

Depressive Symptoms in Fathers and
Their Adolescent Offspring:
Findings From a Multi-Informant
Two-Wave Study

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Acknowledgements

Working on my master's thesis has profoundly broadened my perspectives, both scientifically and personally. Prior to this work, I was not aware of all the fun statistics could be! Moreover, being able to immerse myself in the literature of depression in adolescence and the intergenerational transmission of depression has been very rewarding. I have also learned a lot about myself, and the necessity to find a healthy balance between time devoted to work and personal life. Once again, nature has proved to be the place where I find inner peace and recharge my energy.

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Abstract

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Title: Depressive symptoms in fathers and their adolescent offspring: Findings from a multi-informant two-wave study

Background: The potential adverse effects of paternal depression on offspring mental health have received relatively little attention compared to maternal depression. Few studies have examined the association between paternal and offspring depressive symptoms, especially in adolescence. Of the limited studies available, there is a lack of studies utilizing multi-informant data on adolescent depressive symptoms. The main aim of the current study was to examine the association between paternal and offspring depressive symptoms in middle and late adolescence, using parent reports and self-reports on adolescent depressive symptoms.

Method: Data were taken from two data waves of the prospective community-based study Tracking Opportunities and Problems in Childhood and Adolescence (the TOPP study). The sample comprised Norwegian adolescents aged 14-15 at wave 1 ($N = 454$) and 16-17 at wave 2 ($N = 371$), and their biological parents. Adolescent depressive symptoms were measured by the child and the parent version of the Short Mood and Feelings Questionnaire. Parent depressive symptoms were measured by the depression subscale from the self-report inventory Hopkins Symptoms Checklist-25. Cross-sectional and longitudinal associations were examined using multiple regression analyses, adjusting for maternal depressive symptoms and other relevant confounding variables. All analyses were run separately for each informant's report on adolescent depressive symptoms.

Results: Adolescents' self-reports revealed considerably higher levels of depressive symptoms than parent reports on adolescent depressive symptoms. Significant associations between paternal and adolescent depressive symptoms were found cross-sectionally at both data waves and longitudinally only when fathers reported on adolescent depressive symptoms.

Conclusion: Findings from the current study revealed that the levels of adolescent depressive symptoms, and the strength of the associations between paternal and adolescent depressive symptoms varied depending on which informant was reporting on adolescent depressive symptoms. These findings have important scientific implications, suggesting that future research should use multi-informant data when assessing adolescent depressive symptoms and the effects of paternal depression on adolescent offspring.

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Depression is one of the most common mental disorders worldwide, impairing the lives of millions of people (Murray & Lopez, 1996). During adolescence there is a steep increase in the prevalence of depressive symptoms (Angold & Costello, 2006), and depression is the leading cause of illness and disability for females and males ages 10-19 (World Health Organization, 2014). Adolescent-onset depressive disorders are especially likely to be associated with significant impairment across development (Rudolph & Flynn, 2014), as well as chronicity and relapse into adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999). An improved understanding of the risk factors for adolescent depression is thus vital to inform preventive and treatment programs.

One of the most influential risk factors for depressive symptoms in children and adolescents is parental depression (Thapar, Collishaw, Pine, & Thapar, 2012). Extensive studies demonstrate the effects of maternal depression on offspring psychopathology (for review, see Goodman, 2007), whereas studies on the effects of paternal depression are scarce (Phares, Fields, Kamboukos, & Lopez, 2005). Some recent studies indicate that paternal depression is related to offspring psychopathology, and there is an emerging consensus of the importance of taking into consideration paternal psychopathology in research on child and adolescent psychopathology (Connell & Goodman, 2002; Kane & Garber, 2004). Despite this recognition, there is still limited research on the effects of paternal depression on offspring, especially concerning depressive symptomatology in middle and late adolescence. Further, the findings are somewhat inconsistent, and some of the studies have methodological weaknesses, such as not controlling for maternal depression, and not employing multi-informant data on offspring depressive symptoms (Connell & Goodman, 2002; Kane & Garber, 2004; Reeb et al., 2014). Previous studies have revealed discrepancies between parent and adolescent reports on adolescent emotional problems (Sourander, Helstela, & Helenius, 1999), and that the strength of the association between paternal depression and offspring emotional problems varies depending on which informant's report is being used (e.g. Ringoot et al., 2015). Thus, research on the association between paternal and adolescent depressive symptoms should use multi-informant data on adolescent depressive symptoms.

The current study examined cross-sectional and longitudinal associations between paternal and adolescent depressive symptoms from middle to late adolescence, using data from two waves of a prospective community-based study (the TOPP study). All analyses were conducted separately for self-reports, paternal reports, maternal reports, as well as aggregated scores comprising all three informants' reports on adolescent depressive symptoms.

Depression in Adolescence

Definition and operationalization. One of the main controversies in the field is whether depression is best viewed as a categorical disorder, or on a continuum (Avenevoli, Knight, Kessler, & Merikangas, 2008). According to the categorical approach a person must have a certain number of depressive symptoms, but not a specific constellation of them, to be diagnosed with depression (Ingram, Siegle, & Steidtmann, 2014). This approach is typically used in clinical settings, as well as in epidemiological research. The two most widely used classification systems are the Diagnostic and Statistical Manual of Mental Disorder (DSM-5; American Psychiatric Association, 2013) and the International Classification of Diseases (ICD-10; World Health Organization, 1992). In the DSM-5, adolescent depression is similar to the clinical features of depression in adults, being characterized by emotional (e.g. depressed mood), cognitive (e.g. diminished ability to concentrate) and vegetative (e.g. change in weight and/or appetite) symptoms (American Psychiatric Association, 2013). Unlike depression in adulthood, adolescent depression may also manifest in irritable mood, rather than a predominant depressed mood (American Psychiatric Association, 2013; Kessler, Avenevoli, & Merikangas, 2001).

In contrast, the dimensional approach conceptualizes depression on a continuum with increasing severity of depressive symptoms (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010). This approach is appropriate for research using self-reports of depressive symptoms from a low to high end (e.g. the Child Behavior Checklist; the Short Mood and Feelings Questionnaire). Individuals reporting below the diagnostic threshold for depressive disorders will be identified using the dimensional approach (Gotlib, Lewinsohn, & Seeley, 1995). Studying adolescents who experience subthreshold depression is essential; data collected by the World Health Organizations (WHO) World Health Survey reveals that subthreshold depressive disorders are common all across the world, and that they are not qualitatively different from full-blown depressive diagnoses (Ayuso-Mateos et al., 2010). The dimensional approach to depression was applied in the current study to examine depressive symptoms in a non-clinical sample.

Prevalence. There is a steep increase in the prevalence of depressive symptoms in adolescence, and the peak age of onset for depression occurs around 13 to 15 years of age (Angold & Costello, 2006). Most models aimed to explain the development of depression converge on the idea that the biological transformations of puberty (e.g. changes in brain structure and function) might account for the dramatic rise in depression during puberty

(Rudolph & Flynn, 2014). By mid- to late adolescence the prevalence rates of diagnosable depression are about the same as those in adults (Kaminski & Garber, 2002); the estimated 1-year prevalence of depression among adolescents ages 13-18 is 5.7% (Costello, Erkanli, & Angold, 2006), whereas the three-month prevalence for any depressive disorder for adolescents aged 14 and 16 is 2.7% and 3.1%, respectively (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). In a Norwegian study of adolescents aged 13-17 the prevalence was 9.4% for any current (last two months) depressive disorder, and 23% for life-time depression (Sund, Larsson, & Wichstrøm, 2011).

The prevalence estimates are higher when examining depressive symptoms; a survey revealed that 21% of the North American adolescents had experienced depressive symptoms at least a few days in the last two weeks (Van Voorhees, Melkonian, Marko, Humensky, & Fogel, 2010). In a sample of Norwegian adolescents aged 16-19, 26% had experienced one or more depressive symptoms during the last two weeks (Lundervold, Breivik, Posserud, Stormark, & Hysing, 2013). Because a substantial number of adolescents in the general population report depressive symptoms, research should also be conducted on non-clinical samples when examining risk factors of adolescent depression.

Gender differences. There is greater prevalence of depression among women than men; twice as many women suffer from depression (Grigoriadis & Robinson, 2007). The high female to male sex ratio in the prevalence of depression is one of the most robust findings in epidemiology, and it also exists across most cultures (Grigoriadis & Robinson, 2007; Hilt & Nolen-Hoeksema, 2014). Further, the gender difference is reported both in terms of frequency and severity of symptoms (Hankin, Mermelstein, & Roesch, 2007). The gender difference first emerges in early adolescence at about ages 12-13, and increases throughout adolescence (Hilt & Nolen-Hoeksema, 2014), suggesting that the processes that take place during puberty may have differential effects for females and males (Angold & Costello, 2006). Biological, psychological and social explanations for the gender difference in depression have been proposed (for reviews, see Hankin, Wetter, & Cheely, 2008; Hilt & Nolen-Hoeksema, 2014). Mean levels of depressive symptoms and prevalence estimates of depression were calculated separately for adolescent females and males to examine gender differences in the current study.

Long-term consequences. Depression in adolescence is a strong risk factor for depressive disorders in adulthood, with recurrence rates as high as 70% (Birmaher et al., 1996). Adolescent depression also increases the risk of other adverse health outcomes

(Naicker, Galambos, Zeng, Senthilselvan, & Colman, 2013), and receiving medical benefits lasting into adulthood (Pape, Bjorngaard, Holmen, & Krokstad, 2012). Similarly, adolescents who experience subdiagnostic depression suffer psychosocial impairments, and have heightened risk for future psychiatric diagnoses (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Further, depression in adolescence has detrimental effects not only for the individuals affected, but also for his/her social environment and family (Joormann, Eugène, & Gotlib, 2009). The long-term morbidity and negative consequences associated with adolescent depression highlights the need for research on risk factors for adolescent depression, to inform effective preventive programs.

Intergenerational Transmission of Depression

Previous research have shown that parental depression is associated with an increased risk for internalizing and externalizing problems (for reviews, see Downey & Coyne, 1990; Goodman & Gotlib, 1999; Joormann et al., 2009), and more symptoms of physical illness, greater risk of suicide attempts, academic difficulties and lower social competence in offspring (Lewinsohn, Olino, & Klein, 2005). With regard to depression specifically, offspring of parents with a depressive disorder have a two- to threefold greater risk of developing depressive disorders than offspring of non-depressed parents (Weissman et al., 2006), and are twice as likely to experience an episode of depression than children of parents with other psychiatric or medical conditions (Rice, Harold, & Thapar, 2002). Additionally, having a depressed parent increases the risk for earlier onset and more severe depression (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002), and both recent and prior parental depressive episode are associated with child depressive symptoms (Mars et al., 2012). Findings also indicate an increased risk of psychological distress in offspring even when parents experience mild levels of depressive symptoms (Connell & Goodman, 2002; Mars et al., 2015; West & Newman, 2003). The increased risk of negative health outcomes in offspring of depressed parents is apparent as early as the first months of the child's life, throughout childhood and adolescence, and into adulthood (Gotlib & Colich, 2014; Weissman et al., 2006).

The association between parental and offspring depression can be mediated through both genetics and environmental factors. The integrative model of intergenerational depression by Goodman and Gotlib (1999) illustrates the transmission of depression from mothers to their offspring, but the model might just as well be applied to the effects of paternal depression, with some adjustments made (Connell & Goodman, 2002). The model

posits four general explanations for transmission (Gotlib & Colich, 2014). First, the heritability for major depression has been estimated to approximately 40% (Glowinski, Madden, Bucholz, Lynskey, & Heath, 2003; Sullivan, Neale, & Kendler, 2000), and it appears to increase from childhood to adolescence (Scourfield et al., 2003). Second, prenatal parental depression has been associated with negative effects for children's development (T. Field, 2011; Kvalevaag et al., 2013; Ramchandani et al., 2008). The effects of prenatal parental depression could be due to fetal exposure to neuroendocrine and neurotransmitter correlates of maternal prenatal depression (T. Field, 2011), or the indirect effects of prenatal paternal depression on fetal development through a negative impact on mothers' prenatal mood state (T. Field et al., 2006).

Third, children of depressed parents are exposed to parental maladaptive affect, behavior and cognitions postnatally. This might lead to a more stressful domestic environment for the child, for instance because of more negative and less positive parenting behaviors (for reviews, see Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Wilson & Durbin, 2010). Fourth, parental depression is associated with contextual stressors, such as increased interpersonal problems within the family (e.g. marital conflict), economic pressure and general social disadvantage (Connell & Goodman, 2002; Silberg & Rutter, 2002). In sum, the parent not only passes down the genotype associated with depression; parental depression also influences fetal development and the offspring's rearing environment (Natsuaki et al., 2014).

Underrepresentation of Fathers in Research on Offspring Psychopathology

Findings from decades of extensive research on the effects of maternal depression indicate that maternal depression poses an increased risk for infants, toddlers, younger children and adolescents when it comes to a range of behavioral, emotional and cognitive difficulties, including depression (for reviews, see Goodman, 2007; Goodman & Gotlib, 1999; Gotlib & Colich, 2014; Joormann et al., 2009). In stark contrast to this, research on the effects of paternal psychopathology on their offspring has been mostly overlooked in research until recently, and is thus still poorly understood (Phares, 1992; Phares & Compas, 1992; Phares et al., 2005; Ramchandani & Psychogiou, 2009). Phares and Compas (1992) reviewed studies between 1984 and 1991, revealing that 1% included fathers only, 48% included mothers only, 25% included parent variables but did not separate between the parents, and 26% of the studies analyzed data separately for mothers and fathers. However, a more recent review by Cassano, Adrian, Veits, and Zeman (2006), showed somewhat higher rates of paternal participation in research on child and adolescent psychopathology from 1999 to 2005

than the previous seven-year period (1992-1998). A search in PubMed shows a gradual increase of research on child and adolescent psychopathology including fathers from 2005 to 2015. Despite this increase, the largest share of research still focuses on mothers; in 2014 the number of publications on the association between maternal and offspring psychopathology was 915, whereas it was 185 for paternal psychopathology. A graphical presentation of the number of publications including fathers compared to mothers from 2005 to 2015 (Figure A1), and the list of entry terms can be seen in [Appendix A](#).

There are several possible reasons for why fathers have been left out of the research (for review, see Phares, 1992). First, several influential theories of child development, such as psychodynamic theories and attachment theories, have emphasized the crucial role of mothers in child development (Ramchandani & Psychogiou, 2009). Historically there has been a tendency to attribute child psychopathology to mothers (Phares, 1992). Second, sociocultural norms can also explain the lack of inclusion of fathers. In many societies, mothers are still the primary caretaker in the family during early development, and parenting might be viewed as a female domain (Phares, 1992). In accordance with this, an assumption that fathers are less important to child development might exist (Connell & Goodman, 2002). Third, researchers might believe that fathers are harder to recruit for participation in research than mothers (Cassano et al., 2006), although some prior studies indicate that this is not the case (for review, see Woollett, White, & Lyon, 1982).

Rationale for Including Fathers in Research

Although the base rate of depression is lower among men than women, findings indicate that a substantial number of men experience depression or subclinical elevations in depressive symptoms during their child-rearing years; an estimated 39% of mothers and 21% of fathers had experienced at least one episode of depression by the time their offspring reached 12 years (Dave, Petersen, Sherr, & Nazareth, 2010). Further, it has been suggested that adolescence might not only be a demanding developmental period for adolescents themselves, but for their fathers as well; a previous study revealed increases in paternal depressive symptoms in fathers of adolescent offspring with internalizing problems (Fanti, Panayiotou, & Fanti, 2013). This finding indicates that it might be especially important to investigate the association between paternal and offspring depressive symptoms during adolescence.

Because fathers not only contribute 50% of their children's genes, but also take part in caretaking and contribute to the family context (Connell & Goodman, 2002; Phares &

Compas, 1992), including fathers in research seems pertinent to a better understanding of how parental depression affects the child and adolescent. This is also in line with an ecological perspective which takes into consideration the multiple relationships found within the family environment (Bronfenbrenner, 1986). By excluding fathers from the research some proportion of variance in offspring psychopathology might be unaccounted for, or incorrectly attributed merely to maternal depression, thus leading to a distorted image (Connell & Goodman, 2002). Further, evidence suggests that men tend to experience depression differently to women. For instance, men act out their distress through alcohol or drug use to a greater extent than women (Bronte-Tinkew, Moore, Matthews, & Carrano, 2007), and they are less likely than women to seek help for their health problems, as well as delay seeking help (Galdas, Cheater, & Marshall, 2005). These gender differences might ultimately lead to dissimilar effects of maternal and paternal depression on their offspring, justifying the need for research on the influence of both maternal and paternal depression.

