Conflict with Parents in Adolescent Depression

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Submitted for the degree of philosophiae doctor at the Department of Psychology,
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Series of dissertations submitted to the
Faculty of Social Sciences, University of Oslo
No. 830

ISSN 1564-3991

Cover: Hanne Baadsgaard Utigard.
Print production: Reprosentralen, University of Oslo.

Contents

List of Figures

Acknowledgements

Looking back at eight years of integrated clinical specialist and PhD training, I feel both a sense of gratitude as well as a measure of sadness at closing this chapter of my life. Many people have contributed to these years being both challenging, rewarding and filled with personal growth.

I am grateful to Pravin Israel for involving me in the BUD project, and for giving me a lot of responsibility and opportunity to participate in design and planning of the trial. I have learned a lot both as a researcher and a clinician, even though the running of the trial did not go as well as hoped, and our cooperation had to come to an end.

I have been fortunate to have Luxsiya Waraan as a fellow PhD-candidate in the project, in particular when we have had to face disappointments and frustrations; I could not have wished for a more optimistic and resilient colleague to share such experiences with.

I wish to thank Marianne Aalberg, who took over responsibility for the trial after data collection had ended, and who also agreed to take over as my supervisor. Thank you for light-handed and useful guidance, handling a lot of difficult issues about the project non-dramatically, and for always being enthusiastic about my forays into unknown statistical territory.

I am grateful to Nikolai O. Czajkowski for inadvertently setting me on a path to learning to do Bayesian statistics, for enthusiastically helping me keeping on with that journey, and for eventually agreeing to also formally be my supervisor.

I thank the University of Oslo, Akershus University Hospital and the "National Program for Integrated Clinical Specialist and PhD-training for Psychologists" for the opportunity to develop professionally both as a clinician and as a researcher. Thanks to my fellow travellers in the program, in particular Vidar Ekroll, Nils Martin Sønderland, Sverre U. Johnsen and Kristianne S. Ovenstad, for inspiring discussions and cold swims.

I am also grateful to: The Stan and R projects and all involved for making the free software that has enabled this research. My dear friend Thomas J. Fyhn for shared lunches and fruitful cross-field discussions. Jon T. Monsen, Ole André Solbakken and Ellen Hartmann, both for believing in me and for allowing me to go my own way without bitterness. Tine Jensen for giving me the opportunity to develop as a teacher as well as a researcher. The late Elisabeth Grindheim for once showing me that development is at the heart of clinical psychology. Anne-Stine Meltzer for supporting the research project, and for helping me get the necessary clinical practice with very few hassles, and Rannveig Woll Lothe for continuing what Anne-Stine started.

My heartfelt thanks go to Follo BUP and all my dear colleagues there for being my professional home, safe haven and secure base through all these years. I always feel welcome and valued among you, even after long absences.

I have been privileged to work with many different patients and families through these years, and I thank them all for trusting me, moving me, challenging me and educating me about life.

I have the privilege of being the father of Trygve, Sigurd, Liv and Norea: Thank you for every day reminding me that life is more than work. I thank all my four parents for raising me to never unreasonably doubt my abilities. Finally, to my wife, best friend and favourite colleague Eline: Thank you for patient listening, advice and support, for being proud of me, for sharing equally the work and the joy of our home and family, and for always standing by my side.

Oslo, March 2020
Erling W. Rognli

Summary

Adolescents who are depressed tend to have more conflictual relationships with their parents, and depression and parent-adolescent conflict is thought to have bidirectional association. This thesis presents results from three studies of conflict with parents in adolescent depression. Data was collected as part of a clinical trial comparing Attachment-based Family Therapy to Treatment as Usual for adolescent depression. Analyses were carried out within a Bayesian statistical framework with estimation by Hamiltonian Monte Carlo, and leave-one-out cross-validation and stacking of predictive distributions was used for model evaluation.

The first study analysed outcome data from the trial to evaluate a registered secondary hypothesis of treatment moderation by parent-adolescent conflict, which was partially supported by the results. The overall treatment effect in the trial was small. For adolescent report of conflict with mother, and mother-report of conflict with the adolescent, the data supported a moderator effect in the expected direction. For adolescent report of conflict with father, there was no evidence of a moderator effect. Father-report of conflict with the adolescent was related to substantial differences in the outcome of Treatment as Usual, but not of Attachment-based Family Therapy. The predicted differences in treatment outcome were only of a clinically relevant magnitude in the upper and lower quantiles of the distribution of conflict. Seen together with previous research, this indicates that parent-adolescent conflict may moderate the effectiveness of family- or relationship-focused treatments for adolescent depression when compared with more individually focused treatments.

The second study examined parental characteristics as predictors of adolescent-reported parent-adolescent conflict. Parental depressive symptoms have been found to be associated with increased parent-adolescent conflict. As resolution of conflicts is inevitably an interpersonal situation, an association with parental interpersonal difficulties would also be expected, but this has not previously been studied. Parental depressive symptoms were compared with parental report of interpersonal problems as predictors of conflict. Models with effects varying by parent gender were compared with models with equal effects assumed. Parents who reported problems being too dominant in relationships tended to have adolescents reporting more parent-adolescent conflict, while parents who reported problems with being unassertive and too submissive tended to have adolescents reporting less parent-adolescent conflict. This applied equally for mothers and fathers. For parental depressive symptoms, only a negative association was found for the report of fathers, which was surprising given the existing literature. The findings of the second study suggest that parental interpersonal problems related to dominance and submissiveness is involved in parent-adolescent conflict with depressed

adolescents, perhaps by derailing normative processes related to development of autonomy, or by the way these parents respond to the impaired functioning of the depressed adolescent.

The third study compared discrepancy in the report of adolescents and parents about parent-adolescent conflict to the report of either of them as predictors of adolescent hopelessness. As hopelessness is a predictor of suicidal ideation and poor treatment outcomes in adolescent depression, it is important to understand more about what differentiates depressed adolescents feeling hopeless from those who do not. Unfortunately, rather few studies have investigated this in clinical samples. Discrepancy in reporting is assumed to reflect differences in parent and adolescent representations of the state of their relationship. Based on this, the hypothesis was that large discrepancies can lead to adolescents perceiving a state of conflict as persistent, increasing hopelessness. Results gave preliminary support to the hypothesis. Parents reporting less severe conflict than the adolescent was related to increasing hopelessness, and the absolute level of conflict provided less predictive accuracy than informant discrepancies. The findings warrant attempted replication in a larger sample.

While a small sample size and a large proportion of missing outcome data preclude very strong conclusions, these studies still add to the literature by providing evidence and some new leads for research on conflict with parents in adolescent depression. Discrepant reporting of conflict is shown to be informative and studying change in discrepancy longitudinally may shed further light on its relationship to hopelessness and other clinical characteristics in adolescent depression. Interpersonal theory and the interpersonal circumplex is shown to be a relevant theoretical and measurement framework for studying interaction processes in the family of depressed adolescents. Findings also support further investigation of parent-adolescent conflict as a moderator variable in adolescent depression, adding to existing research suggesting parent-adolescent conflict to be a variable with potential for informing treatment selection.

List of Papers

Paper 1:

Rognli, E. W., Waraan, L., Czajkowski, N. O. & Aalberg, M. (Submitted). Moderation of Treatment Effects by Parent-adolescent Conflict in a Randomized Controlled Trial of Attachment Based Family Therapy for Adolescent Depression.

Computer code for the analysis and other supplementary materials can be found at DOI 10.17605/OSF.IO/KPJC6.
The published version of the paper can be found at DOI 10.21307/sjcapp-2020-011

Paper 2:

Rognli, E. W., Waraan, L., Czajkowski, N. O., Solbakken, O. A., & Aalberg, M. (2020). Conflict with Parents in Adolescent Depression: Associations with Parental Interpersonal Problems and Depressive Symptoms. Child Psychiatry and Human Development. doi:10.1007/s10578-020-00955-0

Computer code for the analysis and other supplementary materials can be found at DOI 10.17605/OSF.IO/D2F8A

Paper 3:

Rognli, E. W., Aalberg, M., & Czajkowski, N. O. (Submitted). Using Informant Discrepancies in Report of Parent-adolescent Conflict to Predict Hopelessness in Adolescent Depression.

Computer code for the analysis and other supplementary materials can be found at DOI 10.17605/OSF.IO/75ZER
The published version of the paper can be found at DOI 10.1177/1359104520969761

Introduction

The parent-child relationship is a cultural icon of unconditional love and devotion, but parent-child relationships are also characterised by conflict and negative emotions (Dix, 1991). This duality of strong emotional bonds and conflict persists into adolescence, with most research showing an average slight increase in conflict, and an average slight decrease in warmth and closeness between parents and adolescents during this period (Laursen & Collins, 2009). An average trend can conceal systematic variability between subgroups of the population studied, and this also appears to be the case for parent-adolescent conflict (Laursen, Coy, & Collins, 1998; Montemayor, 1983). Adolescents suffering from depression is one subgroup often found to have heightened levels of conflict with their parents (e. g. Sheeber, Davis, Leve, Hops, & Tildesley, 2007). Further, parent-adolescent conflict and relationship difficulties have been found to prospectively predict depressive symptoms in adolescents (e. g. Kelly et al., 2016). This makes parent-adolescent conflict among depressed adolescents a relevant topic for further research, to which the present thesis is a contribution.

Adolescent Depression

Depressive episodes are characterised by low mood and/or lack of interest and pleasure that is abnormal for the individual in terms of intensity and persistence, accompanied by cognitive symptoms such as pessimism, hopelessness, low self-esteem and rumination, and somatic symptoms such as fatigue, disturbed sleep and changes in appetite or weight. Difficulty concentrating, psychomotor agitation or retardation, inappropriate guilt, suicidal or self-injurious ideation and behaviour, are also recognised as depressive symptoms. Psychotic symptoms can occur in severe depressive episodes. Panic attacks and various symptoms of anxiety are also prevalent, but not considered depressive symptoms in the major diagnostic systems (American Psychiatric Association, 2000; World Health Organization, 2019). The symptom of hopelessness has a particular significance, as it has consistently been found to be a predictor of suicidal behaviour and is associated with poor treatment outcomes (Asarnow et al., 2011; Weersing, Jeffreys, Do, Schwartz, & Bolano, 2017). Hopelessness has even been proposed as sufficient cause of depressive disorder (Liu, Kleiman, Nestor, & Cheek, 2015). The symptom presentation is generally found to be similar in adolescents and adults, but among adolescents somatic symptoms appear to be especially prevalent, while loss of interest and pleasure is relatively less common (Cole et al., 2011; Rice et al., 2019). Depressive disorders are also differentiated as single episodes of depression, recurrent episodes or chronic depression (Hammen, Brennan, Keenan-Miller, & Herr, 2008). Depression is one of the most prevalent mental health disorders, with a very large burden of disease (Ferrari et al., 2013; Ssegonja et al., 2019). While depressive

disorders do occur in children, there is a well-documented increase of prevalence in adolescence, which is also when the higher prevalence among females appear (Girgus & Yang, 2015). Onset of a depressive disorder in adolescence has been shown to predict poor developmental outcomes across multiple domains, as well as recurrence of depression (Clayborne, Varin, & Colman, 2018; Johnson, Dupuis, Piche, Clayborne, & Colman, 2018). Although there are efficacious treatments available, both psychosocial and psychopharmacological, a considerable number of patients do not respond adequately and calls have been made for improving treatment options (Spielmans, 2020).

The specific aetiology of depression is not known. There is a well-known moderate heritability of depressive disorders, but specific genetic variants accounting for substantial variance in phenotypes have not been reliably identified, suggesting a polygenic and continuous structure of the genetic risk (Flint & Kendler, 2014; Wray et al., 2018). Similarly, no phenotypic or contextual stressors or diatheses have been identified that are either necessary or sufficient, suggesting that adolescent depression should be conceptualised as an equifinal developmental outcome (Cicchetti & Toth, 2009).

Interpersonal stressors and adolescent depression

Although they are neither necessary nor sufficient, it has been shown that interpersonal stressors are more strongly related to depression than other life stressors (Slavich & Irwin, 2014). Further, persons suffering from depression tend to generate more frequent and severe interpersonal stressors through their own behaviour (Liu & Alloy, 2010), giving a bidirectional relationship between interpersonal stress and depression. While much of the literature on this bidirectional relationship between interpersonal stress and depression is built on studies of adult depression, the same associations and patterns seem to extend to adolescent depression (Rudolph et al., 2000).

Parents hold a unique position in the interpersonal context of the adolescent, and adolescents appear to find strains in the parent-adolescent relationship particularly stressful (Persike & Seiffge-Krenke, 2016). Parent-adolescent relationships are obligatory for both parties throughout adolescence, making the parent-adolescent relationship a mostly unavoidable interpersonal context (Laursen & Bukowski, 1997). At the same time the threshold is lowered in obligatory relationships for employing interpersonal behaviours that might lead to the dissolution of non-obligatory relationships, such as coercive or aggressive conflict strategies or interpersonal withdrawal (Laursen & Collins, 2009). Parent-adolescent conflicts are an important kind of interpersonal stressor that is partially dependent on adolescent behaviour, and there

is a robust literature supporting parent-adolescent conflict as an important factor in the development of adolescent depression.

A large number of studies have consistently found parent-adolescent conflict to prospectively predict adolescent self-report of depressive symptoms in community samples (Cohen et al., 2015; Hale 3rd, Nelemans, Meeus, & Branje, 2020; Jones, Beach, & Forehand, 2001; Kelly et al., 2016; Sallinen, Rönkä, Kinnunen, & Kokko, 2016; Sheeber, Hops, Alpert, Davis, & Andrews, 1997; Smith, Nelson, & Adelson, 2019; Yan, Schoppe-Sullivan, & Feng, 2018; Zhang, Baams, van de Bongardt, & Dubas, 2017). One study using a genetically informed design found the effect of parent-adolescent conflict to be moderated by genetic risk, with stronger effects of parent-adolescent conflict at higher levels of genetic risk (Rice, Harold, Shelton, & Thapar, 2006), but another similar study concluded higher genetic risk of depression predicted a relationship trajectory characterised by low support and high conflict, in addition to depression (Brouillard, Brendgen, Vitaro, Dionne, & Boivin, 2018). However, these findings are both completely compatible with a transactional relationship between parent-adolescent conflict and depression, given the temporal resolution of measurements in both. A third genetically informed study found the similar construct of parental hostility, assessed by adolescent as well as parental report, to predict depressive symptoms via an environmental pathway (Lewis, Collishaw, Thapar, & Harold, 2014).

Studies like these, using self-report of depressive symptoms in non-clinical samples, have been criticised for conflating general distress with clinical depression (Coyne, 1994). This is clearly a valid concern, even though the continuity between subclinical and clinical adolescent depression is well supported (Cole et al., 2011; Hankin, Fraley, Lahey, & Waldman, 2005; Klein, Shankman, Lewinsohn, & Seeley, 2009). A converging line of evidence comes from studies comparing clinical and non-clinical samples, suggesting that the association found in community samples extends to the clinical range of depression. Two studies using multimethod and multisource assessments show that clinically depressed adolescents have more conflictual relationships to their parents than healthy controls, and that heightened parent-adolescent conflict is also present among adolescents with subclinical symptom levels (Sheeber et al., 2007; Sheeber & Sorensen, 1998). Parent-adolescent conflict has also been shown to predict recurrence or persistence of depression (Alaie, Laftman, Jonsson, & Bohman, 2019; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000). An observational study comparing problem-solving interactions of depressed and non-depressed adolescents and their parents and using a network approach to model the affective interaction, found evidence of heightened angry affect in the depressed group compared to those who were not depressed (Bodner, Kuppens, Allen, Sheeber, & Ceulemans,

2018). A review of the literature on risk factors for depression among Chinese youth also found support for an association between parent-adolescent conflict and adolescent depression (Tang, Tang, Ren, & Wong, 2019), showing that this association is not necessarily linked to a western cultural context.

Further, there is a body of research indicating that parent-adolescent conflict may play an important role in the intergenerational transmission of depression, as families with member suffering from depression are more likely to have conflictual relationships (Hale 3rd et al., 2020; Hammen, Brennan, & Shih, 2004; Rothenberg, Hussong, & Chassin, 2018; Withers, Cooper, Rayburn, & McWey, 2016). Genetically informed studies, using adoption or children of twins designs, suggest that shared genetic risk only explains some of the intergenerational transmission of depression (Silberg, Maes, & Eaves, 2010; Tully, Iacono, & McGue, 2008), which supports implicating parent-adolescent conflict in a transactional relationship with depression in parents and adolescents, and not as a mere epiphenomenon of depressive states.

Yet, seemingly at odds with this literature, an increase in parent-adolescent conflict in adolescence is found to be normative (Laursen et al., 1998), and probably developmentally functional (Branje, 2018), while adolescent depression is neither, necessitating a more differentiated discussion of parent-adolescent conflict as a phenomenon.

Parent-Adolescent Conflict

A challenge in any study of conflict is the ambiguity of the term itself. Interpersonal conflict can refer to a state of incompatibility of goals, an interpersonal event where such a state shapes the behaviour and perception of those having incompatible goals, or to a particular representation of the state of an interpersonal relationship. In her seminal work on conflicts between children, Shantz (1987) differentiated conflict from aggression, pointing out that aggression can be instrumental, impulsive or even playful, and that the perceived association between aggression and conflict is incidental to the frequent use of aggression as a conflict resolution strategy. She defined conflict as an event involving actors with incompatible and interdependent goal states. This definition usefully differentiates the state of conflict from the behavioural strategies employed to resolve it. However, it fails to capture the interplay over time between how conflict events play out, changing representations of the relationship, and future conflict events. During development the capacity for mentally representing interpersonal relationships increases, and conflict events and their resolution are decreasingly isolated events, but rather continually integrated into the representational part of the relationship (Laursen & Bukowski, 1997; Rueter & Conger, 1995). Multiple conflict events where resolution is not achieved in a satisfactory way

could then lead to expectations of repetition, in turn lowering the threshold for initiating conflict behaviours in both parents and adolescents. Such circular processes have been observed in non-clinical samples (LoBraico, Brinberg, Ram, & Fosco, 2019). A concept of conflict restricted to interpersonal events on the timescale of immediate experience is clearly insufficient for describing parent-adolescent conflict. Parent-adolescent conflict exists as a transactional phenomenon in the interaction over time between incompatible goal states, variations in conflict behaviours and forms of resolution, and the representations of the parent-adolescent relationship in the minds of parents and adolescents.

Incompatible goals are inevitable in parent-adolescent relationships

An argument can be made that incompatible goal states are inevitable between parents and adolescents. Trivers (1974) laid out the foundation of an evolutionary account of parent-offspring conflict. He pointed out how the simple fact that parents are only half as closely related to their children as the children are to themselves will create differences in what level of parental investment is optimal for their inclusive fitness. Parent-offspring conflict theory applies across all species reproducing sexually, including plants, and can explain certain otherwise puzzling phenomena (Godfray, 1995). Schlomer, Del Giudice, and Ellis (2011) present an application of parent-offspring conflict theory to human families. They suggest that human behavioural systems for providing care to children are shaped by evolution to provide different levels of parental care based on the perception of resource scarcity or abundance, offspring quality and viability, future reproductive possibilities for the parent and other offspring available for investment, functioning to maximise the inclusive fitness of the parent. They further argue that reflecting this organisation of parental behaviour, the behavioural systems for seeking care in childhood should be shaped for generally extracting a higher level of investment than the parent would otherwise provide, maximising the inclusive fitness of the child, which is different from that of the parent. Clearly, the developmental history of the individual and the practical, cultural and societal contexts will also impact the behaviour of parents and children, but in complex interaction with these evolved behavioural systems. At the level of the experience of the individual, the operation of such behavioural systems are thought to be observable as patterns of emotional and motivational states conditional on particular stimuli (Bowlby, 1969).

Adolescence is a period of transition, where parental care is gradually withdrawn, and parental control over behaviour is gradually reduced (Laursen & Collins, 2009). Parent-offspring conflict theory predicts that the timing of these transitions should be contentious, and that previously stable resolutions to the conflict about level of parental investment must often be renegotiated. Schlomer et al. (2011) review three reasons for

why parents would be motivated to reduce the level of support provided earlier than adolescents, and to maintain behavioural control for a longer period than adolescents would prefer. Firstly, it is often in the interest of the parent to have the adolescent delay mating. By delaying mating, the adolescent can be induced to invest in siblings, which are twice as highly related to the parent as potential grandchildren are. Secondly, as humans may in this way invest resources in their extended family rather than their own offspring, the preferred level of risk-taking should differ between parents and adolescents. For adolescents, high levels of risky behaviour may be evolutionary adaptive, because risky behaviour may increase access to reproductive opportunity, and the potential fitness cost of lacking reproductive opportunity is extremely high to the individual (Ellis et al., 2012). For parents, investment of resources in the extended family is equally valuable to investment in the particular grandchildren of the child, in terms of fitness gains. Reproductive access is hence less valuable for the parents than for the adolescent, and parents should be selected to limit adolescent risk-taking, as the potential benefit is lower to the parents. Thirdly, the interests of the parent and the adolescent do not align completely in the choice of mate, where genetic quality should be weighted higher by the adolescent, and the ability of a potential mate to invest in the extended family again be weighted higher by the parents (Schlomer et al., 2011).

These differences in evolved propensity for certain goal states, as well as the change in actual dependence on the part of the adolescent create a situation where both parents and adolescents have to make trade-offs in the transitional period from childhood to adulthood. For adolescents, the trade-off is between receiving continued parental support and avoiding parental behavioural control limiting their reproductive options. For parents, the trade-off is between limiting the allocation of resources invested in the adolescent but not triggering an adolescent withdrawal completely ending their behavioural control and influence over adolescent reproductive choice. In parent-offspring conflict theory, there has been a move towards the predicted stable resolutions to conflicts as the observable evidence for the theory (Godfray, 1995). However, in the case of the transition to adulthood in humans, there are probably different conditional strategies that have evolved both in adolescents and parents, with the overall reproductive life-history of each and cues of the environmental context determining the individual parameters of their trade-offs and the selection of strategy (Belsky, Steinberg, & Draper, 1991). This means a single or small set of stable resolutions may not have evolved, with the resolution of conflicting goals happening at the phenotypic level and experienced by the individual.

Adolescence requires renegotiation in the parent-adolescent relationship

This unavoidable incompatibility of goals arising in adolescence necessitates a process of relationship renegotiation between parents and adolescents. The expectancy violation-realignment model proposes that parent and adolescent expectations for their relationship are more frequently violated during adolescence, with these violations leading to a gradual realignment of expectations and hence a changed relationship (Collins, Laursen, Mortensen, Luebker, & Ferreira, 1997; Collins & Luebker, 1994). Arguably, this model describes the same developmental pattern as the one suggested by the previous application of parent-offspring conflict theory, but at the proximate and descriptive level of subjective experience and interaction in the family rather than at the ultimately explanatory evolutionary level. Further, expectations are violated in multiple areas, not only in misaligned interests concerning parental support and control, but also in how interpersonal conflicts between parents and adolescents are resolved. In childhood, resolutions of conflicts between parents and children most frequently happen through assertion of parental power (Laursen & Collins, 2009), but in adolescence the dependency and difference in competence that underlies parental power diminishes (Laursen & Bukowski, 1997), making this form of conflict resolution less stable.

Studies investigating conflict resolution between parents and adolescents in community samples point to how the form of resolution is what is most strongly associated with adolescent adjustment, not the presence of conflict as such (Laursen & Hafen, 2010).

In a large cross-sectional community sample of adolescents researchers found that patterns of conflict resolution reported by adolescents was related to the strength of association between conflicts and depressive symptoms, and that the association was strongest for a pattern of conflict resolution characterised by a combination of engaging in negative conflict behaviours and intermittently submitting to parental dominance and withdrawing without resolution (Branje, van Doorn, van der Valk, & Meeus, 2009). Longitudinal follow-up of the same sample showed a bidirectional prospective relationship between these forms of conflict resolution and conflict frequency (Missotten, Luyckx, Branje, Hale, & Meeus, 2017). Another study used an observational conflict discussion task and a stratified sample of adolescents with non-clinical, sub-clinical and clinical status, finding the way adolescents and parents resolved conflict to be related prospectively to change in adolescent psychopathology at a two-year follow-up, with poor management of conflict predicting exacerbation of symptoms (Marceau et al., 2015). Negative conflict behaviours and withdrawal as a form of resolution was found to be associated with low life satisfaction among Turkish adolescents, lending some

support to the cross-cultural validity of an association between conflict-resolution and adjustment (Dost-Gözkan, 2019).

Conflict resolution renegotiation is an interpersonal process

A reorganization of interpersonal interaction patterns for conflict resolution is necessary for successful resolution of the unavoidable conflicts between parents and adolescents (Laursen & Bukowski, 1997). This renegotiation will not take the form of a completely explicit negotiation process between parents and adolescents. Change in interpersonal interaction patterns is a gradual process over the course of multiple interpersonal events (Pincus & Ansell, 2003). Over time, while resolving specific conflicts, parents and adolescents optimally also renegotiate conflict resolution patterns by gradually changing their way of responding to each other, in an implicit collaborative process (Beveridge & Berg, 2007). When this process is disrupted, persistent unresolved conflicts are likely to occur, as the conflict resolution patterns established in childhood cannot be sustained into adolescence and the development of goal incompatibility is unavoidable between parents and adolescents. The process of renegotiation will likely be dependent on the contribution of the parent, as the previously dominant part of the hierarchical parent-child relationship. Moed et al. (2015) conducted an observational study of parents and young adolescents interacting during discussion of a conflictual topic. They found the length of conflictual interactions and the degree of negative reciprocity to be associated with a higher proportion of adolescent-ended conflicts as well as perceptions of conflict as unresolved, demonstrating the importance of parents facilitating conflict resolution in early adolescence.

The necessity of renegotiating conflict resolution patterns may represent a point in parent-child relationship development where parental interpersonal difficulties can be expected to influence the further relationship trajectory strongly, by causing such disruption. The interpersonal circumplex is a well validated model of individual differences in such interpersonal difficulties (Gurtman, 1996; Monsen, Hagtvet, Havik, & Eilertsen, 2006).

The interpersonal circumplex model is defined by two orthogonal dimensions often termed Agency and Communion[1]. Agency concerns the element of power and autonomy in human relationships, while Communion concerns the element of closeness and bonding. Different interpersonal behaviours, motives and problems can be indexed

[1] The terms Agency and Communion derive from the work of David Bakan and have been widely applied also outside of personality assessment and interpersonal theory (Leonard, 2016). Other terms used for these dimensions are Dominance and Love (e. g. Gurtman, 1992). They have also been identified with the two most interpersonally relevant traits of the five-factor model of personality, Extraversion and Agreeableness (McCrae & Costa, 1989).

along these two dimensions, and related to each other based on their positions in the space defined by the intersection of agency and communion (Gurtman, 1992; Horowitz et al., 2006; Wiggins, Phillips, & Trapnell, 1989). Based on this theoretical model, parents with a rigid tendency towards low communion and/or high agency behaviours should find it particularly difficult to facilitate the renegotiation of conflict resolution processes, as they find it difficult to maintain close bonds and/or often take a dominant stance in interpersonal interactions.

Parent-adolescent conflict ranges from normative to dysfunctional

Taking a transactional view of parent-adolescent conflict in light of the reviewed literature, the kind of conflict resolution achieved in a given conflict event should be influenced by the relationship representations of the adolescent and the parent, their current interpersonal pattern of conflict resolution, as well as the importance to either one of resolving the goal incompatibility. In turn, the kind of conflict resolution achieved should influence the relationship representations, future patterns of conflict resolution as well as the salience of goal incompatibility. Failure of renegotiation of conflict resolution patterns between parents and adolescents during early adolescence would be expected to lead to repeated problems achieving satisfactory conflict resolutions, with the result of persistent unresolved conflict characterizing the state of the parent-adolescent relationship (Rueter & Conger, 1995). Such states of persistent, unresolved conflict may not necessarily be observable only as overt conflictual interactions, but also by interpersonal withdrawal, or avoidance of certain topics and issues, leading to a circumscribed form of parent-adolescent interaction. Parents and adolescents could in this way come to hold increasingly divergent representations of their relationship during adolescence.

This resolves the apparent contradiction between parent-adolescent conflict as a risk factor for depression as well as a normative and functional part of development. When parent-adolescent conflict is found to be associated with poor adjustment and depression it appears to be a form of dysfunctional conflict resulting from the failure of conflict resolution renegotiation processes, relationship representations that were already poor, and/or particularly intense incompatibility of goals. This dysfunctional form of parent-adolescent conflict is characterized by repetitiveness and lack of development towards a stable and power-symmetrical form of conflict resolution, high frequency of conflict behaviours and expressed negative emotion during conflict interactions, negative or diverging relationship representations, and interpersonal withdrawal. Careful attention to these differences between normative increases in parent-adolescent conflict and dysfunctional conflict processes appears crucial to the study of parent-adolescent conflict in adolescent depression.

