sparse with regard to their effects on outcome. Accordingly, a number of important implications stand to follow from an empirical investigation of their mechanistic effects. Most directly, these factors are likely to be associated with improved psychiatric

outcomes, and highlighting their roles in a quantitative manner could lead to subsequent targeted interventions to improve relevant behavior. This process fosters personalized medicine as alliance, motivation, and expectancies could be measured via brief questionnaires during routine clinical care, and patients who have undesirable values on any of these metrics could receive a targeted intervention. At present such judgments (if made at all) are done through clinical judgment, which is an inferior process compared to evaluation by actuarial means (Dawes, 2005). Currently, there exists little guidance beyond clinical intuition regarding how much difference these variables make in pharmacotherapy outcomes for mental disorders in children. Given that psychiatric drug development has lagged behind that of other medication classes (Cowen, 2011), this approach provides a new avenue for gains in symptom reduction, via adjustment of concurrent parameters of therapy. This approach has some parallel, as a number of advances in chemotherapy over the past 40 years have been made by adjusting the administration parameters of existing agents (Roberts & Thomas, 2005). In addition, an increased focus on common therapy factors could facilitate safety monitoring of these psychotropic agents, as improving the patient-provider relationship and patient motivation to engage actively in treatment could reduce acute discontinuation and foster communication channels for reporting of problems before they escalate. It also reintroduces the nature of therapy into psychiatric care, as a brief medication check has become established as a standard of care in pediatric psychiatry (Pruett, Joshi, & Martin, 2010).

Additionally, these findings could be used to improve the training of medical providers. Strong alliances are not inherent to all clinician-patient relationships, and significant variability has been detected among therapists with regard to the quality of alliances (Del Re, Flückiger, Horvath, Symonds, & Wampold, 2012). Challenges also exist in maintaining professionalism

while also forming a strong individual relationship with patients (Priebe & McCabe, 2008), and tension or degradation in the therapeutic relationship (known as "ruptures") in the alliance can impair treatment. However, alliance ruptures also provide the opportunity for skillful clinicians who repair such ruptures in psychotherapy to obtain particularly strong clinical outcomes (Safran, Muran, & Eubanks-Carter, 2011). In this context, a number of psychosocial interventions have been designed to improve adherence in pediatrics, which often focus on CBT and/or motivational interviewing (MI) techniques (Dean, Walters, & Hall, 2010; Haynes, Ackloo, Sahota, McDonald, & Yao, 2008). These interventions can be delivered by allied providers for both psychiatric and non-psychiatric conditions (e.g., Rubio-Valera et al., 2011). There is evidence that MI can be successfully taught to medical providers, resulting in changes in physician behavior and enhanced patient outcomes (Soderlund, Madson, Rubak, & Nilsen, 2011). Specific training to improve alliances with psychiatric medication providers has also improved both adherence and outcome to psychotropic medication interventions in adults (Byrne & Deane, 2011), and expectancy enhancement manuals could also be adapted for child psychiatry. The effects of clinician skills have been shown in psychiatric treatment, as one study has found that a positive response to pharmacotherapy was only seen by patients who had a clinician who was a skilled communicator (van Os et al., 2005). Still, the research base is limited with regard to physician-patient communication in psychiatry, to which the present study can contribute.

Data generated from this investigation can also highlight mechanisms of outcome, as little is known about the process by which the common factors and the placebo process operate in pediatric psychiatry. Given that placebo and common factor effects can account for a significant proportion of pharmacotherapy outcomes, the field currently has a lack of knowledge

about precisely why a number of medications are effective (e.g., SSRIs; Fernandez & Gaspar, 2012). It has been questioned whether the clinician-patient relationship is an adjuvant that fosters the implementation of active medications, or whether it provides an additive effect above and beyond pharmacotherapy (Priebe & McCabe, 2008). The present study is the first in pediatric psychiatry to separate effects due to pharmacotherapy and effects due to common factors elements.

Results from this investigation may also affect the practice of subsequent research. Poor adherence to psychiatric medication can be a common confound in the results of clinical trials (Case, 2011), reducing the ability to accurately identify the degree of symptom change due to medication administration and possibly attenuating efficacy estimates. In another context, a medication may have a large effect, but if the common factors also have large effects, the observed effects of the medication may be washed out in a between-groups comparison. These data would provide an initial metric to estimate how much common factors influence should be observed when planning clinical trials.

The purpose of this study is to evaluate the role of psychosocial variables in pediatric psychiatric practice. It was hypothesized that stronger motivation, expectancies, alliances, and adherence would be associated with reductions in psychiatric symptoms. Specifically, it was predicted that these psychosocial variables would work in tandem, with pretreatment expectancies and motivation being predictive of stronger alliances, which would be predictive of stronger adherence and subsequent outcomes. Understanding psychosocial mechanisms of outcome in pediatric psychiatry highlights a way forward to improve future clinical care, and provides a framework to help understand the assumptions underlying prior research. While medication is often attributed as the active intervention in pediatric psychiatric practice, this

study proposes to reframe contemporary psychiatric practice and partition outcomes into a variety of meaningful components. This investigation stands to open up for consideration significant changes to clinical practice, future research, and the training of medical providers.

Method

Participants

Participants were 159 youth ages 7-17 years presenting for psychiatric treatment (M=11.79, SD=3.10), along with their parents and treating clinicians. Participants were 42.1% female, and the ethnic/racial distribution was 78.6% Caucasian, 1.9% African American, 7.5% Hispanic, 0.6% Pacific Islander, 0.6% as Middle Eastern, 10.1% identifying as "other," and 0.6% not providing information on race/ethnicity. Participants were recruited from one of three sites: The USF Silver Center for Child Development (USF; n=65), the All Children's Hospital Pediatric Psychiatry Clinic (ACH n=63), and the Rothman Center for Pediatric Neuropsychiatry (RCN n=31). The USF and ACH sites are not-for-profit outpatient clinics that serve youth with a broad array of psychiatric problems, while the RCN site is a non-profit specialty clinic that focuses on pediatric OCD and related conditions (e.g., anxiety disorders, tic disorders).

Participating patients were recruited from those referred to board-certified psychiatrists or supervised child and adolescent psychiatry fellows for care as usual, with no circumscribed limit on the number of patients seen per individual provider. Through the normal course of care, clinical diagnoses were established through a clinical interview utilizing all available information, as recommended by Klein, Dougherty, and Olino (2005) as well as Silverman and Ollendick (2005). To add further support for diagnoses, a clinical level of symptomology as indicated by either child or parent via self-report (on the MASC-ADI, CDI-2-SF, YSR, or CBCL) was used as a requirement for study inclusion using a diagnostic "or" rule (Piacentini,

Cohen, & Cohen, 1992). No identifiable data collected from participating youth and parents regarding alliance, motivation, expectancies, or adherence was shared with participating clinicians.

Prescriptions were provided by study clinicians to 127 participants, and a total of 231 observations were available for analysis (1.57 medications prescribed per participant who received medication). Common types of medications provided included stimulants (n=56), serotonin reuptake inhibitors (n=58), alpha-2 agonists (n=24), and atypical antipsychotics (n=17). Common diagnoses assigned to participants included externalizing (n=40), internalizing (n=94), neurodevelopmental (n=38), ADHD (n=87), and tic disorders (n=23). Regarding other interventions received, 39.8% of those who responded to 1-month follow-up calls reported receiving concurrent treatment from another provider by that timepoint, and 52.9% of respondents who responded to 3-month follow-up calls reported receiving concurrent treatment from another provider.

Procedures

After obtaining informed consent, measures pertaining to questionnaires on motivation and expectancies were completed before participants' scheduled initial clinical visit, and questionnaires related to alliance were completed immediately after the clinical session. At one and three months after the initial study intake session, study participants received follow-up assessments via phone. A timetable indicating the timepoint at which each self-report measure was administered (baseline, 1-month followup, or 3-month follow-up) can be found in Table 1.

Measures

All study measures free of non-redistributive copyright can be found in Appendices A-K. Parent and child rated measures.

University of Rhode Island Change Assessment (URICA; McConnaughy et al., 1983).

The URICA is a 32-item measure of motivation for behavior change in mental health treatment. Items are rated on a 5-point Likert scale ranging from "strongly disagree" to "strongly agree." The URICA has four subscales (precontemplation, contemplation, action, and maintenance), and the URICA total score is calculated by subtracting the precontemplation subscale from the sum of the contemplation, action, and maintenance subscales. Higher scores reflect greater readiness for behavior change. Modification of the URICA for different populations is encouraged (Rossi, 1995) and such modification has been successfully employed in other studies (e.g., Dozois, Westra, Collins, Fung, & Garry, 2004; Greenstein, Franklin, & McGuffin, 1999). In the present investigation, minor modifications were made to certain relevant items (e.g., changing the word "psychology" to "psychiatry") on both parent and child forms to properly address their role in treatment. The child form focuses on child readiness for change, and the parent form also focuses on readiness for child behavior change (e.g., what is their perception of the child's problem, are parents contemplating the concept that their child needs to change). Similar modifications have displayed acceptable internal consistency in youth ages 7-17 years (alpha=.71; Keeley, Geffken, Ricketts, McNamara, & Storch, unpublished data), as well as in adults (alpha=.79; Dozois et al., 2004). Pretreatment child URICA scores have been shown to be associated with higher pretreatment child anxiety reported via the MASC (r=.33, Keeley et al., unpublished data), and have been observed to predict positive outcomes in adult psychotherapy (Norcross, Krebs, & Prochaska, 2011).

Clinician rated measures.

Therapeutic Alliance Quality Rating (TAQ-R; Bickman et al., 2010). The TAQ-

R is a standardized clinician rating of therapeutic alliance with other treatment parties (i.e., parents and children). It consists of a 1-item rating for the perceived alliance with each therapeutic party that asks, "In this session, how would you describe your relationship with this youth/caregiver." The TAQ-R is rated on a 5-point Likert scale with responses ranging from "very poor" to "excellent." In the development of the TAQ-R, an initial item pool of 52 items was generated to address the bond, task, and goals elements of therapeutic alliance as specified by Bordin (1979), though it was determined through item response theory and classical psychometrics that single item TAQ-R ratings provided largely redundant information for clinician-rated alliance (Bickman et al., 2010). Variability in TAQ-R scores has been observed for both clinician ratings of alliance with the parent (M=4.22, SD=0.71) and youth (M=3.80, SD=0.82) in a large clinical sample (Bickman, et al., 2010), and TAQ-R scores have been found to be predictive of treatment outcome (Bickman, et al., 2012).

Parent rated measures.

Working Alliance Inventory-Short Form (WAI-SF; Horvath & Greenberg,

1989; Tracey & Kokotovic, 1989). The WAI-SF is a parent-rated measure of therapeutic alliance with the treating clinician. It was originally designed to measure therapeutic alliance in adult psychotherapy, and has been successfully adapted to assess therapeutic alliance of parents of children in psychotherapy (Hawley & Garland, 2008). It consists of 12 items rated on a 1-7 Likert scale ranging from "never" to "always," and focuses on parental agreement on the tasks to be performed in their child's treatment, parental

agreement with the clinician on the goals of their child's treatment, and the therapeutic bond between parent and clinician. When used with parents of children in psychotherapy, it has demonstrated acceptable internal consistency and test-retest reliability (alpha>.93, 6-month testretest r=.77; Hawley & Garland, 2008) and has predicted improvements in youth psychopathology and parent satisfaction during psychosocial treatment (Hawley & Garland, 2008).

Credibility/Expectancy Questionnaire-Parent Version (CEQ-P; Nock, Ferriter, &

Holmberg, 2006). The CEQ-P is a measure of parental outcome expectations and treatment credibility. It consists of 6 items, with factor analysis supporting a two-factor model of 3 items each (falling along the lines of treatment credibility and expectancies). The present study employs the expectancy subscale of the CEQ-P, which has demonstrated strong internal consistency (alpha=.88) and adequate test-retest reliability after 6-8 sessions of psychotherapy (r=.52; Nock, et al., 2006). Scores on the CEQ-P have been associated with parent motivation to participate in treatment, and the CEQ-P expectancy subscale has correlated with parental treatment adherence in pediatric psychotherapy (Nock, et al., 2006).

Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). The CBCL is a parent-report measure of internalizing and externalizing symptoms in children over the past six months. It consists of 118 items rated on a 0-2 Likert scale ranging from "not true" to "very true," and total scores are computed for overall internalizing and externalizing behavior. Based on a normative sample, internal consistency has been observed to be .90, .94, and .97 for the internalizing, externalizing, and total scores, respectively (as measured by Cronbach's alpha), and eight day test-retest reliability has been observed at .91, .92, and .94 for the internalizing, externalizing, and total scores, respectively (Achenbach & Rescorla, 2001). A t-score of 60 or

above has been found to differentiate youths based on treatment referral status, where treatment referral status can be determined with 85% accuracy on the CBCL (Achenbach & Rescorla, 2001).

Brief Progress Monitor-Parent Version (BPM-P; Achenbach, McConaughy,

Ivanova, & Rescorla, 2011). The BPM-P is a measure designed to track changes in child psychopathology throughout treatment. It includes 19 parent-rated items that are scored on the same 0-2 Likert scale as the CBCL. It evaluates internalizing and externalizing psychopathology as well as symptoms of inattentiveness, and consists of a subset of items from the Child Behavior Checklist (Achenbach & Rescorla, 2001) that were identified via factor analysis with the intention of providing a brief, reliable, and valid measure of symptom tracking over time. Internal consistency (as measured by Cronbach's alpha) has been observed to be .80, .85, .88, and .92, for the internalizing, inattentiveness, externalizing, and total scores, respectively (Achenbach, McConaughy, Ivanova, & Rescorla, 2011). Eight day test-retest reliability has been observed to range from .81 to .85 for each of these scores, and the total score has been shown to effectively identify the presence of a psychiatric diagnosis and the use of mental health services in the prior year (Tarren-Sweeney, 2013).