Fathering in Norway

Although the roles of mothers and fathers at the work place and home gradually have been changing for the last decades in western societies such as the USA, mothers often work part-time, whereas fathers are the primary breadwinners in the family (Harrington, Van Deusen, Fraone, & Mazar, 2015). Contemporary fathering in Norway might differ from other comparable industrial countries due to the high gender equality in the Norwegian society (United Nations Development Programme, 2014), and the family-friendly initiatives and welfare rights introduced in Norway in 1970 (Kitterød & Rønsen, 2013). Data from Statistics Norway revealed that Norwegian fathers were more involved in caretaking of their younger children, and took part in household chores to a greater extent in 2010 than they did in 1970 (Kitterød & Rønsen, 2013). It might be that depression in Norwegian fathers to a larger extent influence their offspring compared to other societies in which fathers are less involved in their children's lives. There is a call for more international and cross-cultural research on the association between paternal and adolescent depression in order to clarify these potential societal differences concerning the role of fathers (Ramchandani & Psychogiou, 2009).

Review of Empirical Findings

Findings from the limited research including fathers indicate that there is an increased risk of psychopathology in children and adolescents of fathers suffering from psychopathology (for meta-analyses and reviews, see Connell & Goodman, 2002; Kane & Garber, 2004; Ramchandani & Psychogiou, 2009). A meta-analysis by Connell and Goodman

(2002) revealed that the effect of paternal psychopathology on offspring internalizing disorders was $r = .14$ (weighted mean), representing a small effect. In comparison, the effect of maternal psychopathology on child internalizing disorder was $r = .18$ (weighted mean). These findings suggest that the magnitude of difference between the effects of paternal and maternal psychopathology are quite small, underscoring the importance of studying the effects of psychopathology in both parents.

Research on the effects of paternal psychopathology on offspring has primarily focused on younger children (Reeb et al., 2014); in the meta-analyses by Connell and Goodman (2002) and Kane and Garber (2004) the mean age of children in the studies reviewed was 9.4 and 10.7 years, respectively. Despite sparse research on adolescents, the available evidence suggests that adolescent offspring are more vulnerable to the negative effects of paternal psychopathology than offspring in early and middle childhood (Connell & Goodman, 2002). The same pattern has been found regarding the effects of paternal depression specifically; a study by Weitzman, Rosenthal, and Liu (2011) revealed that offspring aged 12-17 were more strongly affected by paternal depression than offspring aged 5-11. There are several plausible explanations for this age effect. For instance, it has been suggested that fathers spend more time with their children as they get older, thus becoming more salient to offspring development (Lamb & Lewis, 2004; Price-Robertson, 2015). More research on adolescent offspring should be conducted to further investigate this.

Of the limited research available on the effects of paternal depression specifically, significant effects have been found on offspring emotional and behavioral problems in toddlerhood (e.g. Kvalevaag et al., 2013), childhood (e.g. Ramchandani, Stein, Evans, & O'Connor, 2005), and adolescence (e.g. Reeb & Conger, 2009). According to a meta-analysis by Kane and Garber (2004) the mean effect size of paternal depression on internalizing problems in offspring was $r = .24$. The magnitude of the association between paternal depression and internalizing disorders in offspring is in general small, but results from several studies suggest that the effects of maternal and paternal depression on offspring are similar in magnitude (e.g. Amrock & Weitzman, 2014; Jacobs, Talati, Wickramaratne, & Warner, 2015; Kane & Garber, 2004). Nevertheless, there seems to be few studies examining the relationship between paternal depression and offspring depression, especially in middle and late adolescence.

Also, many studies focus on internalizing symptoms or psychological distress (i.e. anxiety and depressive symptoms measured simultaneously), rather than depressive

symptoms specifically. This is reflected in the meta-analyses by Connell and Goodman (2002) and Kane and Garber (2004), in which the focus were not on specific childhood disorders such as depression, but on internalizing problems. Research focusing on broadband syndromes of emotional problems, such as internalizing problems instead of depression specifically, risk to achieve attenuated results because the association between parental and offspring mental health problems might vary across specific disorders (Connell & Goodman, 2002). Moreover, much of the previous research on offspring of depressed parents has been conducted with clinical samples, and the results may not be generalizable to the broader population of depressed individuals, including individuals experiencing subclinical depressive symptoms (Goodman et al., 1997).

Methodological limitations in previous studies. The existing literature on the effects of paternal depression on offspring carries some methodological limitations. First, the majority of studies have only assessed cross-sectional associations (Kane & Garber, 2004), thus making it impossible to examine the direction of causality and to what degree paternal depression influence depression in their offspring over time. Additionally, few of the limited longitudinal studies have adjusted for initial associations and stability of offspring depression (Kane & Garber, 2004; Reeb et al., 2014). By not adjusting for initial levels of offspring depression, one cannot rule out the possibility that the predictive effects of paternal depression was due to a correlation between offspring and paternal depression at baseline (Selig & Little, 2012).

Further, the majority of studies included in the meta-analyses by Connell and Goodman (2002) and Kane and Garber (2004) did not control for maternal psychopathology when examining the link between paternal and offspring psychopathology. If maternal psychopathology is not controlled for in the analyses, one risks to incorrectly attribute the effects to paternal psychopathology. The effect sizes might also be inflated because maternal and paternal variables are often strongly positively correlated (Pleck, 2010). Analyses should be adjusted for maternal depression in order to examine whether paternal depression has independent effects on adolescent depression (Reeb & Conger, 2009).

There are few longitudinal community-based studies on the association between paternal and adolescent depressive symptoms in which adolescents' prior depressive symptoms and maternal depressive symptoms have been controlled for. However, one study should specifically be mentioned; findings from a recent North American study, in which this methodological robust design was applied, indicated that paternal depressive symptoms

predicted depressive symptoms in adolescent offspring one year later (Reeb & Conger, 2009). More studies are needed in order to replicate this finding, as well as to generalize to other cultures in which fathering might differ.

Findings from Norwegian studies. To my knowledge, there are few Norwegian cross-sectional and longitudinal community-based studies that examine the association between paternal and offspring depressive symptoms, especially in middle and late adolescence. Some community-based studies have examined the effects of paternal internalizing symptoms (i.e. anxiety and depressive symptoms) on either internalizing symptoms or depression in adolescent offspring, but the findings are mixed; one study revealed no cross-sectional effect of paternal internalizing symptoms on adolescent depression (Agerup, Lydersen, Wallander, & Sund, 2015), whereas another study indicated a significant cross-sectional association between paternal anxiety/depression on adolescent anxiety/depression (Ranøyen, Klöckner, Wallander, & Jozefiak, 2014). A longitudinal study by Ranøyen, Stenseng, Klockner, Wallander, and Jozefiak (2015), found a predictive effect of paternal anxiety/depression measured when offspring were in preschool age on adolescent offspring anxiety/depression, but this association was entirely mediated by paternal symptoms measured when offspring were adolescents. The associations between paternal anxiety/depression and adolescent offspring anxiety/depression in the study by Ranøyen et al. (2015) were exactly the same as in the study by Ranøyen et al. (2014), because both studies used data from the same community-based longitudinal study (HUNT).

Additionally, some other Norwegian studies have examined the effects of paternal depression, but with samples comprising somewhat younger or older offspring compared to the sample in the current study. For instance, a study by Gere et al. (2013), did not find a significant association between paternal depressive symptoms and depressive symptoms in their offspring aged 7-13, who were referred to child community clinics. Further, a community-based study on the course of offspring depression by Agerup, Lydersen, Wallander, and Sund (2014) revealed a significant association between paternal internalizing problems and adolescent offspring who remained depressed from age 15 to 20, but the association was not significant in the fully adjusted multinomial models. Agerup et al. (2014) used data from the same longitudinal study as Agerup et al. (2015) (the Youth and Mental Health study), in which no cross-sectional effects were found.

In sum, there is limited research on the intergenerational transmission of depression between Norwegian fathers and their adolescent offspring. Although there is some evidence

for significant cross-sectional associations between paternal and adolescent depression, the results are not fully consistent. More studies are thus needed for clarification.

Gender-Specific Patterns

According to some previous studies, daughters are significantly more strongly affected by maternal depression than sons (e.g. Jenkins & Curwen, 2008), whereas paternal depression is significantly more strongly associated with psychopathology in sons compared to daughters (e.g. Weitzman et al., 2011). This same-sex pattern is in accordance with social-learning theory and social-cognitive theory, implying that children are more strongly influenced by models of greater similarity to themselves (e.g. the same gender) (Bandura, 1977; Bussey & Bandura, 1999). It is also suggested that fathers to a larger extent identify with their sons and thus spend more time with them than with their daughters, which might contribute to stronger associations between paternal and sons' psychopathology (Lamb & Lewis, 2004; Ramchandani & Psychogiou, 2009). However, other studies reveal opposite gender patterns from fathers to daughters and mothers to sons (e.g. Ge, Conger, Lorenz, Shanahan, & Elder, 1995; Reeb, Conger, & Wu, 2010), or an universal effects of parental depression on both daughters and sons (e.g. Agerup et al., 2015; Ohannessian et al., 2005; Ranøyen et al., 2014; Reeb et al., 2014). Because of the inconsistent findings in the available literature, more studies are needed for clarification of the potential gender-specific pattern of the intergenerational transmission of depression.

Multi-Informant Data on Adolescent Depressive Symptoms

Previous studies on the effects of paternal depression have used different informants on offspring depressive symptoms (Kane & Garber, 2004). Self-reports are often the only source of information (Hughes & Gullone, 2010a), although some studies use parent reports in addition to self-reports (e.g. Gere et al., 2013; Hughes & Gullone, 2010b; A. J. Lewis et al., 2014), and others only use parent reports (e.g. Amrock & Weitzman, 2014; Fanti et al., 2013; Weitzman et al., 2011). No gold standard exists regarding which informant is the most optimal on child and adolescent psychopathology (De Los Reyes, Thomas, Goodman, & Kunder, 2013; Kraemer et al., 2003; Richters, 1992). Some have argued that children and adolescents are the best informants of their own internalizing symptoms (Bird, Gould, & Staghezza, 1992), although findings indicate that parents provide clinical useful information on depressive symptoms in their adolescent offspring (K. J. S. Lewis et al., 2012).

Informant discrepancies and informant agreement. In community samples, adolescents tend to report higher levels of depressive symptoms than their parents report on

adolescent depressive symptoms (e.g. Sourander et al., 1999). Informant discrepancies between children's/adolescents' self-reports and parent reports in research on child and adolescent psychopathology is a robust finding, even when informants complete identical or parallel measures (Achenbach, 2006, 2011; Achenbach, McConaughy, & Howell, 1987; De Los Reyes, 2011; De Los Reyes et al., 2015; De Los Reyes & Kazdin, 2004, 2005). The tendency for adolescents to rate themselves higher on levels of emotional and behavioral problems than their parents rate them have been found in several societies, including Norway (Rescorla et al., 2013; Sourander et al., 1999).

In line with the informant discrepancies, different informants' reports on children's and adolescent's social, emotional or behavior problems commonly show low to moderate levels of agreement. In a highly cited meta-analysis of 119 studies by Achenbach et al. (1987), findings revealed an average cross-informant correlation of $r = .59$ between maternal and paternal reports on offspring behavioral and emotional problems, whereas the average correlation between parent and offspring reports was $r = .25$. More recent studies have replicated this finding, showing low to moderate correlations between self-reports and parent reports on child and adolescent psychopathology (for meta-analysis, see De Los Reyes et al., 2015). According to the meta-analysis by Achenbach et al. (1987), cross-informant correlations were lower in samples consisting of adolescents (12-19 years) than samples of younger children (6-11 years), and for internalizing disorders compared to externalizing disorders.

Because parent reports and offspring's self-reports on offspring psychopathology diverge, the choice of informant may have essential consequences for the empirical findings (De Los Reyes et al., 2013). Evidence indicates that the magnitude of the associations between parental and offspring psychological problems vary as a function of which informants' report on offspring psychopathology is used (Ge, Conger, Lorenz, & Simons, 1994; Kane & Garber, 2004; Ringoot et al., 2015). Significantly larger effect sizes of parental psychopathology have been found in studies in which the parent reports on offspring internalizing symptoms, compared to studies using offspring's self-reports (for meta-analysis, see Connell & Goodman, 2002). Studies on the association between paternal and adolescent depression using multi-informant data on adolescent depressive symptoms seem to be lacking. The current study attempts to fill a gap in literature by conducting analyses separately for adolescents' self-reports, paternal reports, and maternal reports on adolescent depressive symptoms.

Aggregating scores. Some researchers argue that by aggregating reports from multiple informants, ratings will be more valid and reliable than using reports from only one informant (Bird et al., 1992). There are different ways of combining multiple informants' reports, like: (a) applying a combinational algorithm to the outcome of the reports, (b) averaging scores without giving specific weight to a particular informant, or (c) using more advanced statistical techniques to examine multiple reports in combination (e.g. latent structures in structural equations models) (De Los Reyes et al., 2013; Kraemer et al., 2003; van Dulmen & Egeland, 2011). One commonly used approach rather than simply averaging informants' scores is to create aggregated scores through principal component analysis (PCA), in which the common core of scores provided by multiple informants is extracted (Kraemer et al., 2003). The current study used PCA-aggregated scores comprising adolescents', paternal and maternal reports on adolescent depressive symptoms, to investigate how the research findings were affected by the use of aggregated scores compared to reports from each informant.

The Current Study

The purpose of the current study was two-folded. First, the current study investigated the association between paternal and offspring depressive symptoms in middle (14-15 years) and late (16-17 years) adolescence, both cross-sectionally and longitudinally. In order to meet the methodological shortcomings in previous studies, all analyses were adjusted for maternal depressive symptoms in addition to other relevant confounding variables (i.e. adolescent gender and family demographic variables). Additionally, the longitudinal analyses were also adjusted for adolescents' prior depressive symptoms. The association between maternal and adolescent depressive symptoms were commented on for the purpose of comparison with the association between paternal and adolescent depressive symptoms. However, an elaboration of the association between maternal and adolescent depressive symptoms was beyond the scope of the current study.

Additionally, the current study aimed to extend our knowledge on the use of multiple informants. All analyses of the association between paternal and adolescent depressive symptoms were run separately for self-reports, paternal reports and maternal reports on adolescent depressive symptoms, as well as the aggregated scores comprising all informants' reports. This was done to investigate if the associations were affected by the use of different reports on adolescent depressive symptoms. Also, informant discrepancies and informant

agreement between parent and adolescent reports on adolescent depressive symptoms were examined.

The current study also examined direct gender effects on females' (mothers and adolescent females) and males' (fathers and adolescent males) levels of depressive symptoms, prevalence of adolescent depression, the potential differential effects of paternal depressive symptoms on adolescent females and males, and the stability and change of adolescent depressive symptoms from 14-15 to 16-17 years.

Research Questions and Hypotheses

The following two main research questions and four sub-questions, along with their corresponding hypotheses, were examined in the current study:

Main question 1: *To what degree are paternal and adolescent depressive symptoms associated, and how does this association vary with regard to:*

- a. *Cross-sectional associations at two different developmental stages in adolescence (ages 14-15 and 16-17), before and after adjusting for maternal depressive symptoms and other relevant confounding variables (i.e. adolescent gender and family demographic variables).*
- b. *Longitudinal associations across adolescence from ages 14-15 to 16-17, before and after adjusting for adolescents' prior depressive symptoms, maternal depressive symptoms and other relevant confounding variables (i.e. adolescent gender and family demographic variables).* Due to limited previous research and some inconsistency in the literature, these research questions were exploratory in nature and no hypotheses were stated.

Main question 2: *How do the use of different informants' (i.e. adolescents, fathers and mothers) reports on adolescent depressive symptoms and aggregated scores (comprising all informants' reports) affect the association between paternal and adolescent depressive symptoms?* This research question was exploratory in nature, as there was not sufficient previous empirical basis to formulate hypotheses.