Attachment-Based Family Therapy

This thesis is based on data collected as part of a clinical trial comparing Attachment-based Family Therapy (ABFT) with treatment as usual for adolescent depression. ABFT is a time-limited manualised treatment for adolescent depression and suicidal ideation (Diamond, Diamond, & Levy, 2014). The conceptual and technical origins of ABFT can be found in systemic family therapy, emotion-focused therapy and attachment theory (Diamond, Reis, Diamond, Siqueland, & Isaacs, 2002). It is an experiential therapy, focusing on in-session enactment of new emotional experiences between parents and adolescents as a mechanism of change. ABFT does not assume that conflict or other relational difficulties with parents are the cause of all depressive episodes in adolescents. Rather, the assumption is that depressed adolescents do not use their parents as a source of support and help in managing stressful circumstances in their lives. The first part of the therapy concerns establishing the interactional pattern of a secure attachment relationship, with adolescents actively seeking parental support and advice when they need it, and the parents providing a safe haven and secure base for developing autonomy. The second part of the therapy uses this interactional pattern to help the adolescent make changes that might alleviate depressive symptoms and promote age-appropriate autonomy, such as managing conflicts with parents, peers or school, increasing their activity level, having healthy daily routines, sorting out identity issues and so on. Throughout this phase of therapy, the focus is on reinforcing an interactional pattern of the adolescent defining goals and seeking the advice and support of the parents in refining and achieving these goals, within the context of a validating attachment relationship. By repeated practice with this interaction pattern in sessions, with the support and guidance of the therapist, the family improves their skill at maintaining this interaction on their own, after treatment ends.

ABFT is manualised in the form of treatment tasks, which specify intermediate steps to be completed in therapy before moving on to later tasks. These tasks start with reviewing the depressive episode and its relational context and implementing a relational reframing of the presenting problem. The goal of the first task is to get agreement from both the adolescent and the parents to initially focus treatment on repair of the parent-adolescent relationship. This is accomplished by carefully evoking specific emotional content in the initial session and then refocusing the conversation on the state of the parent-adolescent relationship. If the first task is successfully completed, the therapy moves on to a parallel process with parents and adolescents. The adolescent is assisted in accessing and articulating reasons for not seeking parental support, whether ongoing parental behaviour or previous events, motivated to discuss these issues of distrust, resentment or disappointment frankly with the parent, and helped to

prepare to do so in a mature and regulated manner. The parents on their hand are helped to acknowledge and accept possible ways stressful life circumstances or their own upbringing may have prevented them from being emotionally available and sensitive parents, and to see how this may have impacted their child. Parents are motivated to adopt a validating and explorative attitude to the emotional experiences of their adolescent child and taught some emotional coaching skills. If both tasks with parents and adolescents are successfully completed, the family is brought together for a joint session, where the adolescent shares the issues articulated in the previous task and associated emotions, and the parents validates and explores these, without becoming defensive. This usually leads to a renewed level of trust and emotional closeness in the parent-adolescent relationship. This leads into the fifth task, which is usually the second part of the treatment, promoting adolescent autonomy and reducing depressive symptoms through various collaborative strategies in the family, while reinforcing and practicing new relational skills.

ABFT and dysfunctional parent-adolescent conflict

The change processes proposed in ABFT are likely to be suitable for treating adolescent depression despite high levels of dysfunctional parent-adolescent conflict, although this has not been directly empirically tested. The relational reframe implemented in the first session ensures an early focus on the state of the parent-adolescent relationship. The therapist further actively promotes activation of the complementary attachment and caregiving behavioural systems of the adolescent and parents in sessions (Diamond et al., 2014), thus in a sense regressing the family to a preadolescent stage of relational development, with clearly defined hierarchical roles. From this position, the therapy proceeds by negotiating adolescent autonomy and resolving conflicts. The therapist also explores and recognises the stressors facing the parent, offering emotional support and assisting the parent in accessing other services when appropriate. This would be expected to increase parental motivation to provide support for the adolescent, as the parental trade-off between investment in offspring and other priorities is likely to be sensitive to the perceived level of resources available to the parent (Schlomer et al., 2011). In this view, the change process of ABFT can be conceptualised as restarting the process of parent-adolescent relationship renegotiation when it has been disrupted, which is achieved by resetting the parent-adolescent dyad to their original relational positions.

Current evidence base of ABFT

The efficacy of ABFT has primarily been tested by the group who developed the treatment model. Three clinical trials have been conducted. The first pilot trial enrolled 32 adolescents with a Major Depressive Disorder, who were randomly allocated to 12

weeks of ABFT or a 6-week waitlist control condition. Results gave some support for the efficacy of ABFT, as the proportions of remitters differed between the groups both at post-treatment and at follow-up after 6 months (Diamond et al., 2002).

The next trial conducted by the treatment developers enrolled 66 adolescents with suicidal ideation. Participants were randomly allocated to three months of Enhanced Usual Care (assisted referral to private practice or community mental health centres, along with clinical monitoring sessions) or ABFT. Results supported the hypothesis of ABFT causing more rapid improvement in suicidal ideation, and a larger proportion of remitters, differences which persisted in the follow-up period. There was also a lower rate of drop-out in ABFT. However, the differences in number of sessions attended were considerable between ABFT and Enhanced Usual Care, and the specificity of the effect of ABFT could thus not be definitely established (Diamond et al., 2010). A third trial enrolled 129 adolescents who were randomized to ABFT or Family-enhanced Non-directive supportive therapy, with the same therapists providing both treatments. Results did not support the hypothesis of ABFT superiority. Both groups improved considerably on average, and the proportion of remitters at 16 weeks was around 40 % in both groups (Diamond et al., 2019), which is quite consistent with findings from previous research (Brent et al., 1997; Emslie et al., 2010; Kennard et al., 2009).

A pilot randomized clinical trial comparing ABFT to treatment as usual was later conducted in a Norwegian Child and Adolescent Mental Health Service, enrolling 20 referred adolescents with a diagnosis of Major Depressive Disorder (Israel & Diamond, 2012). Results were inconclusive, given the very small sample size, but suggested that ABFT could feasibly be implemented, was acceptable to Norwegian families, and might be efficacious. These results led to the larger randomized clinical trial which this thesis is based on.

The Norwegian ABFT clinical trial

The clinical trial "Behandling av Ungdom med Depresjon" (BUD) was planned as a multisite pragmatic effectiveness trial, with a target sample size of 120 adolescents. These would be randomized to ABFT or treatment as usual. ABFT therapists were trained among the regular staff at three outpatient clinics within the Department of Child and Adolescent Mental Health Services at Akershus University Hospital. Identification of eligible patients and patient enrolment was integrated with the regular intake procedures of the outpatient clinics, who receive almost all referrals for specialist level mental health care for children and adolescents in their geographical catchment areas.

The BUD clinical trial was unfortunately plagued by inadequate organization and management, as well as a lack of personnel. One of the three outpatient clinics never

enrolled any patients in the trial, and one clinic enrolled very few patients, with a majority of the 61 patients allocated to treatments (51) coming from one of the three planned sites. Follow-up assessments and collection of outcome measures were understaffed and poorly organized. Although the trial had funding to purchase a secure online platform for automatic administration of self-report assessments, respondent compliance with online assessments was low, and there was no staff available to follow up on respondents missing assessments. This led to large proportions of missing data. Primary outcome measurements were conducted by an independent clinical psychologist hired per respondent, which presented some scheduling difficulties, and required respondents to come to a separate appointment for the outcome assessment. This probably contributed to a considerable number of participants declining to participate, which led to an unfortunate 38% proportion of missing outcomes at 16 weeks. Planned follow-up assessments at 26 and 52 weeks were not conducted at all, due to lack of resources and organizational capacity. Supervision of therapists were planned as weekly sessions, and the therapists at the site which included most patients met almost weekly for the entire period of the trial to discuss cases and review video-recordings of therapy sessions (91 supervision meetings over in all 137 weeks including holidays and summer vacations). However, as the trial only had access to one certified ABFT supervisor, who also served as the principal investigator at that time, many of the weekly supervision sessions had to be conducted as peer supervision, when the certified supervisor was occupied or otherwise unavailable. Sometimes, supervision was conducted as a combination of peer supervision and discussion with the certified supervisor by phone. In all 91 sessions were held during the time of the trial, in which 38 (42%) were attended in person by the principal investigator/ABFT supervisor, and 5 (5.5%) conducted by phone. The rate of attendance by the certified supervisor declined across the time of the trial, with a 67% attendance rate in the first year, 28% in the second and 12% in the final six months of the trial.

Overall, the BUD clinical trial was not successfully completed. The target sample size was not achieved, and outcome data was not collected with sufficient fidelity. Further, it is difficult to assess whether ABFT was delivered according to protocol, due to less supervision than planned, and lack of resources and certified personnel to rate video-recordings of therapy sessions. These limitations must be taken into account when interpreting findings.

Aims of the Thesis

This thesis aims to investigate how dysfunctional parent-adolescent conflict in adolescent depression is associated with parental characteristics, treatment outcome and clinical status, using data from a sample of depressed adolescents enrolled in the previously described BUD clinical trial.

Aims of study 1

The first study analyses the available outcome data from the BUD clinical trial, to evaluate the hypothesis that the difference in treatment effect between ABFT and treatment as usual would be larger at higher levels of parent-adolescent conflict, which was registered as the secondary hypothesis of the trial. Response rates to treatment of adolescent depression is unsatisfactory (Eckshtain et al., 2019; Spielmans, 2020) and knowing more about what treatments work for whom may help increase response rates (Kraemer, Wilson, Fairburn, & Agras, 2002). Parent-adolescent conflict has been found to predict poor treatment outcomes (Asarnow et al., 2009; Birmaher et al., 2000; Feeny et al., 2009), but the reverse has also been found for interpersonally focused and family-based treatments (Gunlicks-Stoessel, Mufson, Jekal, & Turner, 2010; Miklowitz et al., 2009; Young, Gallop, & Mufson, 2009). This makes parent-adolescent conflict a plausible moderator of Attachment Based Family Therapy compared to treatment as usual.

Aims of study 2

The second study evaluates whether parental interpersonal problems on the Agency and Communion dimensions (Alden, Wiggins, & Pincus, 1990; Gurtman, 1996) are associated with parent-adolescent conflict in adolescent depression, and if so, whether this association overlaps with the expected association between parental depressive symptoms and parent-adolescent conflict (Cheung & Theule, 2019; Withers et al., 2016). As parent-adolescent conflict has implications for treatment response and the course of depressive disorder, knowing more about the characteristics of parents that contribute to more or less conflict is valuable. As management of conflict inevitably is an interpersonal situation, parental difficulties in interpersonal functioning could also plausibly predict parent-adolescent conflict.

Aims of study 3

The third study compares informant discrepancies in report of parent-adolescent conflict with the reports of different informants as predictors of adolescent hopelessness. Due to a strong association with suicidal behaviour, hopelessness is a particularly important depressive symptom (Wolfe et al., 2019). A previous study found an association between adolescent report of parent-adolescent conflict and hopelessness,

while not finding the same association for parent report on the same constructs (Becker-Weidman et al., 2009). The authors recommended investigating these differences in perception of conflict in future research. Discrepancies in the reports of different informants have been shown to be informative variables in themselves and represent a way of measuring divergence in parent and adolescent representation of the state of their relationship (De Los Reyes, 2011), which may be an index of particularly dysfunctional conflict processes.

Methods

Participants and Procedures

Participants in the BUD clinical trial were recruited among patients referred to two Child and Adolescent Mental Health Services (CAMHS) outpatient clinics. Adolescent participants were required to be between 13 and 17 years of age, and to be currently living with a caregiver who had been their caregiver since before age 4 and was willing to participate in treatment. They had to have a Major Depressive Disorder according to the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV, American Psychiatric Association, 2000), and to score 15 or more on the Grid Hamilton Depression Rating Scale (Williams et al., 2008). Adolescents with a bipolar disorder, severe eating disorder, pervasive developmental disorder, intellectual disability or psychotic disorder were excluded from participating. The study protocol, participant information letters and consent forms were reviewed and approved by the Regional Ethics Committee.

Recruitment and baseline assessment

Trial recruitment procedures were integrated with the regular intake procedures of the participating clinics. Referral letters were screened for mentions of depression or core depressive symptoms. Further, the clinics routinely administers the Youth Self Report (Achenbach, 1991) to referred adolescents, and these were screened for raw scores on the Affective Problems subscale above 6, which would indicate probable depression (Eimecke, Remschmidt, & Mattejat, 2011).

All adolescents either identified as possibly depressed in their referral letters, or with YSR scores indicating possible depression, were offered assessment of eligibility for participation in the trial. Norwegian law gives adolescents the right to confidentiality and to consent to healthcare from the age of sixteen, and adolescents above age fifteen were hence contacted directly by phone. When adolescents were below age sixteen, their parents were contacted first. The study coordinator gave a short, scripted introduction to the study, and if both parents and adolescents expressed interest in participating, the adolescent was asked to complete the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996), as a phone interview. Patients scoring above 17, a threshold expected to maximise sensitivity and specificity (Dolle et al., 2012), were invited to an extended clinical assessment, together with their parents.

At this appointment, adolescents and parents were given complete information about the study and any questions were answered. Adolescents and parents then gave informed consent or assent to participate. Clinical interviews were conducted separately with participating adolescents and the parents. Adolescents and parents also completed self-report measures during the appointment. At the end of the appointment the

clinician determined eligibility for randomization, and then conducted the randomization by opening a sealed, numbered envelope. Randomization was by permuted blocks of four, and stratified by gender, age and severity of depression. Whether or not they were allocated to treatments, families were informed about the assessment results and the diagnostic conclusion. To the extent deemed necessary, the assessing clinician implemented safety monitoring procedures according to clinic guidelines. The flow of participants through the BUD clinical trial is displayed in figure 1. Characteristics of the sample are summarised in table 1.

Diagnoses

A DSM-IV diagnosis of a current Major Depressive episode was a requirement for adolescents to be included in the trial, and some diagnoses were exclusionary criteria. Diagnostic assessments were conducted with the Kiddie-Schedule for Schizophrenia and Affective Disorders (K-SADS, Kaufman et al., 1997). This semi-structured clinical interview has demonstrated good validity for assessment of depressive disorders in adolescents (Lauth et al., 2010). Interviews were conducted with adolescents and parents separately, and information from these interviews combined for a final score using clinical judgement, in accordance with K-SADS guidelines. The interrater reliability of depression diagnoses were assessed by blinded rescoring of 28 interviews. κ for current Major Depression was 0.56, indicating fair interrater reliability (Landis & Koch, 1977).

Adolescent depressive severity and change

The primary outcome measure of the BUD clinical trial was the revised grid version of the Hamilton Depression Rating scale (GRID-HAMD, Williams et al., 2008). Originally published in 1967, this clinician-rated scale for depressive symptoms has been employed as the gold standard in assessment of depressive severity and treatment response for decades, arguably defining these constructs (Bagby, Ryder, Schuller, & Marshall, 2004; Hamilton, 1967). The Hamilton depression rating scale has also been used as an outcome measure in clinical trials of treatments for adolescent depression (Mufson et al., 2004; Mufson, Weissman, Moreau, & Garfinkel, 1999; Santor & Kusumakar, 2001). The GRID-HAMD improves the reliability of scores while maintaining the original scale, by adding an interview guide and allowing clinicians to evaluate the severity and frequency of symptoms separately (Tabuse et al., 2007).

Figure 1: CONSORT Flow Chart for the BUD Clinical Trial

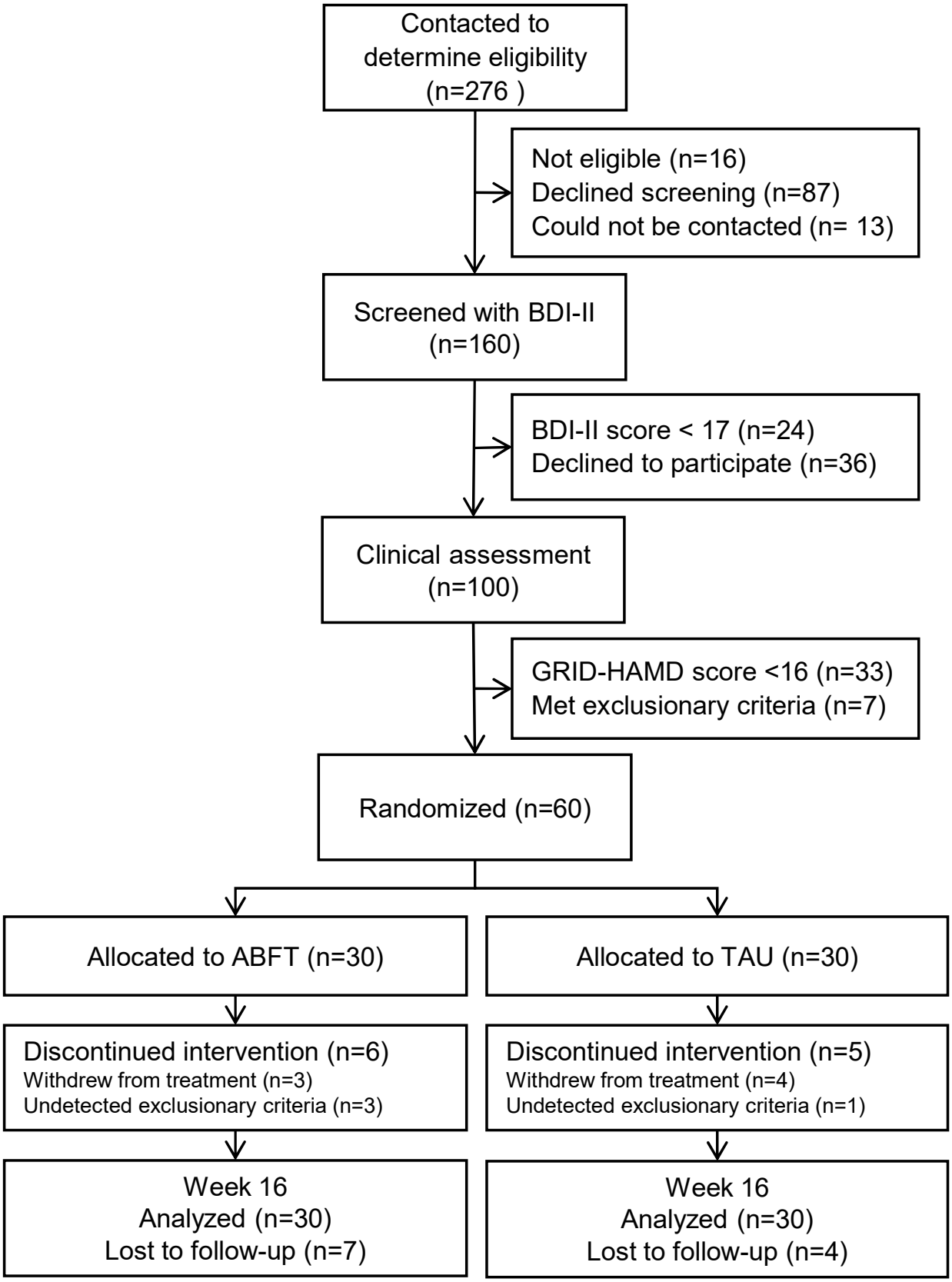


Table 1: Demographic and Clinical Characteristics of the BUD Sample

Variable		Treatment Arm	
		ABFT (n=30)	TAU (n=30)
Age, years (SE)		15.03 (1.35)	14.77 (1.36)
Gender, % (n)	Female	90 (27)	83.3 (25)
Dropout, % (n)	Excluded	7 (2)	3.3 (1)
	Dropped out	10 (3)	13.3 (4)
Ethnicity, % (n)	Norwegian	100 (30)	96.7 (30)
	Scandinavian	0 (0)	3.3 (1)
Living with, % (n)	Both parents	29.6 (8)	36.7 (11)
	Two home family	18.5 (5)	13.3 (4)
	Father (and partner)	18.5 (5)	13.3 (4)
	Mother (and partner)	33.3 (9)	33.3 (10)
	Other	0 (0)	3.3 (1)
Psychiatric	Dysthmia	3.3 (1)	0 (0)
comorbidity, % (n)	Any anxiety disorder	43.3 (13)	46.7 (14)
	Obsessive-Compulsive Disorder	6.7 (2)	6.7 (2)
	Externalizing disorder	0 (0)	13.4 (4)
	PTSD	3.3 (1)	3.3 (1)
	Eneuresis	3.3 (1)	6.7 (2)
	No comorbidity	53.3 (16)	46.7 (14)
Depressive symptoms,	BDI-II	34.23 (7.34)	36.21 (9.84)
mean (SD)	GRID-HAMD	21.87 (4.61)	21.92 (4.07)

Table Note: BDI-II = Beck Depression Inventory II Score; GRID-HAMD = Grid-Hamilton Depression Rating Scale Score; Any anxiety disorder includes social phobia, specific phobia, agoraphobia, generalized anxiety disorder, anxiety disorder NOS and obsessive compulsive disorder; Externalising disorder includes oppositional defiant disorder and attention deficit/hyperactivity disorder.

The inter-rater reliability of the GRID-HAMD was assessed by blinded rescoring of 28 video-recorded interviews. The two-way mixed, consistency, average-measures intraclass correlation coefficient for the total score (McGraw & Wong, 1996) was 0.89, indicating good interrater reliability.

Hopelessness

Although multi-item scales exist for measuring hopelessness, this construct appears to be unidimensional and measurable using very few items (Aish & Wasserman, 2001). As hopelessness is a depressive symptom, it is assessed as part of the K-SADS follow-up interview for depressive disorders and rated on a three-point scale from no hopelessness to a clinically significant level of hopelessness. These ratings were used as the dependent variable in study 3. The interrater reliability for the ordinal Hopelessness item was estimated following a Bayesian approach (Gajewski, Hart, Bergquist-Beringer, & Dunton, 2007). The posterior mean intraclass correlation coefficient was 0.79 (66% and 90% Highest Density Intervals 0.72 – 0.96 and 0.63 – 1)[2] indicating acceptable reliability.

Parent-adolescent conflict

Parent-adolescent conflict was measured using the Conflict Behavior Questionnaire (CBQ, Prinz, Foster, Kent, & O'Leary, 1979). This is a multi-informant measure with a parent form to be completed by parents, and an adolescent form to be completed by the adolescent separately for each parent. The CBQ has two subscales, Perception of the Parent/Adolescent and Perception of the Dyad. The Perception of the Dyad subscale has identical items for all informants, and the item content cover whether the respondent perceives there to be a relational state of frequent and dysfunctional conflict in the parent-adolescent dyad. The Perception of the Adolescent/Parent subscale has some items that differ between the adolescent and parent form, and the item content concern how the respondent perceives the conflict-related behaviour of their counterpart. The CBQ has been widely used, and there is support for its internal consistency and factor structure (Andrews, Lewinsohn, Hops, & Roberts, 1993; Curry et al., 2006; Khan, Malik, & Kamal, 2015; Rengasamy et al., 2013).

For study 2 and 3, the Perception of the Dyad subscale was more appropriate as a measure of parent-adolescent conflict. In study 2, including item content related to how the adolescent perceives the behaviour of the parent could induce or inflate associations with interpersonal problems or depressive symptoms due to construct overlap. In study

[2] The 66% and 90% Highest Density Intervals (see Kruschke, 2018), have the suggested interpretation of the likely and very likely range within which the true parameter value lies, conditional on the model and the data (Mastrandrea et al., 2010).

3, modelling informant discrepancy requires that the respondents are reporting about the same phenomenon, which they would not be if each of them were to report on the perceived conflict behaviour of the other. For consistency, the CBQ Perception of the Dyad subscale was chosen as the moderator variable in study 1 as well, which also allows for conceptually clearer comparison of the report of different informants as moderators.

Parental depressive symptoms and interpersonal problems

Depressive symptoms experienced by parents were measured using a revised subscale for depression from the Symptom Checklist 90-Revised (SCL-90-R, Derogatis & Unger, 2010) developed by Paap et al. (2011) using nonparametric item response theory in a Norwegian sample.

Interpersonal problems were measured using the Inventory of Interpersonal Problems (IIP-32). This is a shortened version of the Inventory of Interpersonal Problems – Circumplex (Alden et al., 1990). The items of this scale were selected from the original item set of the IIP (Horowitz, Rosenberg, Baer, Ureño, & Villaseñor, 1988), to form a measure with subscales assessing the different problem areas defined by the Interpersonal Circumplex. The factor structure of the IIP-32 matches the interpersonal circumplex very well (Gurtman, 1996; Gurtman & Balakrishnan, 1998). This circumplex structure makes it possible to summarise the patterning of responses to the whole scale in a few variables (Gurtman & Balakrishnan, 1998), calculating the position of the individual on the agency and communion dimensions, and the level of general interpersonal distress. The individual subscale scores of the IIP-C are correlated with the subscales neighbouring them in the circumplex, which presents a problem when using these subscale scores together as predictors in a regression analysis. This is a problem the structural summary scores avoid, and these were therefore used in modelling interpersonal problems as predictors of parent-adolescent conflict.

Statistical analysis

The statistical approach in this thesis differs from the usual statistical practice in clinical psychology, by adopting a fully Bayesian approach to estimation and inference (Baldwin & Larson, 2017). In Bayesian statistics, probability distributions are used to quantify uncertainty about quantities of interest. Bayes' theorem is the mathematical equation for conditional probability, and the results of a Bayesian data analysis is the probability of model parameters conditional on the data and the modelling assumptions and external knowledge we have included in the analysis (Gelman & Shalizi, 2013). These probability distributions will sometimes be very wide, showing the uncertainty about that parameter, essentially telling us that not much has been learned from these data (Baldwin & Larson, 2017). However, in some cases our ability to contribute to theoretical development is not dependent on precise point estimates. When little is

known about a phenomenon, and the number of plausible models is large, merely estimating the signs of some model parameters with some degree of certainty can be informative for further theoretical development. Scientific progress in fields such as psychology depends on iterative exploration of the potential model space (Devezer, Nardin, Baumgaertner, & Buzbas, 2019), and expressing the evidence for a model as the quantified uncertainty about its parameters rather than as a dichotomous decision of reject/accept can facilitate such exploration.

Priors

A necessary step in a Bayesian data analysis is the specification of prior distributions for all parameters. The prior distribution functions as the unconditional probability in Bayes theorem, which multiplied by the probability of the data conditional on the parameters and divided by the unconditional probability of the data, yields the conditional probability of the parameters. Specification of priors is probably the most contentious issue in the practice of Bayesian statistics, because there are no objective prior distributions for unknown parameters, and because the prior distribution contributes information to the results of the analysis (Aczel et al., 2020). Some object strongly to the inclusion of information external to the data in the results of a statistical analysis. So called non-informative priors are sometimes advocated, usually in the form of uniform distributions over the possible parameter space, or distributions that are extremely wide. This approach has some well-known problems, as it will often assign non-zero prior probability to parameter values that are known to be completely impossible. In most cases this would misrepresent the prior information that is actually available (Gelman, Simpson, & Betancourt, 2017). By standardizing variables to put them on the same scale, we can in most cases identify what magnitude of parameter values are at all possible. A regression coefficient of 1000 cannot happen with standardized variables, but a uniform prior would place equal prior probability on this as on any other parameter value, which is clearly unreasonable. Such non-informative priors can also lead to bias in estimation, in particular in small samples, where the likelihood of the data then exerts a weaker influence on the posterior distribution (McNeish, 2016).

In hierarchically structured models, hierarchical priors can be specified, where the parameters of some prior distributions are estimated from the data (Gelman et al., 2013). An example of this would be a multilevel regression model with varying intercepts, where the varying intercepts model the clustering of some observations, such as the repeated measures of depression in study 1 or the conflict with parents belonging to the same family as in study 2. We do not know the effect of belonging to a given cluster, so we need to estimate these varying intercepts as parameters, but we usually

lack meaningful information to specify a particular prior distribution for each cluster. However, we usually have more prior information about the reasonable distribution of clustering effects than we have about any particular cluster. This allows us to place meaningful priors on the parameters of the prior distribution of the varying intercepts. For instance, selecting a normal distribution for the varying intercepts, we specify the mean and variance of that distribution as parameters of the model. These parameters (called hyperparameters) would then need (hyper-) priors reflecting our assumptions about the plausible ranges of the mean and variance of the distribution of clustering effects (Gelman et al., 2013).

The general approach taken in these studies have been to specify priors that can be considered weakly informative in that they heavily down-weight or rule out implausible ranges of parameters but otherwise are not likely to shape the posterior much (Gelman et al., 2017), and to use hierarchical priors when relevant, with similar weakly informative hyperpriors. To illustrate, the prior for standardized regression coefficients across all three studies is a standard normal distribution. This gives a prior assuming that the magnitude of any coefficient will most probably be between 0 and 1, no assumptions about the direction of the association or that there is an association at all, and a very low probability of a coefficient larger than 2. Arguably, these assumptions are not very strong in the context of clinical psychology. Complete transparency about the prior distributions should be a standard of reporting (Aczel et al., 2020), and this information has been included in all three papers, either in the article or as supplementary material when necessary due to space restrictions. A complete list of priors used in the analyses can also be found in appendix 1.

Missing data management

A missing observation represents a problem in statistical modelling but is a frequent event in clinical research (Enders, 2017), including in the BUD clinical trial. The goal of missing data management is to preserve in the results of an analysis the increased uncertainty introduced by having unobserved data, as well as minimizing the increase of uncertainty by incorporating all relevant information collected (Enders, 2010).

Disregarding missing observations requires the very strong assumption that the process determining missingness is completely independent of both observed and unobserved variables (missing completely at random). It is also possible that the probability of being missing is systematically associated only with the values of the missing variable (missing not at random). A missing not at random mechanism is not verifiable as the missing values are unobserved by definition. The third possibility is that missingness is partially predictable from observed variables (missing at random), and

this is widely regarded as the most conservative assumption (Van Buuren, 2018). This means that the value of the missing observations can be inferred with some uncertainty from the structure of the dataset that is observed. Missing data in the BUD clinical trial was largely due to participants declining to provide information. This is not a probable mechanism for missing completely at random, as patient characteristics probably relate to the decision to decline providing information (Enders, 2010). Follow-up of participants not coming to appointments for outcome assessments or not answering online assessments was in some cases insufficient. This insufficient follow-up was more likely completely random, but still conditional on the participant deciding to not provide data in the first place.