Brief Medication Questionnaire (BMQ; Svarstad, Chewning, Sleath, &

Claesson, 1999). The BMQ is a parent-rated measure of adherence to pharmacotherapy. The 7-item medication regimen screen portion of the BMQ was employed via phone follow-up. Brief Medication Questionnaire items have shown test-retest stability (Rickles & Svarstad, 2007), have correlated with adherence measurement via electronic monitoring (Shi et al., 2010), and have been used successfully to evaluate psychiatric

medication administration with pediatric caregivers (Dean, Wragg, Draper, & McDermott, 2011). Percentage of medication adherence over the prior week can be calculated following procedures described by Curtin, Keller, and Svarstad (1999), which consist of dividing the number of doses missed over the past week by the number of prescribed doses indicated for the week, and then subtracting this value from 1.

Service Assessment for Children and Adolescents (SACA; Stiffman et al., 2000). The

SACA is a measure of service utilization in children and adolescents. In its original form, it consists of 30 items administered via parent interview. It has demonstrated excellent agreement in service utilization measurement when compared to service records (kappa=.76; Hoagwood et al., 2000), and it is frequently modified to match specific patient populations while maintaining reliability and validity (Stiffman et al., 2005). To reduce participant burden, a 13-item version was used in the present investigation, which combines items regarding outpatient, inpatient, and community-based services while still covering all relevant services received.

Frequency, Intensity, and Burden of Side Effects Rating/Patient Rated Inventory of Side Effects (FIBSER/PRISE; Wisniewski et al., 2006). The PRISE is a checklist of side effects experienced during pharmacotherapy, and the FIBSER is a rating of the frequency, intensity, and burden of side effects experienced from taking psychiatric medication. The PRISE (a 9-item presence/absence checklist) is used to highlight relevant side effects before the administration of the FIBSER (a 3-item measure rated on a 0-6 Likert scale). The FIBSER has demonstrated strong internal consistency (alpha=.91-.93), and higher scores on the FIBSER have been associated with treatment dropout in a randomized controlled trial for adult depression (Wisniewski, Rush, Balasubramani, Trivedi, & Nierenberg, 2006). The FIBSER and PRISE were originally designed for use with adults. They were modified to be used by parents to evaluate

their child's side effects for the present investigation; items have been inspected by a board-certified child and adolescent psychiatrist (Dr. Mark Cavitt at the ACH study site) to ensure item appropriateness for the study population.

Child rated measures.

Therapeutic Alliance Scale for Children-Revised (TASC-R; Shirk & Saiz,

1992). The TASC-R is a measure of youth therapeutic alliance in treatment. Its 12 items are rated on a 4-point Likert scale and assesses bond with the clinician and agreement with the clinician on therapeutic tasks. The TASC-R provides minor wording modifications to the original TASC so that it can address therapeutic alliance at a specific treatment session, whereas the original TASC measures alliance across multiple sessions (Creed & Kendall, 2005). Internal consistency of the TASC-R has been demonstrated for children ages 7-17 (alpha=.88-.92; Creed & Kendall, 2005; Keeley, Geffken, Ricketts, McNamara, & Storch, 2011). Acceptable levels of test-retest reliability have been observed over a period of five psychotherapy sessions (*r*=.60; Keeley et al., unpublished data), and TASC-R scores at session 5 have predicted treatment outcome in CBT for pediatric OCD (Keeley et al., 2011).

Credibility/Expectancy Questionnaire-Child Version (CEQ-C). The CEQ-C is a child-rated assessment of outcome expectancies and treatment credibility. Its 6 items were designed to parallel items on the adult CEQ (Devilly & Borkovec, 2000). It was created for the present investigation due to the lack of empirically validated measures of treatment expectancies for youth. The CEQ was originally developed as a modification to the Expectancies Rating Questionnaire (ERQ; Borkovec & Nau, 1972) to reduce confounding with treatment credibility observed in the ERQ. Similar content is shared

among the measures, and the ERQ has been successfully used with youth ages 7 years and above (Ollendick et al., 2009), suggesting appropriateness of item content for children. The present study employs the expectancy subscale of the CEQ-C, which has three items that are rated on either a 9- or 11-point Likert scale. When used with adults, this subscale has been observed to have adequate internal consistency (alpha=79-.90), with a 1-week test-retest reliability value of r=.82 (Devilly & Borkovec, 2000). The two-factor structure has been supported in adults by confirmatory factor analysis, and expectancy subscale scores have been correlated with reductions in anxiety and global distress during treatment for adult anxiety (Devilly & Borkovec, 2000).

Youth Self Report (YSR; Achenbach & Rescorla, 2001). The YSR is a child-report measure of internalizing and externalizing behavioral problems in children that is intended to parallel the CBCL. It consists of 112 items rated on a 0-2 Likert scale ranging from "not true" to "very true or often true," resulting in total scores for overall internalizing and externalizing behavior. While originally designed for youths ages 11-17, the YSR has demonstrated strong internal consistency for youth ages 7 and above, with Cronbach's alpha values of .88-.89, .88-.89, and .93 observed for the internalizing, externalizing, and total problems scales respectively (Ebesutani, Bernstein, Martinez, Chorpita, & Weisz, 2011). A t-score of 60 or above has been found to differentiate youths based on clinical referral status (Achenbach & Rescorla, 2001). Items focusing on drug use and sexual behavior were not included, as it was not necessary to expose younger participants to these items for the purposes of the present investigation, and adjustments to YSR drug use items have been successfully employed in other investigations (e.g., Substance Abuse and Mental Health Services Administration, 1999).
Multidimensional Anxiety Scale for Children-Anxiety Disorders Index (MASC-ADI; March, Parker, Sullivan, Stallings, & Conners, 1997). The MASC-ADI is brief measure of anxiety symptomology in children. It consists of 10 items rated on a 0-4 scale that ranges from "never true about me" to "often true about me." These items are a subset of items from the 39-item MASC that were identified to best discriminate children with anxiety disorders from nonclinical control participants. Psychometric adequacy has been demonstrated with children ages 7-17 years, with an observed internal consistency of alpha=.74 (Grills-Taquechel, Ollendick, & Fisak, 2008) and three month test-retest reliability of r=.70 (March, Parker, Sullivan, Stallings, & Conners, 1997). When used as a screening instrument to differentiate anxious children from nonclinical controls, the MASC-ADI has displayed sensitivity values of .90-.95, specificity values of .84-.95, an overall correct classification rate of 87-95%, and kappa values of .74-.90 (March, 1997). The MASC-ADI has also been able to distinguish children with anxiety disorders from nonclinical controls when using discriminant function analyses (Grills-Taquechel et al., 2008; Rynn et al., 2006).

Children's Depression Inventory-2nd Edition Short Form (CDI-2-SF; Kovacs, 2011). The CDI-2-SF is a child-rated measure of depressive symptoms intended for children ages 7-17 that was empirically derived from the 28 item CDI-2 long form. Its 10 items are rated on a 0-2 scale and cover the affective, cognitive, and neurovegetative aspects of pediatric depression, and a total score is produced to quantify overall severity of depression. Internal consistency for the CDI-2-SF has been observed at alpha=.82, its two week test-retest reliability has been indicated to be r=.77, and it is strongly correlated with the CDI-2 long form (r=.95, p<.001; Kovacs, 2011). The CDI-2-SF has displayed

sensitivity and specificity values of .84 and .77, respectively, when used to differentiate children meeting DSM-IV criteria for major depressive disorder from matched controls (Kovacs, 2011). For predicting clinician diagnosis, Kovacs (2011) recommends the CDI-2-SF in lieu of the complete CDI, as it has displayed stronger psychometric properties for this purpose than the full CDI-2 form (e.g., better test-retest reliability, better discriminant validity, and improved sensitivity and specificity when using a clinical cutoff score of 6).

Analytic Plan

The present study design reflected multiple levels of nesting. Because multiple medications were allowed for each patient at each timepoint, there were multiple observations per timepoint, multiple timepoints per patient, multiple patients per clinician, and multiple clinicians per site. To identify if method-based adjustments for nested data clustering were necessary, design effects for all dependent variables were considered, where design effects greater than 2 reflect a need to adjust estimates for clustering (Muthén & Satorra, 1995). Among nesting variables (i.e., time, participant, clinician, and site), design effects were greater than 2 only for clinician and site. To address nesting by clinicians, the MLR-complex estimator was employed for all analyses, with clinician specified as the clustering variable. To address nesting by site, dependent variables in all analyses were predicted by dummy-coded site variables before adding predictors and covariates, reflecting a fixed effects approach to clustering (as specified by Cohen, Cohen, West, & Aiken, 2003). The distribution of adherence was nonnormal, with disproportionate amounts of patients displaying either very low or very high adherence. This nonnormality was addressed by use of the MLR-complex estimator.

All bivariate relationships tested as a part of study hypotheses are displayed in Figure 1. Figure 1 shows a pattern of effects that reflect expectancies and motivation as pretreatment

factors, which then affect subsequent alliance; alliance then affects adherence, and adherence then affects treatment outcome. Relationships among variables that were not modeled as unidirectional causes of one another were defined as exogenous, while endogenous relationships were defined when variables were determined by other variables in the system. Relationships among exogenous variables were evaluated by correlational methods, while relationships between exogenous and endogenous variables were evaluated by regression methods. Correlations tested were partial correlations (i.e., correlating the residuals after partialing out the effects of site out of each variable, in order to account for site-based clustering). Regressions were evaluated by first regressing dependent variables onto dummy-coded site, and then adding hypothesized predictors to this model. To evaluate prediction of treatment outcome, 3-month BPM-P scores were first regressed on both site and baseline scores, and then hypothesized predictors were added to this model, reflecting a residualized regression. Outcomes were considered separately for each BPM-P subscale (Internalizing, Externalizing, Attention Problems).

Moderation of bivariate regressions was also considered, with the effects of each individual moderator evaluated in separate models. Moderators included type of medication (including stimulants, SRIs, atypical antipsychotics, alpha-2 agonists, and a comparison was also made between patients who received medication at all and those who did not), disorder class (including internalizing, externalizing, neurodevelopmental, ADHD, and tic-related disorders), patient age, concurrent treatment as reported at 1- and 3-month follow-up, and respective FIBSER items corresponding to frequency, intensity, and interference associated with side effects. Medications were classified based on category definitions established in the Federal Drug Administration National Drug Code Directory (Federal Drug Administration, 2017).

Disorders were classified based on suggestions in the DSM-5 (American Psychiatric Association, 2013), which notes that a series of papers commissioned by Andrews (2009) shaped classification schemes and provided further information regarding the rationale for categorization. When the Andrews (2009) papers disagreed with final DSM-5 classification, disorders in question were evaluated in their own class. This affected ADHD (which is considered with neurodevelopmental disorders in DSM-5, but shows strong relationships with externalizing disorders both conceptually and in the Andrews, 2009 papers) and tic disorders (which were unclassified in the Andrews, 2009 papers and show limited phenomenological overlap with other disorder classes evaluated in this study). Moderators were evaluated in analyses if they were observed more than 15 times in the study sample in order to accommodate central limit theory assumptions (Smith & Wells, 2006). Moderation models were computed by adding the moderator and its product with the main effect to regression models; significant moderation was evaluated based on the product term. All dichotomous moderators were dummycoded, and all continuous predictors were centered prior to creating moderator product terms used in models. To evaluate the results of statistically significant moderation, simple slopes were evaluated following procedures delineated by Preacher, Curran, & Bauer (2006). Simple slopes were evaluated at low, medium, and high levels of study moderators (with medium levels reflecting the mean of the moderator and low and high levels reflecting ± 1 SD, or only with 0 and 1 reflecting low and high levels respectively for dummy-coded moderators). Because moderators reflect post-hoc hypotheses, a Holm-Bonferroni correction (Holm, 1979) was used on all moderation analyses to determine statistical significance. This correction limits familywise Type I error while retaining more power than the traditional Bonferroni correction. As

recommended by Fairchild and McQuillin (2010), significance of simple slopes was tested via unstandardized coefficients. Standardized coefficients are reported to facilitate interpretation.

Figure 1 also implies mediation-based relationships. Indirect effects were tested for all variables that showed significant a and b paths in main effect and/or moderated models (e.g., if alliance significantly predicted adherence and then adherence significantly predicted outcomes in separate bivariate models). Significant direct effects were not required to consider a test of moderation given that a number of processes can lead to significant indirect effects in the absence of direct effects (Hayes, 2013). Moderators that were significant after Holm-Bonferroni correction were also included in respective indirect models (which reflects a process of moderated mediation; please see Figure 2 for a visual depiction of modeled indirect paths). Simple slopes in moderated mediation models were evaluated following procedures delineated by Hayes (2013), and Mplus implementations of these procedures provided by Stride, Gardner, Catley, and Thomas (2015) were employed when available. These procedures focus on evaluating the significance of the product of the indirect paths (i.e., ab for a single moderator or abc for 2 sequential moderators), and also take into account the direct effect of X on Y (c'). Standard errors for tests of indirect effects were calculated based on the delta method. As with other analyses, site-based clustering was accounted for by including dummy-coded site variables on all DVs in indirect effect models. Inconsistent mediation was observed when the indirect effect had the opposite sign of direct effects (MacKinnon, Fairchild, & Fritz, 2007).