Sub-question 1: *To what degree are the informants' reports on mean levels of adolescent depressive symptoms divergent (i.e. informant discrepancies), and how strongly do the informants' reports correlate (i.e. informant agreement)?* It was expected that adolescents report higher levels of depressive symptoms than parents report on adolescent depressive symptoms (H₁). Further, low to moderate informant agreement between parent and adolescent reports on adolescent depressive symptoms was expected (H₂).

Sub-question 2: *Does gender have direct effects on parent and adolescent levels of depressive symptoms?* Females (mothers and adolescent females) were expected to report higher levels of depressive symptoms than males (fathers and adolescent males) (H₃).

Sub-question 3: *Do adolescent depressive symptoms change or remain stable from ages 14-15 to 16-17?* Depressive symptoms were expected to increase from middle to late adolescence, especially for adolescent females (H₄).

Sub-question 4: *Does adolescent gender moderate the association between paternal and adolescent depressive symptoms?* Due to inconsistent findings in previous research, this research question was exploratory in nature and no hypotheses were stated.

Figure 1 displays a general model of the associations examined in the current study. Associations were examined cross-sectionally at ages 14-15 and 16-17, and longitudinally from ages 14-15 to 16-17. Analyses were conducted separately for the different informants' reports on adolescent depressive symptoms, as well as the aggregated score comprising all informants' reports. Associations were examined before and after adjusting for confounding variables. The moderation effect of adolescent depressive symptoms on the association between paternal and adolescent depressive symptoms was also examined.

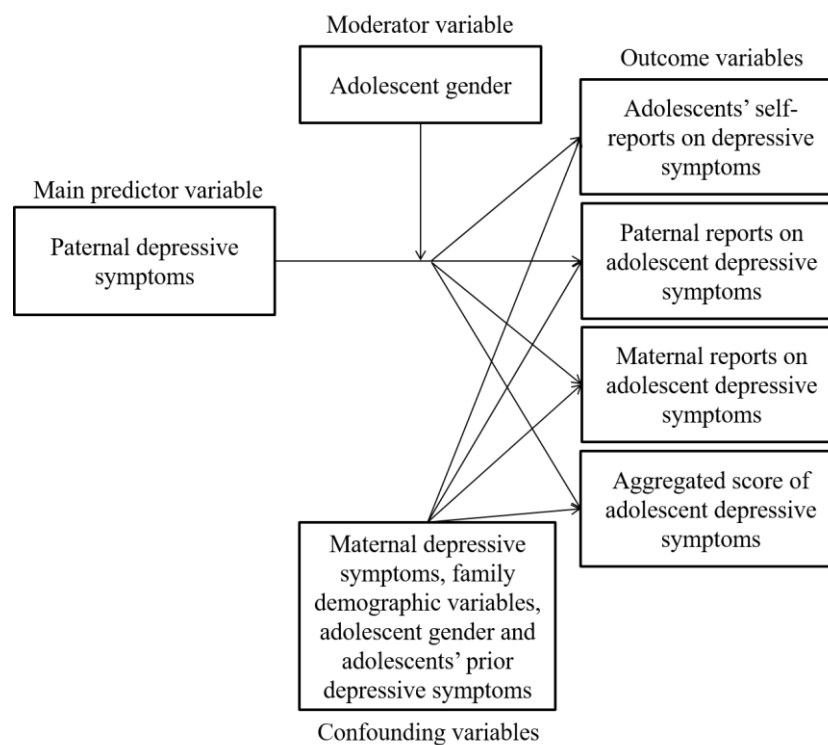


Figure 1. Model of the associations between paternal and adolescent depressive symptoms examined in the current study. Adolescents' prior depressive symptoms were adjusted for only in the longitudinal analyses.

Method

The TOPP Study

The study sample was drawn from the Tracking Opportunities and Problems in Childhood and Adolescence Study (the TOPP study), based at the Norwegian Institute of Public Health. The TOPP study is an eight-wave community-based study following children from they are 18 months (in 1993) to 18.5 years (in 2011) (Mathiesen, Tambs, & Dalgard, 1999). The study investigates factors that could influence the mental health of children, such as parent characteristics and conditions in the child's domestic environment.

Procedure

Participants were recruited from 19 health care areas in Eastern Norway on their visit to public health clinics for the scheduled 18-month vaccinations for the index child in 1993 (t1). In Norway, approximately 95% of the families with infants and toddlers attend to the public health program (Bergsaker et al., 2014). The participants were invited to complete a survey questionnaire, in which 913 (87%) mothers from the 1081 eligible families participated. The families were invited to fill out similar questionnaires at seven subsequent waves, when the children were 2.5 years (t2), 4.5 years (t3), 8.5 years (t4), 12.5 years (t5), 14.5 years (t6), 16.5 years (t7) and 18.5 years (t8). At the first data collection the questionnaires were handed out at the public health clinics, but from the fourth wave the questionnaires were sent by mail. Mothers participated on all waves, fathers filled out separate questionnaires from t6, and adolescents from t5.

Sample

The 19 health care areas in the TOPP study were overall representative of the diversity of communities in Norway; 28% of the families lived in large cities, 55% in small towns and 17% in rural areas (Mathiesen et al., 1999). The current study utilizes data from fathers, mothers and adolescents when the adolescents were 14-15 years, and 16-17 years (i.e. t6 and t7 in the TOPP study). To assist the reader, t6 and t7 will be referred to as wave 1 (w1) and wave 2 (w2) in this thesis. Excerpts from parent and child questionnaires are shown in [Appendix B](#).

The current study focuses on adolescents and their biological fathers. Non-biological fathers ($n = 26$ at w1; $n = 15$ at w2) and adoptive children ($n = 2$) were hence omitted from the analyses. The total sample included 454 adolescents (55.5% females) and 819 parents (41.6% fathers) at w1, and 371 adolescents (58.5% females) and 706 parents (41.1% fathers)

at w2. According to maternal reports, 62% of the families at w1 and 73% at w2 had family income above 550 000 Norwegian kroner (NOK), which was higher than the general population in 2006 and 2008 (Statistisk Sentralbyrå, 2013). At w1, the median education level for fathers and mothers was four years or less from a university or college, indicating that parents in the current sample had somewhat higher levels of education compared to the general population in 2006 (Statistisk Sentralbyrå, 2008). Parents' mean age were 46 years for fathers and 43 years for mothers at w1. Table 1 shows the parents' living arrangements with the adolescent and parental civil status at w1 and w2.

Table 1

Frequency and Percentage of Fathers' and Mothers' Living Arrangements with the Adolescent and Civil Status at Wave 1 and Wave 2

Characteristic	Fathers				Mothers			
	Wave 1		Wave 2		Wave 1		Wave 2	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Living arrangements								
Living with the adolescent full-time	283	82.7	238	81.5	429	89.6	370	88.1
Living with the adolescent less than full-time	59	17.3	54	18.5	50	10.4	50	11.9
Civil status								
Married/live-in partner	268	79.7	262	89.7	328	69.0	349	83.5
Divorced/separated	34	10.1	22	7.5	80	16.8	45	10.8
Not married	33	9.8	7	2.4	64	13.5	22	5.3
Widower/widow	1	.3	1	.3	3	.6	2	.5

Attrition

In longitudinal research, attrition is a threat to the validity of the findings, because it could potentially bias the results (Miller & Wright, 1995). The attrition rate for maternal participation from the first data collection in 1993 to 2006 (i.e. w1 in the current study) was 47.7%, whereas it was 54.4% from 1993 to 2008 (i.e. w2 in the current study). The attrition rate from w1 to w2 was 13% for mothers, 7.6% for fathers, and 18.3% for adolescents. Analyses of sample attrition have previously been conducted to investigate whether the participants who stayed in the TOPP study differed on any measure from those who dropped out of the study (e.g. Gustavson, von Soest, Karevold, & Røysamb, 2012; Karevold, Røysamb, Ystrøm, & Mathiesen, 2009; Nilsen, Gustavson, Røysamb, Kjeldsen, & Karevold, 2013). Nilsen et al. (2013) found that the following three variables measured at the first data collection predicted adolescent participation at the seventh data wave (i.e. w2 in the current

study): female gender, high maternal education and work participation. Maternal age, mothers living alone, family finances, maternal depressive symptoms, parental divorce/separation, and child's internalizing and externalizing problems did not predict adolescent participation.

Gustavson, von Soest, et al. (2012) conducted logistic regression analyses of the data from the first to seventh data waves, revealing that lower maternal educational level at baseline was the only variable that differentiated the families that dropped out of the study from the families that remained. Baseline mental health and relationship variables (maternal variables: age, living alone, family finances, not working, emotionality, sociability, activity, partner support, emotional support from friends and family, chronic stressors, mental distress; child variables: activity, sociability, emotionality and shyness) were not significantly different between the dropouts and the remaining families.

Further, Gustavson, von Soest, et al. (2012) performed a Monte Carlo simulation study showing that while mean estimates became increasingly biased as attrition rates increased, associations between the variables were only minimally affected by attrition. In sum, the attrition analyses indicate that the results from the TOPP study are valid, and that the results are generalizable to similar samples (i.e. samples comprising parents and adolescents with normal functioning).

Ethical Considerations

General ethical guidelines for research have been followed. The data collection was approved by the Data Inspectorate and the Regional Committee for Medical Research Ethics. Participation in the study was voluntary and participants were given written information about the study. The information that was given to the participants about the study emphasized the confidentiality of the participants, the possibility to skip questions, and the right to withdraw from the study at any point. All analyses were conducted on anonymous data.

Measures

Adolescent depressive symptoms. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ) designed for children and adolescents ages 8-18 (Angold et al., 1995). The 13-item questionnaire has a parent version and a self-report version for children and adolescents. At w1, one item («I found it hard to think/concentrate») was omitted due to its resemblance to one item from the Strengths and Difficulties Questionnaire, also filled out by the adolescents (not used in the current study). The 12- and 13-item versions of the SMFQ at w2 correlated highly (self-reports: $r = .99$;

paternal reports: $r = .98$; maternal reports: $r = .99$, all $ps < .001$), and like previously done by others (Nilsen et al., 2013), the original 13-item version was used at w2.

Both parents and the adolescents completed the questionnaires at w1 and w2. Examples of statements from the parent version are: “She/he did not enjoy anything at all” and “He/she cried a lot”. Participants were asked to answer the statements based on the two preceding weeks. Responses were measured on a three-point (0-2) scale, which was slightly differently worded at w1 and w2 (w1: *Seldom true, Sometimes true, Often true*; w2: *Not true, Sometimes true, True*). The statements used in the SMFQ have been carefully translated to Norwegian, back-translated and validated in another Norwegian sample of adolescents aged 13-14 (Sund, Larsson, & Wichstrøm, 2001). Mean scores were calculated to construct indices at each data wave. Total scores were also calculated to inform on prevalence estimates of adolescent depression. A cut-off of 11 on the SMFQ is commonly used in the literature (Turner, Joinson, Peters, Wiles, & Lewis, 2014), and was therefore used in the current study.

Psychometrically sound qualities have been documented for the SMFQ in prior studies of adolescents (e.g. Angold et al., 1995; Turner et al., 2014), including studies on Norwegian adolescents (e.g. Lundervold et al., 2013). The current study’s internal consistency for self-reports on SMFQ was $\alpha = .88$ at w1, and $\alpha = .89$ at w2, similar to former studies using the same data (e.g. Karevold et al., 2009; Nilsen et al., 2013). The internal reliability of both paternal and maternal reports at w1 was $\alpha = .81$, whereas it was $\alpha = .77$ for paternal reports and $\alpha = .85$ for maternal reports at w2.

Parent depressive symptoms. Parent depressive symptoms were assessed at both data waves using the depression subscale from the 25-item version of the self-report instrument the Hopkins Symptoms Checklist (HSCL; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974; HSCL-25; Hesbacher, Rickels, Morris, Newman, & Rosenfeld, 1980; Winokur, Winokur, Rickels, & Cox, 1984). The HSCL-25 measures both anxiety symptoms and depressive symptoms, and like previously done by others (e.g. Gustavson, Røysamb, et al., 2012) the current study used the depression subscale to measure parent depressive symptoms. The subscale originally consists of 15 items, but one item (“Loss of sexual interest or pleasure”) was omitted from the TOPP study on both waves because it was perceived as being too sensitive in a pilot study (Mathiesen et al., 1999).

Parents were asked to indicate to what degree they had experienced a list of symptoms during the last week, such as “Feeling hopeless about the future” or “Worrying too much about things”, on a 4-point scale (from 1 = *not distressed at all* to 4 = *very much distressed*).

Mean scores were calculated to make indices at each wave. The cut-off score derived by Derogatis et al. (1974) for classifying the depression scores as symptomatic using the HSCL-25 (both depression and anxiety) is 1.75 (for mean scores), and this cut-off was used in the current study.

High internal consistency of the HSCL-25 is well established (e.g. Müller, Postert, Beyer, Furniss, & Achtergarde, 2010), also in Norwegian studies (e.g. Strand, Dalgard, Tambs, & Rognerud, 2003; Tambs & Moum, 1993). For the current study, the internal consistency was high for both parents at both waves ($\alpha = .86-.90$).

Confounding variables. Adolescent gender and adolescents' living arrangement with parents (paternal and maternal reports) were significantly associated with the aggregated scores (comprising all informants' reports on adolescent depressive symptoms) at either w1 or w2, and thus included as confounding variables. For more information on the selection of confounding variables, see section "Examination and Selection of Confounding Variables" below. Adolescent gender was coded 0 = *males*, 1 = *females*. Adolescents' living arrangement with parents originally consisted of three response categories (0 = *less than half of the time*, 1 = *half of the time*, 2 = *full time*), which was dummy coded into 0 = *father/mother living with his/her child less than full-time*, and 1 = *father/mother living with his/her child full-time*.

Missing data. To maximize the use of available data and thus increase statistical power, all scales were constructed by using mean score indices if at least 50% of the statements on a questionnaire was answered. Two cases were removed from the sample; one father and one mother had answered less than 50% of the questions on the SMFQ at w1.

Statistics

Statistical analyses were performed in two stages: (a) preliminary analyses, and (b) main statistical analyses, and will be described in the following sections. All statistical analyses were conducted with the computer software IBM SPSS Statistics 22 for Windows. Significance level of .05 (two-tailed tests) was used for all analyses. In this thesis, effect is used in the sense of statistical effect. Effect sizes were measured with Cohen's d for t -tests, and Pearson's r for correlation analyses. Cohen (1988, 1992) provided rules of thumb for interpretation of d and r : small, medium and large effects correspond to $d = 0.20$, $d = 0.50$ and $d = 0.80$, and $r = .10$, $r = .30$ and $r = .50$, respectively. R^2 was reported for the final models of the multiple regression analyses. Standardized beta coefficients were reported in

order to ease the comparison of the importance of the predictors in the regression models (A. P. Field, 2013).

Preliminary analyses.

Assumptions of statistical tests. Preliminary analyses were conducted to assure that the assumptions for multivariate analysis were met (i.e. linearity, normality, statistical independence of the errors, and homoscedasticity), and to examine outliers and multicollinearity between the predictors (A. P. Field, 2013). The analyses revealed that linearity, statistical independence and homoscedasticity were generally acceptable. Further, there was no multicollinearity between the predictors, which is essential when conducting multiple regression analysis (A. P. Field, 2013). Also, the sample size was sufficiently large to obtain a reliable regression model, when following one commonly used rule of thumb (i.e. 10 cases of data for each predictor in the model) (A. P. Field, 2013).

However, some deviations regarding normality and outliers were found, violating the assumption that variables and residuals in multiple regression analyses should be normally distributed in order to draw precise inferences (Tabachnick & Fidell, 2013). The distribution of the residuals in the multiple regression analyses were assessed using histograms and probability plots, revealing deviance from normality. Consequently, the variables' levels of skewness and kurtosis, as well as the variables' frequency histograms were inspected. Z-tests were conducted in order to calculate the significance levels of the variables' skewness and kurtosis. These assessments revealed that all continuous variables were positively skewed, with significant deviance from 0 ($z\text{-value} > 3.29, p < .001$; A. P. Field, 2013). The values of skewness and kurtosis before transformation are shown in Table C1 in [Appendix C](#).