In a Bayesian context the goals of missing data management can be accomplished by treating the missing observations as model parameters to be estimated. This means using the maximal amount of information available to fit the model, while also maintaining the uncertainty related to missing observations in the results (McElreath, 2016), and this is the approach that was taken across all three studies. Missing predictors were imputed using the estimated covariance matrix of the predictors, while missing outcomes were imputed from the likelihood. In study 2, there was a small amount of missing data at the item level in parental responses to the SCL-90 Depression Scale and the IIP-C. Before calculating scale scores, these missing item responses were singly imputed using two-way imputation (Sijtsma & van der Ark, 2003). The variability of the imputations was checked by imputing 10000 datasets and computing the within-person variability of the scale scores across those imputations. As the variability of the scale scores was negligible, single imputations was accepted.

Item response theory

Item response theory (IRT) is an approach to measurement of latent constructs that is distinct from classical test theory, which is still the dominant psychometric theory in psychology (Reise, Ainsworth, & Haviland, 2016). IRT sets up a mathematical model of responses to measurement items, modelling the probability of a given response as a function of the latent trait that is to be measured, and the measurement properties of the item that is responded to (de Ayala, 2009). IRT is an important methodological tool across all analyses in this thesis. In study 1, IRT is used to achieve greater measurement precision for the parent-adolescent conflict predictor variable, by using the latent trait parameter estimates rather than summed scores. In study 2 the outcome variable of the regression model is the latent trait parameter from the IRT model. In study 3, IRT models of both informants are used to obtain latent discrepancy scores, which are measures of discrepancy in reporting that avoid the problems of raw discrepancy scores.

The IRT model used in these studies is a two-parameter logistic model. These models are used for dichotomous items measuring a single latent trait. The probability p of respondent i endorsing item x is modelled as a logistic function of the latent trait parameter θ of the respondent, and two item parameters α and β, according to the following equation:

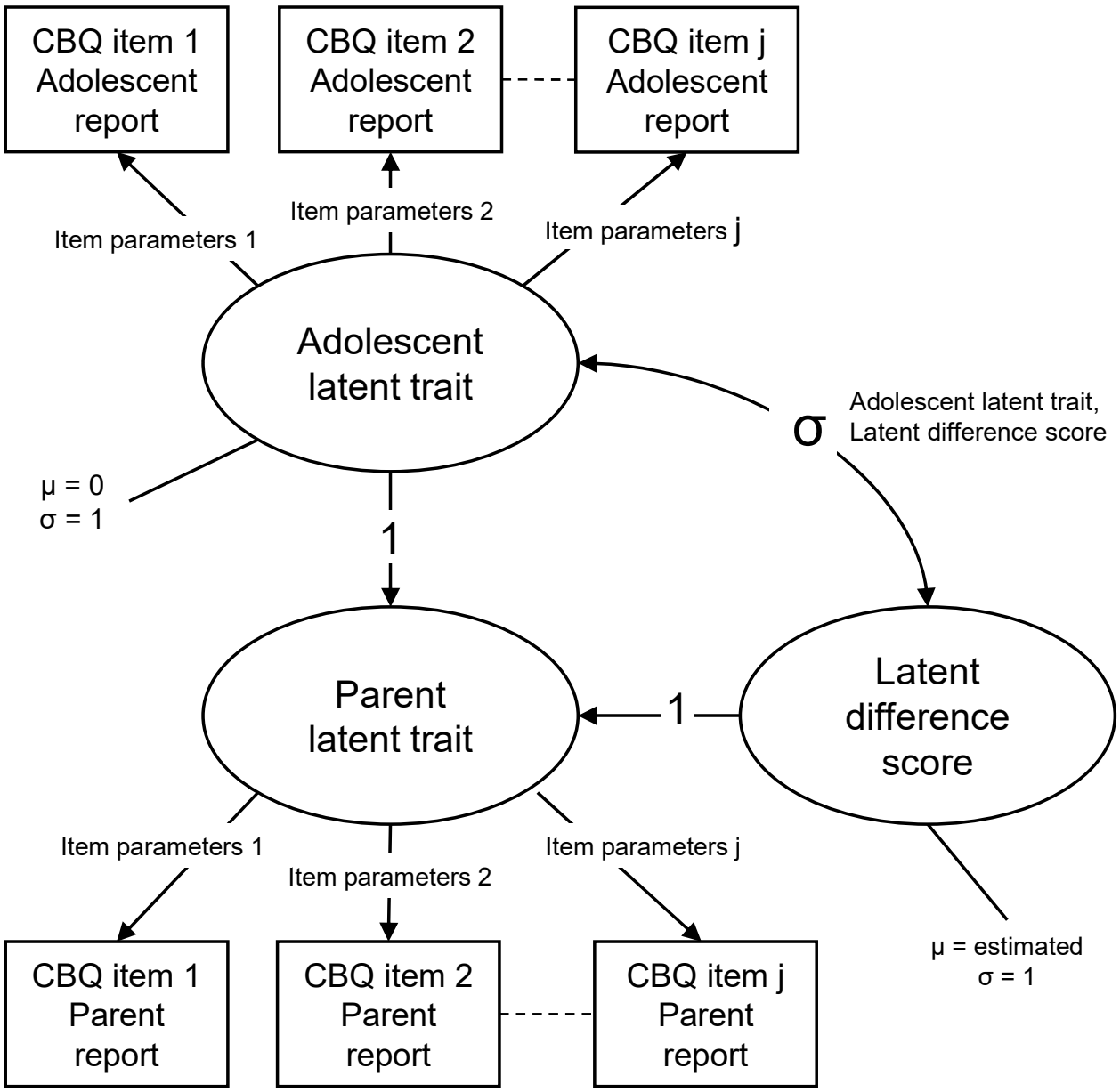
$$p(x_i = 1 \mid \theta_i, \alpha_x, \beta_x) = \frac{e^{\alpha_x (\theta_i - \beta_x)}}{1 + e^{\alpha_x (\theta_i - \beta_x)}}$$

The item threshold or location β, is on the same scale as the latent trait parameter θ. It is equal to the latent trait level where the probability of endorsement for that item is 0.5, the inflection point of the logistic item response function. The second item parameter is the item discrimination α, which defines the rate of change in probability around the inflection point. An item with low discrimination has the probability of endorsement spread more evenly out across the latent trait, and responses hence give less information about the trait level of the respondents. The pattern of item responses across respondents and items makes it possible to estimate the item and person parameters.

Informant discrepancies and latent difference score modelling

Study 3 compares discrepancy in reporting of parent-adolescent conflict to the report of single informants or multiple informants, taking the difference between parent and adolescent report to be a variable of interest in itself. Raw difference scores, obtained by subtracting the sumscore of one informant from that of the other, are for a number of methodological reasons not appropriate for modelling informant discrepancy, but latent difference scores represent a flexible and valid approach (de Haan, Prinzie, Sentse, & Jongerling, 2018). In study 3, latent difference scores representing the difference in reporting between parents and adolescents are used as a predictor variable. These latent difference scores are obtained by fitting an IRT model to the responses of parents and adolescents, and then constraining the latent trait parameter of the parents to be equal to the sum of the latent trait parameter of the adolescent and a latent difference score parameter, as illustrated by figure 2 (de Haan et al., 2018).

Figure 2: Latent Difference Score Model for the CBQ



The adolescent latent trait and the latent difference scores were given a bivariate normal distribution, with the mean of the latent trait set to 0, the variances of both set to 1 and the mean latent difference score and the covariance estimated as model parameters.

Regression modelling

In all three studies in this thesis, some form of regression modelling is used. In studies 1 and 2, these are simple multilevel regression models, with two observations of depressive symptoms over time nested within each adolescent in study 1, and observations of conflict with different parents nested within each adolescent in study 2. Multilevel regression models handle the frequently violated assumption of independent observations by explicitly modelling the clustering of observations (McElreath, 2016).

It is well known that parameter estimates in regression models fitted with an assumption of normally distributed errors are highly sensitive to departures from this assumption, for example outlier observations (Field & Wilcox, 2017). Outliers must be expected in studies of psychotherapy treatment effects, as treatment outcomes are strongly influenced by extratherapeutic factors (Kelley, Bickman, & Norwood, 2010).

One Bayesian approach to achieving a robust regression model is to have a t-distribution as the likelihood, which can have heavier tails than the normal distribution and hence allow for a smaller number of observations deviating from a general trend (Gelman et al., 2013). The degrees of freedom determine the shape of the t-distribution, and this may be fixed at some low number such as 5 (Yang & Yuan, 2016) or estimated as a model parameter. In study 1, outliers were expected, and the amount of data to inform the degrees of freedom parameter was quite low, so the degrees of freedom was fixed at 5. For study 2 the degrees of freedom were estimated as a parameter, with a weakly informative prior, as there was less prior information on the degree of robustness required, and more data to inform the estimation of the degrees of freedom.

In study 3, the dependent variable was ordinal, which was handled by fitting an ordinal probit regression model. This means assuming that the observed distribution of ordinal scores are indicators of a normally distributed latent variable divided by a number of cutpoints equal to one less than the number of ordinal categories. These cutpoints are specified as model parameters, and the likelihood of the data, given a predicted distribution of latent variables, is then defined by these cutpoints and the cumulative normal distribution function (Kruschke, 2015).

Cross-validation, model comparison and model stacking

In all three studies different regression models are compared to answer substantial questions. In all these cases, expected out of sample predictive accuracy has been adopted as the criterion of model performance, and estimated using cross-validation procedures. In these studies, as in most applications, models that faithfully represent the structure of the particular dataset at hand is not the goal. Rather, it is to find models that as well as possible represent how the modelled phenomenon is structured in the population of interest. This distinction between how well a model fits a particular dataset and how well that model represents the generative process of interest, is paralleled in the difference between explained variance and other measures of model fit to data, and expected out-of-sample predictive accuracy estimated by cross-validation (Vehtari & Ojanen, 2012).

Cross-validation in its true form is similar to replication, in that some part of the data are held out from specifying and fitting a model, and the final fitted model is then cross-validated on the new data, to see how much has been learned from the data that

are applicable to new data. For contexts where data are sparse and difficult to collect, approximate forms of cross-validation exist. K-fold and leave-one-out cross-validation is based on the idea that the performance of the model on new data can be approximated by refitting the model several times, each time holding out some of the data, and then checking how well the fitted model is able to predict the held-out data (Vehtari, Gelman, & Gabry, 2017). By combining the measures of predictive accuracy for the each of the data partitions when held out from model fitting, the predictive accuracy to a completely new sample can be approximated. Refitting the model once with each data partition held out is computationally demanding, especially when using Monte Carlo methods to fit the models. Pareto-smoothed importance sampling approximate leave-one-out cross-validation (PSIS-LOO) is a computationally efficient and accurate approximation of cross-validation (Vehtari, Gabry, Yao, & Gelman, 2019; Vehtari et al., 2017). In this thesis, PSIS-LOO was used in study 3, and tried in study 1 and 2. The two latter studies had hierarchical models with very small clusters (the parent(s) of one adolescent or two observations of depressive symptoms) which is a known weakness of PSIS-LOO. As PSIS-LOO diagnostics indicated that estimates of expected predictive accuracy were not reliable for the models of study 1 and 2, exact leave-one-out cross-validation was used instead, leaving out one cluster at a time.

When modelling a phenomenon, selecting a single model is not necessarily sensible. Unless there is reason to believe that one of the models is essentially true, or it is necessary to choose and apply for some practical purpose exactly one of a larger set of models, choosing a single imperfect model among many models imperfect in their own particular ways is neither necessary nor desirable. A weighted composite of different models will often be a better approximation to reality (Yao, Vehtari, Simpson, & Gelman, 2018). In study 2 and 3, model stacking weights are obtained to evaluate the relative contribution made by the different models compared to expected predictive accuracy. The stacking procedure uses the leave-one-out predictive densities of a set of models, and then finds an optimal weighted composite of the models that has the highest expected predictive density (Yao et al., 2018). In study 1 the full moderator model was compared to two simpler models nested within it, and model stacking was hence not relevant in the same way.

Statistical software and model fitting

The models in all three studies were coded in Stan, an open-source and free probabilistic programming language (Carpenter et al., 2017) and fitted with Hamiltonian Monte Carlo, using the RStan interface (Stan Development Team, 2019a) for R (R Core Team, 2019). Convergence of the Hamiltonian Monte Carlo algorithm was validated for all fits by inspecting the various Stan sampling diagnostics as well as effective sample

sizes and Rubin-Gelman statistics for all parameters (Stan Development Team, 2019b). In study 1 and 2, the exact leave-one-out cross-validation was coded in Stan and R. In study 3, PSIS-LOO was used as implemented in the R package loo (Vehtari et al., 2019).

Findings

Study 1

The hypothesis that the outcome of ABFT would be moderated by parent-adolescent conflict was partially supported. For adolescent report of conflict with mother and the reports of both parents a likely moderator effect was found. The posterior distributions of these fitted models are summarised in table 2. The coefficients are standardized. The predicted GRID-HAMD scores are centred by the median (21) and scaled by the median absolute deviation times two (8). Parent-adolescent conflict is the latent standard normal variable from the IRT model. Time is coded 0 for baseline and 1 for outcome. Treatment is coded as 0.5 for ABFT and -0.5 for TAU. This coding of treatment makes all coefficients for terms involving treatment interpretable as the predicted difference between the treatments (Kraemer et al., 2002). The moderator effect is specified as the three-way interaction of treatment, time and conflict.

The posterior distribution can be used to calculate the probability of a regression coefficient that is negative and of a larger magnitude than some threshold for relevance, given the model and the data (Kruschke, 2018). A standardised regression coefficient between -0.5 and 0.5 would have little clinical relevance, corresponding to a predicted difference in outcome of only four points on the GRID-HAMD at a conflict level one standard deviation above average (Moncrieff & Kirsch, 2015). For adolescent report of conflict with mother, the probability of a regression coefficient for the moderator effect below -0.5 was .72, while it was .70 for mother report and .80 for father report. A moderator effect is likely, but far from certain. For adolescent report of father, it was only .09. The models did not support substantial differential effects of treatment, as shown by the treatment by time interactions estimated very near 0.

Table 2: Parameter Estimates from Models of Treatment by Conflict Moderation

Model and Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Adolescent report of conflict with mother						
Intercept	0.09	0.09	0.09	0.00 ; 0.17	-0.05 ; 0.24	11801
Variance of random intercepts	0.36	0.13	0.37	0.28 ; 0.50	0.14 ; 0.56	1764
Variance of errors	0.53	0.08	0.53	0.45 ; 0.59	0.41 ; 0.65	2382
Regression coefficients:						
Time	-0.53	0.15	-0.53	-0.65 ; -0.37	-0.78 ; -0.29	10094
Treatment	0.01	0.18	0.01	-0.16 ; 0.18	-0.29 ; 0.30	10608
Parent-adolescent conflict	0.27	0.29	0.27	-0.01 ; 0.55	-0.21 ; 0.76	7372
Treatment x time	0.07	0.11	0.07	-0.04 ; 0.17	-0.12 ; 0.25	9108
Conflict x time	0.15	0.19	0.15	-0.03 ; 0.31	-0.16 ; 0.47	7425
Treatment x conflict x time	-0.69	0.34	-0.70	-1.01 ; -0.38	-1.25 ; -0.15	5919

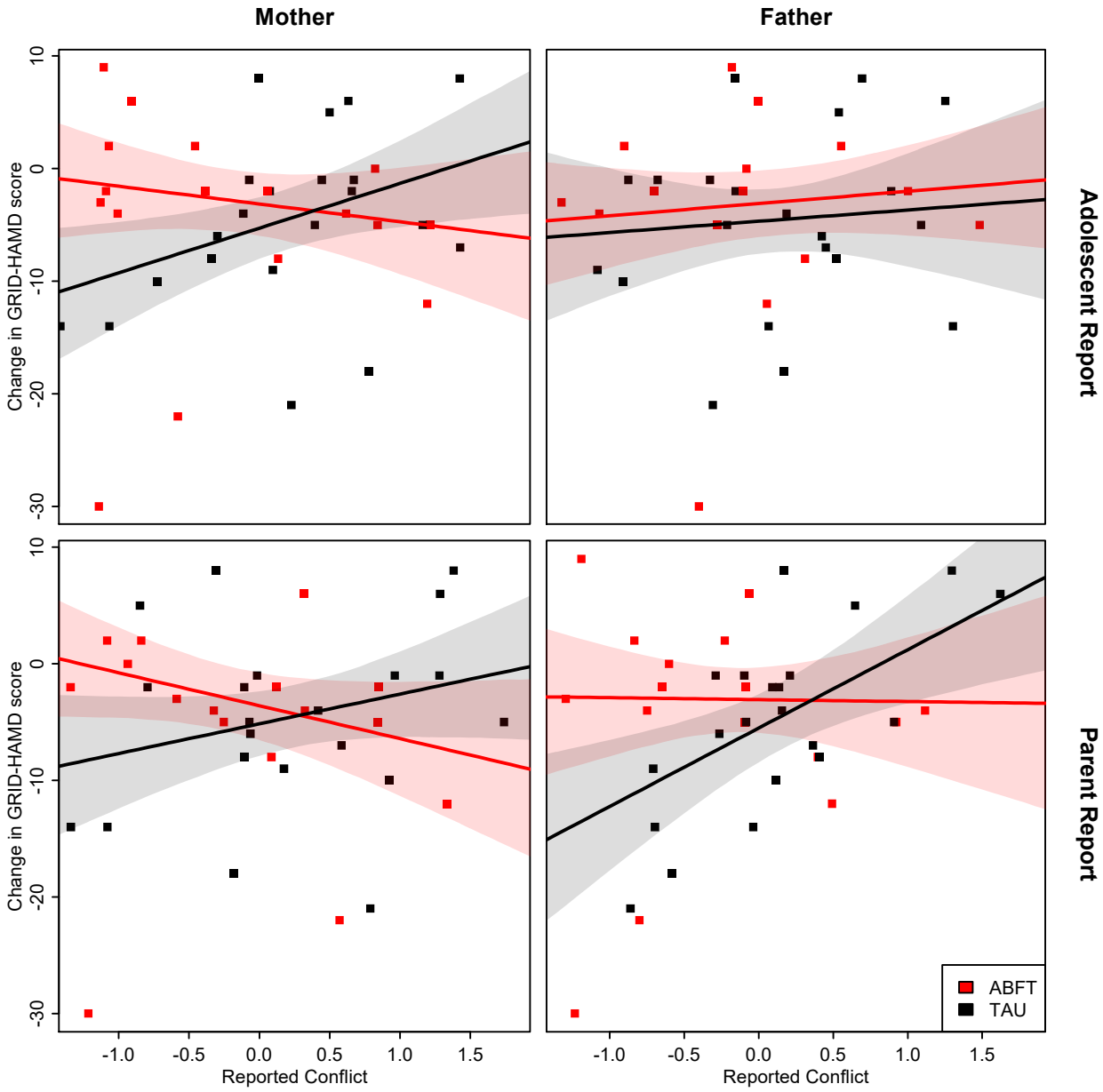
Model and Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Adolescent report of conflict with father						
Intercept	0.09	0.09	0.09	0.00 ; 0.18	-0.07 ; 0.24	12959
Variance of random intercepts	0.36	0.13	0.38	0.27 ; 0.51	0.14 ; 0.58	2059
Variance of errors	0.58	0.08	0.57	0.49 ; 0.64	0.45 ; 0.69	2853
Regression coefficients:						
Time	-0.49	0.16	-0.48	-0.64 ; -0.34	-0.75 ; -0.23	11610
Treatment	0.00	0.19	0.00	-0.19 ; 0.16	-0.30 ; 0.31	11630
Parent-adolescent conflict	0.20	0.31	0.20	-0.07 ; 0.51	-0.29 ; 0.71	11097
Treatment x time	0.04	0.10	0.04	-0.06 ; 0.13	-0.13 ; 0.21	10099
Conflict x time	0.13	0.22	0.13	-0.07 ; 0.34	-0.24 ; 0.47	5198
Treatment x conflict x time	0.01	0.40	0.00	-0.38 ; 0.36	-0.60 ; 0.70	4474
Mothers report of conflict						
Intercept	0.09	0.09	0.09	0.00 ; 0.17	-0.05 ; 0.25	12613
Variance of random intercepts	0.37	0.12	0.39	0.29 ; 0.50	0.18 ; 0.58	2008
Variance of errors	0.54	0.08	0.54	0.46 ; 0.60	0.41 ; 0.66	2488
Regression coefficients:						
Time	-0.55	0.15	-0.54	-0.69 ; -0.40	-0.80 ; -0.30	10341
Treatment	-0.03	0.18	-0.03	-0.21 ; 0.13	-0.33 ; 0.26	11666
Parent-adolescent conflict	0.19	0.30	0.20	-0.07 ; 0.51	-0.29 ; 0.70	8534
Treatment x time	-0.08	0.11	-0.08	-0.18 ; 0.02	-0.26 ; 0.09	9297
Conflict x time	-0.02	0.19	-0.02	-0.19 ; 0.16	-0.33 ; 0.28	6471
Treatment x conflict x time	-0.67	0.34	-0.68	-1.01 ; -0.38	-1.22 ; -0.11	6593
Fathers report of conflict						
Intercept	0.09	0.09	0.09	0.00 ; 0.17	-0.05 ; 0.24	9540
Variance of random intercepts	0.37	0.12	0.38	0.29 ; 0.50	0.17 ; 0.57	1651
Variance of errors	0.51	0.08	0.50	0.43 ; 0.58	0.38 ; 0.64	1740
Regression coefficients:						
Time	-0.54	0.16	-0.54	-0.68 ; -0.38	-0.79 ; -0.27	5165
Treatment	-0.01	0.18	-0.01	-0.18 ; 0.16	-0.32 ; 0.26	10840
Parent-adolescent conflict	0.30	0.30	0.30	0.01 ; 0.59	-0.19 ; 0.81	5729
Treatment x time	-0.03	0.11	-0.03	-0.13 ; 0.08	-0.21 ; 0.16	6466
Conflict x time	0.41	0.24	0.41	0.18 ; 0.61	0.03 ; 0.81	2780
Treatment x conflict x time	-0.86	0.44	-0.87	-1.28 ; -0.46	-1.60 ; -0.17	2701

Table note: Mean = Posterior mean, SD = Posterior standard deviation, Median = Posterior median, 66% and 90% HDI = 66% and 90% Highest density intervals, ESS = Effective sample size, estimates the number of independent draws from the posterior distribution

The magnitudes of the moderator effects are in any case modest, and meaningful only in the upper and lower quantiles of the distribution of conflict. Model comparison with leave-one-out cross-validation showed that the expected out-of-sample fit of the moderator models supporting moderator effects could not be reliably differentiated from

the expected fit of a model with a simple effect of time only, implying that both models may fit the data equally well. In figure 3 the predictions of the moderator models fitted to the four informant perspectives are plotted, along with the observations.

Figure 3: Moderator Model Predictions and Observations



The lines are the posterior mean of the predicted change from baseline to outcome across the range of parent-adolescent conflict. The shading is the 90% Highest Density Intervals of that prediction. For mother-adolescent conflict, the pattern of treatment moderation was similar across both informants, with better outcomes for ABFT at high levels of conflict and better outcomes for treatment as usual at low levels of conflict. Interestingly, the model fitted to father report of conflict showed a different pattern, as it did not predict any change in the outcome of ABFT as a function of the

level of conflict reported, but a large change in the expected outcome of treatment as usual, with poor outcomes associated with high levels of conflict.

Study 2

Parental interpersonal problems on the agency dimension was found to be associated with adolescent report of conflict. When parents reported more interpersonal problems related to being domineering and assertive, their adolescents were predicted to report more conflict in the relationship to that parent. When parents reported more problems related to being unassertive and submissive, adolescents were predicted to report less conflict. The posterior distribution can again be used to calculate the probability of a regression coefficient larger than 0.1, reasoning that a standardized coefficient between -0.1 and 0.1 has little theoretical relevance. The probability of a regression coefficient for agency above 0.1 was .91, suggesting that an association of some theoretically meaningful magnitude is very likely. Comparing models with and without interactions with parent gender using leave-one-out cross-validation showed no support for the associations between agency or other interpersonal problem variables and conflict differing for mothers and fathers.

Contrary to expectations, parental depressive symptoms were not supported as a predictor of parent-adolescent conflict. For fathers, a lower level of depression was associated with increased conflict, while there was no strong association for mothers. There was a considerable range of parental depression in the sample, with some parents reporting clearly clinical levels of depressive symptoms. Model stacking gave a weight of .75 to the model with parental interpersonal problems and .25 to the model with gender-dependent effects of parental depression. Estimates from these two models are summarised in table 3.

Table 3: Parameter Estimates from Models Predicting Parent-adolescent Conflict from Parental Characteristics

Model and Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Interpersonal problems model						
Regression coefficients:						
Agency	0.19	0.07	0.18	0.12 ; 0.24	0.08 ; 0.30	3768
Communion	0.02	0.06	0.02	-0.03 ; 0.07	-0.07 ; 0.11	5126
Elevation	0.08	0.07	0.08	0.02 ; 0.15	-0.02 ; 0.20	6509
Adolescent age	-0.03	0.03	-0.03	-0.05 ; 0.01	-0.08 ; 0.03	7081
Variance of errors	0.27	0.06	0.27	0.21 ; 0.32	0.18 ; 0.37	1016
Variance of random effects	0.21	0.07	0.21	0.15 ; 0.28	0.08 ; 0.34	1214
Degrees of freedom in t-likelihood	21.88	14.15	18.55	4.46 ; 25.64	2.89 ; 41.18	13611
Parental depressive symptoms model						
Intercept	-0.03	0.12	-0.03	-0.14 ; 0.08	-0.23 ; 0.16	840
Regression coefficients:						
Depressive symptoms	-0.16	0.07	-0.16	-0.22 ; -0.09	-0.29 ; -0.05	4719
Dep. sympt. x Mother	0.16	0.09	0.16	0.07 ; 0.24	0.01 ; 0.31	5495
Mother	0.02	0.08	0.03	-0.05 ; 0.10	-0.11 ; 0.16	9331
Adolescent age	-0.03	0.03	-0.03	-0.06 ; 0.00	-0.09 ; 0.02	8449
Variance of errors	0.30	0.06	0.30	0.24 ; 0.35	0.21 ; 0.39	1639
Variance of random effects	0.16	0.08	0.17	0.09 ; 0.25	0.01 ; 0.27	1610
Degrees of freedom in t-likelihood	20.8	13.85	17.46	4.93 ; 24.93	3.08 ; 40.17	14430

Table note: Mean = Posterior mean, SD = Posterior standard deviation, Median = Posterior median, 66% and 90% HDI = 66% and 90% Highest density intervals, ESS = Effective sample size, estimates the number of independent draws from the posterior distribution

Study 3

Informant discrepancies in report of conflict, both mother-adolescent and father-adolescent, was associated with hopelessness. When parents reported less conflict than the adolescent, hopelessness increased. The individual reports of parents or adolescents showed very small associations with hopelessness, while a model with the reports of both informants showed stronger associations, but in the opposite directions. Cross-validation did not definitively support one model over the others, but model averaging via stacking of predictive distributions gave most of the weight to the informant discrepancy models (.88 for mother-adolescent discrepancy and .85 for father-adolescent discrepancy) and some weight to the models with adolescent report (.12 to adolescent report of conflict with mother, and .15 to conflict with father). The estimates from these two models are summarised in table 4. Parent report or multi-informant report received zero weight.