With respect to effect sizes, small, medium, and large effect sizes respectively apply to partial correlations of .1, .3, and .5, and regression model R^2 values of .01, .09, and .25 (Cohen, 1988). For main effects in regression models, R^2 was evaluated based on the amount of variance a main effect accounted for above and beyond site (defined as ME- R^2 for main effects) and for

moderators, R^2 was evaluated based on how much variance was explained by adding the moderator and the moderator product term to the main effect model (defined as ΔR^2). Effect sizes reported for indirect effects were based on recommendations by Wen and Fan (2015) to report standardized estimates of the indirect, direct effect, and total effects. To illustrate, Wen and Fan (2015) provided an example based on standardized estimates, where if the indirect effect is 0.2, the direct effect is 0.3, and the total effect is 0.5, then it can be interpreted that for a change of 1 standard deviation in X, Y will change 0.5 standard deviations, of which 0.2 standard deviations are attributable to the indirect effect and 0.3 standard deviations are attributable to the direct effect. Regarding measure reliability, internal consistency was evaluated through the use of Cronbach's alpha (Cronbach, 1951), with alpha values of .70 or higher considered acceptable (Nunnally, 1978).

Data were missing at a rate of 26.4% on all variables modeled in Figure 1. The principal contributor to these missing data was follow-up response rate, as 52.2% of participants participated in 1-month follow-up and 53.5% of participants participated in 3-month follow-up. In addition, 20 youths among participating families (12.6%) did not provide assent to participation, for reasons including parents not wanting to add additional work to children during a clinic visit (n=4), children preferring to engage in other activity instead of participating (n=3), child/parent expressing concern that the child would be able to comprehend the self-report assessments (n=6), and 7 families did not provide a specific reason for choosing not to provide child assent. In cases of parental consent but lack of child assent, parents were willing to continue to provide behavioral data from themselves, but no child report was provided. To address missing data, several measures were taken. All hypothesis tests were estimated using full-information maximum likelihood estimation in Mplus version 7 (Muthén & Muthén, 2012).

Auxiliary variables were used to aid in the estimation of missing data via a saturated correlates model; auxiliary variables included all common factors as rated by parents, children, and clinicians, as well as baseline scores on all BPM-P subscales. The effects of clinician and site were also included as auxiliary variables in all models (as a part of accounting for their clustering effects in all models). Means and variances of all predictors and covariates were also estimated via FIML estimation (as opposed to assumed as fixed) to aid in parameter estimation in the presence of missing data.

Results

Descriptive Variable Characteristics and Relationships Among Exogenous Variables

Descriptive statistics for the present sample can be found in Table 2; internal consistency for all measures was acceptable. Correlations among exogenous study variables can be found in Table 3. Among pretreatment exogenous variables, child motivation (via the URICA-C) was significantly correlated with all other pretreatment exogenous variables, including the CEQ-C (partial r=0.34, p=.002), the CEQ-P (partial r=0.16, p=.031), and the URICA-P (partial r=0.23, p=.006). Regarding relationships among alliance reporters, clinician-reported alliance with parents was significantly related with parent-rated alliance (via the WAI-SF; partial r=0.26, p<.001) and clinicians' ratings of their alliance with participating children (via the TAQ-R-P; partial r=0.26, p=.008). Additionally, clinician ratings of their alliance with children was significantly correlated with child ratings of alliance (via the WAI-SF; partial r=0.47, p<.001). The correlation among adherence measurements at follow-up at 1-month and 3-months was also significant (partial r=0.55, p<.001).

Bivariate Predictive Relationships

Bivariate predictive relationships for all main effects and significant moderators of main effects can be found in Table 4.

Prediction of alliance by motivation and expectancies. Child-rated alliance showed a significant relationship with child motivation (b=0.52, p<.001, ME-R²=.24). Child-rated alliance had a significant relationship with child expectancies and parental motivation, though these

effects varied in the presence of moderators. Child expectancies predicted child alliance for youth without ADHD (b=0.62, p<.001, ME-R²=0.08, ΔR^2 =0.15). It also predicted child alliance for youth on alpha-2 agonists, though it showed a positive relationship for youth who were not on these medications (*b*=0.31, *p*<.001, ME-R²=0.08, ΔR^2 =0.16) and a negative relationship for youth who received them (*b*=-0.02, *p*<.001, ME-R²=0.08, ΔR^2 =0.15). It also only predicted child alliance for youth who had low (b=1.03, p<.001, ME-R²=0.08, $\Delta R^2=0.26$) and medium (b=0.36, p < .001, ME-R²=0.08, $\Delta R^2 = 0.26$) intensity of side effects. Child alliance was also predicted by parent motivation, but showed a varying relationship based on concurrent treatment received during the 3 months post-baseline; youth who did not receive concurrent treatment during this time period showed a negative relationship between child alliance and parent motivation (b=-0.26, p<.001, ME-R²=0.01, ΔR^2 =0.12), while those who did receive concurrent treatment showed a positive relationship between these variables (b=0.18, p=.012, ME-R²=0.01, $\Delta R^2 = 0.12$). Parent-rated alliance was significantly predicted by parental expectancies (b=0.22, p=.034, ME-R²=0.05) and parent motivation (b=0.30, p<.001, ME-R²=0.09). It was also predicted by child motivation, but only for youth who were prescribed atypical antipsychotics (where it had a negative relationship for these youth; b=-0.21, p<.001, ME-R²=0.00, $\Delta R^2=0.05$).

Among pretreatment exogenous variables, the only one that significantly predicted clinician-rated alliance with children was child motivation (b=0.21, p=.014, ME-R²=0.04). With regard to predicting clinician-rated alliance with parents, child and parent motivation were found to be significant predictors of clinician-rated alliance with parents, though both of these effects were found to be moderated. The effect of child motivation on TAQ-R-P scores was moderated by prescription of atypical antipsychotics, with a stronger relationship for youth who were not prescribed these medications (b=0.27, p=.005, ME-R²=0.04, $\Delta R^2=0.09$), though a significant positive relationship was also found for youth who received these medications (*b*=0.03, *p*<.001, ME-R²=0.04, ΔR^2 =0.09). The effects of parent motivation on TAQ-R-P scores were moderated by externalizing diagnosis; a negative relationship between motivation and alliance was found for youth without externalizing diagnoses (*b*=-0.22, *p*=.021, ME-R²=0.02, ΔR^2 =0.10), whereas a positive relationship was found for those who did have an externalizing diagnosis (*b*=0.22, *p*<.001, ME-R²=0.02, ΔR^2 =0.10). Child and parent expectancies were not significantly related with clinician-rated alliance with parents.

Prediction of medication adherence by motivation, expectancies, and alliance. At 1month follow-up, medication adherence was negatively associated with parental expectancies $(b=-0.34, p<.001, \text{ME-R}^2=0.11)$ and child motivation $(b=-0.28, p=.029, \text{ME-R}^2=0.07)$. It also exhibited a positive relationship with clinician-rated alliance with children for participants who were prescribed atypical antipsychotics $(b=0.14, p<.001, \text{ME-R}^2=0.00, \Delta R^2=0.05)$. All other psychosocial variables were nonsignificant predictors of medication adherence at 1-month follow-up. The only significant predictor of medication adherence at 3-month follow up was parent expectancies, which was again associated with lower adherence $(b=-0.19, p=.014, \text{ME-R}^2=0.05)$.

Prediction of treatment outcome by adherence, motivation, expectancies, alliance, and medication adherence. With regard to predicting change in externalizing symptoms, all significant predictors of BPM-P Externalizing scores reflected a worsening of symptoms. This included child expectancies for youth with tic disorders (*b*=0.04, *p*<.019, ME-R²=0.01, ΔR^2 =0.06), child motivation for youth with tic disorders (*b*=0.14, *p*<.001, ME-R²=0.00, ΔR^2 =0.09), and parent alliance for youth who did not have externalizing disorders (*b*=0.40, *p*<.001, ME-R²=0.01, ΔR^2 =0.03).

With regard to predicting change in internalizing symptoms, clinician-rated alliance with parents was associated with a reduction in symptoms for youth prescribed atypical antipsychotics (*b*=-0.20, *p*<.001, ME-R²=0.00, Δ R²=0.06). An increase in internalizing symptoms was associated with higher levels of child motivation (*b*=0.27, *p*<.001, ME-R²=0.06), child expectancies for youth who were prescribed atypical antipsychotics (*b*=0.13, *p*<.001, ME-R²=0.00, Δ R²=0.04), and those prescribed alpha agonists who had higher levels of medication adherence at 1-month follow-up (*b*=0.83, *p*<.001, ME-R²=0.03, Δ R²=0.07). Higher parent motivation was associated with internalizing symptom reduction in youth who did not receive a prescription at all (*b*=-0.18, *p*=.023, ME-R²=0.00, Δ R²=0.04), but symptom increases in youth who did receive a prescription (relative to no prescription at all; *b*=0.08, *p*<.001, ME-R²=0.00, Δ R²=0.04). Higher child alliance was associated with internalizing symptomic (*b*=-0.22, *p*=.019, ME-R²=0.02, Δ R²=0.09), but symptom increases in youth who were prescription of atypical antipsychotics (*b*=-0.22, *p*=.019, ME-R²=0.02, Δ R²=0.09), but symptom increases in youth who were prescribed atypical antipsychotics (*b*=-0.23, *p*<-.001, ME-R²=0.02, Δ R²=0.09).

With regard to predicting change in attention problems, a reduction in attention problems was associated with child alliance (*b*=-0.15, *p*=.047, ME-R²=0.02) as well as parent expectancies for children who experienced side effects at medium (*b*=-0.24, *p*<.001, ME-R²=0.07, ΔR^2 =0.18) and high (*b*=-0.77, *p*<.001, ME-R²=0.07, ΔR^2 =0.18) levels of intensity. Worsening of attention problems was seen for higher levels of clinician-rated alliance with parents for children who had not received concurrent treatment by 3-month follow-up (*b*=0.42, *p*<.001, ME-R²=0.00, ΔR^2 =0.04).

Relationships of common factors with change in attention problems varied at different levels of moderators. Higher parent alliance was associated with a reduction in attention problems for children who experienced low levels of functional interference due to side effects $(b=-0.54, p=.002, \text{ME-R}^2=0.02, \Delta R^2=0.20)$, but a worsening of symptoms for youth who experienced high levels of functional interference due to side effects $(b=0.75, p<.001, \text{ME-R}^2=0.02, \Delta R^2=0.20)$ and youth who had received concurrent treatment at 3-month follow-up $(b=0.35, p<.001, \text{ME-R}^2=0.02, \Delta R^2=0.16)$. Additionally, greater adherence at 1-month follow-up was associated with a reduction in attention problems for youth with low intensity of side effects $(b=-0.49, p=.015, \text{ME-R}^2=0.01, \Delta R^2=0.17)$, but an increase in symptoms for youth with high intensity of side effects $(b=0.61, p<.001, \text{ME-R}^2=0.01, \Delta R^2=0.17)$.

Indirect Predictive Relationships (Mediation)

Results from analyses of therapeutic processes can be seen in Table 5, which correspond to the models depicted in Figure 2. With regard to the process externalizing symptom change, child motivation had an indirect influence on symptom reduction through parent alliance for youth prescribed atypical antipsychotics (ab=-0.48, p=.013). Inconsistent mediation was observed in this case for children who had tic disorders (i.e., the direct effect was associated with an increase in symptoms for children with tic disorders whereas the indirect effect was associated with a decrease in symptoms; c'=1.11, p<.001). Parent motivation was also indirectly associated through parent alliance with an increase in externalizing symptoms for children who did not have an externalizing diagnosis (ab=0.18, p=.010)

With regard to the process of influence of internalizing symptom change, multiple expectancy factors showed an indirect effect on change through alliance. In particular, the effect of child expectancies on change in internalizing symptoms through child alliance varied depending on moderators. Child expectancies were associated with a worsening of symptoms for youth who were prescribed atypical antipsychotics but did not have an ADHD diagnosis

(*ab*=0.59, *p*<.001). In the context of youth with either/or prescriptions of alpha-2 agonistics or atypical antipsychotics, higher child expectancies were associated with a worsening of symptoms for youth who were prescribed either alpha-2 agonists (*ab*=0.26, *p*=.016) or atypical antipsychotics (*ab*=0.28, *p*=.012), but not for youth who were prescribed both of these medications simultaneously (*ab*=-0.96, *p*=.061) or who were not prescribed either of these medications (*ab*=-0.07, *p*=.079). The indirect effect of child expectancies on internalizing symptom change through child alliance also differed for youth prescribed atypical antipsychotics in the context of side effect intensity. Higher child expectancies through child alliance were associated with symptom reduction for children who were not prescribed atypical antipsychotics and had either a low (*ab*=-0.24, *p*=.015) or medium intensity of side effects (*ab*=-0.11, *p*=.018), but were associated with a worsening of symptoms for youth who did received these medications and also experienced either low (*ab*=0.80, *p*=.010) or medium (*ab*=0.36, *p*=.030) intensity of side effects.

Motivation also had multiple indirect effects on internalizing symptom outcome. Child motivation indirectly predicted through child alliance an improvement of internalizing symptoms for children who were not prescribed atypical antipsychotics (ab=-0.31, p=.004); however, inconsistent mediation was found in this case as the direct effect of child motivation was associated with a worsening of internalizing symptoms (c'=0.57, p<.001). Parent motivation also predicted internalizing symptoms through clinician-rated alliance with parents; a worsening of symptoms was seen for youth who were prescribed atypical antipsychotics and did not have an externalizing diagnosis (ab=0.29, p=.005), but a reduction of symptoms was observed for children who received these medications and did have an internalizing diagnosis (ab=-0.50, p=.004).

Adherence also served as a mediator of treatment process variable effects on internalizing symptoms. Better internalizing outcomes were observed for youth who were prescribed alpha-2 agonists and who also had higher parent expectancies (ab=-2.55, p=.001) and higher child motivation (ab=-2.75, p=.029). This latter effect reflected inconsistent mediation, as the direct effect of child motivation on internalizing symptom change through adherence was associated with a worsening of symptoms (c'=0.47, p=.001). Clinician-rated alliance with children was also indirectly associated through 1-month adherence with a worsening of symptoms for youth who were prescribed both atypical antipsychotics and alpha-2 agonists (ab=6.31, p=.016).