Univariate and multivariate outliers were also investigated during routine preliminary data screening. When inspecting univariate outliers using z-scores, frequency distributions and graphical methods (i.e. histogram and plots) cases with standardized scores in excess of 3.29 ($p < .001$) were detected in all indices.

Data transformation. Transformation was undertaken to improve the normality of distributions and pull univariate outliers closer to the center of the distributions. Transformation is the first option for reducing the impact of univariate and multivariate outliers, and the safest strategy to improve variables' normality (Pallant, 2007; Tabachnick & Fidell, 2013). Natural logarithmic transformation is the recommended procedure when the distribution is substantially positively skewed (Tabachnick & Fidell, 2013), thus this type of transformation was applied to all the continuous variables (i.e. parent and adolescent

depressive symptoms). Although transformation did not result in perfectly normal distributions, it resulted in levels of skewness and kurtosis closer to zero than before transformations were conducted. The values of skewness and kurtosis after transformation are shown in Table C1 in [Appendix C](#). Transformation also reduced the impact of outliers.

Principal component analyses. Principal component analyses (PCA) were performed to create aggregated scores comprising self-reports, paternal and maternal reports on the SMFQ. The transformed SMFQ indices (i.e. mean scores) at w1 and w2 were entered in separate analyses, to create an aggregated score for each data wave. Eigenvalues for the two first factors extracted in the PCA were 1.67 and .72 at w1, and 1.76 and .72 at w2. The factor loadings ranged from .705 to .819, and are indicative of the relative contribution each variable makes to a factor (A. P. Field, 2013). See Table D1 in [Appendix D](#) for more information.

The first component extracted in a PCA is a multi-informant estimate, largely free of the effects from rater-dependent variability and measurement error than any single informant's measure (Kraemer et al., 2003). Because the eigenvalues clearly supported a one-factor solution, the first factor in each PCA were saved as new variables to be used as outcome variables in the statistical analyses. Note that the saved aggregated scores are standardized ($M = 0$, $SD = 1$). The internal reliability of the aggregated scores was $\alpha = .58$ at w1, and $\alpha = .62$ at w2.

Examination and selection of confounding variables. Previous research have shown that the following factors were associated with adolescent depressive symptoms: adolescent gender (i.e. being a female), low family income, low parental education, parental age (i.e. older parents), living arrangements with parents (e.g. father's absence and not living with both biological parents), and parental divorce (e.g. Eley et al., 2004; Mykkestad, Røysamb, & Tambs, 2012; Reinherz et al., 1993; Schraedley, Gotlib, & Hayward, 1999; Størksen, Røysamb, Moum, & Tambs, 2005; Sund, Larsson, & Wichstrøm, 2003; Velez, Johnson, & Cohen, 1989). By not controlling for these possibly confounding variables, significant associations between paternal and adolescent depressive symptoms might actually be caused by a third variable (Reeb et al., 2014). These factors were examined in correlation analyses with the aggregated scores in order to find potential confounders that should be included in the multiple regression analyses. Family demographic variables were reported by both parents. Adolescent gender, adolescents' living arrangement with parents and parental divorce/separation were significantly correlated with the aggregated scores, see Table E1 in [Appendix E](#). The non-significant correlations are for the sake of brevity not reported.

Paternal reported divorce/separation and the aggregated score at w1 were significantly correlated, as well as maternal reported divorce/separation and the aggregated score at w2. Yet, parental divorce/separation was not included as confounding variables in the analyses, due to the probable overlap of parental civil status and adolescents' living arrangements with parents - a possibility which is highly likely because children's living arrangements often depend on whether the parents are married or divorced. Additionally, due to the expectation of rather small effects of paternal depressive symptoms on adolescent depressive symptoms (Connell & Goodman, 2002; Kane & Garber, 2004), it was necessary to preserve statistical power by avoid overfitting the model (Tabachnick & Fidell, 2013).

In sum, adolescent gender and adolescents' living arrangements with parents were included as confounding variables in the multiple regression analyses in addition to maternal depressive symptoms.

Main statistical analyses. To examine how the use of different informants' reports on adolescent depressive symptoms affect the associations (main research question 2), all correlation analyses, moderated regression analyses and multiple regression analyses were conducted separately for four outcomes: (a) self-reports, (b) paternal reports, (c) maternal reports, and (d) the aggregated scores comprising all informant reports.

Descriptive statistical analyses. To inform on central tendency and variability, mean and standard deviation of each continuous variable were assessed prior to the inferential analysis.

Independent and paired samples *t*-tests. All *t*-tests were conducted on the variables before logarithmic transformation was done. Independent-sample *t*-tests were conducted to examine gender effects on adolescent levels of depressive symptoms (sub-question 2), whereas paired-sample *t*-tests were conducted on maternal and paternal self-reports on depressive symptoms to investigate whether levels of depressive symptoms in parents significantly differed (sub-question 2). Paired-samples *t*-tests were also performed in order to examine stability and change of adolescent females' and males' depressive symptoms from ages 14-15 to 16-17 (sub-question 3), and to examine informant discrepancies between the multiple informants' reports on adolescent depressive symptoms (sub-question 1).

Correlation analyses. Bivariate correlation analyses using Pearson's *r* were conducted to examine the cross-informant agreement between the informants' reports on adolescent depressive symptoms (sub-question 1). Further, correlation analyses were performed in order

to investigate the associations between paternal and adolescent depressive symptoms, both cross-sectionally and longitudinally (main research question 1a and 1b). The correlation analyses were conducted with the logarithmic transformed variables. Correlations were not divided by adolescent gender.

Multiple regression analyses. Multiple regression analysis was chosen because it allows for assessment of the relationship between one outcome variable and several predictor variables, while the effects of other confounding variables are statistically eliminated (Tabachnick & Fidell, 2013). Cross-sectional and longitudinal associations between paternal and adolescent depressive symptoms were examined after adjusting for maternal depressive symptoms, adolescent gender and adolescents' living arrangements with parents (research question 1a and 1b). Inspired by autoregressive models for longitudinal data (Selig & Little, 2012), the analyses of the association between paternal depressive symptoms measured at w1 and adolescent depressive symptoms measured at w2 were adjusted for prior levels of adolescent depressive symptoms (measured at w1). All multiple regression analyses were conducted with the logarithmic transformed variables.

Moderated regression analyses. Moderated regression analyses were carried out to investigate whether adolescent gender moderated the association between paternal and adolescent depressive symptoms (sub-question 4). The continuous predictor variables (i.e. paternal depressive symptoms) were grand mean centered before conducting the moderation analyses to avoid multicollinearity (Tabachnick & Fidell, 2013). Interaction terms were constructed in SPSS, by multiplying adolescent gender and paternal depressive symptoms (Tabachnick & Fidell, 2013). Adolescent gender and paternal depressive symptoms were included in the first step of the hierarchical regression analysis, then adding the interaction term in the second step.

Results

Descriptive Statistics

Number of total participants, raw score means and standard deviations for total participants and participants divided by gender are presented in Table 2. Descriptive statistics for the variables after logarithmic transformation can be seen in Table F1 in [Appendix F](#).

Table 2

Descriptive Statistics for Parent and Adolescent Depressive Symptoms for the Total Sample, and Adolescent Females and Males Separately, at Wave 1 and Wave 2

Variable	n	M (SD)		
		Total	Females	Males
Wave 1 (ages 14-15)				
Adolescents' self-reports on depressive symptoms	454	.35 (.37)	.45 (.40)	.23 (.27)***
Paternal reports on adolescent depressive symptoms	340	.19 (.23)	.18 (.20)	.19 (.26)
Maternal reports on adolescent depressive symptoms	476	.17 (.22)	.19 (.24)	.14 (.18)**
Aggregated score of adolescent depressive symptoms	316	.00 (1.0)	.16 (1.04)	-.20 (.91)*
Paternal depressive symptoms	341	1.29 (.32)		
Maternal depressive symptoms	478	1.38 (.38)		
Wave 2 (ages 16-17)				
Adolescents' self-reports on depressive symptoms	371	.46 (.41)	.57 (.43)+++	.29 (.33)***
Paternal reports on adolescent depressive symptoms	290	.19 (.21)	.20 (.22)	.19 (.20)
Maternal reports on adolescent depressive symptoms	419	.23 (.26)	.26 (.29)++	.17 (.21)** +
Aggregated score of adolescent depressive symptoms	261	.00 (1.0)	.20 (1.11)	-.26 (.75)*
Paternal depressive symptoms	290	1.30 (.34)		
Maternal depressive symptoms	416	1.36 (.39)		

Note. Variables shown in the table were based on raw scores (i.e. not logarithmic transformed). Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ; range 0-2). The aggregated scores comprised adolescents' self-reports, paternal and maternal reports on the SMFQ, and these variables are standardized. Parent depressive symptoms were measured by the depression subscale from Hopkins Symptom Checklist 25 (range 1-4). Significant adolescent gender differences at each data wave are marked with * $p < .05$, ** $p < .01$, *** $p < .001$. Significant change in adolescent depressive symptoms from w1 to w2 are marked with + $p < .05$, ++ $p < .01$, +++ $p < .001$.

Raw score mean levels of adolescent depressive symptoms, both self-reported and reported by parents, were in the range of .14 to .57 at w1 and w2 (range of SMFQ: 0-2). The prevalence of adolescent depression at w1 was 10.1% based on self-reports, 1.3% based on paternal reports, and 2.5% based on maternal reports. At w2, the prevalence of adolescent

depression was 14.2% based on self-reports, 1.5% based on paternal reports, and 3.4% based on maternal reports. Prevalence estimates reflect the percentage scoring above the cut-off (11) on the SMFQ, and only the cases in which the adolescent and his/her father and mother participated at the same data wave ($n = 316$ at w1; $n = 261$ at w2) were included in the calculations. Raw score mean levels of parent depressive symptoms were in the range of 1.29 to 1.38 (range of HSCL: 1-4) at w1 and w2. At w1, 9.4% of the fathers and 16.7% of the mothers scored above cut-off (1.75), whereas 8.3% of the fathers and 12.5% of the mothers scored above cut-off at w2.

Gender Effects

Pairwise deletion was used when conducting all paired-sample t -tests in the current study, leading to slightly different means and standard deviations than reported in Table 2. Due to the close similarity of the means and standard deviations, and for the sake of brevity, they are only reported in the table. The t -tests in the current study were also conducted with only the cases in which all three family members participated at the same data wave ($n = 316$ at w1; $n = 261$ at w2), to examine how this would affect the results. Similar patterns were found when using these somewhat lowered samples, thus the results from the additional analyses are not reported.

Levels of parent depressive symptoms. Mothers reported significantly higher levels of depressive symptoms than fathers at w1, $t(329) = 3.00, p = .003, d = 0.23$, representing a small effect size. There was no significant difference at w2, $t(276) = .77, p = .44, d = 0.06$.

Levels of adolescent depressive symptoms. When examining self-reports and maternal reports on adolescent depressive symptoms, adolescent females reported significantly higher levels of depressive symptoms than males at w1 (self-reports: $t(440) = 6.93, p < .001, d = 0.64$; maternal reports: $t(446) = 2.77, p = .006, d = 0.26$) and w2 (self-reports: $t(367) = 7.21, p < .001, d = 0.74$; maternal reports: $t(391) = 3.32, p = .001, d = 0.32$), ranging from small to medium effect sizes. However, this gender difference was not significant using paternal reports on adolescent depressive symptoms at neither w1 ($t(329) = -.48, p = .63, d = -0.05$), nor w2 ($t(283) = .43, p = .67, d = 0.05$). Significant adolescent gender effects are marked with stars in Table 2.

Stability and Change of Adolescent Females' and Males' Depressive Symptoms

Findings revealed a statistically significant increase in adolescent females' depressive symptoms measured by self-reports from w1 to w2, $t(191) = -4.22, p < .001, d = -0.31$,

representing a small effect size. When using maternal reports of adolescent depressive symptoms there were statistically significant increases in symptoms for both genders from w1 to w2 (females: $t(203) = -3.06, p = .002, d = -0.20$; males: $t(156) = -2.31, p = .02, d = -0.17$), representing small effect sizes. Significant increases in depressive symptoms were not found when examining adolescent males' self-reports ($t(132) = -1.45, p = .15, d = -0.13$), and paternal reports on adolescent females' ($t(138) = -1.52, p = .13, d = -0.13$), and males' ($t(103) = -.68, p = .50, d = -0.05$) depressive symptoms. Significant changes in levels of adolescent depressive symptoms from w1 to w2 are marked with crosses in Table 2.

Informant Discrepancies in Reports on Adolescent Depressive Symptoms

At w1, self-reports revealed significantly higher levels of adolescent depressive symptoms than paternal reports ($t(319) = 7.79, d = 0.54$), and maternal reports ($t(436) = 11.23, d = 0.62$), all $ps < .001$, representing medium-sized effects. This pattern was also evident at w2; adolescents reported significantly higher levels of depressive symptoms than paternal reports ($t(266) = 9.68, d = 0.73$), and maternal reports ($t(357) = 11.84, d = 0.69$), all $ps < .001$, representing medium-sized effects. There was no significant difference between paternal and maternal reports of adolescent depressive symptoms at neither w1 ($t(327) = 1.96, p = .05, d = 0.12$), nor w2, ($t(279) = -.610, p = .54, d = -0.03$). A graphical presentation of the informants' mean scores (not divided by gender) on the SMFQ is presented in Figure 2.

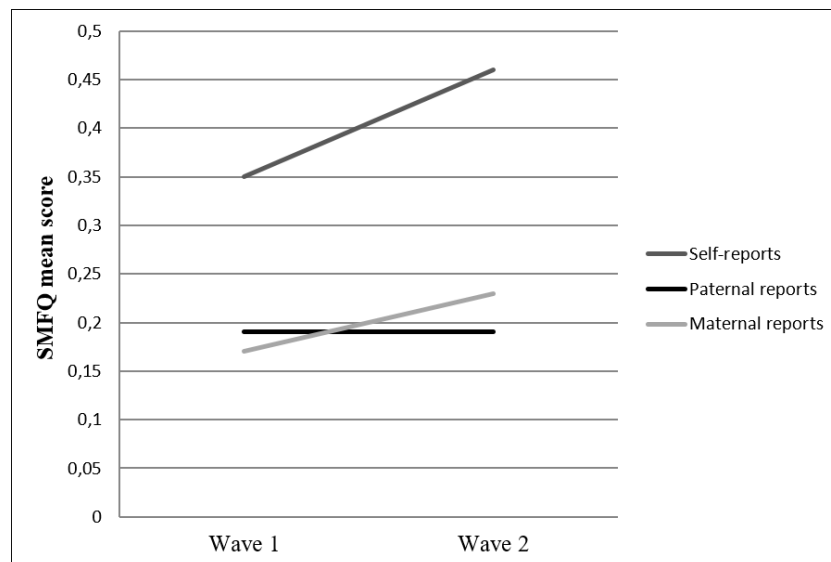


Figure 2. Informant's mean scores on the Short Mood and Feelings Questionnaire, based on raw scores.

Informant Agreement on Adolescent Depressive Symptoms

Cross-informant agreement is expressed as Pearson's correlations, which are shown in Table 3. At w1 and w2, self-reports and parent reports on adolescent depressive symptoms were moderately correlated (range $r = .28-.43$, all $ps < .001$). The cross-informant agreement between mothers and their adolescent offspring was significantly stronger than the cross-informant agreement between fathers and their adolescent offspring at w2 ($p < .05$), but not at w1 ($p > .05$). Paternal and maternal reports were moderately correlated at w1 ($r = .40$) and w2 ($r = .47$) (all $ps < .001$).

Cross-Sectional Associations

Unadjusted cross-sectional associations. Results from the correlation analyses are shown in Table 3. Correlation analyses including only those cases in which all three family members participated at the same data wave ($n = 316$ at w1; $n = 261$ at w2) were also conducted. Because these analyses revealed the same significant associations, the results are not reported any further.

Wave 1 (ages 14-15). The association between paternal and adolescent depressive symptoms at w1 was significant only when using paternal reports of adolescent depressive symptoms ($p < .001$), representing a small-sized effect. Using self-reports, maternal reports and the aggregated score resulted in non-significant associations between paternal and adolescent depressive symptoms (all $ps > .05$). The associations between maternal and adolescent depressive symptoms were significant using all informants' reports, as well as the aggregated score (all $ps < .05$), ranging from small to medium-sized effects.