Table 4: Parameter Estimates from Models Predicting Hopelessness from Adolescent Report and Discrepancy in Reports of Parent-adolescent Conflict

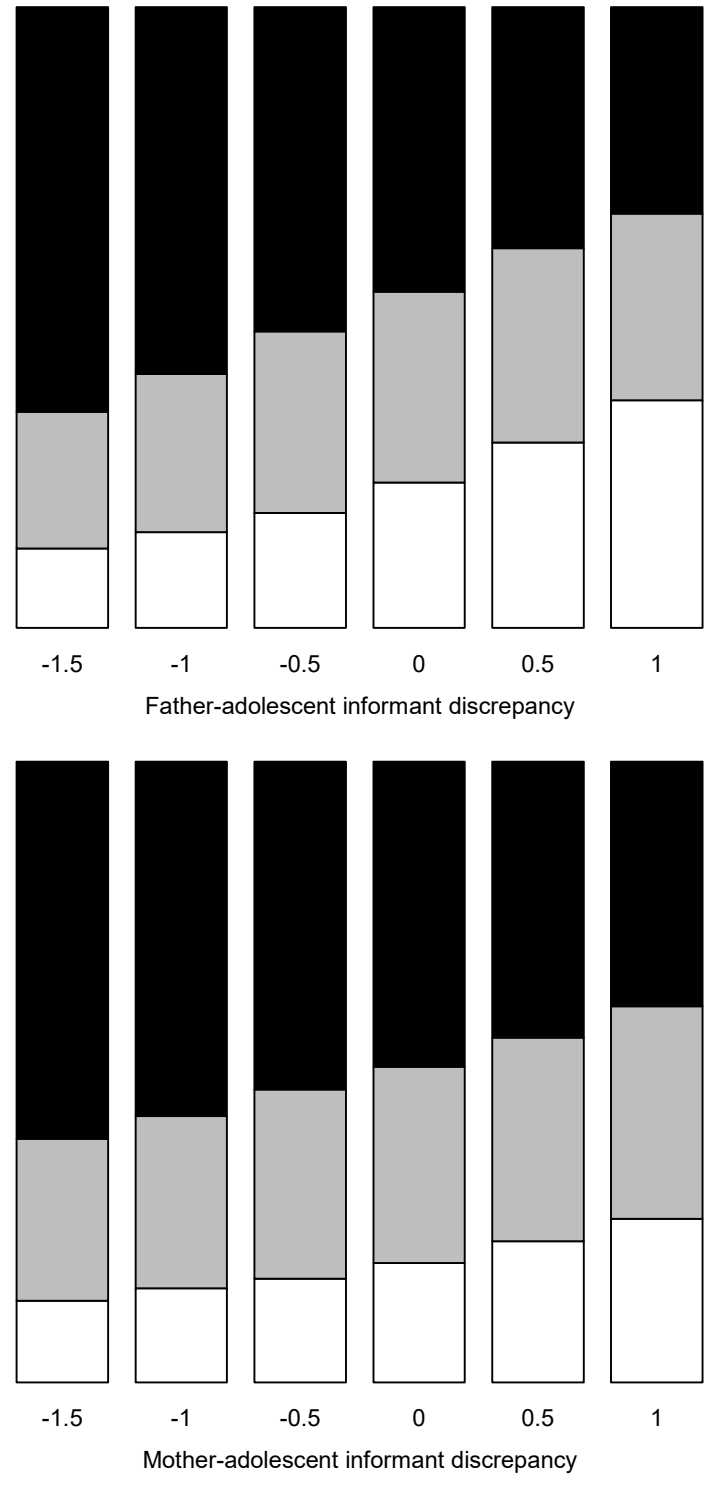
Model and Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Informant discrepancy - mother						
Regression coefficient	-0.25	0.23	-0.24	-0.43 ; -0.01	-0.60 ; 0.13	9087
First Cutpoint	-0.88	0.20	-0.87	-1.07 ; -0.70	-1.19 ; -0.55	16555
Second Cutpoint	0.05	0.17	0.05	-0.12 ; 0.21	-0.25 ; 0.32	11270
Adolescent report - mother						
Regression coefficient	0.08	0.19	0.08	-0.10 ; 0.25	-0.23 ; 0.38	13228
First Cutpoint	-0.87	0.20	-0.86	-1.04 ; -0.66	-1.19 ; -0.55	14336
Second Cutpoint	0.03	0.17	0.03	-0.13 ; 0.20	-0.26 ; 0.31	13092
Informant discrepancy - father						
Regression coefficient	-0.42	0.33	-0.40	-0.68 ; -0.08	-0.96 ; 0.10	7243
First Cutpoint	-0.73	0.23	-0.73	-0.95 ; -0.51	-1.12 ; -0.37	15468
Second Cutpoint	0.13	0.21	0.12	-0.08 ; 0.32	-0.22 ; 0.48	11508
Adolescent report - father						
Regression coefficient	0.06	0.19	0.06	-0.13 ; 0.23	-0.25 ; 0.37	12709
First Cutpoint	-0.76	0.21	-0.75	-0.93 ; -0.53	-1.09 ; -0.40	15386
Second Cutpoint	0.04	0.19	0.04	-0.14 ; 0.22	-0.26 ; 0.37	13631

Table note: Mean = Posterior mean, SD = Posterior standard deviation, Median = Posterior median, 66% and 90% HDI = 66% and 90% Highest density intervals, ESS = Effective sample size, estimates the number of independent draws from the posterior distribution

Calculating the probability of a regression coefficient that is negative and of greater magnitude than -0.1 gives probabilities of .74 for mother-adolescent discrepancy and .85 for father-adolescent discrepancy. A meaningful association is likely, but far from certain.

As parameter estimates from ordinal models can be difficult to interpret directly, figure 4 shows the predicted distributions (weighted composite of the stacked models) of hopelessness at the different levels of informant discrepancy (negative discrepancy means the parent reports less conflict than the adolescent). Overall, the results of the study gave preliminary support to the hypothesis that discrepancy in reports of conflict is related to adolescent hopelessness.

Figure 4: Predicted Distributions of Hopelessness Across Informant Discrepancy



Discussion

Parent-adolescent Conflict as a Moderator of Treatment Outcome

The overall treatment effect was quite disappointing across both treatment arms in the BUD clinical trial, with very few remitters and an average change in GRID-HAMD score smaller than what is considered clinically meaningful (Leucht et al., 2013; Waraan, Rognli, Czajkowski, Aalberg, & Mehlum, 2020, March 3). Some evidence for moderation by parent-adolescent conflict was however found in study 1, though the magnitude of the moderator effect is also quite small.

Kraemer et al. (2002) drew up a conceptual and analytic framework for moderators and non-specific predictors in the context of clinical trials. According to their definition, a moderator is a baseline variable which the effect of treatment allocation on outcome varies across. A non-specific predictor of outcome is a baseline variable that outcome varies across equally for all treatments compared. Parent-adolescent conflict has been found to be such a non-specific predictor of worse treatment outcomes in several earlier trials of psychotherapy for adolescent depression. In a trial of three different psychotherapies for adolescent depression, including one systemic family therapy intervention, higher adolescent-reported parent-adolescent conflict at baseline predicted both treatment non-response and depression recurrence (Birmaher et al., 2000). Adolescent report of more parent-adolescent conflict was also found to be a non-specific predictor of non-responder status in the Treatment of Selective Serotonin Reuptake Inhibitor-Resistant Depression in Adolescents Study (Asarnow et al., 2009). Similarly, in the Treatment of Adolescent Depression Study, mother-report of frequent and intense conflict was found to be a non-specific predictor of worse outcomes in an exploratory analysis (Feeny et al., 2009).

Such non-specific predictor findings reflect one of two actual relationships: Either the variable in question is actually a non-specific predictor of outcome for this patient group, across all treatments, or the finding reflects that the variable would be a moderator if one of the treatments studied were to be compared to the right treatment. The definition given by Kraemer et al. (2002) implies that treatment moderation is a property of the comparison of two or more particular treatments, not the individual treatments or the moderating variable. For example, if a treatment where a negative predictor of outcome is known from earlier trials is compared to a treatment that specifically addresses or targets that negative predictor variable, it is likely that the impact of the negative predictor variable on outcome will be different between those two treatments. In that case, the variable previously found to be a non-specific predictor is a moderator. It is such testing of moderator hypotheses that are well justified theoretically

and based on previous predictor findings that is most likely to yield clinically relevant answers to the important question of what works for whom.

For parent-adolescent conflict in adolescent depression, a number of previous studies suggest that it is not a universal non-specific predictor of outcome. In a trial comparing Interpersonal Therapy for Adolescents (IPT-A) to treatment as usual it was found that adolescents reporting more conflict with mothers at baseline benefited more from Interpersonal Therapy (Gunlicks-Stoessel et al., 2010). A similar finding was obtained for a preventive group intervention based on IPT-A compared with regular school counselling, where the IPT-A based intervention was superior to school counselling only for those adolescents reporting heightened parent-adolescent conflict (Young et al., 2009). A third similar finding was reported from a trial comparing a family focused treatment to enhanced usual care for adolescent bipolar disorder (Miklowitz et al., 2009). The family-focused treatment was only superior to enhanced usual care for adolescents from families reporting heightened expressed emotion. This pattern of findings is however not universal, as the trial of three psychotherapies for adolescent depression mentioned earlier also evaluated parent-adolescent conflict as a possible moderator of the effectiveness of systemic family therapy relative to cognitive-behavioural therapy or non-directive supportive therapy. No evidence was found for this, although systemic family therapy was superior in reducing the level of parent-adolescent conflict (Kolko, Brent, Baugher, Bridge, & Birmaher, 2000). Overall, there is still some evidence that treatments directly addressing the parent-adolescent relationship may be more effective at higher levels of parent-adolescent conflict. This is largely the same finding as in study 1, lending support to its plausibility.

An exploratory analysis of findings from the largest trial of ABFT to date (Diamond et al., 2019) did however not find evidence that the effectiveness of ABFT as a treatment for adolescent suicidal ideation relative to non-directive supportive therapy varied across adolescent-reported family conflict and cohesion (Zisk, Abbott, Bounoua, Diamond, & Kobak, 2019). Whether this finding is directly comparable to the finding in study 1 is debatable, however. The clinical populations studied are overlapping, but not equivalent. The sample in the BUD clinical trial was defined by a diagnosis of Major Depressive Disorder, while only 41% of the other sample received this diagnosis, but were required to have clinically significant suicidal ideation (Diamond et al., 2019). Even more importantly, the variable analysed as a possible moderator in the other study is a composite variable assessing general family climate, which may be a somewhat different construct than the perception of distressing conflict in a specific relationship, which is what is assessed by the CBQ.

Parental Characteristics Associated with Parent-adolescent Conflict

The results of cross-validation and model stacking in study 2 indicated that parental interpersonal problems are relevant predictors of parent-adolescent conflict, compared with parental depressive symptoms. Parent report of interpersonal problems on the agency dimension was related to adolescent report of parent-adolescent conflict, which is consistent with the view that interpersonally complex renegotiation processes between parents and adolescents are necessary to avoid persistent, unresolved conflicts. Cross-validation did not support a model with the association between parental interpersonal problems and parent-adolescent conflict varying by parent gender, suggesting that this applies equally well to fathers and mothers. When parents struggle to adopt a non-dominant interpersonal position when appropriate, as measured by high scores on the Agency dimension (Gurtman, 1996), they will likely find it difficult to transition out of the hierarchical parent-child relationship towards a more symmetrical relationship. As adolescence changes the underlying premises of power in the parent-child relationship (Laursen & Bukowski, 1997), parents persisting in a dominant interpersonal stance will not result in a stable continuation of childhood patterns of conflict resolution. It is more likely to result in repeated failures of conflict resolution, lack of development towards new forms of conflict resolution, and dysfunctional states of parent-adolescent conflict.

The study does not indicate whether this association between parental interpersonal problems on the agency dimension is specific to adolescents in a depressive state, as depression defines the sample. It seems most likely that parental problems with excessive dominance will tend to be associated with more conflict in non-clinical contexts as well, but it is also possible that there are specific characteristics of adolescent depression that are important for this association. Depression involves loss of functioning, likely leading to more frequent violations of parental expectancies for the adolescent (Collins & Luebker, 1994). Parents with dominant interpersonal styles may be attempting to coerce the adolescent to comply with their expectations, generating more frequent conflicts, and find it difficult to engage in re-negotiating their expectancies. In Interpersonal Therapy for Adolescents, an important early therapeutic manoeuvre is the assignment of a limited sick-role, which has the intention of allowing the adolescent to feel less guilt for their low functioning and assume responsibility for working on treatment goals, while also encouraging parents to temporarily suspend their expectations (Mufson, Dorta, Moreau, & Weissman, 2004), illustrating how this is a known clinical issue. Further investigations with non-clinical samples are nevertheless required to assess whether the association is specific to adolescent depression.

The Agency variable ranges from positive scores for problems related to being too dominant and assertive, to negative scores for problems related to being too submissive and non-assertive (Gurtman, 1996). The model predicts lower levels of parent-adolescent conflict for scores in the negative range of Agency, and there were no indicators of the model fitting less well in the lower range of Agency than in the higher range. It appears that lower levels of conflict is more likely when parents report that they find it difficult to be assertive. It should be noted that these parents may very well be experiencing more of other potential difficulties in the relationship to their adolescent children, which were not assessed in this study. Problems related to preoccupation with closeness and care, or with being too withdrawn and finding it hard to feel and express care, was surprisingly not strongly related to parent-adolescent conflict, although it could also very well be related to other difficulties that were not assessed. This supports the central role of issues related to changes in power and autonomy in development of dysfunctional parent-adolescent conflict. Together, this also illustrates an important point about the specificity of different kinds of interpersonal problems. If interpersonal dysfunction is treated as an undifferentiated and global construct, it may obscure specific associations between interpersonal contexts and processes and dimensions of interpersonal problems.

A negative association restricted to the father-adolescent relationship was found for parental depression and parent-adolescent conflict. Not finding a positive association was unexpected, given the extensive literature supporting this (Hammen et al., 2004; Kane & Garber, 2004). Although there are some other discrepant findings (Kim, Thompson, Walsh, & Schepp, 2015; Van Bommel, Van der Giessen, Van der Graaff, Meeus, & Branje, 2019), this discrepancy is still not readily explainable. Differences in measurement and operationalisation could be involved; in a meta-analysis of the association between paternal depression, father-child conflict and child psychopathology, larger effect sizes were found to be associated with community samples and parent-reported measures of parenting behaviours (Kane & Garber, 2004). It could also be that the association is not continuous across different populations, and that the adolescents developing a depressive disorder changes the association between parental depressive symptoms and parent-adolescent conflict. Finally, the expected positive association for mothers is quite improbable given these data and the model, but still not completely ruled out.

Hopelessness and Discrepancy in Report of Parent-adolescent Conflict

The paucity of studies on potentially modifiable predictors of hopelessness in adolescent depression is unfortunate, given the well-established relationship to suicidal ideation and behaviour, and that hopelessness is not likely to simply be a marker of

depressive severity (Wolfe et al., 2019). In clinical samples of adolescents or children there appears to be only two studies directly addressing this question (Becker-Weidman et al., 2009; Kashani, Canfield, Borduin, Soltys, & Reid, 1994). The results of study 3 gave some support to the hypothesis that discrepancy in report of parent-adolescent conflict would be related to hopelessness among depressed adolescents. Compared with study 1 and 2, the uncertainty is even more pronounced in study 3, but given the need for new directions for future research, it is still a relevant finding.

Adolescent report of conflict was correlated with informant discrepancies, such that the probability of a parent reporting lower conflict than the adolescent was higher when adolescents reported higher levels of conflict. This is to be expected, as an adolescent reporting very little or very much conflict can hardly have a parent reporting very much less or very much more, respectively. The stacking procedure favoured the model with informant discrepancies as the predictor. The difference in reporting carries information about these parent-adolescent dyads that is not redundant with the report of either one. This illustrates how the perspective of a single informant may constitute an incomplete perspective on conflict processes, and how averaging or otherwise collapsing different informants may result in the loss of specific information (Korelitz & Garber, 2016).

A question remains about how informant discrepancy relates conceptually to the parent-adolescent conflict variables from which it is derived. It is worth noting that informant discrepancy is not actually a self-reported variable, though it is derived from the combination of two self-reported variables. Convergence in response behaviour, or lack thereof, is taken to indicate something about the relational state that is not necessarily completely known to either party to the relationship. Assuming the transactional view of parent-adolescent conflict advanced in the introduction, informant discrepancies must be related to the representational aspect of parent-adolescent conflict. The actual interactions between parents and adolescents cannot be different for parents compared to adolescents, but the interpretation of conflict events and the representation of the conflictual state of the relationship clearly can. Informant discrepancies are likely to be an indicator of the degree of divergence in such representations. When such divergence develops, subsequent events are also more likely to be interpreted differently, in light of the divergent representations. This difference in interpretation of events can again lead to representations of the relationship diverging even further, allowing for such divergence to persist despite repeated shared experiences.

Across multiple parenting constructs, parents tend to hold a more favourable view than adolescents, though this may be most pronounced in middle adolescence

(Korelitz & Garber, 2016; Mastrotheodoros, Van der Graaff, Dekovic, Meeus, & Branje, 2018). On the other hand, it has been shown that in non-clinical samples, parents tend to be more distressed by conflicts than the adolescent (Steinberg, 2001). The results of study 3 suggest that for depressed adolescents, parents holding a more favourable view of the degree of dysfunctional conflict is related to increased probability of hopelessness. For an adolescent experiencing the parent-adolescent relationship as conflictual in a problematic way, it could be particularly distressing if the parent does not share this view of the relationship. Resolving conflicts and establishing a less hierarchical form of conflict resolution will necessarily be compromised by the parent not acknowledging the severity of conflict experienced by the adolescent, making conflict seem persistent to the latter. As parent-adolescent relationships are largely obligatory, not voluntary relationships, the expected persistent distress related to the relationship can also seem inescapable. Expectations of inescapable, persistent distress is an important aspect of hopelessness (Marchetti, 2018). Further, adolescence is a developmental period where a desired future may be felt to be particularly dependent on supportive close relationships. Adolescents have an increasing capacity for mentally representing concrete long-term life goals, and the transitional process to adulthood make these goals highly salient (Nurmi, 1991). While adolescents are less completely dependent on their parents for practical and emotional support than younger children, it is also clear that the pursuit of such goals is more difficult for adolescents lacking parental support (Laursen & Collins, 2009). Conflict that appears unresolvable can in this way make desired developmental outcomes seem unattainable to the adolescent, further contributing to hopelessness (Marchetti, 2018).

The design of study 3 does not allow for a definitive conclusion about causal directions, and the association between informant discrepancy and hopelessness could also be due to adolescent depressive distortion of conflict events (De Los Reyes, Thomas, Goodman, & Kundey, 2013). The relatively weak relationship between adolescent report of conflict and hopelessness does however argue against this interpretation. Nevertheless, the findings in study 3 are preliminary, and require replication in a larger sample. Longitudinal investigations of change in informant discrepancy and hopelessness would also be useful for clarifying the issue of directionality. Unlike Becker-Weidman and colleagues (2009), adolescent report of conflict was not found to be strongly associated with hopelessness in itself. However, there are several differences in statistical analysis and measurement methodology between these studies that make direct comparisons difficult.

Methodological Considerations

When assessing the methodological quality of these studies, several issues are relevant. Two are related to general problems in clinical psychology: The difficulties of measuring the variables of interest and how the sampled population limits generalizability. Some are more specific to this study, although by no sense unique: The problem of studying fundamentally developmental processes by mainly cross-sectional observation, and the small sample size.

Quality of measurement

In the registration of the BUD clinical trial, the GRID-HAMD was chosen as the continuous measure of depressive severity, with measurement at baseline and after 16 weeks of treatment. Outcome was defined as change in the sum-score of the 17 items in the GRID-HAMD. There are advantages and disadvantages to this. On one hand, the HAMD is the de-facto gold standard for assessing the severity of depression, and the efficacy of many treatments for depression is defined in terms of change in scores on the HAMD (Bagby et al., 2004). The interpretation of scores and change is relatively well established, at least in adults, and corresponds well with clinical global impressions (Furukawa, Akechi, Azuma, Okuyama, & Higuchi, 2007; Leucht et al., 2013). The GRID-HAMD is simply a more reliably scored version of the HAMD (Tabuse et al., 2007). On the other hand, there are a number of well-known psychometric problems with the HAMD, reviewed by Bagby et al. (2004), besides the reliability issues that were somewhat amended by the GRID-HAMD. The HAMD total score is obtained by summing the scores on the individual items. However, the individual items are not all scaled equally, with some being scored from 0-4 and others from 0-2, which means the possible contribution to the total score varies among items. Further, there are varying numbers of items assessing different kinds of symptoms. As an example, sleep disturbances are assessed by three items scored 0-2, while depressed mood is assessed by only one item. Some items assess constructs not considered to be depressive symptoms in current conceptualisations, such as lack of insight and hypochondriasis, while some important depressive symptoms are missing, such as difficulty concentrating. A number of shortened versions of the HAMD have been proposed and tested using item response theory, which are generally found to have better psychometric properties than the full version (Santor, Debrota, Engelhardt, & Gelwicks, 2008; Timmerby, Andersen, Sondergaard, Ostergaard, & Bech, 2017). Consistency with trial registration is required when analysing and reporting the trial, but in hindsight, one of these abbreviated versions would probably have been a better measure.

All three studies also rely on the Conflict Behavior Questionnaire (Prinz et al., 1979) as the measure of parent-adolescent conflict. In all three studies, the Perception of

the Dyad subscale has been used as a measure of frequent and dysfunctional conflict in the parent-adolescent dyad. In study 1 and 3, two items (2 and 4) were removed as analyses indicated a lack of measurement invariance across informants (Verhagen & Fox, 2013). The interpretation of findings depend on the content of the CBQ Perception of the Dyad scale, and the degree to which this actually covers the construct of dysfunctional parent-adolescent conflict. Given the difference between dysfunctional conflict processes and normative increases in parent-child conflict in adolescence, it is important to evaluate to what extent the measure used is likely to tap one or the other. The items of the Perception of the Dyad subscale are:

1	We (my [parent] and I) joke around often.	(Reversed)
2	We do a lot of things together.*	(Reversed)
3	We almost never seem to agree.	
5	At least three times a week, we get angry at each other.	
4	I enjoy the talks we have.*	(Reversed)
6	After an argument which turns out badly, one or both of us apologizes.	(Reversed)
7	We argue at the dinner table at least half the time we eat together.	
8	My [parent] and I compromise during arguments. ("Compromise" means we both give in a little.)	(Reversed)
9	At least once a day we get angry at each other.	
10	The talks we have are frustrating.	
11	My [parent] and I speak to each other only when we have to.	
12	In general, I don't think we get along very well.	
13	We argue at the dinner table almost every time we eat.	
14	We never have fun together.	
15	My [parent] and I have big arguments about little things.	
16	We have enjoyable talks at least once a day.	(Reversed)

*Removed in study 1 and 3.

The parent version is identical, except that the wording refers to the adolescent instead of the parent. These items clearly cover repetitiveness and lack of development (3, 10, 15), high frequency (5, 7, 9, 13), negative relationship representations (1, 12, 14), poor conflict resolution (6, 8, 10), interpersonal withdrawal (11, 14, 16) and to some extent expressed negative emotion (9, 15). Overall, the scale content seems representative of the construct of dysfunctional parent-adolescent conflict.

Sample characteristics and generalisability of findings

Completely general laws cannot largely be expected to hold for social and psychological phenomena across different historical and cultural contexts. Any population sampled from will be particular. The generalisability of findings is necessarily

a matter of degree, and requires theoretical justification in every case. Careful attention to the study population is required to reason about which findings can be generalised to which other contexts and to what degree. It is hence important to accurately characterise the population the sample was drawn from and assess the risk of biased sampling from that population.

The sample studied in this thesis is clearly not representative of the global adolescent population and their families. They are Norwegian and are seeking healthcare for a depressive disorder. Norway is a very wealthy country with a low level of income inequality and a highly developed welfare state (Elstad & Stefansen, 2014). As the composition of sociodemographic risk factors in the Norwegian population is likely to be different from other countries, the composition of the depressed subpopulation may also be different, as some developmental pathways to depression may be relatively less frequently activated (Botter-Maio Rocha et al., 2020).

A large body of work has addressed the question of whether depression is continuous with low mood states within the normal range or represents a distinct disorder with qualitative differences to subsyndromal depression. Findings generally support the dimensional view (Ruscio, 2019), also in the adolescent population (Hankin et al., 2005). The diagnostic category of Major depression used to select this sample hence delineates a group within the depressive spectrum that is recognised to clearly need treatment, but whose symptoms are probably of the same kind as less severe forms of depression.

In the Norwegian public single-payer healthcare system, CAMHS are the primary providers of specialist level mental health services to youth. Referrals for depression or suspected depressive disorder will result in provision of services according guidelines (Helsedirektoratet, 2015). This makes it less likely that there are parts of the population of Norwegian adolescents with depression who receive healthcare elsewhere. However, it is known that a large proportion of depressed adolescents in Norway are not referred for care (Sund, Larsson, & Wichstrom, 2011), and sampling bias due to systematic differences in referral rates between subgroups of adolescents with depression cannot be ruled out. This is a probable explanation for the lack of ethnic minority participants. This could also be the reason for some of the gender imbalance in the sample, where the proportion of male adolescents is only 8 of 60. A marked gender difference in prevalence of depressive disorders appears in adolescence, with approximately double the number of females suffering from depression (Nolen-Hoeksema & Girgus, 1994). Judging from these well-established differences in prevalence, the number of male adolescents is still lower than expected and suggests that there may be gender differences in detection and referral rate to Norwegian CAMHS for adolescents with depression. It is of course also

possible that depressed male adolescents were not identified as such in their referral letters, but as potential participants were also identified through self-report measures, this is not likely to have caused severe bias. Lastly, participation in the trial was by informed consent, which means adolescents strongly opposed to parental involvement may be underrepresented in the sample. Underrepresentation could also apply to parents that hold dismissive attitudes towards mental health care and would not want to be involved in treatment.

In summary, the findings in these studies should most readily be generalizable to clinical populations of depressed non-minority female adolescents in Norway. The findings should also generalize reasonably well to the equivalent populations in countries resembling Norway culturally and in terms of having universal mental health care for youth and a well-developed welfare state, such as the other Scandinavian countries. Generalizing to subclinical populations, ethnic minority adolescents, males or to clinical populations in countries less resemblant of Norway is less justified, but the findings could warrant attempted replication in samples from such populations.

The limitations of a small sample size

The most severe limitation of all three studies is the low sample size of 60 adolescents and their parents. In the case of study 1, the large proportion of missing outcome data contributes further to the lack of data available to draw conclusions. The use of Bayesian modelling mitigates this limitation as much as possible, which is not to say that any increase in sample size would have been valuable and important. The Bayesian approach does not rely on the asymptotic properties of large samples, unlike approaches based on maximum likelihood, and therefore tends to work better in small-sample contexts. Given that proper care is paid to validation of convergence for Markov Chain Monte Carlo methods, and actual specification of priors rather than using non-informative priors or software defaults, Bayesian methods will often perform better than classical approaches with small samples (McNeish, 2016). In Bayesian modelling, a small sample usually leads to a posterior distribution with greater uncertainty, reflecting that less has been learned from seeing the data. The way low sample sizes lead to less certain conclusions is readily apparent across all three studies in this thesis, where there are few definitive findings.

Such uncertain findings arguably still contribute to the scientific literature. Uncertain findings are not random statements, and add something to the gradual accumulation of knowledge on a topic. Further, uncertain findings reported as uncertain do not distort the scientific record in the same way as findings presented with a higher degree of certainty than there are empirical and statistical grounds for. The way uncertainty is given a quantitative expression in the posterior distribution is an

appealing property of the Bayesian approach to estimation and inference. It is a property that may be particularly relevant for fields as psychology, where large sample sizes may be difficult to achieve for many relevant research questions. A decision to pursue a design with a large sample is also a more reasonable investment when it is based on previous findings, even though they are uncertain.

Cross-sectional observations of developmental processes

Study 2 and 3 are also completely cross-sectional and observational, which is a methodological weakness for the topics studied. The studies yield no direct empirical evidence for the causal direction of the associations found, as they lack a temporal sequence of observations. Only theoretical arguments can then be made for the direction of effects.

Two arguments support the interpretation of study 2. Firstly, it is well established that the relationship between dysfunctional forms of parent-adolescent conflict and adolescent depression is bidirectional over time (e. g. Hale et al., 2020). This makes the notion of a simple direction of causality between parent-adolescent conflict and adolescent depression largely irrelevant; these are interacting elements in a developmental process, not a cause and an effect. Secondly, the agency and communion factors of the IIP-C have considerable temporal stability (Renner et al., 2012), and concern how the respondent perceives their interpersonal functioning across relationships, making a strong influence on this measure by current conflict with an adolescent child less plausible. Parental interpersonal styles are hence more likely to be a contributing factor to the developmental process than a product of it.

In the case of study 3, both discrepancies and hopelessness could be due to adolescent depressive distortion, with adolescents representing both their current and future relationships and situations as far worse than the non-depressed parents. However, this explanation is not very consistent with the relatively weak association between adolescent report of conflict and hopelessness.

Ethical Considerations

In the BUD clinical trial, all participation was voluntary, and informed consent was given by all adolescents as well as parents. However, the participation of both parents and adolescents was a requirement for study entry. Making the decision to participate interdependent in this way may have exposed some adolescents or parents to pressure to participate from other members of the family who was particularly motivated to participate. This cannot be ruled out even though study personnel explicitly communicated the voluntary nature of participation to all participants during enrolment in the study, as well as the right to withdraw from further participation for any or no reason and at any time. There are no complaints or other evidence indicating that any

participant involuntarily participated. Still, dichotomising the decision to participate into simple voluntary and involuntary categories would disregard the complexities of family relationships and interdependent decisions, where there is a continuum from volition to coercion. This is an ethical problem that is inherent in research on families, and probably not resolvable. It is assumed that no participant was subjected to an unacceptable level of pressure to participate. In hindsight, eliciting a discussion of participation among the family members as part of obtaining informed consent would have been an extra precaution.

Another ethical issue with the BUD clinical trial is the amount of missing data and the failure to reach the planned sample size. These shortcomings of the trial decrease the scientific value of the results below what it could have been. Considering the amount of resources invested in conducting the trial, both by the sponsoring organisations and funders, as well as by participants volunteering their time for assessments, failing to realise the full scientific potential of the trial is ethically problematic. To the extent that this outcome was predictable when recruitment was initiated, given what was known at the time, not running the trial would probably have been the ethical decision. It is difficult to accurately assess how predictable it was that the trial would not fully accomplish its goals at the time the final decision to run the trial was made. Further, it would have been even less ethically defensible to not analyse and report the findings from the trial when it was in fact conducted. It should serve as an example of how adjusting the level of ambition to the available funding, organizational capacity and personnel is necessary in order to avoid an unethical wastefulness of resources allocated to research.

Conclusions

This thesis presents an investigation of parent-adolescent conflict among adolescents that are depressed. While its contributions to the empirical literature are quite modest, given the uncertainty related to sample size and measurement issues, some overall conclusions may be drawn, and some recommendations for future research can be made.