With regard to the process of influence of attention problems, multiple expectancy and motivation factors showed an indirect effect on change through alliance. Worsening of symptoms was predicted by higher child expectancies through child alliance for youth who were prescribed alpha-2 agonists (ab=0.21, p=.042). Higher parent expectancies were associated with attention problem exacerbation directly through parent alliance for youth who received concurrent treatment by 1-month follow-up (ab=0.25, p=.029); however, these symptoms were inconsistent with direct effect symptom reductions for youth who had medium (c'=-0.53, p < .001) and high (c'=-1.05, p < .001) intensity levels of side effects. Higher levels of parent motivation were associated with worsening of attention problems indirectly through child alliance for youth who were prescribed stimulants (ab=0.05, p=.049) and indirectly thorough parent alliance for youth who received concurrent treatment by 1-month follow-up (*ab*=0.39, p < .001) and who had high levels of functional interference due to side effects (ab=0.30, p=.001). Parent motivation was also associated with worsening of attention systems indirectly thorough clinician-rated alliance with parents for youth who had an externalizing diagnosis and did not receive concurrent treatment by 3-month follow-up (ab=0.32, p=.002).

The indirect effect of child motivation on attention problems varied depending on moderators. For youth who were prescribed atypical antipsychotics, increased child motivation was associated with an increase in attention problems indirectly through parent alliance in youth with low levels of functional interference due to side effects (ab=0.35, p=.029) and a reduction in attention problems in youth who had high levels of functional interference due to side effects (*ab*=-0.66, *p*<.001). It was also associated with an indirect reduction of attention problems through parent alliance for youth who were prescribed antipsychotics and received concurrent treatment by 1-month follow-up (ab=-0.93, p<.001). For youth who did not receive concurrent treatment by 3-month follow-up, it was associated through clinician-reported alliance with parents with an exacerbation of symptoms for youth who were not prescribed atypical antipsychotics (ab=0.27, p=.015), though this reflected inconsistent mediation relative to the direct effect (c'=-0.39, p=.037), and also a reduction of symptoms for these youth was observed when they were prescribed atypical antipsychotics (ab=-0.49, p=.002). Adherence also served as a mediator of treatment process effects on attention problems, as clinician-rated alliance with children through 1-month adherence was indirectly associated with an increase in attention symptoms for youth prescribed atypical antipsychotics and who experienced a high intensity of side effects (ab=0.63, p=.023).

The pattern of significance in bivariate analyses also resulted in one mediation model that had 2 mediators in sequence, which is depicted in Figure 2. From this model, child motivation showed an indirect effect through both clinician-reported alliance with children and adherence at 1-month follow-up; this indirect effect depended on prescription of atypical antipsychotics and intensity of side effects. For youth with high intensity of side effects, a reduction in attention problems was observed when youth were not prescribed atypical antipsychotics (*abc*=-0.12,

p=.039), but an increase in attention problems was observed when atypical antipsychotics were prescribed (abc=0.13, p=.015).

Discussion

The objective of this study was to examine the role of psychosocial variables in pediatric psychiatric treatment, including pretreatment expectancies, motivation for behavior change, therapeutic alliance, and medication adherence. It was anticipated that these variables would have positive relationships with each other and would be associated with symptom reduction. While the psychosocial variables largely showed positive relationships with each other, there were some unexpected inverse relationships, and there were many unexpected associations between psychosocial variables and exacerbated clinical symptoms.

The main theme that encompasses the pattern of observed effects is that they were far less uniform than is usually reported in common factors literature. Usually, common factors are construed as single entities that only show positive relationships with each other and treatment outcome. While this pattern was observed at times, in the present investigation common factors were observed to function in different manners across populations, reporters, and interventions, frequently in a counterintuitive manner. These results suggest that common factors are not as theoretically consistent as is often portrayed in the mental health literature. Or, they may not be as theoretically consistent in child psychiatry as they are in other behavioral and medical fields. Psychosocial correlates of treatment outcome are underexplored in primarily psychiatric settings, which gives little basis for comparison. In pediatric psychotherapy, treatment process variables have been observed to show differing roles across treatment approaches (Hogue et al., 2006; Karver et al., 2008), and perhaps their function may differ even more markedly when

considering psychiatric approaches. It may also be that negative associations with common factors in other studies are weaker than the positive ones (as was observed in the Shirk et al., 2011 meta-analysis for child alliance). In this case, these negative effects may exist, but even then they will be detected and reported less frequently. Given the novel approach of this study and the preliminary nature of its findings, its major contributions are less focused on individual findings. Instead, its primary value lies in highlighting that psychosocial variables have a notable impact on psychopharmacological treatment outcome in children, identifying that their influence may be much more complicated than is commonly portrayed, and identifying hypotheses for the origins of this complexity.

Despite the observed inconsistencies, a number of patterns emerged among results that can be placed in context with prior literature. Regarding exogenous variables, significant correlations were all observed in the expected positive directions. Of note, the majority of nonsignificant correlations reflected cross-informant correlations (i.e., nonsignificant correlations were almost always those between a child-related construct and a parent-related construct). The only exception to this pattern was the nonsignificant correlation between parental motivation and parental expectancies. This disagreement across reporters has been well documented in pediatric mental health research (Martel, Markon, & Smith, 2016) and thus may reflect differences in reporter perspectives in addition to lack of construct overlap. With regard to expectancies and motivation, the most consistent correlate was child motivation, as more motivated children also had higher expectancies and had parents who had higher motivation and expected better treatment outcome. Child motivation is an underexplored construct, as the majority of motivation research in child psychopathology focuses on parental motivation. These findings suggest that it may not only be related to outcomes, but also may serve as a unique indicator of positive ratings

on other psychosocial constructs. One exception to the observed pattern of correlations was that that parent motivation and parental expectancies were not significantly correlated. While adult motivation and expectancies are often related, there are reports of nonsignificant relationships among the constructs (e.g., Vogel et al., 2006). The magnitude of the observed correlation was similar to other significant correlations in this study, reflecting an effect of measurement error on the detection of a significant relationship.

Several patterns also emerged in predictive models, which applied to bivariate approaches as well as moderated and mediated approaches. A very surprising pattern was the multiple associations of therapeutic alliance with an increase in symptoms; a common thread among many of these observations is that such worsening occurred only in the presence of markers of externalizing behavior (e.g., prescription of medications that are used for externalizing conditions such as alpha-2 agonists or atypical antipsychotics). Although alliance is usually associated with improved treatment outcome, with these patients early session alliance has been associated at times with an increase of symptoms in psychotherapy (Florsheim, Shotorbani, Guest-Warnick, Barratt, & Hwang, 2000; Hogue, Dauber, Stambaugh, Cecero, & Liddle, 2006). Others have observed that some of these patients may actually need more clinician authority and boundary setting early on in the therapeutic relationship (Gallagher, Kurtz, & Blackwell, 2010). Different therapist engagement behaviors can be differentially effective depending on the treatment approach (Karver et al., 2008), and youth with externalizing behavior are an identifiable target population for using modified engagement techniques (Karver & Caporino, 2010). Additionally, some of these patients may be adept at positive self-presentation for a limited period of time early in treatment, which may be a marker for poorer outcome (Florsheim et al., 2000). An ideal therapeutic relationship in early sessions may then differ for

these patients, as warmth and emotional support may be more appropriate for patients who are less likely to test clinician boundaries, while an authoritative but empathic early relationship may set the stage for better outcomes for patients who may be more likely to initiate discord. The unexpected relationships between alliance and outcome for these patients was also reflected in observed negative indirect effects, as any positive effects of expectancies/motivation on outcome were reversed when communicated through a negative relationship between alliance and outcome. This indirect effect was observed despite positive direct effects at times (e.g., a positive direct effect of child motivation on symptom reduction), leading to inconsistent mediation. This pattern of moderation was also seen in prediction of alliance, as alliance more frequently had a positive relationship with other common factors for patients who did not match the externalizing/ADHD profile.

Similarly surprising was the frequently negative association between higher expectancies and treatment outcome. While expectancies have often been associated with positive treatment outcome, there has been more variability in the relationship between expectancies and treatment relative to other common factors (Greenberg et al., 2006). In addition, overly high expectancies can be associated with poorer treatment overcome should treatment response not match these high standards of expectations (Constantino et al., 2011; Gaitan-Sierra & Hyland, 2015). Accordingly, clinicians may wish to carefully monitor not only the overall level of expectancies, but also consider whether they are appropriate given the expected outcomes.

Another unexpected result was the pattern of relationships observed with adherence. A limited and confounded set of relationships were detected between adherence and other common factors as well as treatment outcome, as opposed to the uniformly positive associations expected. Most remarkably, stronger adherence was at times associated with poorer outcomes. However,

this was generally in the presence of stronger side effects or medications for youth with externalizing problems, whereas youth with fewer side effects more frequently showed a positive relationship between stronger adherence and positive treatment outcome. It may be that if medications are aversive, adhering to them strongly actually does not help outcome and may be making matters worse in the long run. In this case, monitoring side effects is not just a factor in premature medication discontinuation (Julius et al., 2009) but is also a possible reason for suboptimal outcomes, even in patients who are taking medications appropriately. Yet, given the strong role of adherence in outcome for a number of conditions, it was still unanticipated that adherence would not be significantly related with outcome or other common factors in many instances.

Additionally, when considering relationships between other common factors and adherence, parent expectancies and child motivation were associated with poorer adherence. Only clinician-rated alliance with children was associated with better medication adherence, and in this case only for youth who were prescribed atypical antipsychotics. While overly optimistic expectancies may have the same deleterious effects on adherence as they can on outcome, the relationship between adherence and motivation cannot be explained in the same fashion. Of note, motivation was assessed in the context of motivation for making personal changes in symptomology, not motivation for adherence (which at times has been considered a separate construct; Touré-Tillery & Fishbach, 2014). Children who may be looking to make active behavioral steps toward change may eventually get there, but may find medications to be a less active means to achieve this end (as has been seen in some patient preference studies; e.g., Angelo, Miller, Zoellner, & Feeny, 2008). Given that child motivation was also related to parent expectancies, children who are more motivated to make active changes themselves may have

parents who are overly expectant of change. Moreover, they may also function better with more active methods of change, and may find a pharmacologically-based intervention to be inconsistent with their preferred approach to change.

In contrast to these unpredicted findings, child motivation was more frequently associated with symptom improvement. This is more consistent with the overall common factors literature. In some ways, motivation differs from other common factors with reference to theoretical relationships with treatment outcome and patterns of moderation. Unlike expectancies, motivation is unlikely to show negative effects on outcome when it is too high. Motivation differs from alliance with regard to moderation, as the nature of an appropriate alliance may differ across internalizing and externalizing populations (Zorzella, Muller, & Cribbie, 2015), whereas the function of motivation is more stable across these groups. For instance, pure warmth for an internalizing patient may serve as enabling for an externalizing patient, whereas the nature of patient motivation does not vary in this between-population manner. Instead, withinpopulation variability is more likely to differ across disorders, as internalizing patients are more often self-referring for treatment and less likely to show poor motivation for change (e.g., Duhig & Phares, 2003). Less within-population variability would also be expected for parents, given that they are frequently the ones who make the decision about whether to initiate child treatment (Nock & Kazdin, 2001). Thus by default parents have motivation for child behavior change, leaving more room for variability in children. This decreased variability in parental motivation may reduce the ability to predict outcomes of interest. Given these facets of parent and child motivation, child motivation appears to be a common factor that is uniquely indicative of future symptom reduction, compared to parent motivation which is less variable.

Notably, several hypothesized moderators did not significantly interact with main effects. In particular, significant moderation was not observed for child age and for variables associated with internalizing and neurodevelopmental disorders (including clinician diagnoses of these conditions and prescription of SRIs). While nonsignificance does not mean that these variables do not have an effect on observed relationships, and post-hoc correction reduces the power to detect their specific influence, it does reflect that they had a lesser influence on the common factors process relative to other moderators. It may be that common factors show a more consistent effect in the presence of these constructs. While there have been some suggestions of developmental differences in the presentation of therapeutic alliance in particular (Shirk, Caporino, & Karver, 2010), the nature of common factors may vary more substantially among youth who are less likely to desire treatment themselves (i.e., children with disruptive/externalizing problems). Frequency of side effects also did not significantly moderate outcomes, while intensity and functional interference associated with side effects were significant moderators. Frequency of symptoms has a different and possibly more limited relationship with mental health symptoms than intensity and functional interference, as patients may have one especially impairing symptom which is very bothersome, or conversely may experience multiple low level symptoms that in aggregate are not very bothersome (Jones et al., 2013). Often in these cases overall symptom severity (as rated by perceived intensity and/or interference) is of greater importance.

With regard to overall magnitude of findings, most significant results in bivariate analyses were in the medium range, which is consistent with meta-analytic estimates of common factors in psychotherapy. Several standardized indirect effects also particularly stood out relative to the others (larger than 2), all of which involved the role of 1-month adherence for youth who

were prescribed alpha-2 agonists and the associated effects on internalizing symptoms. The strong effect of this b path was the common element among these indirect effects. It may be that effects of adherence are particularly pronounced for youth taking alpha-2 agonists, where in this instance increased adherence was associated with worsening of internalizing symptoms. Alpha-2 agonists were notable among study medications in that they have uniquely strong effects of acute withdrawal, as rebound hypertension and distress can occur within even one day of sudden discontinuation (particularly with clonidine; Giovannitti, Thoms, & Crawford, 2015). The distribution of adherence in study participants approximated bimodality, especially for poor adherence; that is, patients who had low adherence had generally stopped prescribed medications altogether, while patients who took medication showed more variability in adherence. While strong but imperfect adherence may be adequate for many medications, required adherence levels may be even higher for alpha-2 agonists, and inconsistent adherence to these medications may be associated with an increase in internal emotional distress. Standard errors for these estimates were fairly large as well, resulting in *p*-values that were not as small as many other significant effects observed. Given this, the size of these indirect estimates may have been affected in part by instability associated with the relatively relative small sample size of youth prescribed alpha-2 agonists in the present sample.