Wave 2 (ages 16-17). Like at w1, the association between paternal and adolescent depressive symptoms was significant only when using paternal reports ($p < .001$), representing a medium-sized effect. Further, small-sized effects of paternal depressive symptoms were found when using maternal reports ($p = .028$) and the aggregated score ($p = .006$), in contrast to the non-significant associations at w1. The association between paternal and adolescent depressive symptoms was non-significant using self-reports ($p > .05$), like at w1. The associations between maternal and adolescent depressive symptoms were significant using all informants' reports and the aggregated score (all $ps < .05$), ranging from small to medium-sized effects, consistent with the associations at w1.

Table 3
Correlations of the Continuous Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12
Wave 1												
1. Adolescents' self-reports on depressive symptoms	-											
2. Paternal reports on adolescent depressive symptoms	.28***	-										
3. Maternal reports on adolescent depressive symptoms	.36***	.40***	-									
4. Aggregated score of adolescent depressive symptoms	.72***	.73***	.78***	-								
5. Paternal depressive symptoms	.10	.21***	-.03	.09	-							
6. Maternal depressive symptoms	.14**	.15**	.40***	.26***	.08	-						
Wave 2												
7. Adolescents' self-reports on depressive symptoms	.52***	.18**	.20***	.40***	.05	.07	-					
8. Paternal reports on adolescent depressive symptoms	.29***	.54***	.28***	.46***	.24***	.12	.29***	-				
9. Maternal reports on adolescent depressive symptoms	.33***	.29***	.54***	.48***	.10	.30***	.43***	.47***	-			
10. Aggregated score of adolescent depressive symptoms	.48***	.41***	.44***	.59***	.16*	.18**	.71***	.77***	.82***	-		
11. Paternal depressive symptoms	.05	.30***	.01	.12	.72***	.07	-.03	.33***	.13*	.17**	-	
12. Maternal depressive symptoms	.09	.12*	.35***	.22***	.04	.62***	.24***	.16**	.42***	.32***	.14*	-

Note. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ). The aggregated scores comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. Parent depressive symptoms were measured by the depression subscale from Hopkins Symptom Checklist 25. $N = 216-476$.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Adjusted cross-sectional associations.

Wave 1 (ages 14-15). Results from the multiple regression analyses of the predictor and outcome variables measured at w1 are shown in Table 4. Paternal and adolescent depressive symptoms were significantly associated when paternal reports on adolescent depressive symptoms was used as outcome variable ($p = .001$). The associations between paternal and adolescent depressive symptoms were non-significant when using self-reports, maternal reports and the aggregated score (all $ps > .05$), like the unadjusted associations at w1. The associations between maternal and adolescent depressive symptoms were statistically significant using maternal reports and the aggregated score (all $ps < .001$). Using self-reports and paternal reports resulted in non-significant associations between maternal and adolescent depressive symptoms.

The cross-sectional final models (comprising all predictor variables) at w1 collectively explained 11% of the variance in adolescent depressive symptoms when using self-reports, 6% when using paternal reports, 17% when using maternal reports, and 12 % when using the aggregated score as the outcome variable.

Wave 2 (ages 16-17). Results from the multiple regression analyses at w2 are shown in Table 5. Most of the significant associations found at w1 were also found at w2; however, some new significant associations emerged. In consistence with the results at w1, the association between paternal and adolescent depressive symptoms was significant only when using paternal reports on adolescent depressive symptoms as the outcome variable ($p < .001$). However, the association between paternal and adolescent depressive symptoms was also significant using the aggregated score ($p = .005$), which differed from the results at w1.

Like the results at w1, the association between and adolescent depressive symptoms was significant when using maternal reports and the aggregated score (all $ps < .001$). Unlike the results at w1, there was also a significant association between maternal depressive symptoms and adolescents' self-reported depressive symptoms ($p = .01$). This result is noteworthy, being the only significant association between parent and self-reported adolescent depressive symptoms in all analyses in the current study.

The predictor variables in the final models at w2 collectively accounted for 17% of the variation in adolescent depressive symptoms when using self-reports, 12% when using paternal reports, 19 % when using maternal reports, and 18% when using the aggregated score as the outcome variable.

Table 4

Multiple Regression Analyses of the Cross-Sectional Associations at Wave 1

Variable	Informant on adolescent depressive symptoms							
	Aggregated score		Adolescents		Fathers		Mothers	
	β	p	β	p	β	p	β	p
Paternal depressive symptoms	.08	.143	.10	.072	.18	.001	-.06	.244
Maternal depressive symptoms	.25	< .001	.09	.110	.13	.024	.38	< .001
Adolescent gender ^a	.19	< .001	.28	< .001	.01	.908	.10	.07
Living arrangement with father (paternal reports) ^b	-.11	.049	-.08	.163	-.04	.428	-.10	.060
Total R^2	.12		.11		.06		.17	

Note. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ). The aggregated score comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. Parent depressive symptoms were measured by the depression subscale from the Hopkins Symptom Checklist 25. All predictor and outcome variables were measured at w1. Significant associations ($p < .05$) are in bold. $N = 311$ -322.

^a0 = males, 1 = females. ^b0 = living with his child less than full-time, 1 = living with his child full-time.

Table 5

Multiple Regression Analyses of the Cross-Sectional Associations at Wave 2

Variable	Informant on adolescent depressive symptoms							
	Aggregated score		Adolescents		Fathers		Mothers	
	β	p	β	p	β	p	β	p
Paternal depressive symptoms	.17	.005	-.01	.895	.30	< .001	.08	.158
Maternal depressive symptoms	.27	< .001	.15	.01	.12	.05	.36	< .001
Adolescent gender ^a	.21	< .001	.36	< .001	.03	.618	.10	.096
Total R^2	.18		.17		.12		.19	

Note. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ). The aggregated score comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. Parent depressive symptoms were measured by the depression subscale from the Hopkins Symptom Checklist 25. All predictor and outcome variables were measured at w2. Significant associations ($p < .05$) are in bold. The analyses were also adjusted for *Living arrangement with mother* (maternal reports) and *Living arrangement with father* (paternal reports), but these variables made no significant contributions in the final models, and are thus not reported here. $N = 252$ -269.

^a0 = males, 1 = females.

Longitudinal Associations from Wave 1 to Wave 2

Unadjusted longitudinal associations. Results from the correlation analyses can be seen in Table 3. Paternal depressive symptoms at w1 and adolescent depressive symptoms at w2 were weakly correlated when using paternal reports on adolescent depressive symptoms as outcome variable ($p < .001$), and weakly correlated using the aggregated score ($p = .02$). The associations were non-significant for all other informants' reports (all $ps > .05$). Noteworthy, the same pattern emerged when examining the correlations between maternal depressive symptoms at w1 and adolescent depressive symptoms at w2; the associations were only statistically significant when using maternal reports on adolescent depressive symptoms ($p < .001$), and the aggregated score ($p = .005$), representing small to medium-sized effects.

Adjusted longitudinal associations. Results from the longitudinal multiple regression analyses are shown in Table 6. Paternal depressive symptoms measured at w1 had a significant predictive effect on adolescent subsequent (i.e. measured at w2) depressive symptoms only when using paternal reports on adolescent depressive symptoms as the outcome variable ($p = .034$). Using all other reports, the associations between paternal and adolescent subsequent depressive symptoms were non-significant (all $ps > .05$).

Table 6

Multiple Regression Analyses of the Longitudinal Associations

Variable	Informant on adolescent depressive symptoms							
	Aggregated score		Adolescents		Fathers		Mothers	
	β	p	β	p	β	p	β	p
Paternal depressive symptoms	.10	.065	.01	.921	.12	.034	.10	.07
Maternal depressive symptoms	.01	.849	.01	.926	.09	.105	.00	.97
Adolescents' prior depressive symptoms ^a	.55	< .001	.46	< .001	.53	< .001	.50	< .001
Adolescent gender ^b	.11	.061	.26	< .001	.02	.764	.07	.242
Total R^2	.39		.35		.33		.30	

Note. Adolescent depressive symptoms were measured at w2 by the Short Mood and Feelings Questionnaire (SMFQ). The aggregated score comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. Parent depressive symptoms were measured at w1 by the depression subscale from the Hopkins Symptom Checklist 25. Significant associations ($p < .05$) are in bold. All analyses were adjusted for *Living arrangement with father* (paternal reports at w2) and *Living arrangement with mother* (maternal reports at w2), but these variables made no significant contributions to the final models, and are thus not reported here. $N = 215-238$.

^aIn each analysis, the informant on adolescents' prior depressive symptoms was the same as the informant on the outcome variable. ^b0 = males, 1 = females.

In contrast, maternal depressive symptoms had no predictive effect on adolescent subsequent depressive symptoms in any of the analyses (all $ps > .05$), independent of type of informant and the aggregated score as outcome variables. The predictor variables in the final longitudinal models collectively explained 30-39% of the variance in adolescent depressive symptoms at w2, with the highest score for the analyses using the aggregated score as the outcome variable.

A summary of the cross-sectional and longitudinal associations between paternal depressive symptoms and adolescent depressive symptoms is illustrated in Figure 3.

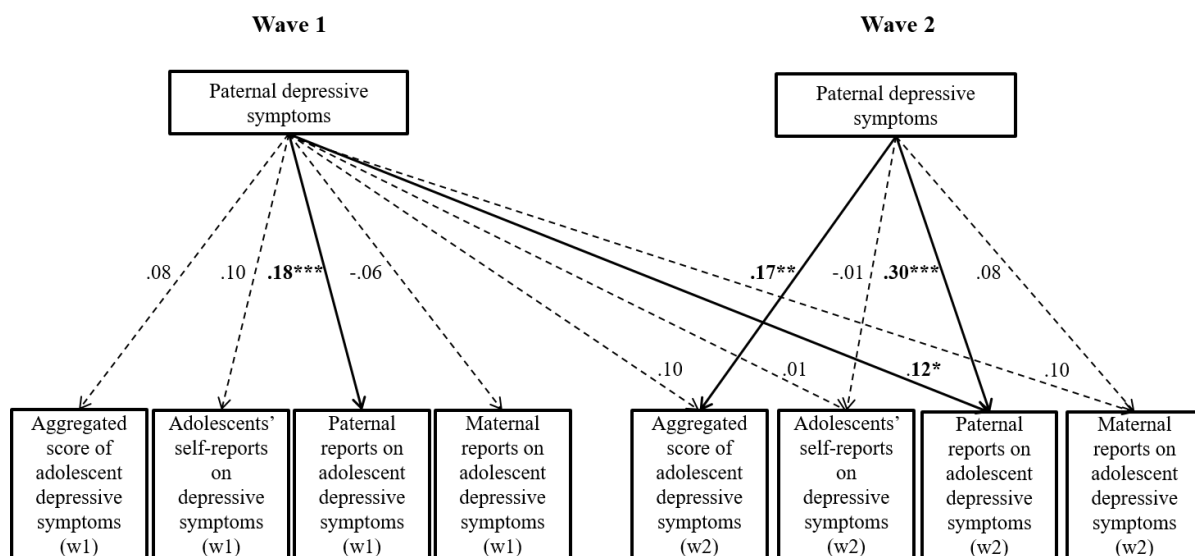


Figure 3. Summary of the adjusted associations cross-sectionally at w1 and w2, and longitudinally from w1 to w2. All analyses were adjusted for adolescent gender and adolescents' living arrangements with parents. The longitudinal analyses were also adjusted for adolescents' prior depressive symptoms. Reported figures are standardized beta coefficients from the multiple regression analyses. Dotted lines = nonsignificant associations. Bold lines = significant associations. $N = 215-322$.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Moderation Effect of Adolescent Gender

Results from the moderated regression analyses revealed no significant moderation effect of adolescent gender on the association between paternal and adolescent depressive symptoms. The results were non-significant in the cross-sectional analyses at w1 and w2 and the longitudinal analyses, for all informants' reports on adolescent depressive symptoms as outcome variables. For the sake of brevity, the non-significant associations are not further reported, and the reader is referred to Table G1 (cross-sectional associations) and Table G2 (longitudinal associations) in [Appendix G](#) for the results.

Discussion

The main purpose of the current study was to examine the association between paternal and adolescent depressive symptoms cross-sectionally and longitudinally using multi-informant data from a community-based prospective study (the TOPP study). The following key findings emerged: (a) The cross-sectional and longitudinal associations between paternal and adolescent depressive symptoms varied depending on which informants' report on adolescent depressive symptoms was used, and the association was only significant when using paternal reports; (b) Adolescents' self-reports revealed higher levels of depressive symptoms than parent reports on adolescent depressive symptoms, and the prevalence estimate of adolescent depression was considerably higher based on adolescents' self-reports compared to parent reports; (c) Correlations between parent and adolescent reports on adolescent depressive symptoms revealed moderate agreement; (d) Higher levels of depressive symptoms for adolescent females than males was found, except when relying on paternal reports; (e) Adolescent females' depressive symptoms significantly increased from ages 14-15 (w1) to ages 16-17 (w2), when relying on self-reports and maternal reports; (f) Adolescent gender did not moderate the association between paternal and adolescent depressive symptoms. The key findings are discussed in the following sections.

Informants' Reports on Adolescent Depressive Symptoms

Informant discrepancies. The levels of adolescent depressive symptoms and prevalence estimates varied considerably between the different informants' reports. At both data waves, self-reports revealed higher levels of adolescent depressive symptoms compared to parent reports. This finding is consistent with previous research using community-based samples, revealing informant discrepancies between adolescents and their parents on adolescent emotional problems (e.g. Sourander et al., 1999). The hypothesis regarding informant discrepancies (H_1) was hence confirmed. There were no significant differences between paternal and maternal reports on adolescent depressive symptoms at either data wave. This finding is not in accordance with another Norwegian study by Gere et al. (2013), who found that mothers reported higher levels of depressive symptoms in their children compared to fathers. However, the children in the study by Gere et al. (2013) were younger (aged 7-13) than the adolescents in the current study, which might explain the inconsistent findings.

The same pattern of informant discrepancies was seen for the prevalence estimates of adolescent depression (last two weeks); the prevalence was remarkably higher when self-reports on adolescent depressive symptoms were used compared to parent reports. At the most, the prevalence estimate was more than nine times higher when using self-reports (14.2%) compared to paternal reports (1.5%) (at w2). This suggests that a considerable share of depressed adolescents is not detected when fathers report on adolescent depressive symptoms. The prevalence of self-reported adolescent depression at ages 14-15 was approximately the same as the prevalence estimate found by another Norwegian study by Sund et al. (2011), in which the prevalence for any current (last 2 months) depressive disorders for adolescents (mean age 14.9) was 9.4%. In the current study, the prevalence of adolescent depression at ages 16-17 was somewhat higher, which is in line with prior literature showing that depressive symptoms increase during adolescence, especially for females (e.g. Dekker et al., 2007; Twenge & Nolen-Hoeksema, 2002).

It should be noted that there is a lack of agreement in the literature regarding the suitable cut-off for depression on the SMFQ (McKenzie et al., 2011; Rhew et al., 2010; Turner et al., 2014). However, the cut-off most commonly used is a total score of 11 (e.g. Thapar & McGuffin, 1998; Turner et al., 2014), although some studies have revealed empirical cut-offs of lower total scores (e.g. McKenzie et al., 2011; Rhew et al., 2010). The cut-off being used in the current study was 11, resulting in more conservative prevalence estimates than one would get if a lower cut-off was used. Importantly, scoring above cut-off on the SMFQ does not imply the existence of a clinical depression diagnosis.

There are several possible reasons for the discrepancies between parent reports and self-reports on adolescent depressive symptoms (for more information, see Achenbach, 2006; E. T. Barker, Bornstein, Putnick, Hendricks, & Suwalsky, 2007; De Los Reyes, 2011; Kraemer et al., 2003; Richters, 1992; Treutler & Epkins, 2003). Parents might not have accurate information about the depressive symptoms their child is experiencing, because thoughts and emotions are less obvious to the parent than overt behavior (van der Ende, Verhulst, & Tiemeier, 2012). As children enter adolescence, they spend increasingly more time outside the home, making it even more difficult for parents to observe them (Sourander et al., 1999). Further, adolescents might avoid talking with their parents about personal problems (Sourander et al., 1999). This could possibly be due to family conflicts or strained parent-adolescent relationships, which are likely to occur in adolescence because of the relational challenges associated with this developmental period (E. T. Barker et al., 2007).