When benchmarked against the results of clinical trials and assessments of the outcome of treatment as usual in the community (Bear, Edbrooke-Childs, Norton, Krause, & Wolpert, 2019; Eckshtain et al., 2019), the results of the BUD clinical trial show a worrying lack of average treatment effectiveness for adolescent depression, irrespective of trial arm. There is unfortunately no reason to believe these results may not be largely representative of Norwegian CAMHS. There is hence a pressing need for the Norwegian CAMHS sector to investigate and benchmark treatment effectiveness for this patient group, and consider implementation of more effective treatments.

Parent-adolescent conflict has been included as a variable in several studies of adolescent depression treatment, as well as in study 1 of this thesis (Asarnow et al., 2009; Brent et al., 1998; Curry et al., 2006; Gunlicks-Stoessel et al., 2010; Young et al., 2009; Zisk et al., 2019). Unfortunately, this literature does not distinguish explicitly between parent-adolescent conflict as a process on the level of the family system and on the level of dyadic relationships within the family system. Neither has sufficient attention been paid to the complexity of measuring parent-adolescent conflict. Frequency of conflictual interactions, affective intensity of interactions, form of resolutions, persistence of conflict topics and negative representations of the relationship have generally not been differentiated in the conceptualisation of parent-adolescent conflict in clinical trials, meaning that some aspects are included in some conceptualisations but left out in others. This may diminish the power of studies to detect actual relationships, and confuse comparisons across studies. The field would likely benefit from articulating more clearly what aspect or aspects of parent-adolescent conflict at what level of observation are expected to be relevant for the clinical processes under study, and be more careful and specific about measurement of the kind of parent-adolescent conflict that is of interest.

This thesis nevertheless supports further study of dysfunctional states of parent-adolescent conflict as an important complicating factor in adolescent depression. The results show that parent general interpersonal problems contribute to dysfunctional conflict, perhaps by derailing normative developmental processes related to development of autonomy. The results suggesting that adolescents feeling conflicts to be more severe than parents is related to hopelessness, in effect a more complicated presentation of depression, further strengthen the case for attending to the state of communication and representations of relationships in the family. Finally, finding dysfunctional forms of parent-adolescent conflict to be a likely moderator of the effectiveness of ABFT relative to treatment as usual adds to the existing literature suggesting that parent-adolescent conflict may indeed be one factor that differentiates what works for whom in the treatment of adolescent depression. Further investigations of parent-adolescent conflict as a moderator of treatment outcome should be conducted, for ABFT compared to other treatments in particular, and more generally when comparing family- or relationship-oriented treatments to more individually focused treatments.

While adolescent depression is too often experienced in solitary despair, it unfolds in the context of interpersonal relationships, and will therefore be unavoidably intertwined in the fundamental interpersonal relationships of growing up. Continued

attention to the family context in treatment and clinical management of adolescent depression is clearly necessary.

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Paper 1

Moderation of Treatment Effects by Parent-adolescent Conflict in a Randomised Controlled Trial of Attachment Based Family Therapy for Adolescent Depression

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Acknowledgements

The authors thank the staff of the outpatient clinics Follo and Øvre Romerike at the Department of Child and Adolescent Mental Health Services, Akershus University Hospital, for contributing their time as therapists and assisting in enrolment of participants. We also gratefully acknowledge the invaluable contribution of the participating adolescents and their parents.

Abstract

Conflict with parents is frequent in adolescent depression, and has been shown to predict poor treatment outcomes. Attachment-based Family Therapy (ABFT) is a manualised treatment for adolescent depression that may be robust to parent-adolescent conflict. Parent-adolescent conflict was hypothesised as a moderator of treatment outcome in a randomised trial comparing 16 weeks of ABFT to treatment as usual, in Norwegian Child and Adolescent Mental Health Services. Change in depression ratings from baseline to week 16 was modelled using linear mixed models, and a three-way interaction of time, treatment allocation and parent-adolescent conflict was fitted to estimate a moderator effect. The moderator model was compared to simpler models using leave-one-out cross-validation. Results showed better outcomes predicted for Attachment-based Family Therapy at high levels of mother-adolescent conflict, and for treatment as usual at low levels of mother-adolescent conflict, giving preliminary support to the moderator hypothesis. Findings for father-adolescent conflict were less conclusive. High levels of conflict predicted worse outcomes in treatment as usual across three of four informants. Cross-validation did not clearly support the moderator model over a simple effect of time, indicating that the replicability of these findings is uncertain. The efficacy of Attachment-based Family Therapy may depend more on heightened parent-adolescent conflict than previously thought. The results suggest that parent-adolescent conflict should be further studied as a moderator of outcome in Attachment-based Family Therapy. The trial did not meet its recruitment target and had high attrition, limiting the conclusions that may be drawn. ClinicalTrials.gov Identifier: NCT01830088, registered 12 April 2013.

Keywords: Attachment Based Family Therapy, adolescent depression, randomised controlled trial, moderator, parent-adolescent conflict, Bayesian data analysis

Adolescents who suffer from depression report experiencing more conflict and less support in the relationship with their parents [1]. The transition into adolescence is normatively accompanied by increases in parent-child conflict [2], but not all parent-adolescent dyads manage these conflicts equally well [3]. Parent-adolescent conflict has been linked to onset of adolescent depressive symptoms in multiple studies [4-6]. Parent-adolescent conflict has further been found to predict recurrence of depression in adulthood [7], and depression has been found to mediate intergenerational continuity in high-conflict family environments [8].

Parent-adolescent Conflict and Treatment of Adolescent Depression

Because parent-adolescent conflict has shown a consistent association with the development and course of adolescent depression, it has also been studied as a potential predictor or moderator of outcome in multiple clinical trials of adolescent depression treatments. Moderators in the context of clinical trials have been defined as baseline variables across which the effect of treatment allocation on treatment outcome varies. Baseline variables that are associated with treatment outcome independently of treatment allocation are referred to as non-specific predictors [9]. Adolescent-reported parent-adolescent conflict was found to be a non-specific predictor of both treatment nonresponse and depression recurrence in a trial of different psychotherapies for adolescent depression [10]. In the Treatment of Selective Serotonin Reuptake Inhibitor-Resistant Depression in Adolescents Study, adolescent report of more parent-adolescent conflict was also found to be a non-specific predictor of nonresponse [11]. Mother-report of frequent and intense conflict was similarly found to be a non-specific predictor of poor outcomes in an exploratory analysis of data from the Treatment of Adolescent Depression Study [12]. However, in a trial comparing Interpersonal Therapy to treatment as usual, adolescents reporting more conflict with mothers at baseline benefited more from Interpersonal Therapy, which is a moderator effect [13]. Similarly, in a preventive group intervention based on Interpersonal Therapy with school counselling, the intervention was superior to school counselling only for those adolescents who reported heightened parent-adolescent conflict [14], also showing moderation by parent-adolescent conflict for the interpersonally focused treatment. Another comparable finding was reported from a trial comparing family-focused treatment to enhanced usual care for adolescents with a bipolar disorder [15]. For adolescents from families reporting heightened expressed emotion, the family focused treatment was superior to enhanced usual care, but this was not the case for adolescents from families with lower levels of expressed emotion.

Seen together, these findings suggest that while parent-adolescent conflict can impede treatment, treatments gains can perhaps be made in these cases by focusing treatment on family-related issues, making treatment effectiveness conditional on the level of conflict or family distress.

Attachment Based Family Therapy for Adolescent Depression

Attachment Based Family Therapy (ABFT) is a manualised family therapy for adolescent depressive symptoms and suicidal ideation [16]. ABFT initially focuses on identification and repair of relational ruptures between depressed adolescents and their parents. Building on reduced conflict and renewed trust in the parent-adolescent relationship, the family is then guided in collaborative work to reduce depressive symptoms and improve functioning. The developers of the intervention have conducted several clinical trials and other program evaluations [17], and ABFT has been designated a probably efficacious treatment [18]. Still, in the largest randomised controlled trial conducted to date, ABFT was not found to be superior to family-enhanced non-directive supportive therapy for reducing adolescent suicidal ideation [19]. A secondary analysis of that trial found that observations of less cooperative family communication, as well as non-white race and lower income-to-needs ratio predicted higher treatment benefit in both trial arms [20].

In Norway, an initial study found ABFT implementation in Norwegian public child and adolescent mental health services to be feasible and the treatment to be acceptable to Norwegian families [21]. A larger Norwegian randomised controlled trial comparing 16 weeks of ABFT to treatment as usual for adolescent depression was conducted to follow up on these findings. Contrary to the primary hypothesis of the trial, ABFT was not found to be superior to treatment as usual [22]. While the findings from these recent trials do not provide evidence that ABFT on average is more effective than treatment as usual or other active comparisons in treating adolescent depression or suicidal ideation, ABFT is a treatment where moderation of effectiveness by parent-adolescent conflict is highly plausible. In line with this, the registered secondary hypothesis of the Norwegian clinical trial was that the difference between ABFT and treatment as usual would be larger at higher levels of parent-adolescent conflict, that is, that treatment effects would be moderated by parent-adolescent conflict [9]. In this study, we will present results of these planned moderator analyses.

Methods

Participants

Participating families were recruited among adolescents referred to two Child and Adolescent Mental Health Services (CAMHS) in South-eastern Norway. The clinics were

publicly funded, and all treatments were provided free of charge to the patients and their families, within the framework of the universal health insurance system of Norway. During pre-specified recruitment periods, all referral letters for adolescents (13 - 17 years) were examined for mentions of depression or core depressive symptoms (depressed mood, anhedonia, or fatigue). The CAMHS routinely administered the Youth Self Report [23], and these were screened for raw scores on the Affective Problems subscale above 6 to find depressed adolescents not identified as such in their referral letters [24]. Eligible adolescents or their parents, depending on adolescent age, were contacted by telephone and invited to participate in a randomised trial of family therapy for adolescent depression. Participants were required to be currently living with an adult who had become a caregiver for them before age four, and willing to have this adult participate in treatment. Interested adolescents meeting these criteria were screened with Beck Depression Inventory-II [25] over telephone and invited for an assessment session if they scored above 17, a threshold expected to maximise sensitivity [26]. Adolescents were included in the study if they scored above 15 on the Grid Hamilton Depression Rating scale [GRID-HAMD, 27] and met diagnostic criteria for a current Major Depressive Disorder [28]. Adolescents meeting criteria for a psychotic disorder, anorexia nervosa, bipolar disorder, intellectual disability or pervasive developmental disorder were excluded from the study. In a small number of cases, exclusionary criteria (psychotic disorder or atypical anorexia nervosa) were not detected at baseline but uncovered during treatment. One family withdrew consent shortly after randomisation and are not included in any analyses. 60 participants were randomised, of which 52 (87%) were female. Figure 1 shows the flow of participants through the study.

[**Fig 1:** CONSORT Flow-chart for Study Participants]

Procedures

Participating adolescents and their parents met with a study-affiliated clinical psychologist (the first or second author) at the CAMHS and written informed parental consent and adolescent assent was obtained. Adolescents and parents were then interviewed separately with a semi-structured diagnostic interview. All interviews were video-recorded. Both parents and adolescents completed self-report measures. If the adolescent met inclusion criteria, the assessing clinician conducted randomisation by opening a sealed, numbered envelope containing the treatment allocation. Randomisation was stratified by site, age (13-15 years and 16-17 years), gender (male and female), and severity of depression (GRID-HAMD score of ≤ 24 and ≥ 25). Parents and adolescents were given feedback on diagnosis and treatment allocation at the end of the assessment session. The assessing clinician

answered questions from parents and the adolescent concerning the assessment and implemented standard safety monitoring procedures to the extent deemed necessary. CAMHS staff were then informed of treatment allocation and given a report of the assessment findings.

Treatment

Both ABFT and treatment as usual were provided for a minimum of 16 weeks but could be extended if deemed necessary by the therapist. ABFT consisted of weekly sessions as well as extra parent sessions in the early part of therapy. ABFT was delivered according to an available draft of the treatment manual [16]. Treatment as usual was not manualised, and the therapists were free to provide the treatment they considered most appropriate. When adolescents were assessed to be at high risk of self-harm or suicide, the therapist assigned to the case was immediately notified by study staff.

Clinician training and supervision

Therapists were trained in ABFT for the purpose of the trial. Training consisted of a day-long introductory seminar, followed by a three-day workshop, as well as reading the treatment manual. Therapists were required to have completed one case of ABFT under supervision before treating patients allocated to ABFT in the trial. All ABFT sessions were videotaped for supervision purposes. Weekly supervision by an experienced ABFT therapist was intended, but not achieved in practice. For the duration of the trial the therapists met nearly weekly and did peer supervision, and 42% of these sessions were also attended by a certified ABFT therapist and trainer. Clinicians in the treatment as usual arm were recruited from the regular staff of the CAMHS, and treated patients in the trial as part of their regular patient workload. Access to supervision varied by clinical experience, but all had access to discussing cases in multidisciplinary teams.

Changes to the protocol

According to the registered protocol, our primary and secondary outcome measures were supposed to be measured at 12, 24 and 48 weeks after treatment start. A four week waiting period from randomisation to treatment start was planned, but this was not feasible due to the severity of the depression for many patients and Norwegian standards of care. Consequently, the treatment period was extended from 12 to 16 weeks.

Assessment

For the duration of the treatment, patients were asked to complete self-report measures every other week. Most data were collected electronically using a secure online platform. Some self-report measures were administered as paper booklets by the treating

clinicians. Post-treatment assessment at 16 weeks was conducted by an independent clinical psychologist blinded to treatment allocation.

Measures

Diagnosis

Diagnostic evaluations were conducted with the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version [29]. Interrater reliability of the diagnosis of Major Depressive Disorder was established by blinded rescoring of a subsample of 28 interviews, including both excluded and included patients. κ for current Major Depression was 0.56, indicating fair interrater reliability [30].

Treatment Outcome

The primary outcome measure of the clinical trial was the total score on the GRID-HAMD, which is a version of the Hamilton Rating Scale for Depression that includes a structured interview guide, and scoring guidelines for weighing severity and frequency of symptoms to a composite score [27]. The GRID-HAMD has previously shown excellent interrater reliability [31]. Interrater reliability was assessed by blinded rescoring of a subsample of 28 interviews, including both excluded and included patients. The two-way mixed, consistency, average-measures intraclass correlation coefficient for the total score [32] was 0.89, indicating good interrater reliability.

Parent-adolescent Conflict

Parent-adolescent conflict was measured with the Perception of the Dyad subscale of the Conflict Behavior Questionnaire [CBQ, 33]. This scale consists of 16 items rated true or false concerning current conflict in a parent-adolescent relationship and was completed by parents as well as the adolescents separately for each parent. The CBQ was translated to Norwegian for this study, and a blind reverse translation was approved by the original author.

Analysis plan

Analyses included all patients randomised to treatment regardless of adherence to study treatment or procedures, in accordance with intent-to-treat principles. One patient withdrew consent and was omitted from all analyses. We conducted analysis within a Bayesian modelling framework, with estimation by Hamiltonian Monte Carlo as implemented in the statistical modelling platform Stan, using the RStan package [version 2.19.2, 34] for R [version 3.6.1, 35]. The results of a Bayesian analysis are distributions that show the probability of different model parameter values, conditional on the data and the model. For readers unfamiliar with Bayesian statistics, Baldwin and Larson [36] provide a very accessible introduction to the use of Bayesian linear regression in clinical psychology.

Analysing a Multi-informant Measure of Conflict

To improve the measurement precision of the hypothesised moderator variable we used a latent variable rather than raw scores, which we obtained by fitting a two-parameter logistic item response model to the CBQ data. The Stan platform is well suited for estimating IRT models, which can be embedded in a larger model of interest [37]. Adolescents completed the CBQ separately for each parent, and each parent completed the CBQ for their relationship to the adolescent. In the majority of cases this gave four different ratings of the degree of parent-adolescent conflict, two by the adolescent for each parent, and one by each parent. We chose to model all four ratings as potential moderators, fitting these models separately. We used the report of all four informants to fit the item response model, specifying the four latent conflict variables to have a multivariate normal distribution, with means of 0 and standard deviations of 1, and constraining item parameters to be equal across informants, assuming measurement invariance. Checking this assumption resulted in removal of two items [38]. Visual inspection of the posterior distributions of item characteristic curves plotted against the data indicated good fit for the remaining items. These plots as well as further details concerning checking of measurement invariance are available online at DOI: 10.17605/OSF.IO/KPJC6.

Robust Modelling of Treatment Moderation

We specified a hierarchical linear regression model with pre- and post-treatment GRID-HAMD scores nested within patients as the outcome variable, and a random intercept for each patient. The model included terms for the predictor variables time, treatment allocation and parent-adolescent conflict, and interaction terms for treatment by time, conflict by time and a three-way interaction of treatment by conflict by time. The three-way interaction estimates the moderator effect of interest, while the conflict by time interaction estimates a non-specific predictor effect. Treatment allocation was coded as -0.5 for treatment as usual and +0.5 for ABFT, which allows the coefficients for treatment or interactions with treatment to be interpreted as the predicted difference between the treatment groups, with the sign indicating the direction of the difference [9]. We standardised the GRID-HAMD scores by subtracting the median score across both time points of 21 and dividing by two times the median absolute deviation of 8, as standardisation can improve Hamiltonian Monte Carlo estimation and simplifies specification of reasonable priors [37].

Regression models with normally distributed errors are sensitive to outliers [39]. Psychotherapy outcome is known to be influenced strongly by extratherapeutic factors [40], which increases the probability of having multivariate outliers. To avoid having outliers

influencing slope estimates disproportionally to the bulk of observations we specified a Students' t-distribution with five degrees of freedom to the errors, giving a robust estimation of regression coefficients [37,41].

After fitting the model, we used exact leave-one-out cross-validation (LOO-CV), leaving out one patient at a time, to compare the model to three less complex models, repeating this across all four informant perspectives on conflict. LOO-CV estimates the expected log posterior density (ELPD), indicating how well the model is expected to fit new data from the same distribution [42]. The first of these three models had the term for the moderator effect removed, making it a model with conflict as a non-specific predictor of outcome. The second had all terms involving conflict removed, making it a model of different effects of treatment allocation over time. The third had all terms involving both conflict and treatment removed, making it a model of the effect of time alone. For clarity, we will term these four models "Moderator", "Non-specific predictor", "Treatment" and "Time" when comparing them.

Missing Data Management

There was a substantial number of patients missing outcome data (38 %). Bayesian data analysis provides a natural way to handle such missing observations, by estimating these as unobserved parameters of the model, which ensures that the loss of information due to missing data is reflected in the posterior distribution as increased uncertainty of model parameters such as regression coefficients [43]. When single items were missing from the CBQ, we estimated the latent variable of the IRT model from the observed items. In some cases, the entire CBQ was missing, and in these cases the latent variable was also estimated as a model parameter. This applied to 12% of adolescent reports of conflict with father, 28% of father reports of conflict, 3% of adolescent report of conflict with mother and 5% of mother reports of conflict.

Prior Distributions

In a Bayesian data analysis, a prior distribution must be assigned to all model parameters, representing our prior knowledge of these parameters. This prior distribution is combined with the likelihood of the data to produce the posterior distribution of the parameters. This allows us to include information and assumptions on what ranges of a parameter are at all reasonable. When reading the results of a Bayesian data analysis, the reader should examine the prior distributions used and assess whether they are reasonable assumptions, and these should hence always be reported. The prior distributions used in this analysis and their justifications are summarised in table 1.

TABLE 1 HERE

Estimation and Validation of Convergence

We fitted all models using four chains with the default Stan algorithm, 1000 warmup iterations and drawing 3500 samples from each chain. Gelman-Rubin statistics ($\hat{R}$) were below 1.01 for all parameters. Other Stan convergence diagnostics also indicated convergence for all chains and valid sampling from the posterior.

Results

Table 2 summarises posterior estimates from the moderator model with the different informants. The coefficient for the three-way interaction between treatment, time and parent-adolescent conflict is interpretable as the predicted difference in outcome (in units of 8 points on the GRID-HAMD) between ABFT and treatment as usual associated with a level of parent-adolescent conflict one standard deviation above the sample mean. The sign of the coefficient signifies the direction of the difference, with a negative coefficient being a difference favouring ABFT when conflict increases.

TABLE 2 HERE

We report the 66% and 90% Highest Density Intervals [44] of the marginal posterior distributions of each parameter. The choice of intervals is arbitrary, but .66 and .90 correspond to probabilities that have been described as likely and very likely, respectively [45]. These intervals show that there is considerable uncertainty, in particular for the coefficient for the moderator effect, and these data do not completely rule out an effect close to 0. Still, the main weight of the evidence is on a difference between ABFT and treatment as usual in the expected direction for three of the informants. The posterior probabilities of a coefficient for the moderator effect below 0 is .98 for adolescent report of conflict with mother, .50 for adolescent report of conflict with father, .97 for mother report of conflict and .97 for father report of conflict. Correlations between the latent conflict variables of the different informants are summarised in table 3.

TABLE 3 HERE

Visualised Model Predictions

To understand the implications of a fitted model, plotting its predictions across the range of a predictor variable can be helpful. Figure 2 shows the predicted change in GRID-HAMD score from baseline to outcome across the range of parent-adolescent conflict for all four informants, with the different lines representing the two treatment conditions (red for ABFT and black for treatment as usual). The uncertainty of the prediction is visualised by shading showing the 90% HDI. The points are the observations used to fit the model.

[**Fig 2:** Model Predictions Across the Range of Conflict for Different Informants]

Given the uncertainty in these estimates, they must be interpreted cautiously. For conflict with mother, the pattern is similar for both adolescent and mother report, with better outcomes predicted for ABFT relative to treatment as usual at high levels of conflict, and the opposite at low levels of conflict. This is not the case for conflict with father. For adolescent report the posterior distribution of the regression coefficient for a moderator effect has a mean of approximately 0, implying no moderation. For father report, the moderator model implies that parent-adolescent conflict is associated with worse or better outcomes in treatment as usual only, but that outcome in ABFT does not vary over father-reported conflict.

Using Cross-validation to Evaluate Expected Out-of-Sample Model Fit

Table 4 shows the differences in ELPD (expected log posterior density, obtained by LOO-CV) between the four models that were compared. It should be noted that the standard errors of these differences are known to be optimistic, especially in small samples, and a difference of four standard errors or more has been recommended for selection of one model over the other [46,42].

TABLE 4 HERE

Cross-validation clearly shows that the model "Time" has a better expected out-of-sample fit than "Treatment", with a difference in ELPD larger than ten times the standard error. The model "Time" also fits better than the model "Non-specific predictor" across all four informants, with differences in ELPD of more than five standard errors. The picture is less clear for the comparison of the "Moderator" model to "Time". For adolescent report of conflict with father "Time" is clearly better, with a difference larger than ten times the standard error. For father-report of conflict, and for adolescent and mother report of conflict with mother, the differences are too small relative to their standard errors to conclude with certainty that one model is better than the other.

Discussion

Our findings have considerable uncertainty, and the predicted differences in treatment outcome related to parent-adolescent conflict are clinically meaningful only in the higher and lower quantiles of the conflict distribution. This is not surprising, given the overall small average treatment effect in the trial [22]. For mother-adolescent conflict, there is some evidence of a moderator effect. The posterior distributions of the regression coefficients indicate that a moderator effect is more probable than equal effects of treatment across the range of mother-adolescent conflict, regardless of adolescent or parent report, and while

cross-validation does not fully support a moderator effect, it does not rule it out either. Adolescent report of conflict with father does not appear to be associated with treatment outcome. For father report of conflict, the fitted model implies an association with outcome restricted to treatment as usual, with the same uncertain results of cross-validation.

Earlier studies have found parent-adolescent conflict, in particular with mother, to negatively impact outcomes of various treatments for adolescent depression [10-12]. Our findings are similar for treatment as usual in two Norwegian CAMHS, giving further evidence for parent-adolescent conflict as a negative predictor of outcome in treatment of adolescent depression, although in our case not for adolescent report of conflict with father. Further, we found some evidence that in a family-based treatment, the reverse association may hold, in particular for mother-adolescent conflict. This is also in line with the findings from other studies [14,15,13], although findings from a secondary analysis of the largest trial conducted by the ABFT treatment developers did not show this pattern [20]. That study found adolescent report of family conflict and cohesion to be a non-specific predictor of reduction in suicidal ideation, but no evidence of moderation. However, those findings may not be directly comparable to the ones presented here, as their measure of general perceptions of family climate arguably assesses a different construct than the CBQ, which assesses the perception of distressing conflict in a specific dyadic relationship [33].

Does ABFT Depend on Heightened Parent-adolescent Conflict?

The pattern of moderation implied by the fitted model is worth noting, as the predicted outcomes of the two treatments compared appear to show roughly opposite associations with mother-adolescent conflict. This is an example of a moderator effect one would not necessarily suspect by looking at the residuals of a simpler model, as the error variances would not differ substantially between treatment groups even with a stronger moderation effect. Although this is speculative, such a pattern of moderation could perhaps explain the unexpected findings of the two latest trials of ABFT [19,22], where ABFT did not perform better than active comparisons, even though previous findings have been promising [17]. In developing and early testing of ABFT, the patient group have been predominantly composed of youth from disadvantaged neighbourhoods, with many families suffering from financial strain [47]. Financial strain has been shown to increase the frequency and severity of parent-adolescent conflict [e.g. 48], and was found to predict higher treatment benefit of ABFT and family-enhanced non-directive supportive therapy [20]. The degree of variation in and level of parent-adolescent conflict among patients participating in early development of ABFT has not been reported, as far as we know. It is therefore possible that the effectiveness

of ABFT is more dependent on the presence of parent-adolescent conflict and other relationship difficulties than previously thought. The findings presented in the present paper are too uncertain to permit a definitive conclusion, but indicate that this issue would bear further investigation.

Limitations and Strengths

This study has multiple limitations that must be taken into account. Firstly, the trial did not meet its planned sample size, and the resulting sample is small. Further, the proportion of missing outcome data was considerable. This lack of data is well reflected in the uncertainty of the reported results. Secondly, the number of male adolescents in the sample is very low, and the results cannot be generalised to the male adolescent population. A third limitation is that the adherence and competence of the ABFT therapists was not systematically assessed. Without systematic ratings of adherence and competence, we cannot conclude with certainty that the treatment provided in the ABFT treatment arm was according to the treatment manual [16]. The therapists had training and some supervision from an experienced and certified ABFT therapist. All had completed one case under supervision previous to treating randomised cases, in addition to their previous general clinical experience. The trial as such represents a reasonably realistic test of the efficiency of ABFT when implemented in a Norwegian context, but the therapists lacked extensive experience with the ABFT treatment model, which is both technically and personally demanding [16], and had less supervision than in other ABFT clinical trials [19,47,49]. Strengths of the study are reporting on a planned moderator analysis with a clear theoretical justification and employing modern estimation and modelling techniques.

Conclusion

The secondary hypothesis of the Norwegian ABFT clinical trial was that the treatment effect of ABFT relative to treatment as usual would be moderated by parent-adolescent conflict. These findings give some support to that hypothesis, but is of insufficient certainty to inform clinical practice. Further studies should investigate whether parent-adolescent conflict and other strains in the parent-adolescent relationship moderate the effectiveness of ABFT relative to other treatments. Given recent findings that suggest the average effect of ABFT to differ little from treatment models that may be less demanding to implement [19], it would be important to know whether subgroups of depressed adolescents could benefit relatively more from ABFT, in particular subgroups known to be doing poorly in other treatments.

Declarations

Funding

This research was partially funded by the South-Eastern Norway Regional Health Authority (grant 2013008). The funding body did not participate in or influence study design, data collection, statistical analyses, interpretation of results, or writing of this manuscript.

Conflicts of interest

The authors declare that they have no conflict of interest.

Ethics approval

All procedures performed were in accordance with the ethical standards of the national research committee and with the 1964 Helsinki declaration and its later amendments. The study protocol, participant information letters and consent forms were reviewed and approved by the Regional Committee for Medical and Health Research Ethics for Eastern Norway (REK Øst).

Consent to participate

Informed consent was obtained from all individual participants included in the study, or their legal guardians, in which case assent to be included in the study was obtained from the underage participant.

Consent for publication

Not applicable.

Availability of data and materials

The data that support the findings of this study are available from Akershus University Hospital, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are, however, available from the authors upon reasonable request and with permission of Akershus University Hospital and the Regional Committee for Medical Research Ethics, South-East Norway.

Code availability

Stan and R code from the analysis, as well as the fitted models (including posterior samples) and results of LOO-CV is available at doi:10.17605/OSF.IO/KPJC6.