As a whole, the observed patterns of results (and their exceptions) merit replication and evaluation in future research. As such, it is premature to make strong and conclusive inferences from individual findings, but rather the present models suggest new hypotheses that have often not been considered in common factors research (e.g., alliance being associated with increases in symptoms under some conditions, common factors having heterogeneous effects across conditions and interventions). Additional possible reasons for the observed exceptions could

include confounding variables that were unaccounted for (Moore, Neugebauer, van der Laan, & Tager, 2012). For instance, there is some evidence of reciprocal influence of alliance and symptom change in pediatric psychological treatment (Labouliere, Reyes, Shirk, & Karver, 2015) and this longitudinal influence may confound results observed at 3 months after treatment initiation. Oppositional youths in particular may show more instability in the therapeutic relationship throughout the course of treatment (Rauktis, Vides de Andrade, Doucette, McDonough, & Reinhart, 2005). While initial status of psychosocial variables frequently has a substantial influence on subsequent treatment outcome, perhaps consideration of their continued role throughout treatment can serve to further explain the observed results in future research. This concept would be reflected in part by the relative lack of prediction of 3-month adherence relative to the more robust set of associations with 1-month adherence. Perhaps pretreatment common factors are more strongly related to more proximal outcomes and as the outcomes become increasingly distal, changes in psychosocial variables show a more dynamic influence (e.g., alliance ruptures; Safran & Kraus, 2014).

Several limitations of this study are to be noted. First, there were missing data at all time points, with a substantial amount of missing data at follow-up timepoints in particular. Multiple efforts were made to address this missingness that have been shown to produce substantial improvement in parameter estimation in real-world research (e.g., FIML estimation, including auxiliary covariates that are predictive of missingness). Such methods can produce estimates that match population parameters. These missing data were a trade-off for obtaining a naturalistic sample that resembles treatment-seeking patients from a variety of backgrounds. Other studies with real-world populations have been successfully conducted with even higher levels of missing data by using similar analytical approaches (Dong & Peng, 2013). Second, given that inclusion

criteria were very broad with treatment-seeking patients, there is substantial heterogeneity in psychopathology and psychopharmacology among participants. Common factors have generally been seen to have influence across populations, but rarely are they evaluated in a sample that is representative of such a broad set of problems. This broad sample permitted comparisons of the role of common factors across these populations and allowed for the generation of a number of unforeseen findings, which would be not be possible with more limited inclusion criteria. Nevertheless, this heterogeneity, along with lack of randomization, precludes the identification of causal mechanisms and a priori evaluation of differences across populations. This study is a first step towards evaluating the role of multiple psychosocial variables in tandem with psychopharmacological intervention, and as such serves as both a test of prior hypotheses and as a means to generate new ones for further investigation. Third, measurement of symptom change was restricted to self-report, and there was not a single best outcome measure for each patient. Future work would benefit from independent evaluation of diagnosis and symptom severity. With multiple measures of outcome (i.e., externalizing, internalizing, attention problems), this study is able to capture the different targets of treatment relevant to each patient. One concern about using these multiple measures of outcome is that symptom measurements were included for non-principal treatment targets (e.g., measuring internalizing symptoms in a child with ADHD and few internalizing problems). While the analytic models employed adjust estimates for pre-treatment scores, this process can serve to reduce the average magnitude of change. However, there were many participants with externalizing, internalizing, and attention problem symptoms, providing sufficient variability to evaluate symptom change outcomes for these domains. This method is consistent with other studies in pediatric psychopathology, which frequently eschew a single total combined total score and rather divide behavior problems into

subgroups such as internalizing and externalizing symptoms (Lindhiem, Bennett, Orimoto, & Kolko, 2016). Symptom increases in non-principal treatment targets are also possible and often are overlooked in clinical work, and this multi-domain approach to outcome measurement permitted evaluation of this hypothesis. Finally, a large number of hypothesis tests were performed. Post-hoc hypotheses involving moderators were controlled using strong post-hoc corrections, but the chance for Type I error still remains.

Clinically, these results suggest that common factors impact treatment outcome, even in psychopharmacological interventions. While they show notable influence across a number of populations, their impact may not be as straightforward as is often portrayed. There may not be a single "optimal patient" who receives better outcomes due to intrinsic motivation, a positive outlook towards treatment, and strong alliance with the clinician. In fact, being too strong in some of these areas may actually attenuate outcome in some circumstances. Moreover, the process of how these factors work concurrently throughout treatment may also be counterintuitive at times. Changes in the function of common factors during the full course of treatment has also been seen in psychotherapy; for example, early alliance may be facilitated by warmth and empathy, whereas alliance later on in treatment may be better developed by directiveness for patients who are looking for continued change (Keijsers et al., 1995). For these reasons, careful monitoring of both the level and specific role of common factors throughout treatment is warranted. One option to implement this type monitoring is through brief assessments in therapy progress notes (De Nadai et al., 2017). In pediatric interventions, the presence of multiple therapeutic stakeholders means this monitoring involves several dimensions, as parents, children, and clinicians often do not agree on common factors (e.g., Bickman et al., 2012). Monitoring of therapeutic alliance for children with ADHD and

externalizing disorders may deserve special focus. A positive initial alliance can be associated with poorer long-term outcomes for these patients, which is contrary to conventional wisdom.

Theoretically, these results suggest that psychosocial variables serve as common factors across patient populations and treatment interventions in pediatric psychiatry, but the nature of their influence on outcome may not be as common as originally thought. They are generally considered to have a consistently positive impact on symptom outcome and are conceptualized in this manner across all symptom types and patient populations. While not frequently considered, possible symptom exacerbations based on psychological interventions have been overlooked (Barlow, 2010). Not only are the effects of psychosocial variables not uniformly common across situations, but they may not be common across reporters as well. Contradictory effects were observed at times depending on the reporter in the present sample. Reporting agreement in child mental health treatment is often highly variable further examination among reporter agreement and discrepancies may provide further insight (De Los Reyes et al., 2015). Methods that unify the input of multiple reporting parties such as the Alliance Observational Coding System (AOCS, Karver, Shirk, Day, Field, & Handelsman, 2003) or factor analytic modeling (Bauer et al., 2013) may also provide novel insight regarding how common factors function within child common factors research.

Results from this investigation suggest that the process of how common factors work together can lead to unexpected changes in results. Many investigations focus on the early session role of common factors or aggregate measures of common factors throughout treatment, and rarely do they go beyond bivariate associations with outcome. Given how the function of psychosocial variables can change throughout treatment, continual measurement of these variables in clinical investigations may be necessary to elucidate their effects. For example,

interpersonal processes between clinician and patient have predicted subsequent outcome expectancies even when controlling for baseline differences in expectancies (Ahmed, Westra, and Constantino, 2012), and dyadic reciprocity in alliance has been observed in adult patients (Marcus, Kashy, Wintersteen, & Diamond, 2011). How common factors vary among each other and across time will be necessary to understand their role in treatment outcome.

Overall, the impact of psychosocial variables spans across pediatric psychiatric treatments, but may not function in a "one size fits all" fashion across different therapeutic populations and approaches. The present findings suggest that the process of how common factors work in pediatric psychiatry may be much more complicated than is often suggested. Psychosocial variables can cut across treatments as elements that are common to all psychiatric interventions, but the nature of their effects on outcome may actually not be common. Careful consideration of the differences in roles of psychosocial variables across patient populations and interventions may help to better explain treatment effects in clinical and research settings, with particular focus on how they are impacting treatment for both better and worse.

Tables and Figures

	Baseline	M1	M3
Parent			
Parent Alliance (WAI-SF)	Х		
Parent Expectancy (CEQ-P)	Х		
Parent Motivation (URICA-P)	Х		
Parent-Reported Adherence (BMQ)		Х	
Parent-Rated Symptoms (CBCL)	Х		
Parent-Reported Outcome (BPM-P)	Х		Х
Parent Report of Services (SACA)		Х	Х
Parent-Rated Side Effects (FIBSER/PRISE)		Х	
Youth			
Youth Alliance (TASC-R)	Х		
Youth Expectancy (CEQ-C)	Х		
Youth Motivation (URICA-C)			
Youth Anxiety and Depression (MASC-ADI/CDI-2-SF) ^a	Х		
Youth Symptoms (YSR) ^a	Х		
Clinician			
Clinician Alliance (TAQ-R)	Х		

 Table 1. Timetable of self-report assessments administered

Note. M1=1-month follow-up; M3=3-month follow-up; WAI-SF=Working Alliance Inventory-Short Form; CEQ-P=Credibility/Expectancy Questionnaire-Parent Version; URICA-P= University of Rhode Island Change Assessment-Parent Version; BMQ=Brief Medication Questionnaire; CBCL=Child Behavior Checklist; BPM-P=Brief Progress Monitor-Parent Version; SACA= Service Assessment for Children and Adolescents; FIBSER=Frequency, Intensity, and Burden of Side Effects Rating; PRISE=Patient Rated Inventory of Side Effects; TASC-R=Therapeutic Alliance Scale for Children-Revised; CEQ-C=Credibility/Expectancy Questionnaire-Child Version; URICA-C= University of Rhode Island Change Assessment-Child Version; MASC-ADI=Multidimensional Anxiety Scale for Children-Anxiety Disorders Index; CDI-2-SF=Children's Depression Inventory-2nd Edition Short Form; YSR=Youth Self Report; TAQ-R=Therapeutic Alliance Quality Rating

^aThe MASC-ADI and CDI-2-SF were administered to 21 participants who had anxiety and/or depression diagnoses; all other participants received the YSR

Variable	Mean	SD	Alpha
CEQ-C	16.62	5.94	.84
CEQ-P	17.16	4.83	.85
URICA-C	7.70	2.58	.87
URICA-P	10.36	1.29	.71
TASC-R	37.77	6.94	.86
WAI-SF	71.61	9.67	.90
TAQ-R-Child	3.57	0.70	N/A
TAQ-R-Parent	3.98	0.68	N/A
MASC-ADI ^a	14.50	6.21	.75
CDI-2-SF ^a	5.95	3.76	.74
CBCL Externalizing	15.98	10.80	.92
CBCL Internalizing	16.43	9.39	.87
YSR Externalizing ^a	20.76	9.50	.88
YSR Internalizing ^a	21.19	11.75	.90
BMQ (M1)	70.25	42.15	N/A ^b
BMQ (M3)	68.14	44.47	N/A ^b
FIBSER-Frequency	2.33	2.25	N/A ^b
FIBSER-Intensity	1.98	1.67	N/A ^b
FIBSER-Interference	1.33	1.52	N/A ^b
BPM-P Externalizing (Baseline)	5.81	3.47	.82
BPM-P Externalizing (M3)	5.16	3.58	.86
BPM-P Internalizing (Baseline)	4.52	2.92	.76
BPM-P Internalizing (M3)	5.06	3.28	.81
BPM-P Attention Problems (Baseline)	6.86	3.25	.81
BPM-P Attention Problems (M3)	5.98	3.37	.83

Table 2. Descriptive statistics for the present sample

Note. M1=1-month follow-up; M3=3-month follow-up; CEQ-C=Credibility/Expectancy Questionnaire-Child Version; CEQ-P=Credibility/Expectancy Questionnaire-Parent Version; URICA-C= University of Rhode Island Change Assessment-Child Version; URICA-P= University of Rhode Island Change Assessment-Parent Version; TASC-R=Therapeutic Alliance Scale for Children-Revised; WAI-SF=Working Alliance Inventory-Short Form; TAQ-R=Therapeutic Alliance Quality Rating; MASC-ADI=Multidimensional Anxiety Scale for Children-Anxiety Disorders Index; CDI-2-SF=Children's Depression Inventory-2nd Edition Short Form; CBCL=Child Behavior Checklist; YSR=Youth Self Report; BMQ=Brief Medication Questionnaire; FIBSER=Frequency, Intensity, and Burden of Side Effects Rating; BPM-P=Brief Progress Monitor-Parent Version

^aThe MASC-ADI and CDI-2-SF were administered to 21 participants who had anxiety and/or depression diagnoses; all other participants received the YSR

^bCronbach's alpha could not be calculated for this measure because only one item is used to create the total score