Consistent with these explanations, parents may be less accurate reporters of adolescent depressive symptoms, a hypothesis which has been supported in previous studies (e.g. A. J. Lewis et al., 2014). However, informant discrepancies have recently been understood as something more than measurement error, namely that multiple informants provide different, but legitimate, perspectives on psychopathology in children and adolescents (De Los Reyes, 2011; De Los Reyes et al., 2015; De Los Reyes et al., 2013). In accordance with this standpoint, there is an increasing agreement about collecting data from an array of informants in order to get a more valid picture (Achenbach, 2006; Kraemer et al., 2003).

Informant agreement. The cross-informant agreement on adolescent depressive symptoms between fathers and mothers in the current study was somewhat lower than the mean correlation between parents in a meta-analysis by Achenbach et al. (1987) ($r = .59$). Further, findings from the current study show that parent reports and self-reports on adolescent depressive symptoms were moderately correlated, confirming the hypothesis regarding informant agreement (H_2). The cross-informant agreement between fathers and their adolescent offspring in the current study ($r = .28-.29$) is in accordance with the findings from the meta-analysis by Achenbach et al. (1987), in which the mean correlation between children's self-reports and parent reports was $r = .25$. However, findings from a Norwegian twin study utilizing multi-informant data from adolescents, mothers and fathers on adolescent depressive symptoms revealed a cross-informant agreement between fathers and their adolescent offspring of $r = .36$ (Ask, Waaktaar, Seglem, & Torgersen, 2015), indicating that the cross-informant agreement in the current study might be somewhat low.

At 14-15 years of age, there was a tendency for stronger cross-informant agreement between mothers and their adolescent offspring than between fathers and their adolescent offspring, although not a significant difference. At 16-17 years of age, however, the cross-informant agreement between mothers and their adolescent offspring was significantly stronger than between fathers and their adolescent offspring. This finding suggests that mothers to a greater extent than fathers agree with their adolescent offspring on adolescent levels of depressive symptoms. It might be that mothers have a closer relationship with their adolescent offspring, possibly resulting in more open communication about feelings. Consequently, mothers may have a greater awareness of their child's emotional state than fathers. Future studies should investigate to what degree the parent-child relationship influence the agreement between parents and adolescents on adolescent depressive symptoms.

Gender Effects

Adolescent depressive symptoms. Adolescent females reported higher levels of depressive symptoms at ages 14-15 and 16-17 compared to adolescent males, as expected in the third hypothesis (H₃) of the current study. This gender difference was also present when examining maternal reports and the aggregated score. The finding is consistent with several previous studies (e.g. Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Kiss et al., 2007), including other Norwegian community-based studies of adolescents (e.g. Agerup et al., 2014; Lundervold et al., 2013). Several explanations have been proposed to explain the gender difference in the prevalence of depression, such as gender-related hormonal and biological changes during puberty, different coping patterns, amount and type of stress encountered, and socialization of affective and cognitive vulnerabilities in females (for reviews, see Goodman & Tully, 2006; Grigoriadis & Robinson, 2007; Hilt & Nolen-Hoeksema, 2014). Interestingly, when paternal reports on adolescent depressive symptoms were examined, no gender difference between adolescent females and males emerged. This finding shows that fathers not only report lower levels of adolescent depressive symptoms than adolescents' self-reports, they also do not report any gender difference between levels of depressive symptoms in their adolescent offspring.

Further, findings from the current study revealed that adolescent depressive symptoms significantly increased from ages 14-15 to 16-17 for adolescent females, based on self-reports and maternal reports. This is in line with the findings from a meta-analysis revealing that females' depression scores increased between the ages of 12 and 16, whereas males' scores remained stable after age 13 (Twenge & Nolen-Hoeksema, 2002). Paternal reports of adolescent depressive symptoms in the current study indicated no significant increase in adolescent females' depressive symptoms from ages 14-15 to 16-17. Again, paternal reports of adolescent depressive symptoms differed from adolescents' self-reports. When mothers reported on adolescent depressive symptoms in the current study, a significant increase in depressive symptoms for adolescent males was also found. An increase in adolescent males' self-reported depressive symptoms was also found, but the increase was not statistically significant. The fourth hypothesis of the current study was confirmed when relying on self-reports and maternal reports, but not paternal reports.

Parent depressive symptoms. Gender differences between the levels of parent depressive symptoms were also evident; mothers reported significantly higher levels of depressive symptoms compared to fathers when offspring were 14-15 years. This finding is in

line with prior studies on self-reported depressive symptoms in Norwegian adults (e.g. Gere et al., 2013; Strand et al., 2003). However, there was no significant gender difference between parents' levels of depressive symptoms when offspring were 16-17 years, hence the hypothesis about gender effects (H₃) was only partly confirmed. One possible explanation for the lack of parental gender effects at w2 is that mothers with the highest levels of depressive symptoms may have dropped out of the study from w1 to w2, reducing the difference between paternal and maternal levels of depressive symptoms at w2. The attrition rate for mothers from w1 to w2 was almost twice as large as the paternal attrition rate, which may have affected the results.

Some of the parents reported elevated levels of depressive symptoms, scoring above the cut-off on the HSCL; 9.38% of the fathers, and 16.73% of the mothers scored above cut-off when their offspring were 14-15 years. When adolescents were 16-17 years, 8.27% of the fathers and 12.5% of the mothers scored above cut-off. These findings indicate that a substantial number of parents were depressed when their offspring were in middle and late adolescence. Another Norwegian community-based study by Strand et al. (2003) revealed that 4% of the adult men, and 13.6% of the adult women scored above cut-off on the HSCL-25. The prevalence of maternal depression in the current study is in line with the prevalence of depression in adult women in the study by Strand et al. (2003), whereas the prevalence of paternal depression in the current study was more than twice as large as the prevalence of depression for the adult men in the study by Strand et al. (2003). It should be noted that the entire HSCL-25 (i.e. depression and anxiety subscales) was used in the study by Strand et al. (2003), thus limiting the comparison of the studies. Also, the prevalence estimates in the current study might be somewhat overestimated, because the cut-off applied is made for the entire HSCL-25 (i.e. both the depression and anxiety subscale), whereas only the depression subscale was used in the current study.

Moderation effect of adolescent gender. In the current study, there was no significant interaction effect of adolescent gender on the association between paternal and adolescent depressive symptoms, neither cross-sectionally or longitudinally, independent of which informants' report on adolescent depressive symptoms was used. Due to inconsistent findings in previous studies, the research question on the moderating effect of adolescent gender was exploratory and no hypothesis was stated. Findings from the current study are in accordance with findings from previous studies in which no moderation effect of adolescent gender was found (e.g. Ranøyen et al., 2014), but not consistent with results from other

studies revealing a significant moderation effect of adolescent gender (e.g. Reeb & Conger, 2009). There are several possible explanations for the absence of a moderating effect of adolescent gender in the current study. It might be due to the lack of statistical power in the current study, because more power is needed to detect significant interactions than significant main effects (Aguinis & Stone-Romero, 1997). Further, it could also be due to the increasing gender equality in Norway, which possibly increases the similarity of the ways parents treat their children, thus minimizing the differences between how adolescent females and males are affected by parental depression.

The Association between Paternal and Adolescent Depressive Symptoms

The main purpose of the current study was to examine the association between paternal and adolescent depressive symptoms, and how the use of different informants' reports on adolescent depressive symptoms affect this association. These research questions were exploratory in nature and no hypotheses were stated. Findings from the current study indicate that there are some tendencies for paternal depressive symptoms to be associated with adolescent depressive symptoms; paternal depressive symptoms were consistently associated with adolescent depressive symptoms when fathers reported on their own and their offspring's depressive symptoms. The significant associations were present both cross-sectionally at ages 14-15 and 16-17, and longitudinally from ages 14-15 to 16-17. These associations remained significant when adjusting for relevant confounding variables (i.e. maternal depressive symptoms, adolescent gender, adolescents' living arrangement with parents and adolescents' prior depressive symptoms). The findings are in accordance with the growing evidence indicating an increased risk for depression among offspring of depressed fathers (for meta-analysis, see Kane & Garber, 2004).

However, when using the other informants' reports (i.e. self-reports and maternal reports) on adolescent depressive symptoms and the aggregated scores, significant associations between paternal and adolescent depressive symptoms were in general not found. Most importantly, when adolescents reported on their own depressive symptoms, no significant effects of paternal depressive symptoms was found, neither cross-sectionally at ages 14-15 and 16-17, nor longitudinally from ages 14-15 to 16-17. This finding is in accordance with the results from another Norwegian community-based study by Agerup et al. (2015), in which no significant association between paternal internalizing problems and adolescent depression was found. It should be noted that findings from the current study are not directly comparable with the findings from the study by Agerup et al. (2015), because

they measured paternal internalizing problems (and not depressive symptoms specifically), and because adolescents were categorized based on clinical depression diagnoses.

Nevertheless, findings from the current study might suggest that the findings from the study by Agerup et al. (2015) also apply to subdiagnostic depression in community-based samples.

Findings from the current study are inconsistent with previous studies demonstrating significant effects between paternal and adolescent offspring depressive symptoms. With regard to Norwegian studies, findings from the current study are not in accordance with the findings from a community-based study by Ranøyen et al. (2014), revealing a significant association between paternal internalizing problems (i.e. anxiety/depression) and adolescents' self-reported internalizing problems. A considerable strength of their study was the large sample comprising 5732 adolescents, substantially increasing the power to detect significant effects. Because the study by Ranøyen et al. (2014) did not measure depressive symptoms specifically, the comparison with findings from the current study is somewhat limited.

Further, findings from the longitudinal analyses in the current study are not in accordance with previous longitudinal studies on adolescent offspring using similar methodological design as the current study (i.e. adjusting for maternal depressive symptoms and adolescents' prior depressive symptoms, using approximately the same sample size). One study is particularly relevant to the current study; a community-based study by Reeb and Conger (2009) found a significant association between paternal depressive symptoms and self-reported adolescent depressive symptoms one year later. The contrasting findings between the North American study by Reeb and Conger (2009) and the current study raise questions about cultural differences in fathering.

Effects of maternal depressive symptoms. Although not the main focus of the current study, findings revealed that maternal and adolescent depressive symptoms were significantly associated when adolescents' reported on their own depressive symptoms at ages 16-17. This finding is noteworthy, because it was the only significant relationship found between parent depressive symptoms and adolescents' self-reported depressive symptoms in the current study. The finding is in accordance with a meta-analysis by Connell and Goodman (2002), revealing stronger associations between maternal and offspring psychopathology than between paternal and offspring psychopathology, as well as another Norwegian study in which only maternal, and not paternal, internalizing problems were associated with adolescent depression (Agerup et al., 2015). However, there was no significant association between maternal and adolescent depressive symptoms at ages 14-15, and no significant predictive

effect of maternal depressive symptoms from ages 14-15 to 16-17 when using adolescents' self-reports.

Although Norwegian fathers are more involved in caretaking of their younger children today compared to earlier decades (Kitterød & Rønsen, 2013), it could be that mothers are more involved in the lives of their children in late adolescence than fathers. For this reason, maternal depression might influence offspring's mental health to a greater extent than paternal depression. Future studies should investigate potential mechanisms underlying the significant association between maternal and offspring depressive symptoms at ages 16-17. Also, future studies on the association between parent and adolescent depressive symptoms should use more recent birth cohorts, because parent roles and other relevant family variables might have changed from the 2000s (in which the data used in the current study was collected) to the 2010s.

Inconsistencies in Associations When Using Different Informants' Reports

Significant associations when using paternal reports. There are several explanations for the inconsistent findings on the association between paternal and adolescent depressive symptoms when different informants' reports on adolescent depressive symptoms were used as outcome variables. First and foremost, it is possible that the significant relationship between paternal and adolescent depressive symptoms when fathers reported on adolescent depressive symptoms reflect a bias. The magnitude of the correlation coefficients for the associations between paternal and adolescent depressive symptoms were in some instances more than twice as large when fathers reported on adolescent depressive symptoms compared to adolescents' self-reports, suggesting that the estimations could be inflated. One potential reason for this is that the depressed fathers might report higher levels of offspring depressive symptoms than non-depressed fathers. This is in accordance with the depression-distortion hypothesis (Richters, 1992), suggesting a negative bias in reporting on the child's problems due to the parent's level of depressive symptoms. Previous studies have supported this hypothesis, revealing that depressed mothers (Kiss et al., 2007; Youngstrom, Loeber, & Stouthamer-Loeber, 2000) and depressed fathers (e.g. Treutler & Epkins, 2003) over-report problems in their offspring. Paternal depressive symptoms could account for the systematic variance between paternal and adolescent depressive symptoms which is unrelated to the true score variance between these two variables (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003).

The stronger associations found when fathers reported on adolescent depressive symptoms could also be explained by shared method variance, which occurs when the same informant reports on both the predictor and outcome variable. Some portion of the variance between the predictor and outcome variable might thus be the result of some characteristics of the informant, like the informant's mood state (Podsakoff et al., 2003; Podsakoff, MacKenzie, & Podsakoff, 2012; Ringoot et al., 2015). Indeed, previous studies have found that the association between paternal and offspring depression is inflated when fathers report on both their child's depression and their own depression (e.g. Ringoot et al., 2015). Inflated associations possibly due to shared method variance were not specific for fathers in the current study, but were also observed for maternal depressive symptoms. It should be noted that the effect of shared method variance was reduced in the longitudinal analyses, because of the temporal separation of the predictor and the outcome variable (Podsakoff et al., 2003); parental mood state (e.g. depressive symptoms) might have changed from w1 to w2, potentially changing parents reports on their own and offspring depressive symptoms.

Lack of significant associations when using adolescents' self-reports. There are several potential reasons for the lack of significant association between paternal and adolescent depressive symptoms when using adolescents' self-reports in the current study. The lack of findings is interesting and perhaps somewhat surprising, given the that the heritability of depressive symptoms in adolescence is around 40% (for review, see Rice, 2010). However, it should be noted that some studies have found lower heritability for depressive symptoms among adolescents, such as a Norwegian twin study revealing that the heritability was 25% (Ask et al., 2015), indicating that shared and non-shared environmental factors play a larger role than shared genetic liabilities. Moreover, partly different genetic factors might influence adolescent depressive symptoms (e.g. at age 15) and parental depressive symptoms (e.g. at age 45), thus implying lower parent-offspring associations than would be expected from the heritability estimates. Indeed, findings indicate that new genetic influences emerge and others are attenuated during life (Kendler, Gardner, & Lichtenstein, 2008). In theory, epigenetic mechanisms may also play a role in the development of depressive tendencies (for review, see Lau, Lester, Hodgson, & Eley, 2014), and possibly contribute to reduced paternal-offspring associations in the current study.

It might be that depressive symptoms in Norwegian adolescents to a larger extent are associated with other factors than paternal depressive symptoms. Certainly, a substantial proportion of the variance in adolescent depressive symptoms remained unexplained by the

predictor variables and confounding variables, especially in the cross-sectional analyses. The final models, independent of informant and type of design (i.e. cross-sectional or longitudinal), explained 6-39% of the variation in adolescent depressive symptoms, indicating that other important predictors of adolescent depressive symptoms exist. When children enter adolescence, less time is spent with the family (Larson, Richards, Moneta, Holmbeck, & Duckett, 1996), possibly making psychosocial factors related to social arenas of friends, school and leisure activity more influential on psychological well-being and distress in adolescence (Mykkestad et al., 2012). In line with this, a Norwegian study by Mykkestad et al. (2012) revealed that psychological distress (i.e. anxiety and depressive symptoms) among adolescents was more strongly related to academic-related problems and being bullied at school than parental psychological distress. The greater influence of psychosocial factors compared to parental psychopathology might explain the relatively low degree of variance in adolescent depressive symptoms explained by the final models in the current study.