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Fig 1: CONSORT Flow-chart for Study Participants

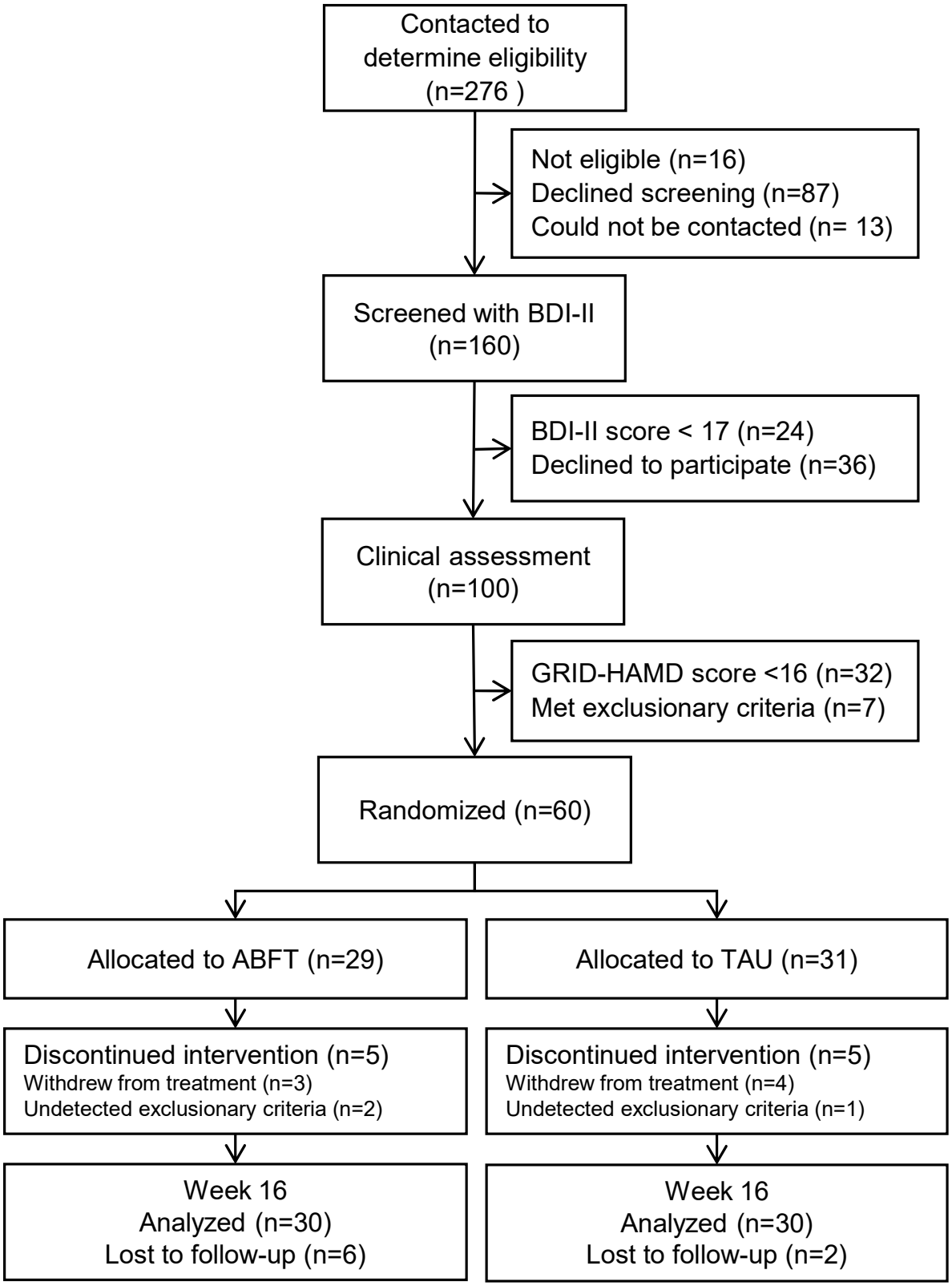

Fig 2: Model Predictions Across the Range of Conflict for Different Informants

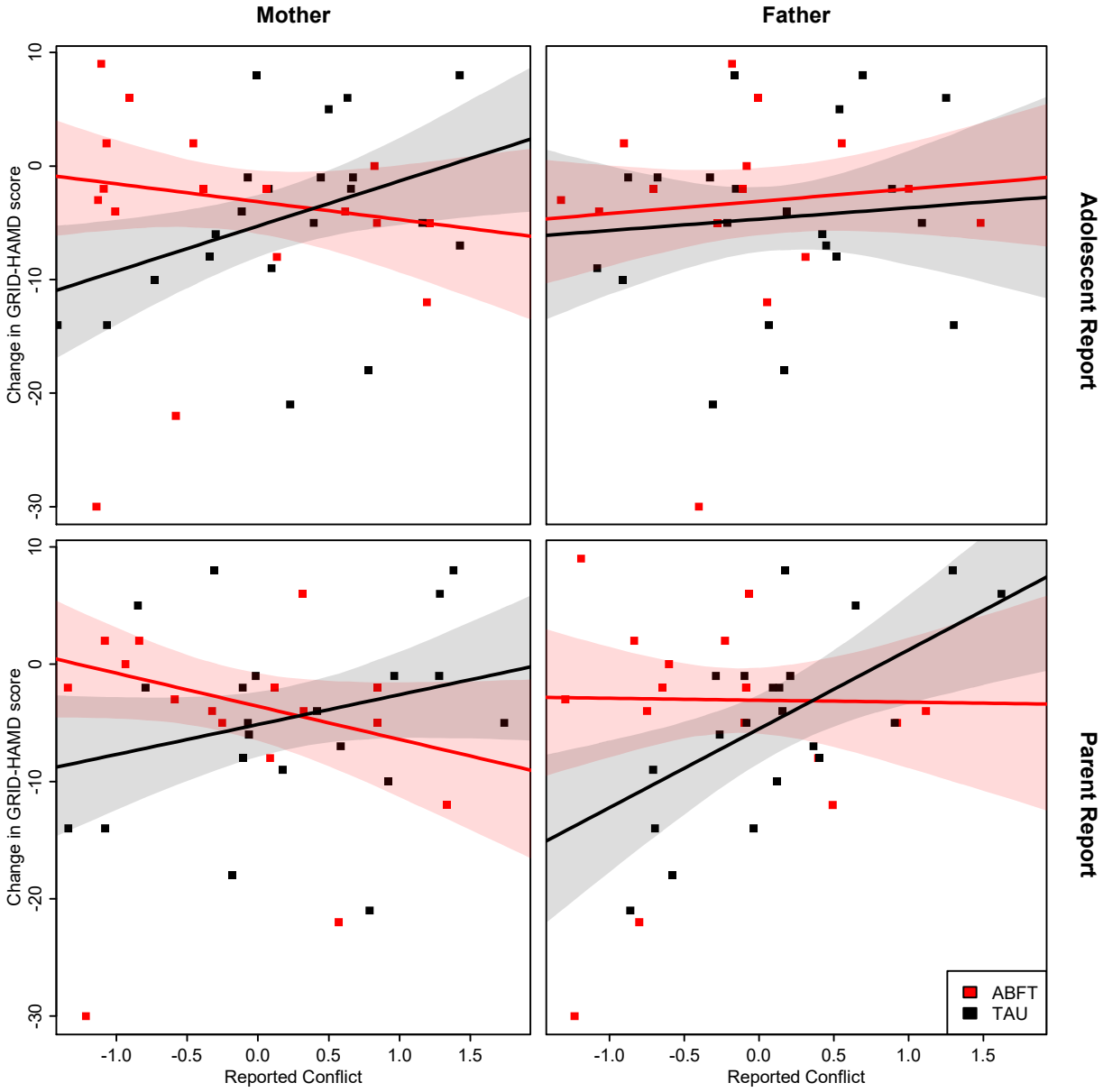

Table 1: Prior Distributions and Reasoning for Choices of Prior

Parameter	Prior Distribution	Reasoning
Regression coefficients	Normal (0, 1)	Weakly informative prior as the dependent variable is centred on the median score and scaled by twice the median absolute deviation.
Error variance	Half-student's t (3, 0, 1)	Weakly informative prior on the error variance, putting most of the prior weight on errors between 0 and 1, but with heavy tails allowing for a much higher error variance.
Random intercepts	Hierarchical normal prior, with location 0 and a Half-student's t (3, 0, 1) prior on the scale.	Defines random intercepts as deviations from the intercept of the whole sample, and estimates the variance of the random intercepts from the data, with a weakly informative hyperprior.
Latent variables for CBQ IRT model	Multivariate normal (0, 1) with an LKJ (2) prior on the standardised covariance matrix.	Defines the latent variables for parent-adolescent conflict as four correlated Normal (0,1) variables with a weakly informative prior on the correlation coefficients.
Item thresholds for CBQ IRT model	Hierarchical normal prior with hyperpriors Normal (0, 3) for location and Half-students' t (3, 0 ,1) for scale.	Weakly informative hierarchical prior for the item thresholds, as these are interdependent with the defined latent variable.
Item discrimination for CBQ IRT model	Gamma (2, 0.5)	Places most of the prior weight on discrimination between 1 and 5, which is the most probable range for items of an established instrument, but does not rule out higher or lower values.

Table 2: Parameter Estimates from Moderator Models

Conflict Report and Model Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Adolescent report of conflict with mother						
Intercept	0.09	0.09	0.09	0.00 ; 0.17	-0.05 ; 0.24	11801
Variance of random intercepts	0.36	0.13	0.37	0.28 ; 0.50	0.14 ; 0.56	1764
Variance of errors	0.53	0.08	0.53	0.45 ; 0.59	0.41 ; 0.65	2382
Regression coefficients:						
Time	-0.53	0.15	-0.53	-0.65 ; -0.37	-0.78 ; -0.29	10094
Treatment	0.01	0.18	0.01	-0.16 ; 0.18	-0.29 ; 0.30	10608
Parent-adolescent conflict	0.27	0.29	0.27	-0.01 ; 0.55	-0.21 ; 0.76	7372
Treatment x time	0.07	0.11	0.07	-0.04 ; 0.17	-0.12 ; 0.25	9108
Conflict x time	0.15	0.19	0.15	-0.03 ; 0.31	-0.16 ; 0.47	7425
Treatment x conflict x time	-0.69	0.34	-0.70	-1.01 ; -0.38	-1.25 ; -0.15	5919
Adolescent report of conflict with father						
Intercept	0.09	0.09	0.09	0.00 ; 0.18	-0.07 ; 0.24	12959
Variance of random intercepts	0.36	0.13	0.38	0.27 ; 0.51	0.14 ; 0.58	2059
Variance of errors	0.58	0.08	0.57	0.49 ; 0.64	0.45 ; 0.69	2853
Regression coefficients:						
Time	-0.49	0.16	-0.48	-0.64 ; -0.34	-0.75 ; -0.23	11610
Treatment	0.00	0.19	0.00	-0.19 ; 0.16	-0.30 ; 0.31	11630
Parent-adolescent conflict	0.20	0.31	0.20	-0.07 ; 0.51	-0.29 ; 0.71	11097
Treatment x time	0.04	0.1	0.04	-0.06 ; 0.13	-0.13 ; 0.21	10099
Conflict x time	0.13	0.22	0.13	-0.07 ; 0.34	-0.24 ; 0.47	5198
Treatment x conflict x time	0.01	0.40	0.00	-0.38 ; 0.36	-0.60 ; 0.70	4474
Mothers report of conflict						
Intercept	0.09	0.09	0.09	0.00 ; 0.17	-0.05 ; 0.25	12613
Variance of random intercepts	0.37	0.12	0.39	0.29 ; 0.50	0.18 ; 0.58	2008
Variance of errors	0.54	0.08	0.54	0.46 ; 0.60	0.41 ; 0.66	2488
Regression coefficients:						
Time	-0.55	0.15	-0.54	-0.69 ; -0.4	-0.80 ; -0.30	10341
Treatment	-0.03	0.18	-0.03	-0.21 ; 0.13	-0.33 ; 0.26	11666
Parent-adolescent conflict	0.19	0.30	0.20	-0.07 ; 0.51	-0.29 ; 0.70	8534
Treatment x time	-0.08	0.11	-0.08	-0.18 ; 0.02	-0.26 ; 0.09	9297
Conflict x time	-0.02	0.19	-0.02	-0.19 ; 0.16	-0.33 ; 0.28	6471
Treatment x conflict x time	-0.67	0.34	-0.68	-1.01 ; -0.38	-1.22 ; -0.11	6593
Fathers report of conflict						
Intercept	0.09	0.09	0.09	0 ; 0.17	-0.05 ; 0.24	9540
Variance of random intercepts	0.37	0.12	0.38	0.29 ; 0.5	0.17 ; 0.57	1651
Variance of errors	0.51	0.08	0.50	0.43 ; 0.58	0.38 ; 0.64	1740
Regression coefficients:						
Time	-0.54	0.16	-0.54	-0.68 ; -0.38	-0.79 ; -0.27	5165

Conflict Report and Model Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Treatment	-0.01	0.18	-0.01	-0.18 ; 0.16	-0.32 ; 0.26	10840
Parent-adolescent conflict	0.30	0.30	0.30	0.01 ; 0.59	-0.19 ; 0.81	5729
Treatment x time	-0.03	0.11	-0.03	-0.13 ; 0.08	-0.21 ; 0.16	6466
Conflict x time	0.41	0.24	0.41	0.18 ; 0.61	0.03 ; 0.81	2780
Treatment x conflict x time	-0.86	0.44	-0.87	-1.28 ; -0.46	-1.60 ; -0.17	2701

Table note: Mean = Posterior mean, SD = Posterior standard deviation, Median = Posterior median, 66% and 90% HDI = 66% and 90% Highest Density Intervals, ESS = Effective Sample Size, estimates the number of independent draws from the posterior distribution

Table 3: Correlation Coefficients for Latent Conflict Variables (Posterior means and 90% CI)

	Adolescent on father	Adolescent on mother	Father on adolescent
Adolescent on mother	0.20 (-0.06 ; 0.46)		
Father on adolescent	0.59 (0.39 ; 0.79)	0.33 (0.04 ; 0.62)	
Mother on adolescent	0.03 (-0.21 ; 0.27)	0.49 (0.28 ; 0.73)	0.50 (0.26 ; 0.75)

Table 4: Model Comparison with Leave-one-out Cross-validation

Reporter and Models Compared	Difference	SE
Adolescent Report of Conflict with Mother		
Time	0	0
Moderator	-1.86	1.36
Treatment	-1.87	0.14
Non-specific predictor	-3.91	0.71
Adolescent Report of Conflict with Father		
Time	0	0
Treatment	-1.87	0.14
Non-specific predictor	-3.74	0.31
Moderator	-5.57	0.40
Mother Report of Conflict		
Time	0	0
Treatment	-1.87	0.14
Moderator	-3.02	1.32
Non-specific predictor	-4.47	0.30
Father Report of Conflict		
Time	0	0
Treatment	-1.87	0.14
Moderator	-2.34	1.86
Non-specific predictor	-4.54	0.77

Table note: Difference = Difference in expected log posterior density to the best-fitting model of those compared; SE = Standard error of the difference

Paper 2

Child Psychiatry & Human Development
https://doi.org/10.1007/s10578-020-00955-0

ORIGINAL ARTICLE

Conflict with Parents in Adolescent Depression: Associations with Parental Interpersonal Problems and Depressive Symptoms

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Abstract
Conflict with parents is common among depressed adolescents, interferes with treatment, and may increase risk of recurrence. Parental depressive symptoms have been shown to predict conflict with adolescent children, but an important role for different kinds of parental interpersonal problems, as described by interpersonal circumplex, is also plausible. This study compared parental interpersonal problems to parental depressive symptoms as predictors of parent-adolescent conflict reported by a depressed adolescent child, using multilevel linear regression, leave-one-out cross-validation and model stacking (N = 100 parents, 57 mothers and 43 fathers, of 60 different adolescents). Cross-validation and model stacking showed that including parental interpersonal problems contributes to accurate predictions. Parents reporting more interpersonal problems related to excessive dominance or submissiveness was associated with increased or decreased conflict, respectively. Parental depressive symptoms were found to be negatively associated with parent-adolescent conflict only in father-adolescent relationships.

Keywords Parent-adolescent conflict · Adolescent depression · Parental depression · Interpersonal circumplex · Bayesian data analysis

Introduction

Parent-adolescent conflict is common between depressed adolescents and parents of both genders [1, 2]. High levels of parent-adolescent conflict predicts the development of adolescent depression [3–5], appears to interfere with treatment [6, 7], and increases the risk of recurrence in adulthood [8]. Both maternal and paternal depression are well established as predictors of parent-adolescent conflict [9–11], but as managing and resolving conflict is inevitably an interpersonal situation, an association with parental difficulties in interpersonal functioning is also plausible.

Interpersonal Theory and the Interpersonal Circumplex

A prominent approach to individual differences in interpersonal functioning is interpersonal theory, originating in the work of Sullivan [12]. This line of research has identified two fundamental dimensions of interpersonal phenomena, termed agency and communion. These two dimensions and the interpersonal circumplex they define when combined has shown good fit to variation in observed interpersonal behaviour, as well as interpersonal styles and individual differences in interpersonal functioning [13, 14]. As an interpersonal disposition, agency concerns being predominantly dominant or submissive across interpersonal situations, while the dimension of communion in a similar manner refers to being predominantly nurturing and warm or more distant and cold. The interpersonal circumplex has the advantage of not assuming interpersonal difficulties to be unidimensional, allowing for the impact on functioning to differ across kinds of interpersonal situations.

Electronic supplementary material The online version of this article (doi:https://doi.org/10.1007/s10578-020-00955-0) contains supplementary material, which is available to authorized users.

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Published online: 18 January 2020

Interpersonal Problems and Parent-Adolescent Conflict

In adolescence, the development of age-appropriate autonomy requires gradual renegotiation of patterns of interaction, and parent-adolescent conflicts are suggested to play an important role in this reorganization of the parent–child relationship [15, 16]. In interpersonal theory terms, adolescents will tend to assume high-agency interpersonal behaviours across an increasing range of interpersonal situations with their parents, both conflictual and non-conflictual. Interpersonal theory predicts that if parents reciprocate with low-agency behaviours to an appropriate degree, the result is a transitory increase in interpersonal conflict, before a new pattern of interaction is established [14]. Such patterns of transitory increase in conflict and subsequent realignment of the relationship has been observed in non-clinical samples [16]. The functional impairment and cognitive and affective symptoms of adolescent depression will tend to increase the frequency of potential conflicts in the parent-adolescent relationship, as adolescents are unable to meet parental expectations and behave in ways parents might find unacceptable [17]. Parental difficulties in interpersonal functioning could then lead to a cascade of parent-adolescent conflict, first making parents more prone to escalate potential conflicts, and then increasing the probability of negative resolution and persistence of these conflicts.

The Present Study

Interpersonal theory is a conceptually rich and well-developed theoretical framework for studying parent-adolescent interaction [18], but has not yet been applied to the study of parent-adolescent conflict in adolescent depression. The aim of the present study is to evaluate whether parental interpersonal problems are associated with parent-adolescent conflict reported by their depressed adolescent children and investigate whether the strength of the association varies across the interpersonal circumplex. We will also assess whether any such association has incremental predictive value compared with the expected association between parental depression and parent-adolescent conflict that has been found in previous research in related populations [10, 19–21].

Methods

Participants

The data analysed in this study are from baseline assessments in a randomized controlled trial (clinicaltrials.gov identifier NCT01830088) comparing Attachment-Based Family Therapy [22] to treatment as usual [23], manuscript in preparation]. Participating families were recruited among adolescents referred to two Child and Adolescent Mental Health Services (CAMHS) in South-eastern Norway. During pre-specified recruitment periods, all referral letters for adolescents (13–17 years) were examined for mentions of depression or core depressive symptoms (depressed mood, anhedonia, or fatigue). The CAMHS routinely administered the Youth Self Report [24], and these were screened for raw scores on the Affective Problems subscale above six to find depressed adolescents not identified as such in their referral letters [25]. Eligible adolescents or their parents, depending on adolescent age, were contacted by telephone and invited to participate in a randomized trial of family therapy for adolescent depression. 276 adolescents were contacted. Participants were required to be currently living with an adult who had become a caregiver for them before age four, and willing to have this adult participate in treatment. Interested adolescents meeting these criteria (160 of 276) were screened with Beck Depression Inventory-II [26] over telephone and invited for an assessment session if they scored above 17, a threshold expected to maximize sensitivity [27]. Of those screened with the BDI-II, 136 scored above the threshold, and 100 of these agreed to meet with study personnel for a clinical assessment. Adolescents were included in the study if they scored above 15 on the Grid Hamilton Depression Rating scale [28] and met Diagnostic and Statistical Manual of Mental Disorders [29] criteria for a current major depressive episode. Adolescents meeting criteria for a psychotic disorder, eating disorder, bipolar disorder, intellectual disability or pervasive developmental disorders were excluded from the study. One family withdrew consent after assessments had been completed. In all 60 adolescents were included (55 female, 5 male), with 43 fathers and 57 mothers, among whom there were 19 intact couples.

Procedures

Participating adolescents and their parents met with a study-affiliated clinical psychologist (the first or second author) at the CAMHS for an assessment and written informed parental consent and adolescent assent was obtained. Adolescents and parents were then interviewed separately. All interviews were video recorded. Self-report measures collected from parents and adolescents were completed during the appointment.

Measures

Parent-Adolescent Conflict

Parent-adolescent conflict was measured by the report of the adolescent on the Perception of the Dyad subscale of the Conflict Behavior Questionnaire [CBQ, 30], separately for each parent. This scale consists of 16 items rated true or false concerning current conflict in a parent-adolescent

relationship. The CBQ has been widely used as a measure of parent-adolescent conflict in depressed adolescents [e. g. 31, 32]. The CBQ was translated to Norwegian for this study, and a blind reverse translation was approved by the original author.

Parental Interpersonal Problems

Parental interpersonal problems were measured by parents completing the Inventory of Interpersonal Problems—Circumplex [IIP-C, 32 item version, 33, 34]. The items of IIP-C map onto the interpersonal circumplex, and is well established as a valid and structurally sound measure of problems in interpersonal functioning [35, 36]. We computed scores for each parent on the two main orthogonal factors Agency and Communion, and the general interpersonal distress factor Elevation, according to the method described by Gurtman and Balakrishnan [37], using available Norwegian norms for standardizing the scores [38]. An unofficial Norwegian translation of the IIP-C was used, with some items deviating slightly from the official Norwegian translation. We carefully examined item-scale correlations and found that the circumplex structure of the instrument was not compromised.

As a norm-adjusted standardized variable, Agency runs from negative scores for more problems than the mean of the normative sample related to being interpersonally submissive, through zero for the mean level of interpersonal difficulty, to positive scores for more problems related to being interpersonally dominant. Similarly, Communion runs from negative scores for problems related to being withdrawn and cold, to positive scores for more problems related to being preoccupied with caring and maintaining interpersonal closeness. These main factors of the IIP-C are stable measures of a trait-like interpersonal style [39]. Elevation is a measure of a more state-like general level of interpersonal distress [37].

Parental Depressive Symptoms

Parental depressive symptoms were measured by parental responses to 17 items from the Symptom Checklist 90—Revised [SCL-90-R, 40], which comprise the revised depression subscale developed by Paap and colleagues [41] using nonparametric item response modelling and a large Norwegian outpatient sample.

Adolescent Depression Severity and Diagnosis

Diagnostic assessments were conducted with the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version [42]. The severity of adolescent depressive symptoms were further assessed with the clinician-rated Grid-Hamilton Depression Rating Scale [28].

Analysis Plan

We conducted analysis within a Bayesian modeling framework, with estimation by Hamiltonian Monte Carlo (HMC) as implemented in the Stan programming language, using the RStan package version 2.18.2 [43], for R version 3.5.1 [44]. The results of a Bayesian analysis are distributions that show the probability of different model parameter values, conditional on the data and the model. For the reader unfamiliar with Bayesian statistics, Baldwin and Larson [45] provide a very accessible introduction to the use of Bayesian linear regression in clinical psychology. Bayesian modelling is also well suited to small sample sizes, as long as proper caution is paid to choice of priors and validation of convergence [46]. Stan and R code for the analysis, as well as the sets of samples drawn from the posterior distribution and used for inference, has been made available at https://doi.org/10.17605/OSF.IO/D2F8A.

Modelling Predictors of Parent-Adolescent Conflict

Our overall analytic approach was multiple regression modelling, with adolescent report of parent-adolescent conflict as the dependent variable, and a simple multilevel structure with parents nested within adolescents and a random intercept for each adolescent [47]. The regression models were specified with a latent dependent variable, obtained by fitting a two-parameter logistic item response model to the responses on the CBQ Perception of the Dyad scale. Stan is well suited for estimating item response theory (IRT) models, and these can be incorporated as part of a larger model of interest [48]. Our aim in doing IRT modelling was not to develop a revised measure, only to extract a continuous and more reliable dependent variable. Another advantage of item response models is that the reliability of the scale can be evaluated across the range of the latent trait, showing at what ranges the scale provides most information, and hence highest precision, given an item response models that fits the data [49].

Given that parent-adolescent conflict is clearly a multidetermined phenomenon, we expected observations that deviated substantially from the predicted value based on a limited set of predictors. We therefore aimed for robust estimation of the regression model, by defining a t-distribution for the likelihood, with the degrees of freedom estimated as a parameter [50]. This allows the model to adapt the level of robustness to the data, and hence avoid letting such outlier observations influence the slope too much.

Bayesian analysis requires specification of a prior distribution for all parameters (priors), representing our

assumptions and knowledge about the model parameters independently of the data. For example, if a standardized beta coefficient from a linear regression model could not reasonably be expected to be greater than 2 or smaller than −2, and would most likely fall between −1 and 1, as would often be the case in clinical psychology, this knowledge can be encoded by a Normal (0,1) prior distribution. A reader evaluating the results of a Bayesian analysis should consider the priors specified and decide whether they are reasonable, and priors should hence always be reported [45]. The priors for this analysis are summarized in Table 1.

Missing Data Management

The CBQ had 0.7% data missing as single items. For the cases with items missing on the CBQ, we estimated the latent variable based on the observed items. There was 0.4% data missing as single items from the IIP-C, and 0.1% from the SCL-90 Depression Scale. For the individual IIP-C scales and the SCL-90 Depression scale we singly imputed missing responses to items by two-way imputation [51], using the 'twoway' function from the R-package 'mokken' version 2.8.11 [52], before calculating scale scores. To verify that single imputation was appropriate, we multiply imputed 1000 datasets using two-way imputation and calculated the variables Agency, Communion, Elevation and Parental depressive symptoms in each dataset for all respondents with missing responses. This allowed us to assess to what extent the calculated summary variables of interest to us varied across imputations. The standard deviations of the standardized summary variables calculated across imputations and within each respondent ranged from 0.02 to 0.04 for Agency, 0.02 to 0.04 for Communion, 0.01 to 0.02 for Elevation and < 0.01 to 0.04 for Parental depressive symptoms, indicating that imputations varied minimally, and that single imputation was unlikely to bias results severely.

In two cases, the complete CBQ was missing, in three other cases the complete SCL-90-R, and in one of these three cases the IIP-C was also missing. For these we used Bayesian imputation, treating the missing observations as unknown parameters of the model, which preserves the uncertainty due to not having made these observations in the posterior distribution [53]. For the missing observations of IIP-C and SCL-90-R, we specified a multivariate normal distribution for the complete predictor matrix, composed of observed data and parameters for the missing observations. This allows us to use any information available in the other predictor variables to inform the estimates for the missing observations.

Estimation and Evaluation of Convergence

All posterior samples used for inference were drawn using four markov chains in Stan with the NUTS algorithm, 1000 warmup iterations, and 3500 samples from each chain. There were no divergent iterations or other Stan indicators of biased inference. Gelman-Rubin statistics [50], and effective sample size estimates (see Table 4), indicated convergence for all parameters.

Table 1 Prior distributions and reasoning for choices of prior

Parameter	Prior distribution	Reasoning
Random intercepts	Hierarchical normal prior, with location 0 and a Half-student's t (3, 0, 1) hyperprior for scale	Defines random intercepts as deviations from the sample mean of 0, and estimates the variance of the random intercepts from the data, with a weakly informative hyperprior
IRT-theta (Conflict level)	Normal (0,1)	Fixes the location and scale of the latent conflict variable for model identifiability, and to ensure a standardized dependent variable for interpretability
IRT-beta (Item difficulty)	Hierarchical normal prior with hyperpriors Normal (0, 3) for location and Half-student's t (3, 0, 1) for scale	Weakly informative hierarchical prior, as the interdependent IRT-theta parameter has fixed location and scale
IRT-alpha (Item discrimination)	Gamma (2, 0.5)	Item discrimination parameters for the CBQ assumed to lie between 0 and 10, as the item characteristic curve does not change meaningfully across alphas larger than 10
Error variance in regression model	Half-student's t (3, 0, 1)	Regularizing prior on the error variance, which still allows for large estimates if warranted by the data
Degrees of freedom in Student's t-distributed likelihood	Gamma (2, 0.1) Constrained to be ≥ 1	Degrees of freedom for the likelihood between 1 and about 30, allowing for the likelihood to be very near normal, or have a large degree of robustness, as required
Regression coefficients	Normal (0, 1)	Regularizing prior on the regression coefficients

Evaluating Hypotheses Through Cross-validation and Model Stacking

Our research question can be framed as a question of comparative predictive value of different models. Does parent report of interpersonal problems contribute unique information to predicting adolescent report of parent-adolescent conflict, when compared with a model predicting conflict from parental report of depressive symptoms? The predictive precision of models can be compared by estimating their expected fit to new data. We estimated this using exact leave-one-out cross-validation. This is conducted by refitting the model once for each observation (or cluster of observations in hierarchical models, if predictive precision for new clusters is what is of interest) with one observation left out for each refitting. The log-likelihood of the held-out data given the refitted model is saved for each refitting, and together estimates the expected log predictive density, a measure of the expected fit of the model to new data from the same distribution [54]. Different models can then be compared on their expected log predictive density values.

The results of leave-one-out cross-validation can also be used for model stacking, a procedure that takes a set of models and gives the weighted combination of these that has the highest expected predictive accuracy [55]. The obtained stacking weights are interpretable as the contribution of each model to predictive accuracy when combined with the other models entered in the stacking procedure.