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. CEQ-C	-	0.07	0.34**	0.03	0.27**	0.11	0.08	0.10	-0.08	-0.12	0.06	0.11	0.16	0.12	-0.16	0.10	-0.02	-0.09	0.05
2. CEQ-P	-	-	0.16*	0.17	0.17	0.22*	0.13	0.04	-0.30	-0.26**	-0.24	-0.33**	-0.32**	-0.10	0.05	-0.09	-0.20	-0.10	-0.32**
3. URICA-C	-	-	-	0.23**	0.49**	-0.05	0.21*	0.19*	-0.24	0.03	0.03	-0.11	-0.02	-0.09	0.18*	-0.06	-0.13	0.34**	-0.19
4. URICA-P	-	-	-	-	0.06	0.30**	-0.06	0.14*	0.22	0.11	0.08	0.25	0.29**	0.08	0.18*	-0.04	0.09	0.12	0.02
5. TASC-R	-	-	-	-	-	-0.01	0.47**	0.12	-0.07	0.10	0.08	-0.21*	-0.34**	-0.07	-0.18**	0.05	-0.13	-0.23*	-0.11
6. WAI-SF	-	-	-	-	-	-	0.01	0.26**	0.00	-0.03	0.08	0.15	0.31**	0.02	0.08	0.05	0.11	0.10	0.17
7. TAQ-R-C	-	-	-	-	-	-	-	0.26**	-0.09	-0.10	0.29*	-0.07	-0.17	-0.26*	-0.09	-0.14	-0.03	-0.08	-0.02
8. TAQ-R-P	-	-	-	-	-	-	-	-	0.09	-0.02	0.08	0.06	0.08	-0.23*	0.02	0.01	-0.15	0.03	0.07
9. BMQ (M1)	-	-	-	-	-	-	-	-	-	0.55**	0.24	0.30	0.11	-0.14	-0.20	-0.06	0.04	-0.17	-0.04
10. BMQ (M3)	-	-	-	-	-	-	-	-	-	-	0.09	0.05	0.10	0.04	-0.01	-0.04	-0.06	-0.12	-0.03
11. FIBSER-1	-	-	-	-	-	-	-	-	-	-	-	0.79**	0.53**	-0.09	-0.02	0.01	0.13	-0.21**	0.22*
12. FIBSER-2	-	-	-	-	-	-	-	-	-	-	-	-	0.80**	-0.05	0.08	-0.03	0.19	-0.07	0.27*
13. FIBSER-3	-	-	-	-	-	-	-	-	-	-	-	-	-	0.23*	0.12	0.16	0.43**	0.10	0.41**
14. BPM-P Ext (B)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-0.07	0.48**	0.70**	0.02	0.32**
15. BPM-P Int (B)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-0.22*	-0.05	0.60**	-0.05
16. BPM-P Att (B)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.50**	-0.06	0.72**
17. BPM-P Ext (M3)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.17	0.62**
18. BPM-P Int (M3)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.13
19. BPM-P Att (M3)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table 3. Bivariate correlations among psychosocial variables

Note.* *p*<.05, ** *p*<.01

Note. B=Baseline; M1=1-month follow-up; M3=3-month follow-up; CEQ-C=Credibility/Expectancy Questionnaire-Child Version; CEQ-P=Credibility/Expectancy Questionnaire-Parent Version; URICA-C= University of Rhode Island Change Assessment-Child Version; URICA-P= University of Rhode Island Change Assessment-Parent Version; TASC-R=Therapeutic Alliance Scale for Children-Revised; WAI-SF=Working Alliance Inventory-Short Form; TAQ-R-C=Therapeutic Alliance Quality Rating-Child Version; TAQ-R-P=Therapeutic Alliance Quality Rating-Parent Version; BMQ=Brief Medication Questionnaire; FIBSER-1=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 1 (Frequency); FIBSER-2=Frequency, Intensity, Itemsity, Itemsity

Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 3 (Interference); BPM-P Ext=Brief Progress Monitor-Parent Version (Externalizing); BPM-P Int=Brief Progress Monitor-Parent Version (Internalizing); BPM-P Att=Brief Progress Monitor-Parent Version (Attention Problems)

			Moderator		
DV	Predictor	Moderator ^a	Level ^b	b (Slope) ^c	R ^{2d}
TASC-R	CEQ-C			0.28**	0.08
TASC-R	CEQ-C	Alpha-2	Low	0.31**	0.16
TASC-R	CEQ-C	Alpha-2	High	-0.02**	0.16
TASC-R	CEQ-C	ADHD	Low	0.62**	0.15
TASC-R	CEQ-C	ADHD	High	0.18	0.15
TASC-R	CEQ-C	FIBSER-2	Low	1.03**	0.26
TASC-R	CEQ-C	FIBSER-2	Med	0.36**	0.26
TASC-R	CEQ-C	FIBSER-2	High	-0.32	0.26
TASC-R	CEQ-P			0.18	0.03
TASC-R	URICA-C			0.52**	0.24
TASC-R	URICA-P			0.07	0.01
TASC-R	URICA-P	CTM3	Low	-0.26*	0.12
TASC-R	URICA-P	CTM3	High	0.18*	0.12
TASC-R	URICA-P	Stimulant	Low	0.16	0.04
TASC-R	URICA-P	Stimulant	High	-0.01*	0.04
WAI-SF	CEQ-C			0.11	0.01
WAI-SF	CEQ-P			0.22*	0.05
WAI-SF	URICA-C			-0.05	0.00
WAI-SF	URICA-C	Atypical	Low	-0.01	0.05
WAI-SF	URICA-C	Atypical	High	-0.21**	0.05
WAI-SF	URICA-P			0.30**	0.09
TAQ-R-C	CEQ-C			0.08	0.01
TAQ-R-C	CEQ-P			0.13	0.02
TAQ-R-C	URICA-C			0.21*	0.04
TAQ-R-C	URICA-P			-0.06	0.00
TAQ-R-P	CEQ-C			0.11	0.01

 Table 4. Residualized regression relationships for all main effects and significant moderators of main effects

 Table 4. (continued)

	liucu)				
DV	Predictor	Moderator ^a	Moderator Level ^b	<i>b</i> (Slope) ^c	R ^{2d}
TAQ-R-P	CEQ-P			0.04	0.00
TAQ-R-P	URICA-C			0.20**	0.04
TAQ-R-P	URICA-C	Atypical	Low	0.27**	0.09
TAQ-R-P	URICA-C	Atypical	High	0.03**	0.09
TAO-R-P				0.1//*	0.02
TAO-R-P	URICA-P	Fxt Dx	Low	-0.22*	0.02
TAO-R-P	URICA-P	Ext Dx	High	0.22	0.10
mų ni	oncerti	LAT DA	111,511	0.22	0.10
BMQ (M1)	CEQ-C			-0.15	0.03
BMQ (M1)	CEQ-P			-0.34**	0.11
BMO(M1)				-0.28*	0.07
	UNICA-C			-0.20	0.07
BMQ (M1)	URICA-P			0.26	0.07
BMQ (M1)	TASC-R			-0.02	0.00
	WALCE			0.00	0.00
BMQ (M1)	WAI-5F			0.00	0.00
BMQ (M1)	TAQ-R-C			-0.03	0.00
BMQ (M1)	TAQ-R-C	Atypical	Low	-0.07	0.05
BMQ (M1)	TAQ-R-C	Atypical	High	0.14**	0.05
BMQ (M1)	TAQ-R-P			0.12	0.02
BMO (M3)	CEO-C			-0.11	0.03
				0.11	0.05
BMQ (M3)	CEQ-P			-0.19*	0.05
BMQ (M3)	URICA-C			-0.03	0.02
BMO (M3)				-0.05	0.02
				-0.03	0.02
BMQ (M3)	TASC-R			0.06	0.02
BMQ (M3)	WAI-SF			-0.15	0.04

Table 4. (continued)

× *	,		Moderator		
DV	Predictor	Moderator ^a	Level ^b	<i>b</i> (Slope) ^c	R ^{2d}
BMQ (M3)	TAQ-R-C			0.04	0.02
BMQ (M3)	TAQ-R-P			0.00	0.02
BPM-P Ext	CEQ-C			-0.10	0.01
BPM-P Ext	CEQ-C	Tic Dx	Low	-0.18	0.06
BPM-P Ext	CEQ-C	Tic Dx	High	0.04*	0.06
BPM-P Ext	CEQ-P			-0.14	0.02
BPM-P Ext	URICA-C			-0.05	0.00
BPM-P Ext	URICA-C	Tic Dx	Low	-0.16	0.09
BPM-P Ext	URICA-C	Tic Dx	High	0.14**	0.09
BPM-P Ext	URICA-P			0.03	0.00
BPM-P Ext	TASC-R			-0.07	0.01
BPM-P Ext	WAI-SF			0.08	0.01
BPM-P Ext	WAI-SF	Ext Dx	Low	0.40**	0.03
BPM-P Ext	WAI-SF	Ext Dx	High	-0.02	0.03
BPM-P Ext	TAQ-R-C			0.15	0.02
BPM-P Ext	TAQ-R-P			0.01	0.00
BPM-P Ext	BMQ (M1)			0.05	0.01
BPM-P Ext	BMQ (M3)			-0.06	0.01
BPM-P Int	CEQ-C			0.00	0.00
BPM-P Int	CEQ-C	Atypical	Low	-0.04	0.04
BPM-P Int	CEQ-C	Atypical	High	0.13**	0.04
BPM-P Int	CEQ-P			-0.14	0.02
BPM-P Int	URICA-C			0.27**	0.06
			Moderator		
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DV	Predictor	Moderator ^a	Level ^b	b (Slope) ^c	R ^{2d}
BPM-P Int	URICA-P			0.01	0.00
BPM-P Int	URICA-P	No Prescription	Low	-0.18*	0.04
BPM-P Int	URICA-P	No Prescription	High	0.08**	0.04
BPM-P Int	TASC-R			-0.13	0.02
BPM-P Int	TASC-R	Atypical	Low	-0.22*	0.09
BPM-P Int	TASC-R	Atypical	High	0.03**	0.09
BPM-P Int	WAI-SF			0.05	0.00
BPM-P Int	TAQ-R-C			-0.03	0.00
BPM-P Int	TAQ-R-P			0.01	0.00
BPM-P Int	TAQ-R-P	Atypical	Low	0.05	0.06
BPM-P Int	TAQ-R-P	Atypical	High	-0.20**	0.06
BPM-P Int	BMQ (M1)			-0.18	0.03
BPM-P Int	BMQ (M1)	Alpha-2	Low	-0.18	0.07
BPM-P Int	BMQ (M1)	Alpha-2	High	0.83**	0.07
BPM-P Int	BMQ (M3)			-0.11	0.01
BPM-P Att	CEQ-C			-0.03	0.00
BPM-P Att	CEQ-P			-0.27*	0.07
BPM-P Att	CEQ-P	FIBSER-2	Low	0.29	0.18
BPM-P Att	CEQ-P	FIBSER-2	Med	-0.24**	0.18
BPM-P Att	CEQ-P	FIBSER-2	High	-0.77**	0.18
BPM-P Att	URICA-C			-0.16	0.02
BPM-P Att	URICA-P			0.05	0.00
BPM-P Att	TASC-R			-0.15*	0.02

Table 4. (continued)

			Moderator		
DV	Predictor	Moderator ^a	Level ^b	b (Slope) ^c	R ^{2d}
BPM-P Att	WAI-SF			0.14	0.02
BPM-P Att	WAI-SF	FIBSER-3	Low	-0.54**	0.20
BPM-P Att	WAI-SF	FIBSER-3	Med	0.11	0.20
BPM-P Att	WAI-SF	FIBSER-3	High	0.75**	0.20
BPM-P Att	WAI-SF	CTM1	Low	-0.10	0.16
BPM-P Att	WAI-SF	CTM1	High	0.35**	0.16
BPM-P Att	TAQ-R-C			0.07	0.01
BPM-P Att	TAQ-R-P			0.06	0.00
BPM-P Att	TAQ-R-P	CTM3	Low	0.42**	0.04
BPM-P Att	TAQ-R-P	CTM3	High	0.02	0.04
BPM-P Att	BMQ (M1)			0.08	0.01
BPM-P Att	BMQ (M1)	FIBSER-2	Low	-0.49*	0.17
BPM-P Att	BMQ (M1)	FIBSER-2	Med	0.06	0.17
BPM-P Att	BMQ (M1)	FIBSER-2	High	0.61**	0.17
BPM-P Att	BMQ (M3)			-0.09	0.01

Table 4. (continued)

*Note.** *p*<.05, ** *p*<.01

Note. M1=1-month follow-up; M3=3-month follow-up; TASC-R=Therapeutic Alliance Scale for Children-Revised; WAI-SF=Working Alliance Inventory-Short Form; TAQ-R-C=Therapeutic Alliance Quality Rating-Child Version; TAQ-R-P=Therapeutic Alliance Quality Rating-Parent Version: BMO=Brief Medication Questionnaire; BPM-P Ext=Brief Progress Monitor-Parent Version (Externalizing); BPM-P Int=Brief Progress Monitor-Parent Version (Internalizing); BPM-P Att=Brief Progress Monitor-Parent Version (Attention Problems); CEQ-C=Credibility/Expectancy Questionnaire-Child Version; CEQ-P=Credibility/Expectancy Questionnaire-Parent Version; URICA-C= University of Rhode Island Change Assessment-Child Version; URICA-P= University of Rhode Island Change Assessment-Parent Version; TASC-R=Therapeutic Alliance Scale for Children-Revised: WAI-SF=Working Alliance Inventory-Short Form; TAQ-R-C=Therapeutic Alliance Quality Rating-Child Version; TAQ-R-P=Therapeutic Alliance Quality Rating-Parent Version; BMQ=Brief Medication Questionnaire; Alpha-2=Alpha-2 agonists; FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); CTM1=Concurrent treatment received by 1-month follow-up; CTM3=Concurrent treatment received by 3-month follow-up; Atypical=Atypical antipsychotic; Ext Dx=Externalizing Diagnosis; Tic Dx=Tic Disorder Diagnosis; FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 3 (Interference)

^aModerators were included in table if they were significant after Holm-Bonferroni correction

Table 4. (continued)

^bFor continuous moderators, low, medium, high values reflect -1 SD, the mean, and +1 SD respectively for the corresponding measure. For dichotomous moderators, low values reflect the absence of the moderator (i.e., 0 in dummy-coding), and high values reflect presence of the moderator (i.e., 1 in dummy-coding).

^cSlopes reflect standardized beta weights for main effects and standardized simple slopes for moderators evaluated at the respective moderator level. Significance was evaluated based on unstandardized analyses as recommended by Fairchild and McQuillin (2010), and standardized results are presented to facilitate comparison across models.