The lack of significant findings might also be explained by the design of the current study, and the characteristics and size of the sample. First, the two-year time span for the longitudinal analyses might not be the optimal time span to detect predictive effects of paternal depressive symptoms on offspring. Parents were asked to report the presence of depressive symptoms during the last week, whereas adolescents and parents were asked to report the presence of adolescent depressive symptoms during the last two weeks. It is possible that the reports of depressive symptoms reflected rather transient mood states, which could explain why paternal depressive symptoms did not have an effect on adolescent self-reported depressive symptoms measured two years later. If paternal depressive symptoms have short-term effects on offspring depressive symptoms in middle and late adolescence, a shorter time-span could possibly reveal different results. However, the effects of paternal depressive symptoms might not be detected in cross-sectional analyses if paternal depressive symptoms only have been present for a short period (e.g. the past week). Findings from a North American study by Reeb and Conger (2009) revealed significant effects of paternal depressive symptoms on adolescent depressive symptoms one year later, suggesting that one year might be a more optimal follow-up time.

Also, it might be that significant long-term effects of paternal depressive symptoms could have been found if paternal depressive symptoms were measured during an earlier developmental stage than adolescence. A study by Nilsen et al. (2013), also using data from the TOPP study, revealed that maternal distress (i.e. anxiety and depressive symptoms)

measured in early childhood significantly predicted adolescent depressive symptoms, whereas maternal distress in preadolescence did not predict adolescent depressive symptoms. This long-term effect of maternal distress might reflect heritability, or imply that that younger childhood is a specific vulnerable period for later development of depressive symptoms (Nilsen et al., 2013). It might be that paternal depression in younger childhood have the same long-term effects as maternal distress, being evident when the offspring reach adolescence. The TOPP study did not include fathers until offspring were 14-15 years of age, thus an investigation of the long-term effects of paternal depressive symptoms has not been possible. However, a longitudinal Norwegian study by Ranøyen et al. (2015) examining the predictive effect of paternal anxiety/depression measured when offspring were in preschool age, revealed that the effect on adolescent offspring anxiety/depression was entirely mediated by paternal symptoms measured when offspring were adolescent. Because of the limited studies of the long-term effects of paternal depressive symptoms, more studies should be conducted to replicate the findings by Ranøyen et al. (2015).

Second, a predominance of low levels of depressive symptoms among the parents and adolescents in the current study might explain the null-findings. Although some of the participants in the current sample reported elevated levels of depressive symptoms as reflected by scores above cut-off on the SMFQ and the HSCL, mean levels were generally low. Previous studies have shown that the association between depression in fathers and offspring is limited to the more severe cases of depression in offspring (e.g. Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005). It is possible that the adolescents and fathers whose symptoms would meet the diagnostic criteria for a depressive disorder dropped out of the study from w1 to w2, or did not participate in the first place. This might have reduced the chances of achieving significant effects.

Third, the number of fathers included in the current study might have been too low to achieve significant effects, as more cases are needed to demonstrate small effects (Tabachnick & Fidell, 2013). Indeed, in some of the analyses, the association between paternal and adolescent depressive symptoms nearly reached significant level, for instance in the cross-sectional analyses at ages 14-15 when using self-reports on adolescent depressive symptoms ($\beta = .10, p = .072$).

Fourth, it is possible that adolescents are more able to avoid the stressors associated with parental depression, or to seek social support from peers or other adults than younger children (Nilsen, 2012), which can counteract the negative effects of having a depressed

parent. Indeed, findings from a Norwegian study by Mykkestad et al. (2012) demonstrated that social support from friends and spending time with friends were the strongest protective factors for psychological distress. Moreover, Nilsen (2012) found that lack of friend support predicted depressive symptoms in adolescent females. Future research should further examine if social support protects adolescents from the negative effects of paternal depression.

Lastly, lack of significant associations between paternal and self-reported adolescent depressive symptoms could be due to some other protective factors such as positive paternal characteristics. In a study by E. D. Barker, Copeland, Maughan, Jaffee, and Uher (2012), 37% of the association between maternal depression and internalizing disorders in their offspring was explained by increased exposure to risk factors such as low socioeconomic status and low educational attainment. Parents in the current sample reported having somewhat higher levels of education and family income compared to the general population, suggesting that the parents in the current study were quite well-functioning. Although some of the fathers reported elevated levels of depressive symptoms, it might be that they had good coping strategies, thus reducing the adverse effects of paternal depressive symptoms on their offspring. The influence of paternal functional status on the intergenerational transmission of depression from fathers to their offspring should be examined in future research.

Strengths and Limitations

Strengths. The current study fills a gap in the research field, by investigating the associations between depressive symptoms in Norwegian fathers and their adolescent offspring in middle and late adolescence, using a community-based sample. A considerable strength of the current study was the methodologically sound design in which associations were examined both cross-sectionally and longitudinally, before and after adjusting for maternal depressive symptoms, adolescent gender, adolescents' living arrangement with parents, as well as adolescents' prior depressive symptoms in the longitudinal analyses. Further, the measures used in the current study (i.e. the SMFQ and the depression subscale from HSCL-25) had acceptable to good internal consistency at both data waves.

The current study also addressed issues related to the use of different informants on adolescent depressive symptoms (i.e. informant discrepancies and informant agreement), by demonstrating considerable discrepancies between parent and adolescent reports on the levels of adolescent depressive symptoms and prevalence estimates of adolescent depression. Further, by running separate analyses for self-reports, paternal and maternal reports on

adolescent depressive symptoms, the current study illustrated that the strength of association between paternal and adolescent depressive symptoms varied depending on which informants' report was used.

Limitations. Some methodological limitations of the current study must be addressed. First, the current study did not address the reversed direction of effects in the longitudinal analyses. It is possible that adolescent depressive symptoms increase the risk of depressive symptoms in their fathers. This would be in line with the transactional model by Sameroff and MacKenzie (2003), positing continuous reciprocal influences between children and their context. Indeed, some recent evidence indicates that paternal depressive symptoms and internalizing problems in adolescent offspring are reciprocally related (e.g. Fanti et al., 2013).

Further, aggregating informants' reports using principal component analysis (PCA) may not be the optimal way to combine scores from multiple informants. One reason is that PCA-aggregated scores might reflect non-random measurement error (Kim & Mueller, 1978). For instance, if paternal reports of adolescent depressive symptoms are significantly associated with paternal depressive symptoms due to shared method variance, this bias would also be present in the analyses using the PCA-aggregated score because this score is a "summary" of the informants' reports. Further, there should be substantial agreement between informants for the PCA-aggregated score to be a meaningful expression of all informants' reports, although a scientific consensus on the accepted level of agreement does not exist (De Pauw et al., 2009). The agreement between informants' reports in the current study was moderate, but the internal reliability of the aggregated scores was below the conventionally acceptable level of alpha ($\alpha < .70$) (DeVellis, 2003), raising questions about the usefulness of the aggregated scores in the current study.

Lastly, the participants in the current study generally reported low levels of depressive symptoms, as well as higher parent educational levels and family income than the general population. Findings from the current study might thus not generalize to clinical populations, and populations with lower socioeconomic status. Replication with more diverse samples is necessary in order to increase the confidence in the external validity of the results. Future studies should to a larger extent strive to include social groups known to be at risk for higher levels of depressive symptoms, such as families with low socioeconomic status and ethnic minority groups (Sund et al., 2003).

Implications and Directions for Future Research

Scientific implications. Findings from the current study have some important scientific implications. First, the findings demonstrated that the levels of adolescent depressive symptoms varied depending on which informants' report was examined; adolescents' reports at ages 14-15 and 16-17 revealed higher levels of depressive symptoms than parent reports on adolescent depressive symptoms. The discrepancy between informants' reports was even more apparent when examining the prevalence estimates of adolescent depression; prevalence estimates were considerably higher when adolescents reported on their own depressive symptoms compared to parent reports. This finding is very important, because it shows that a substantial share of depressed adolescents may not be detected if one relies on parent reports. Thus, researchers who are examining adolescent depressive symptoms in the general population should keep in mind that parent and adolescent reports might be discrepant. Preferably, reports should be obtained from both the adolescent and his/her parents.

Second, findings revealed in the current study contribute to the interpretation of previous research, and the conduction of future research on the intergenerational transmission of depression from fathers to their offspring. The results revealing that the strength of association between paternal and adolescent depressive symptoms varied depending on which informants report on adolescent depressive symptoms was used, might explain why some studies find significant associations and others don't. Findings from the current study also suggest that the association might be inflated if fathers report on their own depressive symptoms and offspring depressive symptoms, possibly due to over-reporting by depressed fathers or because of shared method variance. Thus, when reading and evaluating research literature on the effects of paternal depression, one should consider who is reporting on offspring depressive symptoms. Based on findings from the current study, researchers should avoid using single informant data, and rather utilize multi-informant data on offspring depressive symptoms.

Clinical implications. The findings also have some clinical implications. The knowledge gained from the current study has the potential to help mental health professionals identify those likely to be at greater risk of developing depressive symptoms in adolescence. Findings from the current study add to the findings from previous studies showing that adolescent females in the general population report higher levels of depressive symptoms than

adolescent males, and that adolescent females' self-reported depressive symptoms increase from ages 14-15 to 16-17. Mental health professionals should be aware of the higher prevalence of depression in adolescent females when conducting diagnostic screening. Further, findings from the current study indicate that maternal depressive symptoms are associated with adolescent depressive symptoms at ages 16-17. Taking the results from the analyses using paternal reports on adolescent depressive symptoms into account as well, findings suggest that mental health professionals should evaluate both parents' mental health status when adolescents ages 14 to 17 are referred to mental health clinics for depressive symptoms. Additionally, when parents are receiving treatment for depressive symptoms, standard procedure should be to initiate preventive measures to avoid the development of depressive symptoms in their offspring.

Future research. Findings from the current study indicate some directions for future research. First, future studies should examine the long-term effects of exposure to paternal depressive symptoms in early childhood, the effects of repeated exposure to paternal depressive symptoms during childhood and adolescence, and the short-term effects of paternal depressive symptoms in adolescence. Because there are some inconsistencies in the existent research literature, more studies are needed for clarification. Future research should utilize multi-informant data to reduce the influence of shared method variance, and to further investigate how the use of parent and offspring reports on offspring depressive symptoms affects the strength of the association between paternal and offspring depression. Also, studies should be conducted in different societies to investigate whether there are cultural differences in the intergenerational transmission of depression from fathers to their offspring.

Second, studies should also address the issue on how to handle informant discrepancies between reports from multiple informants. To date, there is little or no consensus on a general framework for understanding, interpreting and aggregating divergent reports from multiple informants' reports within a study (Bird et al., 1992; De Los Reyes et al., 2013; Kraemer et al., 2003). Confirmatory factor analyses (CFA) or structural equation modeling (SEM) have been suggested as means to combine multi-informant reports, because the CFA/SEM framework "allows one to extract and directly model the non-random measurement error without it contaminating the loadings on the latent variable" (van Dulmen & Egeland, 2011, p. 90). Hence, researchers should consider using a latent variable constructed through CFA/SEM when aggregating discrepant multi-informant data on offspring mental health. Also, future studies could include an independent criterion (e.g. a

clinical interview) assessed at a later time-point, to investigate which informant or multi-aggregated score is better at predicting offspring subsequent depressive symptoms measured by the independent criterion (van Dulmen & Egeland, 2011).

Third, given the tendency for significant associations between paternal and adolescent depressive symptoms when fathers are reporting on adolescent depressive symptoms, future studies should attempt to identify the potential mechanisms underlying this association. Optimally, one should use genetically sensitive designs that are able to separate the environmental factors from the genetic factors (Natsuaki et al., 2014). Identification of the environmental mechanisms that operate in intergenerational transmission of depression is important, because these mechanisms can be targeted by intervention programs for families of depressed parents, and to design effective preventive programs (Natsuaki et al., 2014). In general, more research is needed to fully understand the genetic liability for depression and the specific biological, psychological, and social mechanisms that could be involved in the intergenerational transmission of depression (Rudolph & Flynn, 2014).

Finally, future studies should examine the direction of causality in the longitudinal analyses. Relatively few studies have examined child effects on parents, or the bidirectionality of effects (e.g. Hughes & Gullone, 2010b; Kouros & Garber, 2010). Longitudinal research incorporating a full reciprocal design (e.g. panel models) allows for examination of relations in both directions, and the assessment of the relative strength of the cross-lagged effects (Selig & Little, 2012).

Conclusion

The current study is one of the few studies on the association between paternal and offspring depressive symptoms in middle and late adolescence, using a community-based sample. Findings from the current study revealed that paternal depressive symptoms were significantly associated with adolescent depressive symptoms cross-sectionally at ages 14-15 and 16-17, and longitudinally from ages 14-15 to 16-17 only when fathers reported on adolescent depressive symptoms. However, the associations might be inflated when fathers reported on their own depressive symptoms and adolescent depressive symptoms, because of shared method variance. These findings highlight the importance of considering which informant is reporting on adolescent depressive symptoms when interpreting previous research on the association between paternal and offspring depressive symptoms. Moreover, the findings suggest that single-informant data should be avoided in research examining the association between paternal and offspring depression. The lack of significant association

between paternal depressive symptoms and adolescents' self-reported depressive symptoms is in accordance with some previous studies but not others, suggesting that more studies are needed for clarification of the inconsistencies in the existing research literature.

Further, findings from the current study complement previous research showing that adolescents in the general population report higher levels of depressive symptoms compared to their parents' reports on adolescent depressive symptoms. Moreover, there was also considerable discrepancy between prevalence estimates of adolescent depression depending on whether self-reports or parent reports on adolescent depressive symptoms were examined. These findings indicate that the levels of adolescent depressive symptoms and the prevalence of adolescent depression might be underestimated if parent reports are the only source in clinical and research assessments of adolescent depressive symptoms. Thus, researchers and mental health professionals should strive to obtain information on adolescent depressive symptoms from both the parents and the adolescent.

Lastly, findings from the current study is in line with previous research demonstrating that adolescent females report higher levels of depressive symptoms than adolescent males, and that adolescent females' self-reported depressive symptoms increase from middle to late adolescence. Adolescent gender did not moderate the association between paternal and adolescent depressive symptoms in the current study, which is in accordance with some previous studies, but not others. Consequently, more studies are needed for clarification.

In conclusion, future research should further examine the potential adverse effects of paternal depressive symptoms on adolescent offspring, with regard to both short-term and long-term effects. Researchers should take into account the substantial informant discrepancies between parents' reports and adolescents' self-reports on depressive symptoms demonstrated in the current study, by utilizing multi-informant data on adolescent depressive symptoms. Future research should further elucidate how the use of different informants' reports affects the strength of the associations between paternal and offspring depressive symptoms.

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Appendix A

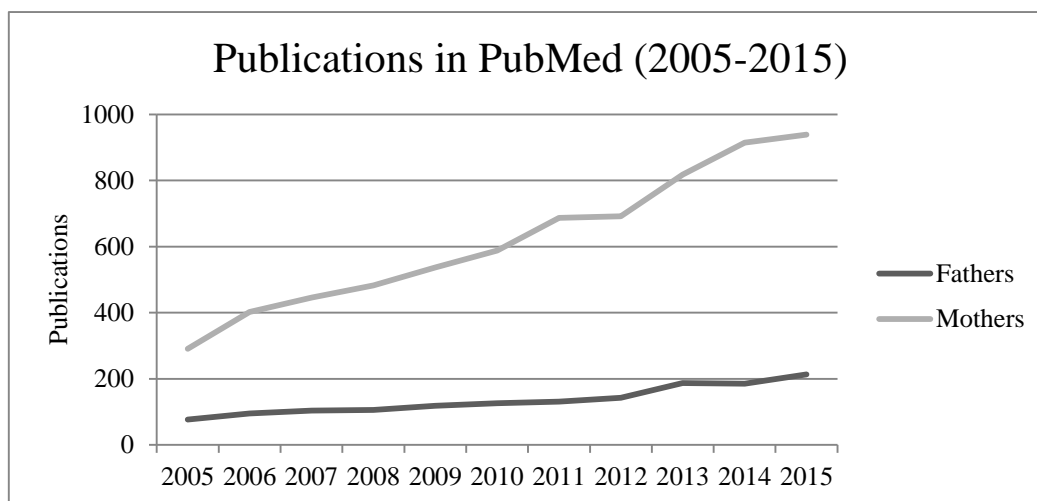


Figure A1. Number of publications on child and adolescent psychopathology from 2005 to 2015 including fathers or mothers.