We fitted, cross-validated and stacked four models. As our model had a hierarchical structure with parents nested within adolescents, we left one family out at a time. To calculate the pointwise log-likelihood, we took the summed log-probability mass of the observed item responses to the CBQ conditional on the expected value from the regression and the item parameter estimates. The first model had parental depressive symptoms as the predictor. The second had parental depressive symptoms, parent gender and their interaction as predictors. The third had the three parent interpersonal problem variables agency, communion and elevation as predictors. The fourth had the three parent interpersonal problem variables, parent gender, and interaction terms between each interpersonal problem variable and parent gender. We also included adolescent age in years, centred on age 15, as a covariate in all four models, as age has been shown to be associated with parent-adolescent conflict [56].

By cross-validating and stacking these models, we can obtain an estimate of the relative predictive value of parental interpersonal problems and parental depressive symptoms for predicting parent-adolescent conflict for a new depressed adolescent, and assess whether any associations are conditional on parent gender, by comparing the fit of models with interaction terms to models without. Due to the low number of male adolescents in the sample, we did not fit models with adolescent gender.

Results

Distribution of Predictor Variables

The mean scores on the IIP-C variables were: agency −0.31 (SD 0.63, range −2.25; 1.36), communion 0.23 (SD 0.54, range −1.89; 1.88) and elevation 0.22 (SD 0.69, range −1.37; 1.66), showing a considerable variation in the degree and kind of interpersonal problems reported by the parents in this sample. On the SCL-90-R revised depression scale (items rated 1–5), the mean item score was 1.94 (SD 0.78, range 1; 4). Some parents reported clearly clinical levels of depressive symptoms: 39 (40.2%) were at or above the mean raw score of a clinical outpatient sample [41]. Posterior estimates of the predictor variable correlation matrix are displayed in Table 2.

Item Response Modelling of the CBQ—Perception of the Dyad

Inspection of item characteristic curves and the observed data indicated adequate fit. These plots can be found in the Supplementary material. Figure 1 shows the test information function, which indicates that the scale has most information about above-average levels of conflict, but covers the relevant range reasonably well.

Evaluating Models by Leave-one-out Cross-validation and Model Stacking

The four models and the differences in expected log posterior density are displayed in Table 3, along with the stacking weights obtained from the stacking_weights() function from the R package loo [57].

Observing the expected log posterior densities and their standard errors, several conclusions may be drawn. Firstly, the data do not support modelling an interaction between parent gender and interpersonal problems, given the difference in

Table 2 Estimated predictor correlation coefficients (posterior means and 93% CI)

	Parental depression	Agency	Communion
Agency	−.42 (−.55; −.27)		
Communion	−.06 (−.26; .15)	−.22 (−.45; .05)	
Elevation	.58 (.45; .68)	−.54 (−.67; −.36)	−0.18 (−.43; .12)

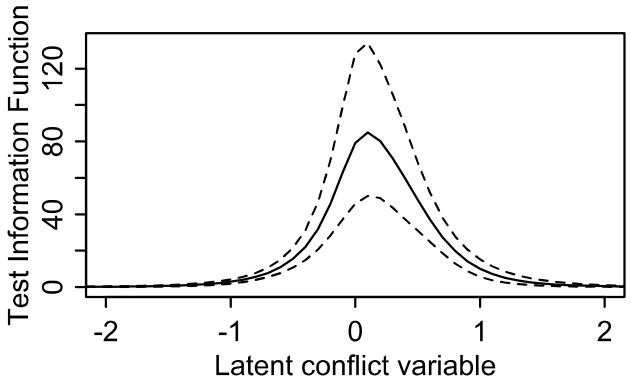

Fig. 1 Test information function for the conflict behaviour. Questionnaire—perception of the Dyad

Table 3 Results of leave-one-out crossvalidation and model stacking

Model	Difference	SE	Stacking weight
Parental interpersonal problems	0	0	0.75
Parental interpersonal problems with parent gender interaction	3.89	1.58	0
Parental depressive symptoms	8.35	6.21	0
Parental depressive symptoms with parent gender interaction	10.07	7.28	0.25

Difference = Difference in expected log posterior density to model with highest expected log posterior density; SE = Standard error of the difference; Stacking Weight = Model weight in stacking procedure

expected log posterior density and the stacking weights. Secondly, the data supports both parent interpersonal problems and parental depressive symptoms as predictors of parent-adolescent conflict. Though the difference between the model with parental interpersonal problems and the models with parental depression is larger than the standard deviation, it is not by much. The stacking weights imply that a combination of the model with parental interpersonal problems and the model with parental depression and an interaction with parent gender gives the highest expected predictive accuracy, but with most weight given to parental interpersonal problems.

Regression Parameter Estimates

The regression model parameter estimates from the two models given a positive stacking weight are displayed in Table 4. Both models also have a large number of hierarchical parameters (such as IRT item parameters and hyperparameters, latent trait estimates, and random intercepts per adolescent). These parameters are summarised in the Supplementary material.

The regression parameter estimates show a positive association between parental agency-related interpersonal problems and parent-adolescent conflict. The positive sign of the coefficient implies that as parents report more problems related to being too interpersonally domineering, their adolescents will tend to report more conflict. The posterior distribution of regression coefficient values (summarised in the table by its mean, standard deviation and the 3.5th and 96.5th percentiles) shows that the data are not at all consistent with a negative association under this model. The data are also not very consistent with a near-zero association, with only a 0.08 probability of a standardised regression coefficient smaller than 0.1.

The posterior distribution for Communion is symmetric around 0, which means the data are most consistent with no strong association between parent-adolescent conflict and parents reporting difficulties either being too cold and distant or overly concerned with maintaining relationships. It is worth noting that in a Bayesian data analysis, an estimate of 0 is no less certain than any other estimate, unlike in classical hypothesis testing, where failure to reject the null hypothesis cannot be interpreted as evidence for the null hypothesis being true [58].

The posterior mean estimate for Elevation, the interpersonal problem variable measuring general interpersonal distress, is weakly positive, but there is considerable uncertainty in this estimate. An association near zero (between -0.1 and 0.1) is quite consistent with the data, with a probability of 0.60, but any association is probably positive, with a 0.89 probability of a regression coefficient larger than 0. This means there may be an association between parental general interpersonal distress and parent-adolescent conflict, and that any association is probably positive and of small magnitude, but that the data does not provide conclusive evidence.

For parental depression, the coefficients show a negative association for fathers only, as the positive coefficient for the interaction with dummy-coded parent gender is of similar magnitude as the coefficient for parental depression. The coefficient for paternal depressive symptoms is below -0.1 with a 0.81 probability. The posterior distribution of the total coefficient for maternal depressive symptoms (obtained by elementwise addition of the posterior samples for the two coefficients) shows evidence for no strong association between maternal depressive symptoms and parent-adolescent conflict, with a 0.92 probability of a coefficient between -0.1 and 0.1. Both the regression coefficient for parent gender and the intercept (necessary in a model with a dummy-code, to estimate the effect of belonging to the reference category, in this case a father-adolescent relationship), is estimated very close to 0, implying that there are probably no large differences in reported conflict level between mother-adolescent dyads and father-adolescent dyads as groups. The coefficient for adolescent age is also very close to 0 in both models.

Table 4 Regression model parameter estimates

Parameters	Mean	SD	93% CI	ESS	R̂
Interpersonal problems model					
Agency	0.19	0.07	0.07; 0.31	3768	1
Communion	0.02	0.06	−0.08; 0.12	5126	1
Elevation	0.08	0.07	−0.04; 0.21	6509	1
Adolescent age	−0.03	0.03	−0.09; 0.04	7081	1
Variance of errors	0.27	0.06	0.18; 0.39	1016	1
Variance of random effects	0.21	0.07	0.05; 0.34	1214	1
Degrees of freedom in t-likelihood	21.88	14.15	4.84; 53.70	13,611	1
Parental depressive symptoms model					
Intercept	−0.03	0.12	−0.24; 0.18	840	1
Depressive symptoms	−0.16	0.07	−0.30; −0.04	4719	1
Depressive symptoms × mother	0.16	0.09	0; 0.33	5495	1
Mother	0.02	0.08	−0.12; 0.17	9331	1
Adolescent age	−0.03	0.03	−0.10; 0.03	8449	1
Variance of errors	0.30	0.06	0.20; 0.41	1639	1
Variance of random effects	0.16	0.08	0.02; 0.30	1610	1
Degrees of freedom in t-likelihood	20.8	13.85	4.57; 51.59	14,430	1

Mean = Posterior mean; SD = Posterior standard deviation; 93% CI = 3.5th and 96.5th percentiles of the posterior distribution; ESS = Effective Sample Size, refers to the effective number of samples from the posterior distribution; R̂ = Gelman-Rubin Statistic, indicates convergence of HMC chains at 1

In summary, there are two main findings: Adolescent reported conflict is predicted to be higher when parents report more problems than average related to being too interpersonally domineering, and lower when parents report more problems than average being interpersonally submissive, and this applies regardless of parent gender. Given the model weighting, this interpersonal tendency has higher predictive utility than parental depressive symptoms. Conflict is also predicted to be higher with fathers who report less than average depressive symptoms, and lower when fathers report more depressive symptoms, while the depressive symptoms of mothers do not appear to be strongly associated with parent-adolescent conflict.

Discussion

The aim of our analysis was to evaluate to what extent the interpersonal problems reported by parents are associated with parent-adolescent conflict reported by their depressed adolescent children, and whether these associations varied across the interpersonal circumplex. We also wanted to assess whether any such associations have incremental predictive value compared with the expected association between parental depression and parent-adolescent conflict that has been found in previous research in related populations [10, 19–21]. Our results indicate that parental agency-related interpersonal problems are associated with parent-adolescent conflict, and that parent interpersonal problems does add predictive value.

Parent-Adolescent Conflict is Associated with Parental Agency-Related Problems

Our results suggest an association between interpersonal problems on the agency-dimension and parent-adolescent conflict. In childhood and early adolescence, resolution of parent-adolescent conflict is mainly by parental power assertion, or reciprocal withdrawal [59]. It has been suggested that conflicts and renegotiation of interaction patterns for conflict resolution is an important mechanism of change in parent-adolescent relationships [15]. Finding parental problems with being too dominant and assertive to be related to increased parent-adolescent conflict is consistent with this view. The Agency variable of the IIP-C indexes difficulties in assuming an interpersonally submissive or dominant position when needed [37]. Parents scoring high on the Agency variable would be expected to struggle with accepting and encouraging age-appropriate adolescent autonomy, and to find the normative transition to increasing interpersonal equality in parent–child conflicts [60], to be particularly challenging. It is worth noting that parents scoring in the negative range on the Agency variable are also reporting above average levels of interpersonal problems, but their problems concern being too submissive and unassertive. These are predicted to have lower than average levels of parent-adolescent conflict, and the model appears to fit equally well across the range of the Agency variable. This means that parental report of more severe difficulties with an unassertive interpersonal style is associated with

lower levels of parent-adolescent conflict. While this is not theoretically surprising in itself it demonstrates an important point: If interpersonal difficulties are not differentiated in measurement and modelling, it may obscure specific associations between different interpersonal processes and different dimensions of interpersonal difficulties, such as those described by the interpersonal circumplex. Though they are found to have less conflicts with their depressed adolescent, it is entirely possible that these parents find other aspects of the parent-adolescent relationship, such as limit-setting, more difficult than parents reporting less such problems.

It is also notable that problems relating to preoccupation with closeness and care, or with being withdrawn and detached, do not appear to be strongly related to the level of parent-adolescent conflict. This suggests that the way in which parents respond to the developing autonomy of the adolescent may be more important for the level of parent-adolescent conflict than how they handle closeness and warmth in the parent-adolescent relationship. Still, parental interpersonal problems on the communion dimension may very well be associated with other difficulties in the parent-adolescent relationship that were not assessed in this study.

Paternal but not Maternal Depressive Symptoms are Associated with Less Conflict

Not finding parental depressive symptoms to be positively associated with parent-adolescent conflict was surprising, given the literature supporting this association, for both parent genders [e. g. 20, 61]. However, there are other discrepant findings in the literature, such as a longitudinal study of an at-risk sample which did not find parental depression to predict conflict trajectory membership [62], and a longitudinal study of mother-adolescent conflict interactions where maternal internalising symptoms was not associated with maternal conflict behaviour [63]. Any explanation for this unexpected finding will nevertheless be speculative. It might be due to differences in measurement and operationalisation of parent-adolescent conflict. In a meta-analysis of the association between paternal depression, father–child conflict and child psychopathology, larger effect sizes were found to be associated with community samples and parent-reported measures of parenting behaviours [20]. A second possibility is discontinuity of the association across populations and contexts, with the dynamics of parental depression and parent-adolescent relationships changing when adolescents themselves develop a depressive disorder. Lastly, although a positive association for both parent genders is quite improbable given these data and the model, improbable is still not impossible, and the sample may simply be unrepresentative.

Strengths, Limitations and Recommendations for Future Research

This study has several limitations. The sample size is small, but this was somewhat mitigated by making the individual parent the unit of analysis in a multilevel model, and then fitting and comparing models where all predictors interacted with parent gender. As the number of male adolescents in the sample is minimal, replication is necessary to generalise the findings to depressed adolescent males. Further, the study design is cross-sectional. A longitudinal design would have allowed for stronger inferences concerning the direction of effects. However, as the agency and communion factors of the IIP-C has considerable temporal stability [64], and concerns how the respondent perceives their interpersonal functioning across relationships, a strong influence on this measure by current conflict with their depressed adolescent is less plausible.

The study is strengthened by clinical assessment of a major depression diagnosis, by not relying on a single informant, having a large proportion of participating fathers and employing powerful and modern modelling and estimation techniques.

These findings add to the literature by demonstrating how parental interpersonal dispositions are related to parent-adolescent conflict in adolescent depression. They demonstrate the utility of interpersonal theory and the IIP-family of measures for studies of conflict processes in adolescent depression. While not carrying the weight of evidence necessary for any clinical recommendation, we would suggest future studies on conflict processes in adolescence consider including an IIP measure such as the brief IIP-C-IRT [65] as a theoretically rich and differentiated measure of parent and adolescent interpersonal styles.

Summary

Parent-adolescent conflict is common among depressed adolescents and their parents. High levels of parent-adolescent conflict can interfere with treatment and may increase risk of recurrence. Parental depressive symptoms have been shown to predict conflict with adolescent children, but as management of conflicts is inevitably an interpersonal situation, parental difficulties in interpersonal functioning could also play an important role. Interpersonal theory suggests that variation in interpersonal difficulties have two main dimensions, termed agency and communion. The present study compared these dimensions of parental interpersonal problems to parental depressive symptoms as predictors of adolescent-reported parent-adolescent conflict, in a sample of 100 parents of 60 adolescents with a Major depressive disorder (92% female). We employed Bayesian multilevel

modelling, leave-one-out cross-validation and model stacking to compare and weight different models. These were models predicting parent-adolescent conflict from parental depressive symptoms and from parental interpersonal problems, with and without interactions with parent gender. Results suggest that including parental interpersonal problems contributes substantially to accurate predictions of parent-adolescent conflict, and that these associations do not depend on parent gender. When parents reported more interpersonal problems related to excessive dominance or submissiveness, adolescent report of conflict tended to be higher or lower, respectively. Parental interpersonal difficulties related to the communion dimension was not associated with parent-adolescent conflict. Parental depressive symptoms were found to be negatively associated with parent-adolescent conflict in father-adolescent relationships only. These findings support the view that parental difficulties in negotiating the normative transition to a less hierarchical parent–child relationship may be related to heightened parent-adolescent conflict in adolescent depression. The study is limited by a small sample size and low number of male adolescents. Future studies on parent-adolescent conflict should consider using the interpersonal circumplex and related measures, as a theoretically rich and differentiated model of parent and adolescent interpersonal styles.

Acknowledgements Open Access funding provided by Oslo University & Oslo University Hospital.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments. The study protocol, participant information letters and consent forms were reviewed and approved by the Regional Committee for Medical and Health Research Ethics for Eastern Norway (REK Øst).

Informed Consent Informed consent was obtained from all individual participants included in the study, or their legal guardians, in which case assent to be included in the study was obtained from the underage participant.

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Paper 3

Using Informant Discrepancies in Report of Parent-adolescent Conflict to Predict Hopelessness in Adolescent Depression

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Abstract

Hopelessness is an important symptom of adolescent depression, being associated with both risk of suicide and poor treatment response, but predictors of hopelessness are understudied. Conflict with parents is common in adolescent depression, but parents and adolescents often disagree when reporting conflict severity. Discrepancy in reporting may be an indicator of the parent-adolescent dyad lacking a shared representation of the state of their relationship. This could make conflicts seem unresolvable to the adolescent, leading to expectations of persistent stress and lack of support, increasing hopelessness. This study employed latent difference scores, ordinal regression and cross-validation to evaluate the hypothesis that discrepancy in report of parent-adolescent conflict would predict hopelessness among depressed adolescents. Parents reporting less conflict than the adolescent was associated with increased adolescent hopelessness, giving preliminary support to the hypothesis.

Hopelessness is a state where a person expects negative events to occur, feels important goals cannot be achieved, and that they are powerless to improve their future, either by their own agency or by the help of others (Marchetti, 2018). As a symptom of adolescent depression, hopelessness is particularly important, as it is a well-established predictor of adolescent suicidal behaviour (Wolfe et al., 2019), and has been shown to predict poor treatment response (Emslie, Kennard, & Mayes, 2011). There is also some evidence that hopelessness is implicated in the development of depression (Alford, Lester, Patel, Buchanan, & Giunta, 1995).

Predictors of Hopelessness

Not all depressed adolescents experience hopelessness (Yorbik, Birmaher, Axelson, Williamson, & Ryan, 2004), and very few studies has previously investigated predictors of hopelessness in clinical samples. Kashani, Canfield, Borduin, Soltys, and Reid (1994) found a lack of supportive relationships to predict hopelessness in a sample of preadolescent inpatients with various diagnoses. Becker-Weidman et al. (2009) modelled both cognitive and socio-environmental variables as possible predictors of hopelessness in a large clinical sample of adolescents with a diagnosed depressive disorder. They found cognitive distortions in view of self and the world, an internal attributional style and need for social approval, as well as family conflict, to predict hopelessness.

Predictors of adolescent hopelessness as a dimensional construct have also been studied in community samples. Studies in disadvantaged samples have suggested that hopelessness is related to family dysfunction and weak social support networks, as well as to exposure to traumatic events (Bolland, Lian, & Formichella, 2005; Duyan, 2016; Perez-Smith, Spirito, & Boergers, 2002). A longitudinal study of an at-risk sample found that maternal parenting style predicted development of cognitive vulnerabilities for hopelessness,

which showed an interaction with negative life events in predicting development of hopelessness (Garber & Flynn, 2001).

The reviewed literature indicates that difficulties in the parent-adolescent relationship may be related to hopelessness in adolescents, besides cognitive variables relatively conceptually close to the phenomenon of hopelessness itself. In addition to mediation by development of cognitive vulnerabilities, as suggested by the work of Garber and Flynn (2001), difficulties in the parent-adolescent relationship could increase the risk of hopelessness through other pathways as well. Parent-adolescent relationships are obligatory, not voluntary relationships (Laursen & Collins, 2009), and distressing aspects of the relationship may hence be experienced as inescapable. The ability to mentally represent increasingly concrete long-term goals for an adult life also develops in adolescence, and the transitional process to adulthood make these goals highly salient (Nurmi, 1991). At the same time, the adolescent is still dependent on practical and emotional support from their caregivers to be able to approach these goals (Laursen & Collins, 2009). This makes adolescence a developmental period where a positive future is more acutely felt to be dependent on supportive close relationships.

Parent-adolescent Conflict and Discrepancies in Reporting

Conflict between parents and adolescents is common and to some extent normative (Laursen & Collins, 2009), but the form of conflict resolution achieved and the conflict behaviours that parents and adolescents engage in are systematically related to adolescent adjustment. Repetitive conflict interactions that lead to withdrawal rather than resolution, lack of negotiation and aggressive conflict tactics place adolescents at risk (Branje, van Doorn, van der Valk, & Meeus, 2009). Between depressed adolescents and their parents, such dysfunctional forms of conflict are more frequent (Bodner, Kuppens, Allen, Sheeber, & Ceulemans, 2018; Sheeber, Davis, Leve, Hops, & Tildesley, 2007), and a negative predictor

of depressive disorder course as well as treatment outcome (Alaie, Laftman, Jonsson, & Bohman, 2019; Asarnow et al., 2009; Feeny et al., 2009).

Discrepancies between the reports of different informants are common when measuring the level of parent-adolescent conflict, as is usual in multi-informant assessment (De Los Reyes, Thomas, Goodman, & Kundey, 2013). Such discrepancies can be due to differences in access to information about what is reported on, and merely indicate that the phenomenon varies across the contexts in which the informants observe or experience it. This is not likely to be the case with parent-adolescent conflict, where the context is necessarily shared between informants, and neither parents nor adolescents can be regarded as an informant with access to objective information (De Los Reyes et al., 2013). Rather, their reports reflect their individual representation of the conflict state of the relationship, which is built on how they have perceived and interpreted previous conflict situations and how these have been distributed in time (Adams & Laursen, 2001). Informant discrepancies in reports of conflict may therefore represent information about something subtly different than conflict itself, by indicating to what extent the parent-adolescent dyad lack a shared representation of the current state of their relationship. A number of studies have found informant discrepancies to be related to adjustment in children and adolescents (e. g. Nelemans et al., 2016; Ohannessian, Lerner, Lerner, & von Eye, 2016; Van Heel et al., 2019), supporting the study of informant discrepancies as a variable in itself.

The Present Study

Becker-Weidman and colleagues (2009) noted how adolescent report of conflict, but not parental report, was associated with hopelessness in their sample. Arguing for further research on the specific role of family conflict in development and maintenance of hopelessness, they also recommended investigating how other family members share the adolescents' perception of their family. Conflict that is unacknowledged by parents may

appear unresolvable to the adolescent, giving an expectation of uncontrollable, persistent stress and lack of social support, leading to hopelessness. Discrepancies in report of conflict is one way of operationalising this relational state between adolescents and their parents. This led us to hypothesize that discrepancy between adolescent and parent report of conflict would predict hopelessness among depressed adolescents more than the absolute level of conflict. To evaluate this hypothesis, we compare models predicting hopelessness from the conflict reports of single or multiple informants to a model predicting hopelessness from the level of informant discrepancy.

Methods

Participants

We collected data as part of baseline assessments for a randomized controlled trial (clinicaltrials.gov identifier NCT01830088). Participating families were recruited among adolescents referred to two Child and Adolescent Mental Health Services (CAMHS) in South-eastern Norway. During pre-specified recruitment periods, referral letters for adolescents (13 - 17 years) were examined for mentions of depression or core depressive symptoms (depressed mood, anhedonia, or fatigue). The CAMHS routinely administered the Youth Self Report (Achenbach, 1991), and these were screened for raw scores on the Affective Problems subscale above 6 to find depressed adolescents not identified as such in their referral letters (Eimecke, Remschmidt, & Mattejat, 2011). Eligible adolescents or their parents, depending on adolescent age, were contacted by telephone and invited to participate in a randomized trial of family therapy for adolescent depression. 276 patients were contacted. Participants were required to be currently living with an adult who had become a caregiver for them before age four, and willing to have this adult participate in treatment. Interested adolescents meeting these criteria were screened with Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) over telephone and invited for an assessment session if they

scored above 17, a threshold expected to maximize sensitivity (Dolle et al., 2012). 160 of the 276 contacted were screened with the BDI-II and 100 of these met with study personnel for a clinical assessment. Adolescents were included in the study if they scored above 15 on the Grid Hamilton Depression Rating scale (GRID-HAMD, Williams et al., 2008) and met Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association, 2000) criteria for a current major depressive episode assessed with the Schedule for Affective Disorders and Schizophrenia for School-age Children - Present and Lifetime Version (K-SADS-PL, Kaufman et al., 1997). Exclusion criteria were psychotic disorders, eating disorders, bipolar disorder, intellectual disability or pervasive developmental disorders. One family withdrew consent after assessment. In all 60 adolescents were included (55 female, 5 male), with 43 fathers and 57 mothers participating.

Procedures

Eligible adolescents and their parents met with a study-affiliated clinical psychologist at the CAMHS for an assessment. Parents and adolescents aged 16 or above gave written informed consent to be included in the study, and adolescents below age 16 gave their assent to be included. Adolescents and parents were then interviewed separately. All interviews were video-recorded. Both parents and adolescents completed self-report measures during the appointment.

Ethical approval

All procedures performed were in accordance with the 1964 Helsinki declaration and its later amendments. The study protocol, participant information letters and consent forms were reviewed and approved by the Regional Committee for Medical and Health Research Ethics for Eastern Norway (REK Øst).

Measures

Hopelessness

There is support for hopelessness as a unidimensional construct (Aish & Wasserman, 2001). We operationalized adolescent hopelessness as the clinician rated hopelessness item in the follow-up interview for Depressive disorders in the K-SADS, scored after interviewing both the adolescent and the parents (Kaufman et al., 1997). This item is scored from 1 to 3, with 1 indicating the absence of hopelessness, 2 indicating a subclinical degree of hopelessness, and 3 indicating a clinical degree of depressive hopelessness. The interrater reliability of the scores was assessed by blinded re-scoring of a random sample of 20 interviews. We estimated the intraclass correlation coefficient for the Hopelessness item following the Bayesian approach of Gajewski, Hart, Bergquist-Beringer, and Dunton (2007). The posterior mean intraclass correlation coefficient was 0.79 (66% and 90% Highest Density Intervals 0.72 – 0.96 and 0.63 – 1)[1] indicating acceptable reliability. Computational details are in the supplementary material.

Parent-adolescent Conflict

Parent-adolescent conflict was assessed with the Perception of the Dyad subscale of the Conflict Behavior Questionnaire (CBQ, Prinz, Foster, Kent, & O'Leary, 1979). The CBQ has seen wide use as a multi-informant measure of parent-adolescent conflict among depressed adolescents (e. g. Curry et al., 2006; Sheeber & Sorensen, 1998), and the Perception of the Dyad subscale was among the candidate predictors of hopelessness investigated by Becker-Weidman et al. (2009). Items were translated to Norwegian, and the original author approved a blind reverse translation. Parents completed the measure for their

[1] We generally choose to report the 66% and 90% Highest Density Intervals (see Kruschke, 2018), with the suggested interpretation of the likely and very likely range within which the true parameter value lies (Mastrandrea et al., 2010).

relationship to the adolescent, and the adolescent completed the measure separately for each participating parent.

Analysis plan

All modelling was conducted in the programming language Stan with the RStan interface (version 2.19.2, Stan Development Team, 2019a) for R (version 3.6.1, R Core Team, 2019). Stan allows for Bayesian inference with estimation by Hamiltonian Monte Carlo (HMC), works well with high-dimensional models, and has sensitive diagnostics for biased or unreliable estimation.

Modelling informant discrepancies

Informant discrepancies can be modelled using Latent Difference Scores (de Haan, Prinzie, Sentse, & Jongerling, 2018). Latent Difference Scores are obtained by fitting a latent variable model to the responses of both informants and constraining the latent trait variable of one informant to be equal to the sum of a freely estimated latent difference variable and the latent trait variable of the other informant. We implemented this by specifying a two-parameter logistic item response model (IRT model) to the four sets of parent and adolescent responses to the Perception of the Dyad subscale of the CBQ. In the IRT model we specified the latent trait parameter of each parent to be equal to the sum of the latent trait parameter of the adolescent reporting about that parent and a latent difference score parameter. The latent traits of the adolescent and the latent difference scores were specified to have a bivariate normal distribution, with variances constrained to 1, the latent trait mean constrained to 0, and the mean of the latent difference scores and the covariance as estimated parameters. Figure 1 illustrates the structure of the latent discrepancy score model.

FIGURE 1 HERE

Latent discrepancy score modelling assumes measurement invariance across the kinds of respondents whose discrepant reports are to be quantified (de Haan et al., 2018). The latent

trait estimates from an IRT model has measurement invariance across different kinds of respondents if the model fits equally well to all of them (Verhagen & Fox, 2013). To evaluate whether the assumption of measurement invariance was satisfied, we fitted models with item parameters freely estimated for mothers, fathers, and for adolescent report on mother and father separately, and compared these to the fit of models with item parameters constrained to be equal for all four kinds of respondent, following the procedure described by Verhagen and Fox (2013). To compare these models we used approximate leave-one-out cross-validation (PSIS-LOO, Vehtari, Gelman, & Gabry, 2017). This a computationally efficient way of estimating the expected fit of a model to future data, similarly to the Deviance Information Criterion used by Verhagen and Fox (2013), but which also has the advantage of sensitive diagnostics for when estimates are likely to be biased (Vehtari et al., 2017). Item-wise PSIS-LOO showed that the models with equal item parameters had better fit with the exception of two items. These were the reverse scored "2: We do a lot of things together." and "4: I enjoy the talks we have.". These were omitted when estimating the latent difference scores. Visual inspection of the posterior distributions of item characteristic curves plotted against the data indicated good fit for the remaining items. These plots are available in the supplementary material.