 ${}^{d}R^{2}$ values reflect amount of variance attributable specifically to the predictor in main effect models, and reflect the amount of additional variance attributable to adding the moderator and its interaction term in moderation models

Table 5. Numerical results for indirect effect models tested

							Std. Indirect	Std. Direct	Std. Total
X	Μ	Y	W ^a	V ^a	U ^a	Moderator Level ^b	Effect ^c	Effect ^c	Effect ^c
Expectanci	es/Motivation	n Through Alli	iance to Outc	ome					
CEQ-C	TASC-R	BPM-P Att	ADHD			Low W	-0.13	0.00	-0.13
						High W	-0.01	0.00	-0.01
CEQ-C	TASC-R	BPM-P Att	Alpha-2			Low W	-0.06	0.01	-0.05
						High W	0.21*	0.01	0.22
CEQ-C	TASC-R	BPM-P Att	FIBSER-2			Low W	-0.17	0.03	-0.14
						Med W	-0.08	0.03	-0.05
						High W	0.01	0.03	0.04
URICA-C	TASC-R	BPM-P Att					-0.07	-0.18	-0.24
URICA-P	TASC-R	BPM-P Att	CTM3			Low W	0.05	0.09	0.14
						High W	-0.07	0.09	0.02
URICA-P	TASC-R	BPM-P Att	Stimulant			Low W	-0.04	0.08	0.04
						High W	0.05*	0.08	0.14
CEQ-P	WAI-SF	BPM-P Att		CTM1	FIBSER-2	Low V, Low U	0.01	-0.01	0.00
						High V, Low U	0.25*	-0.01	0.25
						Low V, Med U	0.01	-0.53**	-0.52**
						High V, Med U	0.25*	-0.53**	-0.27
						Low V, High U	0.01	-1.05**	-1.04**
						High V, High U	0.25*	-1.05**	-0.79*

Table	5. (continu	(ed)
I ante	U • 1	continu	icu,

							Std.	Std.	Std.
							Indirect	Direct	Total
Χ	Μ	Y	$\mathbf{W}^{\mathbf{a}}$	V ^a	U ^a	Moderator Level ^b	Effect ^c	Effect ^c	Effect ^c
URICA-P	WAI-SF	BPM-P Att		CTM1		Low W	-0.04	0.07	0.03
						High W	0.39**	0.07	0.46**
URICA-C	WAI-SF	BPM-P Att	Atypical	CTM1		Low W, Low V	0.00	-0.19	-0.19
						High W, Low V	0.06	-0.19	-0.13
						Low W, High V	-0.04	-0.19	-0.23
						High W, High V	-0.93**	-0.19	-1.13**
URICA-P	WAI-SF	BPM-P Att		FIBSER-3		Low V	-0.16	0.01	-0.15
						Med V	0.07	0.01	0.08
						High V	0.30**	0.01	0.31
URICA-C	WAI-SF	BPM-P Att	Atypical	FIBSER-3		Low W, Low V	0.01	-0.21	-0.20
						High W, Low V	0.35*	-0.21	0.14
						Low W, Med V	0.00	-0.21	-0.21
						High W, Med V	-0.16	-0.21	-0.37
						Low W, High V	-0.02	-0.21	-0.23
						High W, High V	-0.66**	-0.21	-0.87**
URICA-C	TAQ-R-P	BPM-P Att	Atypical	CTM3		Low W, Low V	0.27*	-0.39*	-0.13
						High W, Low V	-0.49**	-0.39*	-0.88**
						Low W, High V	0.00	-0.39*	-0.39*
						High W, High V	-0.01	-0.39*	-0.40

Table 5. (continued

							Std.	Std.	Std.
							Indirect	Direct	Total
Χ	Μ	Y	$\mathbf{W}^{\mathbf{a}}$	V ^a	$\mathbf{U}^{\mathbf{a}}$	Moderator Level ^b	Effect^c	Effect^c	Effect ^c
URICA-P	TAQ-R-P	BPM-P Att	Ext Dx	CTM3		Low W, Low V	-0.18	0.23	0.05
						High W, Low V	0.32**	0.23	0.54**
						Low W, High V	0.04	0.23	0.27
						High W, High V	-0.07	0.23	0.15
CEQ-P	WAI-SF	BPM-P Ext		Ext Dx		Low V	0.18	-0.51**	-0.32
						High V	-0.04	-0.51**	-0.54**
URICA-P	WAI-SF	BPM-P Ext		Ext Dx		Low V	0.18*	-0.09	0.09
						High V	-0.05	-0.09	-0.14
URICA-C	WAI-SF	BPM-P Ext	Atypical	Ext Dx	Tic Dx	Low W, Low V, Low U	-0.01	-0.39*	-0.39
						Low W, Low V, High U	-0.01	1.11**	1.11**
						Low W, High V, Low U	0.00	-0.39*	-0.38
						Low W, High V, High U	0.00	1.11**	1.12**
						High W, Low V, Low U	-0.48*	-0.39*	-0.86**
						High W, Low V, High U	-0.48*	1.11**	0.64
						High W, High V, Low U	0.21	-0.39*	-0.18
						High W, High V, High U	0.21	1.11**	1.32**
CEQ-C	TASC-R	BPM-P Int	ADHD	Atypical	Atypical	Low W, Low V and U	-0.19	0.06	-0.13
						High W, Low V and U	-0.02	0.06	0.04
						Low W, High V and U	0.59**	0.46**	1.05**
						High W, High V and U	0.05	0.46**	0.51*

							Std.	Std.	Std.
							Indirect	Direct	Total
Χ	Μ	Y	$\mathbf{W}^{\mathbf{a}}$	V ^a	$\mathbf{U}^{\mathbf{a}}$	Moderator Level ^b	Effect ^c	Effect^c	Effect^c
CEQ-C	TASC-R	BPM-P Int	Alpha-2	Atypical	Atypical	Low W, Low V and U	-0.07	-0.02	-0.09
						High W, Low V and U	0.26*	-0.02	0.24
						Low W, High V and U	0.28*	0.56**	0.83**
						High W, High V and U	-0.96	0.56**	-0.40
CEQ-C	TASC-R	BPM-P Int	FIBSER-2	Atypical	Atypical	Low W, Low V and U	-0.24*	-0.03	-0.27**
						Med W, Low V and U	-0.11*	-0.03	-0.14*
						High W, Low V and U	0.03	-0.03	0.00
						Low W, High V and U	0.80*	0.53**	1.34**
						Med W, High V and U	0.36*	0.53**	0.89**
						High W, High V and U	-0.09	0.53**	0.45*
URICA-C	TASC-R	BPM-P Int		Atypical		Low V	-0.31**	0.57**	0.26*
						High V	0.30	0.57**	0.87**
URICA-P	TASC-R	BPM-P Int	CTM3	Atypical		Low W, Low V	0.05	0.05	0.10
						High W, Low V	-0.05	0.05	-0.01
						Low W, High V	-0.22	0.05	-0.17
						High W, High V	0.23	0.05	0.28
URICA-P	TASC-R	BPM-P Int	Stimulant	Atypical		Low W, Low V	-0.05	0.05	-0.01
						High W, Low V	0.07	0.05	0.10
						Low W, High V	0.15	0.05	0.19
						High W, High V	-0.24	0.05	-0.21

Table 5. (continued)

							Std.	Std.	Std.
							Indirect	Direct	Total
Χ	Μ	Y	$\mathbf{W}^{\mathbf{a}}$	V ^a	$\mathbf{U}^{\mathbf{a}}$	Moderator Level ^b	Effect ^c	Effect^c	Effect ^c
URICA-C	TAQ-R-P	BPM-P Int	Atypical	Atypical		Low V	0.00	0.29**	0.29**
						High V	0.73**	0.29**	1.02**
URICA-P	TAQ-R-P	BPM-P Int	Ext Dx	Atypical		Low W, Low V	-0.02	0.05	0.03
						High W, Low V	0.03	0.05	0.08
						Low W, High V	0.29**	0.05	0.34**
						High W, High V	-0.50**	0.05	-0.45**
Expectanci	es/Motivation	Through Ad	herence to Oi	itcome					
CEO-P	BMO (M1)	BPM-P Att		FIBSER-2	FIBSER-2	Low V and U	0.15	0.11	0.27
				1 12 0 211 2		Med V and U	0.02	-0.47**	-0.45**
						High V and U	-0.12	-1.05**	-1.17**
URICA-C	BMQ (M1)	BPM-P Att		FIBSER-2		Low V	0.17	-0.44*	-0.27
						Med V	-0.01	-0.44*	-0.45
						High V	-0.19	-0.44*	-0.63*
CEQ-P	BMQ (M1)	BPM-P Int		Alpha-2		Low V	0.14	-0.31	-0.18
						High V	-2.55**	-0.31	-2.86**
URICA-C	BMQ (M1)	BPM-P Int		Alpha-2		Low V	0.03	0.47**	0.50**
						High V	-2.75*	0.47**	-2.28

							Std.	Std.	Std.
							Indirect	Direct	Total
Χ	Μ	Y	$\mathbf{W}^{\mathbf{a}}$	$\mathbf{V}^{\mathbf{a}}$	$\mathbf{U}^{\mathbf{a}}$	Moderator Level ^b	Effect^c	Effect^c	Effect ^c
Alliance Th	rough Adher	ence to Outco	<u>me</u>						
TAQ-R-C	BMQ (M1)	BPM-P Att	Atypical	FIBSER-2		Low W, Low V	0.03	0.14	0.17
						High W, Low V	-0.43	0.14	-0.29
						Low W, Med V	-0.01	0.14	0.13
						High W, Med V	0.10	0.14	0.24
						Low W, High V	-0.04	0.14	0.10
						High W, High V	0.63*	0.14	0.77*
TAQ-R-C	BMQ (M1)	BPM-P Int	Atypical	Alpha-2		Low W, Low V	0.01	-0.06	-0.05
-	- · ·		•••	-		High W, Low V	-0.23	-0.06	-0.29
						Low W, High V	-0.30	-0.06	-0.36
						High W, High V	6.31*	-0.06	6.24*

Note.* *p*<.05, ** *p*<.01

Note. M1=1-month follow-up; M3=3-month follow-up; CEQ-C=Credibility/Expectancy Questionnaire-Child Version; URICA-C= University of Rhode Island Change Assessment-Child Version; URICA-P= University of Rhode Island Change Assessment-Parent Version; CEQ-P=Credibility/Expectancy Questionnaire-Parent Version; TAQ-R-C=Therapeutic Alliance Quality Rating-Child Version; TASC-R=Therapeutic Alliance Scale for Children-Revised; WAI-SF=Working Alliance Inventory-Short Form; TAQ-R-P=Therapeutic Alliance Quality Rating-Parent Version; BMQ=Brief Medication Questionnaire; **Y**: BPM-P Att=Brief Progress Monitor-Parent Version (Attention Problems); BPM-P Ext=Brief Progress Monitor-Parent Version (Externalizing); BPM-P Int=Brief Progress Monitor-Parent Version (Internalizing); Alpha-2=Alpha-2 agonists; Atypical=Atypical antipsychotic; FIBSER-2=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 2 (Intensity); FIBSER-3=Frequency, Intensity, and Burden of Side Effects Rating-Item 3 (Interference); CTM1=Concurrent treatment received by 2-month follow-up; CTM3=Concurrent treatment received by 3month follow-up; Ext Dx=Externalizing Diagnosis; Tic Dx=Tic Disorder Diagnosis

^aW reflects moderator of X-M relationship, V reflects moderator of M-Y relationship, and U reflects moderator of c'

Table 5 (continued.)

^bFor continuous moderators, low, medium, high values reflect -1 SD, the mean, and +1 SD respectively for the corresponding measure. For dichotomous moderators, low values reflect the absence of the moderator (i.e., 0 in dummy-coding), and high values reflect presence of the moderator (i.e., 1 in dummy-coding).

^cSlopes reflect standardized effects. Significance was evaluated based on unstandardized analyses as recommended by Fairchild and McQuillin (2010), and standardized results are presented to facilitate interpretation as recommended by Wen and Fan (2015).



Figure 1. *Hypothesized model of psychosocial variables and treatment outcome in pediatric psychiatry, reflecting all bivariate relationships tested in the present study.*



Figure 2. *Graphical depiction of moderated mediation models tested, including the overall framework (top) and specific 2-mediator model (bottom)*

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APPENDIX A:

URICA-P

The following questions address how you view symptoms of depression and anxiety for your child.

Item	1 Strongly Disagree	2 Disagree	3 Undecided	4 Agree	5 Strongly Agree
 As far as I'm concerned, my child doesn't have any problems that need changing. 					
2) I think my child might be ready for some improvement.					
 I am doing something about the problems that had been bothering my child. 					
4) It might be worthwhile to work on my child's problem.					
5) My child isn't the problem one. It doesn't make much sense for him/her to be here.					
6) It worries me that my child might slip back on a problem he/she has already changed, so I am here to seek help for him/her.					
7) I am finally doing some work on my child's problem.					
8) I've been thinking that I might want to change something about my child.					
 My child has been successful in working on his/her problem but I'm not sure I can keep up the help on my own. 					
10) At times my child's problem is difficult, but I'm working to help.					
11) Being here is pretty much a waste of time because the problem doesn't have to do with my child.					

12) I'm hoping this place will help me to better understand my child's problem.			
13) I guess my child has faults, but there's nothing that he/she really needs to change.			
14) I am really working hard to help my child.			
15) My child has a problem and I really think I should work to help.			
16) I'm not following through with helping my child as well as I had hoped, and I'm here to prevent a relapse of the problem.			
17) Even though I'm not always successful helping my child, I am at least working on his/her problem.			
18) I thought once I had resolved my child's problem I would be free of it, but sometimes I still find myself struggling to help.			
19) I wish I had more ideas on how to solve my child's problem.			
20) I have started working on my child's problems but I would like help.			
21) Maybe this place will be able to help my child.			
22) I may need a boost right now to help me maintain the changes I've already made in helping my child.			
23) I may be able to deal with my child's problem better, but I don't really think so			
24) I hope that someone here will have some good advice for my child.			
25) Anyone can talk about helping their children; I'm actually doing something about it.			
26) All this talk about psychiatry is boring. Why can't people just forget about their problems?			
27) I'm here to prevent my child from having a relapse of his/her problem.			