A systematic search was conducted 02.10.2015 in PubMed with the following search strategy for research including fathers:

(((((paternal[Title/Abstract]) OR dad[Title/Abstract]) OR father[Title/Abstract]) OR dads[Title/Abstract]) OR fathers[Title/Abstract]))

AND

(((((mental health[Title/Abstract]) OR depression[Title/Abstract]) OR depressive symptoms[Title/Abstract]) OR psychopathology[Title/Abstract]) OR psychological distress[Title/Abstract]) OR mental distress[Title/Abstract]) OR internalising[Title/Abstract]) OR internalizing[Title/Abstract]) OR emotional difficulties[Title/Abstract]) OR common mental disorder[Title/Abstract]))

AND

(((((offspring[Title/Abstract]) OR child[Title/Abstract]) OR adolescent[Title/Abstract]) OR infant[Title/Abstract]) OR intergenerational[Title/Abstract]) OR son[Title/Abstract]) OR daughter[Title/Abstract]) OR sons[Title/Abstract]) OR daughters[Title/Abstract]) OR children[Title/Abstract]) OR adolescents[Title/Abstract]))

When conducting the same search on research including mothers, the following entry words were used in the first line:

(((((maternal[Title/Abstract]) OR mom[Title/Abstract]) OR mother[Title/Abstract]) OR moms[Title/Abstract]) OR mothers[Title/Abstract]))

Appendix B

Excerpts from the paternal and adolescent questionnaire used in the TOPP study at wave 2 are shown on the next four pages. The excerpts show the questions from the parent and child/adolescent version of the Short Mood and Feelings Questionnaire (SMFQ), and the depression subscale from the Hopkins Symptoms Checklist (HSCL-25). The questions used at wave 1 are identical, except for one question from the SMFQ which was removed (for more information, see “Measures” in the method section of the thesis).

Hvilke områder opplever du som vanskelig for deg nå for tiden (sett *en ring* rundt et tall for å indikere hvor enig du er i utsagnet):

		Ikke vanskelig	1	2	3	4	5	Veldig vanskelig
53	Forholdet til foreldrene dine	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
54	Forholdet til venner eller andre personer	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
55	Parforhold, kjæreste eller forelskelser	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
56	Opplevelse av egen kropp eller utseende	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
57	Forholdet til tobakk, alkohol eller rus	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
58	Skolen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
59	Tanker om noe vondt som har skjedd	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
60	Tanker om noe vondt som kan skje	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

PLAGSOMME FØLELSER OG TANKER

Her følger en liste over forskjellige følelser og tanker man av og til kan ha. Tenk på **de to siste ukene** og kryss av for hvor ofte du har følt eller tenkt noe av det som står nedenfor (sett *kun ett* kryss på hver linje):

		2	1	0
		Stemmer	Stemmer noen ganger	Stemmer ikke
61	Jeg var lei meg eller ulykkelig	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
62	Jeg følte meg så trøtt at jeg bare ble sittende uten å gjøre noen ting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
63	Jeg var veldig rastløs	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
64	Jeg var ikke glad for noe	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
65	Jeg følte meg lite verdt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
66	Jeg gråt mye	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
67	Jeg tenkte at livet ikke var verdt å leve	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
68	Jeg synes det var vanskelig å tenke klart eller konsentrere meg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
69	Jeg hatet meg selv	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
70	Jeg tenkte at jeg aldri kunne bli så god som andre ungdom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
71	Jeg følte meg ensom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
72	Jeg tenkte at ingen egentlig var glad i meg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
73	Jeg følte meg som et dårlig menneske	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

		2	1	0
		Stemmer	Stemmer noen ganger	Stemmer ikke
74	Jeg syntes jeg gjorde alt galt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
75	Jeg tenkte at fremtiden ikke hadde noe positivt å by meg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
76	Jeg tenkte på å ta livet mitt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Les gjennom alle utsagnene og kryss av for å vise i hvor stor grad du føler at utsagnet passer for deg **den siste uken**. Det er ingen svar som er riktige eller gale.

		0	1	2	3
		Passer ikke i det hele tatt	Passer til en viss grad, eller noe av tiden	Passer godt, eller en god del av tiden	Passer best, eller mesteparten av tiden
77	Jeg merket at jeg var tørr i munnen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
78	Jeg hadde pustevansker (f.eks. pustet altfor fort, eller ble andpusten uten fysisk anstrengelse)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
79	Jeg følte meg skjelven (f.eks. følte at bena kom til å gi etter under meg)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
80	Jeg opplevde situasjoner som gjorde meg så engstelig at jeg ble utrolig lettet når de var over	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
81	Jeg følte at jeg kom til å besvime	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
82	Jeg svettet mye (f.eks. i hendene) uten at det var varmt og uten fysisk anstrengelse	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
83	Jeg følte meg redd uten å ha særlig grunn til det	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
84	Jeg hadde problemer med å svelge	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
85	Jeg var oppmerksom på hjerterytmen min uten at jeg hadde vært i fysisk aktivitet (f.eks. følelse av økt hjerterytme, eller at hjertet hoppet over et slag)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
86	Jeg følte at jeg var nær ved å få panikk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
87	Jeg var redd for at selv en enkel, triviell oppgave kunne bringe meg ut av fatning	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
88	Jeg var livredd	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
89	Jeg bekymret meg for å komme opp i situasjoner der jeg kunne få panikk og dumme meg ut	<input type="checkbox"/>	<input type="checkbox"/>	<input checked="" type="checkbox"/>	<input type="checkbox"/>
90	Jeg skalv ofte (f.eks på hendene)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
91	Jeg unngikk aktiviteter hvor jeg var i sentrum for andres oppmerksomhet	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
92	Jeg unngikk å gjøre ting eller snakke til andre av redsel for å bli flau	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

52 **Når du sammenligner ungdommen din med ungdommer flest, vil du si at hun/han jevnt over er:**

1 Klart lettere å ha med å gjøre

2 Litt lettere å ha med å gjøre

3 Omtrent vanlig

4 Litt vanskeligere å ha med å gjøre

5 Klart vanskeligere å ha med å gjøre

PLAGSOMME FØLELSER OG TANKER

Her følger en liste over forskjellige følelser og tanker man av og til kan ha. Tenk på de to siste ukene og kryss av for hvor ofte du tror 16-17 åringen har følt eller tenkt noe av det som står nedenfor. (Sett **kun ett** kryss på hver linje.)

		0	1	2
Han/hun:		Stemmer ikke	Stemmer noen ganger	Stemmer
53	var lei seg eller ulykkelig	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
54	følte seg så trøtt at han/hun bare ble sittende uten å gjøre noen ting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
55	var veldig rastløs	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
56	var ikke glad for noe	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
57	følte seg lite verdt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
58	gråt mye	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
59	tenkte at livet ikke var verdt å leve	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
60	syntes det var vanskelig å tenke klart eller konsentrere seg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
61	hatet seg selv	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
62	tenkte at han/hun aldri kunne bli så god som andre ungdommer	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
63	følte seg ensom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
64	tenkte at ingen egentlig var glad i han/henne	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
65	følte seg som et dårlig menneske	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
66	syntes han/hun gjorde alt galt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
67	tenkte at fremtiden ikke hadde noe positivt å by meg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
68	tenkte på å ta livet sitt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>



DIN OPPLEVELSE AV STRESSPLAGER SISTE UKE

Nedenfor er en liste over problemer eller plager folk kan ha. Vurder hvor mye av de følgende plager eller ulemper du har eller har hatt siste uke (til og med i dag). (Sett ett kryss på hver linje.)

	1	2	3	4
	Ikke i det hele tatt	Litt	En god del	Svært mye
106	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
107	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
108	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
109	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
110	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
111	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
112	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
113	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
114	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
115	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
116	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
117	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
118	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
119	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
120	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
121	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
122	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
123	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
124	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
125	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
126	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
127	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
128	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
129	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>



Appendix C

Table C1

Skewness and Kurtosis Before and After Logarithmic Transformation of the Predictor and Outcome Variables

Variable	Before transformation		After transformation	
	Skewness	Kurtosis	Skewness	Kurtosis
Wave 1 (ages 14-15)				
Adolescents' self-reports on depressive symptoms	1.68	2.89	1.08	.66
Paternal reports on adolescent depressive symptoms	1.70	3.23	1.22	1.05
Maternal reports on adolescent depressive symptoms	2.13	5.33	1.56	2.47
Paternal depressive symptoms	1.76	3.86	1.08	.87
Maternal depressive symptoms	1.45	2.34	.82	.13
Wave 2 (ages 16-17)				
Adolescents' self-reports on depressive symptoms	1.40	1.81	.81	.06
Paternal reports on adolescent depressive symptoms	1.60	3.46	1.07	.99
Maternal reports on adolescent depressive symptoms	2.37	8.62	1.43	2.71
Paternal depressive symptoms	2.14	6.68	1.20	1.61
Maternal depressive symptoms	1.89	4.93	1.07	.91

Note. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire. Parent depressive symptoms were measured by the depressive subscale from Hopkins Symptom Checklist 25. $N = 290-478$.

Appendix D

Table D1

Principal Component Analyses of the Informants' Reports on the Short Mood and Feelings Questionnaire (Mean Scores) at Wave 1 and Wave 2

Variable	Factor loadings	
	Factor score w1	Factor score w2
Adolescents' self-reports on depressive symptoms	.718	.705
Paternal reports on adolescent depressive symptoms	.734	.769
Maternal reports on adolescent depressive symptoms	.784	.819
Variance explained (%)	55.6	58.6

Note. Two separate principal component analyses were run; one for mean scores at w1, and one for mean scores at w2. $N = 316$ at w1, and $N = 261$ at w2.

Appendix E

Table E1

Bivariate Correlations between the Aggregated Scores and Confounding Variables at Wave 1 and Wave 2

Confounding variable	Aggregated score at w1	Aggregated score at w2
Adolescent gender ^a	.19***	.24***
Living arrangement with father (paternal reports) ^b	-.15**	-.17**
Living arrangement with mother (maternal reports) ^b	-.04	-.13*
Divorce/separation (paternal reports) ^c	.11*	.03
Divorce/separation (maternal reports) ^c	.10	.15*

Note. The aggregated scores comprised adolescents' self-reports, paternal and maternal reports on the Short Mood and Feelings Questionnaire. The confounding variables and the aggregated scores were measured at the same data wave. $N = 226-316$.

^a0 = males, 1 = females. ^b0 = living with his/her child less than full-time, 1 = living with his/her child full-time. ^c0 = married/live-in partner; not married/single; widow, 1 = divorced/separated.

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Appendix F

Table F1

Descriptive Statistics for Parent and Adolescent Depressive Symptoms for the Total Sample, and Adolescent Females and Males Separately, at Wave 1 and Wave 2 (After Transformation)

Variable	n	M (SD)		
		Total	Females	Males
Wave 1 (ages 14-15)				
Adolescents' self-reports on depressive symptoms	454	.27 (.24)	.34 (.25)	.19 (.19)
Paternal reports on adolescent depressive symptoms	340	.16 (.17)	.15 (.16)	.16 (.19)
Maternal reports on adolescent depressive symptoms	476	.14 (.17)	.16 (.18)	.12 (.14)
Aggregated score of adolescent depressive symptoms	316	.0 (1.0)	.17 (1.03)	-.22 (.91)
Paternal depressive symptoms	341	1.23 (.22)		
Maternal depressive symptoms	478	1.29 (.25)		
Wave 2 (ages 16-17)				
Adolescents' self-reports on depressive symptoms	371	.34 (.26)	.42 (.25)	.23 (.22)
Paternal reports on adolescent depressive symptoms	290	.16 (.16)	.17 (.16)	.16 (.15)
Maternal reports on adolescent depressive symptoms	419	.19 (.18)	.21 (.20)	.15 (.16)
Aggregated score of adolescent depressive symptoms	261	.0 (1.0)	.21 (1.09)	-.28 (.79)
Paternal depressive symptoms	290	1.24 (.23)		
Maternal depressive symptoms	416	1.27 (.25)		

Note. The variables in the table were transformed by logarithmic transformation. Adolescent depressive symptoms were measured by the Short Mood and Feelings Questionnaire (SMFQ). The aggregated scores comprised adolescents' self-reports, paternal and maternal reports on the SMFQ, and these variables are standardized. Parent depressive symptoms were measured by the depression subscale from Hopkins Symptom Checklist 25.

Appendix G

Table G1

Moderated Regression Analyses of the Effect of Adolescent Gender on the Cross-Sectional Associations between Paternal and Adolescent Depressive Symptoms at Wave 1 and Wave 2

Variable	Aggregated score β (<i>p</i>)	Informant on adolescent depressive symptoms		
		Adolescents β (<i>p</i>)	Fathers β (<i>p</i>)	Mothers β (<i>p</i>)
Wave 1 (ages 14-15)				
Model 1				
Paternal depressive symptoms	.11 (.06)	.12 (.03)	.22 (.00)	-.02 (.72)
Adolescent gender ^a	.20 (.00)	.30 (.00)	.00 (.95)	.11 (.05)
<i>R</i> ²	.05	.10	.05	.01
Model 2				
Paternal depressive symptoms	.18 (.03)	.18 (.03)	.31 (.00)	.07 (.43)
Adolescent gender ^a	.20 (.00)	.30 (.00)	.00 (.97)	.11 (.05)
Interaction term (Paternal depressive symptoms x Adolescent gender)	-.11 (.21)	-.08 (.31)	-.12 (.13)	-.12 (.17)
<i>R</i> ²	.05	.10	.05	.02
Wave 2 (ages 16-17)				
Model 1				
Paternal depressive symptoms	.20 (.00)	.01 (.80)	.33 (.00)	.15 (.01)
Adolescent gender ^a	.27 (.00)	.39 (.00)	.07 (.25)	.19 (.00)
<i>R</i> ²	.10	.15	.11	.05
Model 2				
Paternal depressive symptoms	.21 (.02)	.01 (.94)	.30 (.00)	.16 (.06)
Adolescent gender ^a	.27 (.00)	.39 (.00)	.07 (.26)	.19 (.00)
Interaction term (Paternal depressive symptoms x Adolescent gender)	.00 (.98)	.01 (.90)	.04 (.67)	-.01 (.87)
<i>R</i> ²	.10	.15	.11	.05

Note. Reported figures are standardized beta coefficients from regression analysis, *p* values in brackets. The outcome variables are mean scores of the Short Mood and Feelings Questionnaire. The aggregated score comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. The continuous predictor variable "paternal depressive symptoms" was grand mean centered before conducting the analysis. All predictors and the outcome variable were measured at w1 for the cross-sectional analyses at w1, and all predictors and the outcome variable were measured at w2 for the cross-sectional analyses at w2. *N* = 258-329

^a0 = males, 1 = females.

Table G2

Moderated Regression Analysis of the Effect of Adolescent Gender on the Longitudinal Associations between Paternal and Adolescent Depressive Symptoms

Variable	Informant on adolescent depressive symptoms			
	Aggregated score	Adolescents	Fathers	Mothers
	β (<i>p</i>)	β (<i>p</i>)	β (<i>p</i>)	β (<i>p</i>)
Model 1				
Paternal depressive symptoms	.18 (.01)	.07 (.21)	.24 (.00)	.11 (.07)
Adolescent gender ^a	.26 (.00)	.37 (.00)	.07 (.29)	.15 (.01)
<i>R</i> ²	.09	.14	.06	.03
Model 2				
Paternal depressive symptoms	.15 (.13)	.06 (.48)	.21 (.02)	.08 (.36)
Adolescent gender ^a	.26 (.00)	.37 (.00)	.07 (.30)	.15 (.01)
Interaction term (Paternal depressive symptoms x Adolescent gender)	.04 (.66)	.01 (.88)	.04 (.68)	.04 (.69)
<i>R</i> ²	.09	.14	.06	.03

Note. Reported figures are standardized beta coefficients from regression analysis, *p* values in brackets. The outcome variables are mean scores of the Short Mood and Feelings Questionnaire. The aggregated score comprised adolescents' self-reports, paternal and maternal reports on the SMFQ. The continuous predictor variable "paternal depressive symptoms" was grand mean centered. All predictor variables were measured at w1. *N* = 226-278.

^a0 = males, 1 = females