Modelling predictors of hopelessness and evaluating model fit

To estimate the associations between the latent conflict traits or latent difference scores and the ordinal hopelessness variable, we used ordinal probit regression (Kruschke, 2015). We specified four different models, regressing the ordinal distribution of hopelessness scores on the estimated latent trait of the adolescent (adolescent report), the estimated latent trait of the parent (parent report), both latent traits (multi-informant report), and the estimated latent difference scores (informant discrepancy). We fitted each of these four models simultaneously to both the mother-adolescent data and the father-adolescent data, in order to

share item parameters for the IRT models. We compared these four models using approximate leave-one-out cross-validation (PSIS-LOO). Estimates of the leave-one-out predictive density from PSIS-LOO can also be used for stacking of predictive distributions (Yao, Vehtari, Simpson, & Gelman, 2018). This is a procedure that given a set of models finds the weighted combination of models that has a predictive distribution that is closest to the data generating process, allowing model weights to be interpreted as the contribution to predictive accuracy gained from each model if these were combined as one single model (Yao et al., 2018).

Prior distributions, estimation and validation of convergence

In Bayesian data analysis, prior distributions must be specified for all model parameters, representing our assumptions and knowledge about the parameters irrespective of the data (Gelman et al., 2013). The weakly informative prior distributions used in this analysis and the reasoning behind choosing them can be found in the supplementary material, and we encourage the reader to review them and consider whether they are reasonable.

We estimated all models running four Markov chains with the standard algorithm, with 1000 warmup iterations and drawing 2500 samples from each chain (Stan Development Team, 2019b). Rubin-Gelman statistics were below 1.01 for all parameters, and there were no negative Stan convergence diagnostics, indicating valid sampling from the posterior distribution.

Results

Sample characteristics and latent variable distributions

The distributions of K-SADS Hopelessness scores (1/2/3) were 11/17/23 for adolescents with mothers reporting and 10/11/21 for the adolescents with fathers reporting. The posterior means of the latent trait for adolescent report of conflict had a range of -1.48 to 2.53 for father-adolescent conflict and -1.39 to 1.53 for mother-adolescent conflict.

The posterior means of the latent difference scores had a range of -1.42 to 1.56 for father-adolescent conflict, and -1.75 to 1.73 for mother-adolescent conflict. The correlation of the latent difference scores and the latent traits had a posterior mean of -0.34 (SD 0.19) for father-adolescent conflict and a posterior mean of -0.33 (SD 0.19) for mother-adolescent conflict.

Cross-validation and stacking of models

We then used the R-package loo (Vehtari, Gabry, Yao, & Gelman, 2019), to compare models using PSIS-LOO. We also calculated model stacking weights (Yao et al., 2018). Results are displayed in table 1.

TABLE 1 HERE

For both the mother-adolescent and the father-adolescent data, the models with latent difference scores as the only independent variable are better than the models with the reports of multiple informants. The differences in predictive accuracy to the models with single informants is within the standard error of the estimate. The stacking procedure does however give most weight to the model with latent difference scores as the only independent variable, for both mothers and fathers, and some weight to the models with adolescent report as the independent variable. This means that once informant discrepancies are taken into account, there is not much predictive accuracy to gain from information about the absolute level of conflict reported by the adolescent, and when both of these are taken into account, there is nothing to gain from the information reported by the parents.

Regression model parameter estimates and model predictive distributions

The parameter estimates of the fitted regression models are summarised in table 2. The full sets of samples drawn from the posterior distributions of all four models, and the Stan model code are available at [DOI withheld for blinding].

TABLE 2 HERE

The 90% and 66% HDIs are quite wide, showing the considerable uncertainty in the estimates. If we reason that a standardised regression coefficient between -0.1 and 0.1 is practically close enough to 0 to be of little theoretical interest in this case, we can use the posterior distribution to calculate the probability of a regression coefficient that is negative and of a larger magnitude than -0.1 (Kruschke, 2018). For the model with informant discrepancies these probabilities are .74 for mother-adolescent conflict and .85 for father-adolescent conflict. There seems to be a difference in the magnitude of the association between mothers and fathers, but the posterior distributions of the regression coefficients overlap considerably. The mean posterior difference between the mother-adolescent regression coefficient and the father-adolescent regression coefficient is 0.18, with a standard deviation of 0.40. The probability of the father-adolescent regression coefficient having a larger negative magnitude than the mother-adolescent regression coefficient is 0.67 – probable, but far from certain.

Regression coefficient estimates can be difficult to interpret directly, in particular in ordinal regression, and visualisation of the predictive distribution of the model can be helpful to see the implications of a model fit. Choosing some values for the independent variables, we can make repeated draws from the predicted distributions of the dependent variable at those levels of the independent variable, with the drawn distributions of dependent variable values containing the uncertainty of the model fit.

In figure 2, we have plotted the distributions of hopelessness values predicted by the stacked models (combining draws according to the stacking weights) for both father-adolescent conflict and mother-adolescent conflict, at different levels of informant discrepancy, holding adolescent report of conflict constant at the mean.

FIGURE 2 HERE

With the uncertainty in the model fit preserved in the plotted distributions, the main weight of the evidence is still on the frequency of clinically significant hopelessness increasing when parents report less conflict than the adolescent, in particular for father-adolescent informant discrepancy.

Discussion

Given the clinical importance of hopelessness in adolescent depression, it is unfortunate that predictors of adolescent hopelessness have received relatively little research attention. In this study we found preliminary support for our hypothesis that parent-adolescent informant discrepancy in report of conflict would be associated with hopelessness among depressed adolescents. The present findings suggest that informant discrepancies capture information about some way parent-adolescent dyads differ that is distinct from the level of conflict, and which is related to adolescent hopelessness.

We found evidence for a relationship between hopelessness and parents reporting less conflict than the adolescent. The same pattern of discrepancy, but concerning family routines and chaos rather than conflict, was found to predict development of depressive symptoms in a longitudinal study of a community sample (Human, Dirks, DeLongis, & Chen, 2016). Although speculative at present, it is possible that informant discrepancies, across different variables, all indicate similar negative family processes. Such family processes could be difficult to assess accurately using self-report, in which case informant discrepancies would have potential for clinical assessment. Further research is needed to evaluate whether there are common family processes that predict informant discrepancies, and for what classes of variables. As noted by De Los Reyes et al. (2013), the meaning of informant discrepancies will differ when what is reported on is part of a context shared by the informants, and when it is not. It is likely that discrepancies due to different access to information is less indicative of

negative family processes than mismatched perceptions of shared contexts like family routines, family chaos and parent-adolescent conflict.

Another recent longitudinal study of a community sample found discrepancy in reports of negative interactions between fathers and adolescents, but not mothers, to predict development of depressive symptoms (Nelemans et al., 2016). This is in line with our finding that the association might be stronger for the father-adolescent relationship.

Unlike Becker-Weidman and colleagues (2009), we did not find that adolescent report of conflict was strongly associated with hopelessness in itself. However, there are several differences in statistical analysis and measurement methodology between these studies that make direct comparisons difficult.

Limitations

This study is limited by a small sample size, which is reflected in the uncertainty of the posterior estimates. Applying Bayesian data analysis is an advantage in such cases, as the uncertainty is preserved and visible in the results, and inference does not rely on asymptotic properties of the sampling distribution. Careful attention to choice of priors and validation of convergence is crucial in such cases (McNeish, 2016), and this has been observed in the present analysis. Considering the sample size and the uncertainty of the posterior, we view the present findings as an interesting lead, deserving attempted replication. Replication in a sample with a larger proportion of male adolescents would help clarify whether such an association is gender specific, as the number of male adolescents in this sample was low. The cross-sectional design also limits the causal inferences that may be drawn. It cannot be ruled out that hopelessness is related to adolescents overestimating the level of conflict relative to the parents (i.e. depressive distortion, De Los Reyes et al., 2013), although the low probability of a strong positive association between adolescent report of conflict and hopelessness does make this interpretation less reasonable.

Conclusion

That informant discrepancies appear to have a stronger association with hopelessness than adolescent report alone, is a reminder of why multi-informant assessments are vital in the study of relational phenomena. Relying on a single informant or on analysing the reports of multiple informants separately can probably conceal or misrepresent associations, as would have been the case if we had only analysed adolescent report of conflict. If replication should support these preliminary findings, it would imply that clinicians working with depressed adolescents and their families need to be attentive not only to conflict in the family, but also to differences in the perception of conflict. When adolescents find parent-adolescent conflict more severe than their parents, it seems to indicate different family processes than heightened conflict alone, and this may have implications for intervention.

These results further demonstrates how latent difference scores (de Haan et al., 2018) can be combined with item response theory for studying informant discrepancies, and how Stan (Stan Development Team, 2019b) is a powerful and flexible computational framework for such analyses. Change in informant discrepancy should also be considered for inclusion as a mediator variable treatment studies, in particular those involving family-oriented interventions.

Funding

Data collection was partially funded by the South-Eastern Norway Regional Health Authority (grant 2013008).

Declaration of Conflicting Interests

The Authors declare that there is no conflict of interest.

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Table 1: Comparison of Models with PSIS-LOO and Stacking

Model	Difference (SE)	P-loo (SE)	Stacking weight
Conflict with Mother			
Informant discrepancy	-	4.5 (0.4)	0.88
Adolescent report	-0.5 (1.2)	3.4 (0.4)	0.12
Parent report	-0.6 (0.9)	3.6 (0.4)	0
Multi-informant report	-1.1 (0.3)	5.7 (0.6)	0
Conflict with Father			
Informant discrepancy	-	6.1 (0.6)	0.85
Adolescent report	-0.8 (1.6)	3.5 (0.4)	0.15
Parent report	-0.8 (1.5)	3.8 (0.5)	0
Multi-informant report	-1.1 (0.3)	7.2 (0.8)	0

Table note: Difference (SE) = Difference to model with highest estimated predictive accuracy; P-loo (SE) = Effective number of parameters, an estimate of model complexity; Weight = Model weight from the stacking procedure.

Table 2: Regression parameter estimates from informant discrepancy and adolescent report models

Parameter	Mean	SD	Median	66% HDI	90% HDI	ESS
Informant discrepancy - mother						
Regression coefficient	-0.25	0.23	-0.24	-0.43 ; -0.01	-0.60 ; 0.13	9087
First Cutpoint	-0.88	0.20	-0.87	-1.07 ; -0.70	-1.19 ; -0.55	16555
Second Cutpoint	0.05	0.17	0.05	-0.12 ; 0.21	-0.25 ; 0.32	11270
Adolescent report - mother						
Regression coefficient	0.08	0.19	0.08	-0.10 ; 0.25	-0.23 ; 0.38	13228
First Cutpoint	-0.87	0.20	-0.86	-1.04 ; -0.66	-1.19 ; -0.55	14336
Second Cutpoint	0.03	0.17	0.03	-0.13 ; 0.20	-0.26 ; 0.31	13092
Informant discrepancy - father						
Regression coefficient	-0.42	0.33	-0.4	-0.68 ; -0.08	-0.96 ; 0.10	7243
First Cutpoint	-0.73	0.23	-0.73	-0.95 ; -0.51	-1.12 ; -0.37	15468
Second Cutpoint	0.13	0.21	0.12	-0.08 ; 0.32	-0.22 ; 0.48	11508
Adolescent report - father						
Regression coefficient	0.06	0.19	0.06	-0.13 ; 0.23	-0.25 ; 0.37	12709
First Cutpoint	-0.76	0.21	-0.75	-0.93 ; -0.53	-1.09 ; -0.4	15386
Second Cutpoint	0.04	0.19	0.04	-0.14 ; 0.22	-0.26 ; 0.37	13631

Table note: Mean = Posterior mean, SD = Posterior Standard deviation, Median = Posterior median, 66% and 90% HDI = The 66% or 90% Highest Density Interval, ESS = Effective Sample Size, the estimated number of effectively independent draws from the posterior distribution

Figure 1: Latent discrepancy score model

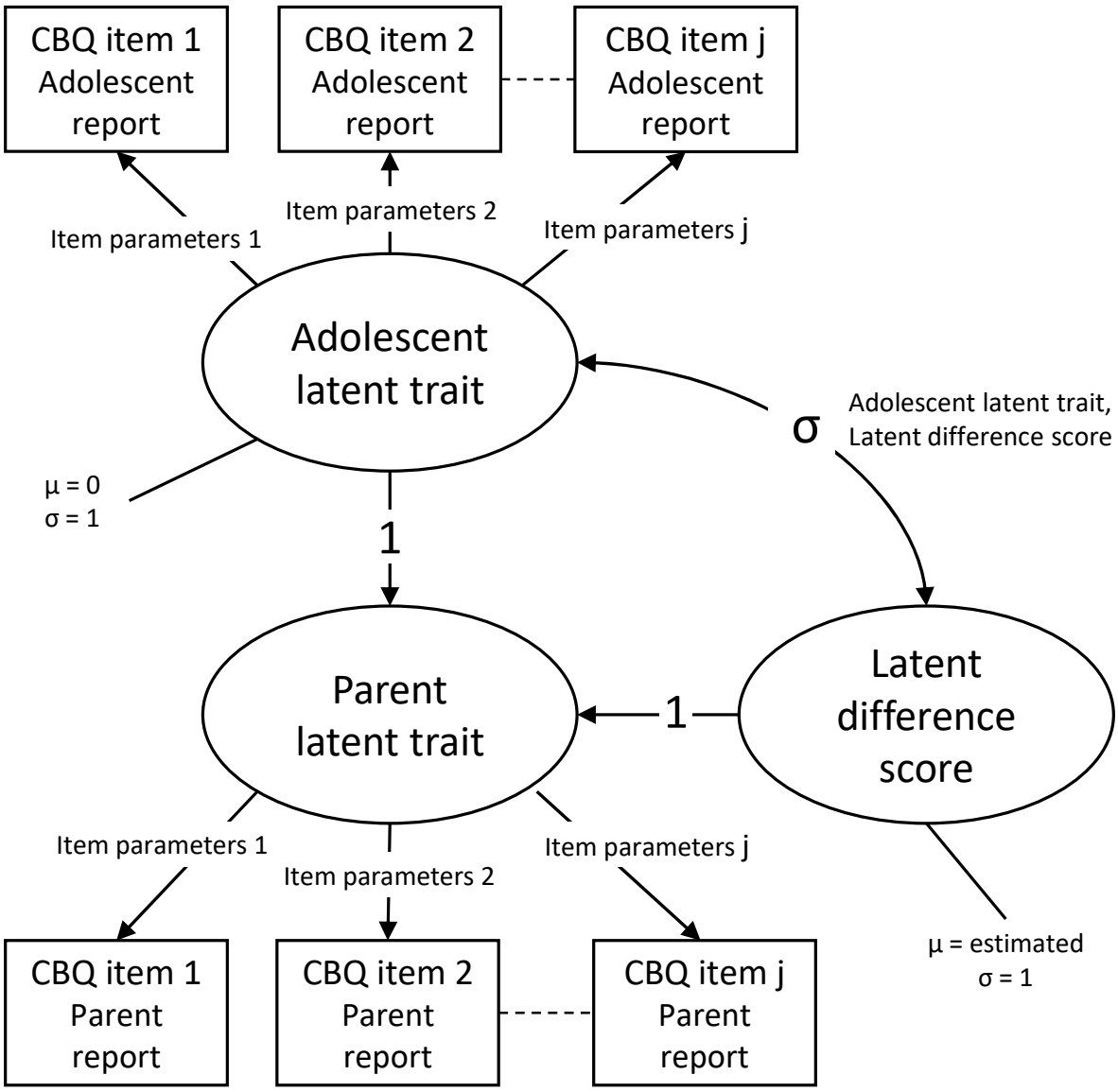
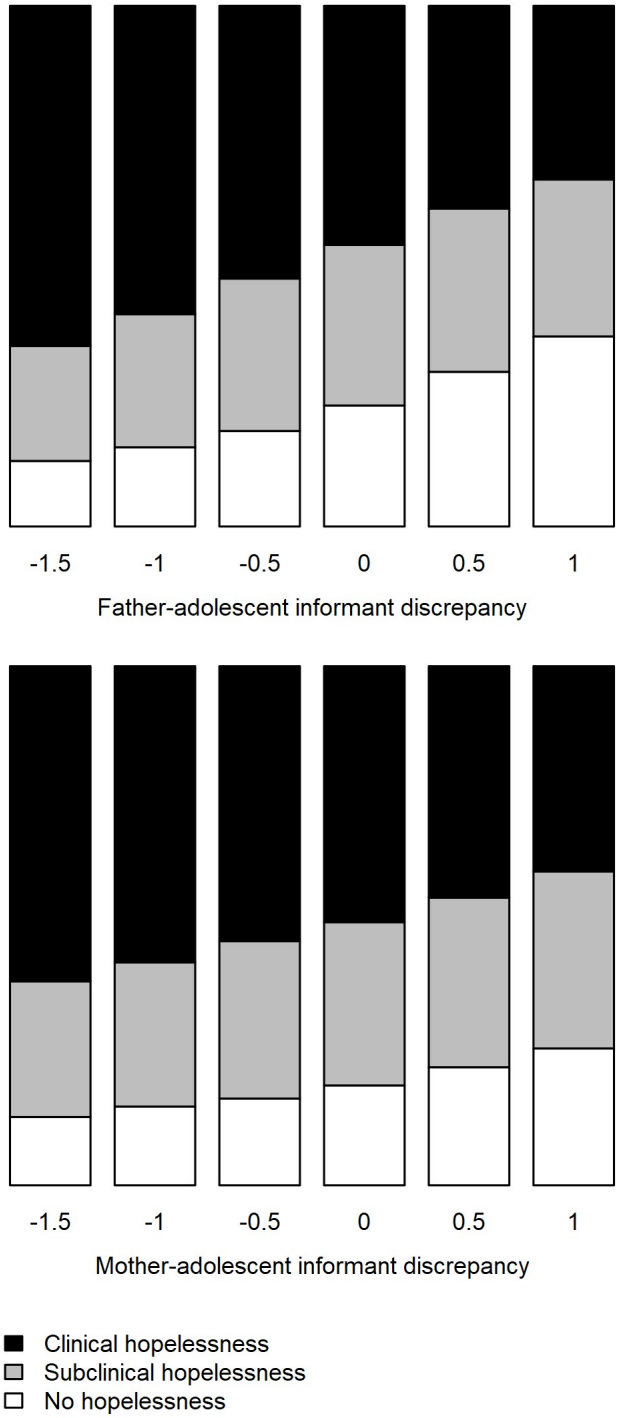


Figure 2: Predicted distributions of hopelessness across informant discrepancy



Appendix 1: Tables of Prior Distributions

Study 1:

Parameter(s)	Prior Distribution	Reasoning
Regression coefficients	Normal (0, 1)	Weakly informative prior as the dependent variable is centred on the median score and scaled by twice the median absolute deviation.
Error variance	Half-student's t (3, 0, 1)	Weakly informative prior on the error variance, putting most of the prior weight on errors between 0 and 1, but with heavy tails allowing for a much higher error variance.
Random intercepts	Hierarchical normal prior, with location 0 and a Half-student's t (3, 0, 1) prior on the scale.	Defines random intercepts as deviations from the intercept of the whole sample, and estimates the variance of the random intercepts from the data, with a weakly informative hyperprior.
Latent variables for CBQ IRT model	Multivariate normal (0, 1) with an LKJ (2) prior on the standardized covariance matrix.	Defines the latent variables for parent-adolescent conflict as four correlated Normal (0,1) variables with a weakly informative prior on the correlation coefficients.
Item thresholds for CBQ IRT model	Hierarchical normal prior with hyperpriors Normal (0, 3) for location and Half-students' t (3, 0 ,1) for scale.	Hierarchical prior with weakly informative hyperpriors, estimating the distribution of item thresholds from the data. Wide hyperprior on the location of the distribution, as the interdependent latent variables are constrained to standard normal.
Item discrimination for CBQ IRT model	Gamma (2, 0.5)	Places most of the prior weight on discrimination between 1 and 5, which is the most probable range for items of an established instrument, but does not rule out higher or lower values.

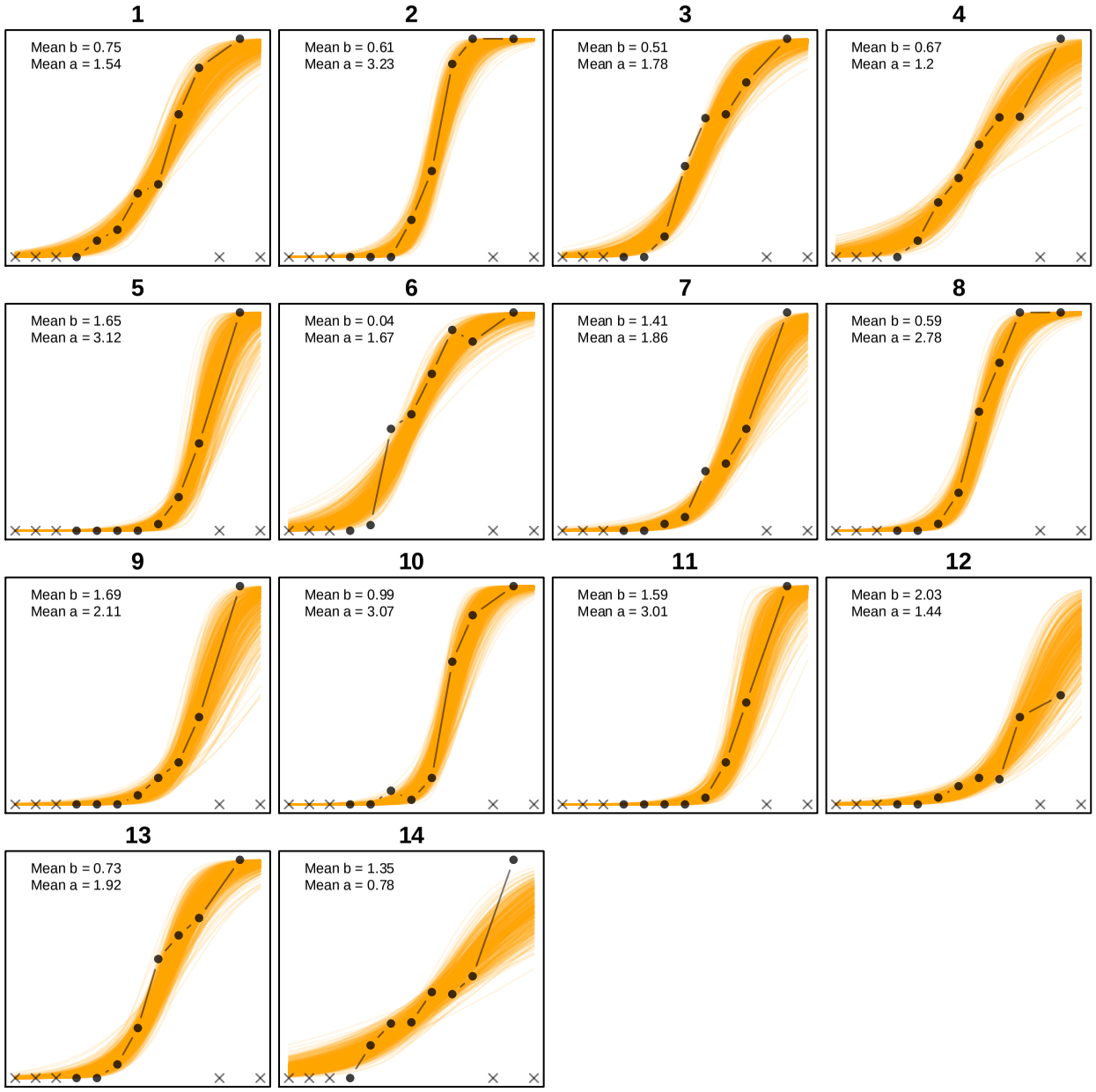
Study 2:

Parameter	Prior Distribution	Reasoning
Random intercepts	Hierarchical normal prior, with location 0 and a Half-student's t (3, 0, 1) hyperprior for scale.	Defines random intercepts as deviations from the sample mean of 0, and estimates the variance of the random intercepts from the data, with a weakly informative hyperprior.
IRT-theta (Conflict level)	Normal (0,1)	Fixes the location and scale of the latent conflict variable for model identifiability, and to ensure a standardized dependent variable for interpretability.
IRT-beta (Item difficulty)	Hierarchical normal prior with hyperpriors Normal (0, 3) for location and Half-student's t (3, 0, 1) for scale.	Weakly informative hierarchical prior, as the interdependent IRT-theta parameter has fixed location and scale.
IRT-alpha (Item discrimination)	Gamma (2, 0.5)	Item discrimination parameters for the CBQ assumed to lie between 0 and 10, as the item characteristic curve does not change meaningfully across alphas larger than 10.
Error variance in regression model	Half-student's t (3, 0, 1)	Regularizing prior on the error variance, which still allows for large estimates if warranted by the data.
Degrees of freedom in Student's t-distributed likelihood	Gamma (2, 0.1) Constrained to be ≥ 1	Degrees of freedom for the likelihood between 1 and about 30, allowing for the likelihood to be very near normal, or have a large degree of robustness, as required.
Regression coefficients	Normal (0, 1)	Regularizing prior on the regression coefficients.

Study 3:

Parameter(s)	Prior Distribution	Reasoning
Regression coefficients	Normal (0, 1)	Weakly informative, given that the latent independent variables are standardized and that large regression coefficients would be very surprising.
Thresholds for probit regression	Normal (0, 3)	Weakly informative, given an approximately standard normal latent variable and two thresholds.
Latent traits and latent difference scores	Multivariate normal with location 0 for latent traits, a normal (0,1) hyperprior on the mean of the latent difference scores, both variances constrained to 1, and a LKJ (2) hyperprior on the correlation matrix.	Defines latent trait as a standard normal variable for model identifiability and interpretability, estimates the mean of latent difference scores, and the correlation between latent difference scores and latent traits, and restricts the latent traits and the latent difference scores to have the same scale.
Item thresholds for CBQ IRT model	Hierarchical normal prior with the hyperpriors Normal (0,3) for location and Half-students' t (3,0,1) for scale.	Hierarchical prior with weakly informative hyperpriors, estimating the distribution of item thresholds from the data. Wide hyperprior on the location of the distribution, as the interdependent latent variables are constrained to standard normal.
Item discrimination for CBQ IRT model	Gamma (2, 0.5)	Places most of the prior weight on discrimination between about 1 and 5, which is the most probable range for items of an established instrument, but does not rule out higher or lower values.

Appendix 2: Conflict Behavior Questionnaire Item Characteristic Curves


1
Mean b = 0.75
Mean a = 1.54
2
Mean b = 0.61
Mean a = 3.23
3
Mean b = 0.51
Mean a = 1.78
4
Mean b = 0.67
Mean a = 1.2
5
Mean b = 1.65
Mean a = 3.12
6
Mean b = 0.04
Mean a = 1.67
7
Mean b = 1.41
Mean a = 1.86
8
Mean b = 0.59
Mean a = 2.78
9
Mean b = 1.69
Mean a = 2.11
10
Mean b = 0.99
Mean a = 3.07
11
Mean b = 1.59
Mean a = 3.01
12
Mean b = 2.03
Mean a = 1.44
13
Mean b = 0.73
Mean a = 1.92
14
Mean b = 1.35
Mean a = 0.78

Appendix 3: Bayesian Computation of the Intraclass Correlation Coefficient for Ordinal Ratings

The Bayesian approach to calculating Intraclass Correlation Coefficients (ICCs) described in the work of Gajweski and colleagues (2007) concern pooling information across ratings at different sites, which was not relevant for our application. However, they develop the idea of using a hierarchical ordinal probit model, with ratings by different raters nested within rated subjects. The observed ratings are taken to indicate a latent normal variable, defined as the sum of subject effects and rater errors. The posterior distribution of the variances of the subject effects and rater errors can then be used to calculate the posterior distribution of the two-way, random effects, single measures ICC (McGraw & Wong, 1996). An advantage of this approach is that it avoids treating ordinal ratings as continuous variables.

The following Stan program implements this approach with a non-centered parametrization. We have specified half-t distributions on the variances, with the degrees of freedom parameter estimated, to allow for heavy-tailed distributions of errors. The gamma (2, 0.1) prior on the degrees of freedom allows for anything from a half-cauchy to a practically half-normal distribution. The normal (0, 10) prior on the cutpoints is very weakly informative with a standard normal latent variable.

Gajewski, B. J., Hart, S., Bergquist-Beringer, S., & Dunton, N. (2007). Inter-rater reliability of pressure ulcer staging: ordinal probit Bayesian hierarchical model that allows for uncertain rater response. Statistics in Medicine, 26(25), 4602-4618. doi:10.1002/sim.287

McGraw, K. O., & Wong, S. P. (1996). Forming inferences about some intraclass correlation coefficients. Psychological Methods, 1(1), 30-46. doi:10.1037/1082-989x.1.1.30

```
data{

  int subjects;
  int raters;
  int categories;
  int scores[subjects*raters];
  int subject_index[subjects*raters];
}

parameters{

  real<lower=0> sigma_effects;
  real<lower=0> sigma_errors;
  real<lower=1> nu_effects;
  real<lower=1> nu_errors;
  vector[subjects] effects_raw;
  vector[subjects*raters] errors_raw;
  ordered[categories-1] cutp;
}

model{

  vector[subjects] effects = effects_raw * sigma_effects;
  vector[subjects*raters] errors = errors_raw * sigma_errors;
  cutp ~ normal(0,10);

  effects_raw ~ std_normal();
  nu_effects ~ gamma(2,0.1);
  sigma_effects ~ student_t(nu_effects,0,1);

  errors_raw ~ std_normal();
  nu_errors ~ gamma(2,0.1);
  sigma_errors ~ student_t(nu_errors,0,1);

  scores ~ ordered_probit(effects[subject_index] + errors, cutp);
}

generated quantities{

  real<lower=0, upper=1> icc = sigma_effects/(sigma_effects +
sigma_errors);
}
```