28) It is frustrating, but I feel my child might be having a recurrence of a problem I thought had been resolved.			
29) I have worries about my child's symptoms but so does the next person. Why spend time thinking about them?			
30) I am actively working on my child's problem.			
31) I would rather cope with my child's faults than try to change them.			
32) After all I had done to try to change my child's problem, every now and again it comes back to haunt me.			
APPENDIX B:

URICA-C

The following questions address how you view your thoughts and feelings of being down or nervous.

Item	1	2	3	4	5
	Strongly	Disagree	Undecided	Agree	Strongly
	Disagree				Agree
1) As far as I'm concerned, I					
don't have any problems that					
need changing.					
2) I think I might be ready for					
some self-improvement.					
3) I am doing something about					
the problems that had been					
bothering me.					
4) It might be worthwhile to					
work on my problem.					
5) I'm not the problem one. It					
doesn't make much sense for					
me to be here.					
6) It worries me that I might slip					
back on a problem I have					
already changed, so I am here					
To seek help.					
/) I am finally doing some work					
On my problem.					
8) I ve been thinking that I hight					
about myself					
0) I have been successful in					
yorking on my problem but					
I'm not sure I can keen un the					
effort on my own					
10) At times my problem is					
difficult but I'm working					
on it.					
on it.					

11) Being here is pretty much a waste of time for me because			
the problem doesn't have to do			
12) I'm hoping this place will help			
me to better understand			
myself.			
there's nothing that I really			
need to change.			
14) I am really working hard to			
change.			
15) I have a problem and I really			
think I should work at it.			
16) I'm not following through			
with what I had already			
hoped and I'm here to prevent			
a relapse of the problem.			
17) Even though I'm not always			
successful in changing, I am			
at least working on my			
problem.			
18) I thought once I had resolved			
my problem I would be free of			
it, but sometimes I still find			
myself struggling with it.			
19) I wish I had more ideas on			
now to solve the problem.	 	 	
problems but I would like			
help.			
21) Maybe this place will be able			
to help me.			
22) I may need a boost right now			
to help me maintain the			
changes I've already made.			
problem better, but I don't			
really think so.			
24) I hope that someone here will			
have some good advice for			
me.			

25) Anyone can talk about changing; I'm actually doing something about it.			
26) All this talk about psychiatry is boring. Why can't people just forget about their problems?			
27) I'm here to prevent myself from having a relapse of my problem.			
28) It is frustrating, but I feel I might be having a recurrence of a problem I thought I had resolved.			
29) I have worries but so does the next person. Why spend time thinking about them?			
30) I am actively working on my problem.			
31) I would rather cope with my faults than try to change them.			
32) After all I had done to try to change my problem, every now and again it comes back to haunt me.			

APPENDIX C:

TAQ-R

All questions below refer to the session that you just completed with this family. Please select one answer for each question.

Did the youth participate in the session that you just had for him or her?

Yes	

□ No

> □ No

Did the youth's caregiver participate in the session that you just had for this youth?

Ye	s

		Very Poor	Poor	Satisfactory	Good	Excellent
1.	In this session, how would you describe your relationship with this YOUTH?					
2.	In this session, how would you describe your relationship with this CAREGIVER?					

APPENDIX D:

WAI-SF

Following are sentences that describe some of the different ways a person might think or feel about his or her family's counselor. Using the following 7-point scale, <u>please respond to every item with your first impressions of your counselor</u>:

	Never	Rarely	Occasionally	Sometimes	Often	Very Often	Always
1. The counselor and I agree about the things that my family needs to do in therapy to help improve our situation.	1	2	3	4	5	6	7
2. What we are doing in counseling gives me new ways of looking at my family's problems.	1	2	3	4	5	6	7
3. I believe the counselor likes my family.	1	2	3	4	5	6	7
4. The counselor does not understand what my family is trying to accomplish in counseling.	1	2	3	4	5	6	7
5. I am confident in the counselor's ALLIANCE AND MEDICATION ADHERENCE to help my family.	1	2	3	4	5	6	7
6. The counselor and my family are working towards mutually agreed upon goals.	1	2	3	4	5	6	7

7. I feel that the counselor appreciates my family.	1	2	3	4	5	6	7
8. We agree on what is important for my family to work on.	1	2	3	4	5	6	7
9. My family has built a mutual trust with the counselor.	1	2	3	4	5	6	7
10. My family and the counselor have different ideas on what our real problems are.	1	2	3	4	5	6	7
11. We have established a good understanding of the kind of changes that would be good for my child/family.	1	2	3	4	5	6	7
12. I believe the way we are working with my family's problem is correct.	1	2	3	4	5	6	7

APPENDIX E:

CEQ-P

The following questions address how you believe this treatment will affect your child's troublesome symptoms of depression and anxiety.

1. By the end of the therapy period, how much improvement in your child's problem behavior do you really feel will occur?

0% 10% 40% 50% 60% 70% 80% 90% 100% 20% 30% 2. By the end of therapy, how much improvement in your child's behavior do you think will have occurred? 2 3 4 5 6 7 8 9 1 Not at all Somewhat Very much 3. At this point, take a minute to think about how much do you really feel that therapy will help to reduce your child's problem behaviors?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

APPENDIX F:

BMQ

1. Please list below all of the medications your child took in the **PAST WEEK**. For each medication you list, please answer each of the questions in the box below.

If he/she has stopped medication completely for these conditions since he/she first saw the psychiatrist, please write "Stopped" after listing the medication and the reason for stoppage (for example, side effects).

Medication name and strength	How many days did	How many times per day did	How many pills did	How many times did he/she miss
(if stopped, please	he/she take it?	he/she take it?	he/she take	taking a pill?
provide the reason)			each time?	

APPENDIX G:

SACA

In the last 3 months, has your child received outpatient or inpatient help from any of the following sources?

			IF YES: COL A NUMBER OF HOURS OR DAYS OF SERVICE	IF YES: CHECK TYPES OF SERVICES GIVEN:
1.	Community mental health center or other outpatient mental health clinic	NO YES DK	hrs.	<pre>assessmentindividual treatment/therapygroup treatmentfamily/parent treatment/edmedicationeducation/trainingcase management</pre>
2.	Professional like a psychologist, psychiatrist, social worker, or family counselor not part of a service or clinic already mentioned	NO YES DK	hrs.	<pre>assessmentindividual treatment/therapygroup treatmentfamily/parent treatment/edmedicationeducation/trainingcase management</pre>
3.	Partial hospitalization or day treatment program	NO YES DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training
4.	Drug or alcohol clinic	NO YES DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training
5.	Therapist or counselor or family preservation worker who came to your home	NO YES DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training case management
6.	Emergency room for problems with behaviors or feelings	NO YES DK	hrs.	assessment individual treatment/therapy family/parent treatment/ed medication
7.	Pediatrician or family doctor for problems with behaviors or feelings	NO YES DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training

8.	Probation or juvenile corrections officer or a court counselor	NO YES	DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training
9.	Priest, Minister or Rabbi for problems with behaviors or feelings	NO YES	DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed education/training
10.	Acupuncturist/Chiropractor	NO YES	DK	hrs.	assessment individual treatment/therapy group treatment family/parent treatment/ed medication education/training
11.	Crisis hotline	NO YES	DK	hrs.	
12.	Any self-help group like Alcoholics Anonymous or peer counseling	NO YES	DK	hrs.	
13.	Other: describe	NO YES	DK	hrs	

APPENDIX H:

FIBSER

INSTRUCTIONS: Select the best response for the following three questions.

 Choose the response that best describes the frequency (how often) of the side effects of the medication you have taken within the past week for your depression. Do not rate side effects if you believe they are due to treatments that you are taking for medical conditions other than depression or anxiety. Rate the frequency of these side effects for the past week.

No side	Present	Present	Present	Present	Present	Present all
effects	10% of the	25% of the	50% of the	75% of the	90% of the	the time
	time	time	time	time	time	
0	1	2	3	4	5	6

2) Choose the response that best describes the intensity (how severe) of the side effects that you believe are due to the medication you have taken within the last week for your depression or anxiety. Rate the intensity of the side effect(s), when they occurred, over the last week.

No side	Trivial	Mild	Moderate	Marked	Severe	Intolerable
effects						
0	1	2	3	4	5	6

3) Choose the response that best describes the degree to which antidepressant medication side effects that you have had over the last week have interfered with your day to day functions.

No	Minimal	Mild	Moderate	Marked	Severe	Unable to
impairment	impairment	impairment	impairment	impairment	impairment	function
						due to
						impairment
0	1	2	3	4	5	6

APPENDIX I:

PRISE

Please indicate all symptoms you have experienced in the past week. These symptoms may or may not have been caused by your treatment.

1. GASTROINTESTINAL	4. NERVOUS SYSTEM
1.1 Check ALL symptoms that you have	4.1 Check ALL symptoms that you have
experienced during the past week regardless	experienced during the past week regardless
of cause:	of cause:
🗌 Diarrhea	Headache
Constipation	Tremors
Dry Mouth	Poor coordination
Nausea/vomiting	Dizziness
No symptoms in this category	No symptoms in this category
1.2 If you had any symptoms over the last	4.2 If you had any symptoms over the last
week, how bad was your WORST symptom?	week, how bad was your WORST symptom?
Tolerable	Tolerable
Distressing	Distressing
2. HEART	5. EYES/EARS
2.1 Check ALL symptoms that you have	5.1 Check ALL symptoms that you have
experienced during the past week regardless	experienced during the past week regardless
of cause:	of cause:
Palpitation (skipping a beat)	Blurred Vision
Dizziness on standing	Ringing in ears
Chest pain	No symptoms in this category
No symptoms in this category	5.2 If you had any symptoms over the last
2.2 If you had any symptoms over the last	week, how bad was your WORST symptom?
week, how bad was your WORST symptom?	Tolerable
☐ Tolerable	Distressing
Distressing	
3. SKIN	6. GENITAL/URINARY
3.1 Check ALL symptoms that you have	6.1 Check ALL symptoms that you have
experienced during the past week regardless	experienced during the past week regardless
of cause:	of cause:
Rash	Difficulty urinating
Increased perspiration	Painful Urination
Itching	Frequent urination
Dry skin	Menstrual irregularity
No symptoms in this category	No symptoms in this category

3.2 If you had any symptoms over the last	6.2 If you had any symptoms over the last
week, how bad was your WORST symptom?	week, how bad was your WORST symptom?
☐ Tolerable	
Distressing	Distressing
7. SLEEP	8. OTHER
7.1 Check ALL symptoms that you have	8.1 Check ALL symptoms that you have
experienced during the past week regardless	experienced during the past week regardless
of cause:	of cause:
Difficulty sleeping	Anxiety
Sleeping too much	Poor concentration
No symptoms in this category	General malaise
7.2 If you had any symptoms over the last	Restlessness
week, how bad was your WORST symptom?	Fatigue
☐ Tolerable	Decreased energy
Distressing	Other
	No symptoms in this category
	8.2 If you had any symptoms over the last
	week, how bad was your WORST symptom?
	Tolerable
	Distressing

APPENDIX J:

TASC-R

Please read the sentences below about this meeting you just had with your therapist. After reading each sentence, decide how much the sentence is like you. There are no right or wrong answers for this questionnaire, just how you feel.

1. I liked sper	nding time with my tl	herapist.	
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
2. I found it h	ard to work with my	therapist on solving	problems in my life.
I Not Like Me	2 A Little Like Me	3 Mostly Like Me	4 Very Much Like Me
3. I felt like m	y therapist was on n	ny side and tried to he	elp me.
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
4. I worked w	ith my therapist on s	solving my problems.	
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
5. When I wa	s with my therapist, I	I wanted the session t	o end quickly.
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
6. I look forw	ard to meeting with	my therapist again.	
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
7. I felt like m	y therapist spent too	much time working	on my problems.
	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
8. I'd rather l	nave done something	other than meet with	my therapist.
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me

9. I used my t	ime with my therapi	st to make changes in	my life.
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
10. I like my t	herapist.		
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
11. I would ra	ther have not worke	ed on my problems wi	th my therapist.
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me
12. I think my	therapist and I wor	ked well together on	dealing with my problems.
1	2	3	4
Not Like Me	A Little Like Me	Mostly Like Me	Very Much Like Me

APPENDIX K:

CEQ-C

These questions address how you believe this treatment will affect your troublesome thoughts and feelings of being sad, down, or nervous.

1. By the end of the therapy period, how much improvement in your symptoms do you think will occur? 0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100% 2. At this point, how much do you really feel that therapy will help to reduce your symptoms? 2 3 5 8 9 1 4 6 7 Not at all Somewhat Very much 3. By the end of the treatment period, how much improvement do you really feel will occur? 0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

APPENDIX L:

Documentation of Institutional Review Board Approval

JHM ACH IRB ACH Box #9496 FWA# 00000977 IRB# 00001642 727.767.4275



April 27, 2016

Alessandro De Nadai 501 6th Ave. South St. Petersburg, FL 33701

Dear Mr. De Nadai,

Your Final Report for the protocol entitled "Alliance and Mechanisms of Medication Adherence in Pediatric Psychiatric Practice" IRB# 11-0431, Ref# 110244 was reviewed and accepted under expedited review. This will be reported at the 05/11/2016 meeting of the JHM ACH Institutional Review Board. This action fits the criteria for expedited review under research category 45 CFR 46.110 (b)(1).

As Principal Investigator of this protocol, it is your responsibility to maintain the documentation and records per the federal requirements under the Health Insurance Portability and Accountability Act of 1996 (HIPAA) and Title 45 CFR 46.

Thank you for your participation in the JHM ACH Institutional Review Board research process.

Sincerely,

VJorgensen

Signature applied by Verena Jorgensen on 04/27/2016 12:50:42 PM EDT

E. Verena Jorgensen, M.D. Chair, JHM ACH IRB

EVJ:se