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Depression and Adolescent Sexual Activity in Romantic and Nonromantic Relational Contexts: A Genetically-Informative Sibling Comparison

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Adolescent dating and sexual activity are consistently associated with risk for depression, yet the pathways underlying this association remain uncertain. Using data on 1,551 sibling pairs (ages 13–18) from the National Longitudinal Study of Adolescent Health, the current study utilized a sibling comparison design to assess whether adolescent dating, sexual intercourse with a romantic partner, and sexual intercourse with a nonromantic partner were associated with higher levels of depressive symptoms independent of familial factors. Results indicated that adolescent dating, in and of itself, was not associated with depressive symptoms. The association between depressive symptoms and sexual activity with a romantic partner was fully accounted for by between-family genetic and shared environmental confounds. In contrast, sexual activity with a nonromantic partner was significantly associated with both mean levels of depressive symptoms and clinically severe depression, even within sibling dyads. This relationship was greater for younger adolescents (<15 years). These results are consistent with a growing body of research demonstrating that relationship contexts may be critical moderators of the psychosocial aspects of adolescent sexual experiences.

Keywords: depression, adolescence, behavior genetics, relationships

A hallmark of adolescent social development is interest in dating and the formation of romantic relationships. By the end of high school, more than 70% of adolescents report involvement in a romantic relationship (Carver, Joyner, & Udry, 2003). Despite their prevalence, these relationships correlate with psychological distress, with adolescents who date consistently reporting greater levels of depressive symptoms than adolescents who do not (e.g., Davila, 2008; Davila, Steinberg, Kachadourian, Cobb, & Finchman, 2004; Joyner & Udry, 2000). Dating, moreover, is often accompanied by increasing physical intimacy and sexual initiation, with more than one half of American adolescents losing their virginity during high school (Allan Guttmacher Institute, 1999). As with dating, adolescents who are sexually active tend to display a range of psychosocial difficulties, including depression, poor academic achievement, low self-esteem, and substance use compared to adolescents who are not sexually active (Hallfors, Waller, Bauer, Ford, & Halpern, 2005; Joyner & Udry, 2000; Lynch, 2001; McGue & Iacono, 2005; Rector, Johnson, & Noyes, 2003;Spriggs & Halpern, 2008). In the current article, we leverage a behavioral genetics approach to address two major challenges to understanding the mechanisms by which adolescent dating, sexual activity, and depressive symptoms are associated: (a) the diversity of relationship contexts that adolescents experience, and (b) the role of common genetic and shared environmental factors.

Dating and Depressive Symptoms

As noted in Davila (2008), the association of adolescent dating and depression is a finding which is comparatively recent but which "cannot be denied" (p. 26). Perhaps the most pervasive explanation is *the stress and coping model* (e.g., Larson, Clore, & Wood, 1999), which suggests the intense emotions which accompany dating inherently provoke psychological challenge and stress. Adolescent relationships are often marked by lability, break-ups, unmet needs for intimacy, and feelings of rejection, and they may require sophisticated conflict management skills few adolescents have mastered. Individuals vary in their coping strategies and resources, and those adolescents who either lack or do not utilize effective coping mechanisms to navigate romantic difficulties may be particularly at risk for depression. In support of this, the association between romantic experiences and adolescent depression is heightened among adolescent girls prone to "corumination," with a tendency to discuss their relationships and associated negative emotions repetitively and extensively with each other (Starr & Davila, 2009).

A similar perspective, known as *the individual differences theory* (Davila, 2008) suggests that variations in personality, social

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history, and interpersonal style account for relations between dating and depression. Levels of neuroticism, social competence, rejection sensitivity, and attachment style, for example, differ across individuals and may contribute to likelihood of engagement in a relationship, the way relationship events may be perceived, and the extent to which a relationship may emerge as depressogenic. Indeed, the association of dating with depressive symptoms seems strongest among adolescents whose relationships are characterized by insecure—and particularly preoccupied—styles of attachment (Davila et al., 2004), suggesting that vulnerability to dating-related dysphoria is related to interpersonal variability and comfort within relationships.

Lastly, *the attention impairment theory*, proposes an indirect effect of adolescent dating on depression (Davila, 2008; Joyner & Udry, 2000). Dating is a time-consuming activity which shifts the focus and attention of adolescents away from domains such as school, family, and friendships. The resulting decrements in these important areas of life may precipitate depression (Joyner & Udry, 2000), particularly when coupled with the tumultuous nature of adolescent relationships. Teenagers may be left with reduced social support and unanticipated academic difficulties when a relationship ends, or they may alternatively find their loyalties to a romantic partner strained by problems integrating that person into their existing social world. Consistent with this, poor school performance and family dysfunction have been shown to mediate associations of adolescent dating with depressive symptoms (Joyner & Udry, 2000).

Sexual Activity and Depressive Symptoms

One of the distinguishing aspects of different adolescent relationships is the presence and nature of sexual activity. Adolescents vary in their engagement and interest in sexual behaviors, but nearly 65% of adolescents initiate sex over the course of a romantic relationship (Grello, Welsh, Harper, & Dickson, 2003). Alternatively, adolescents may also be sexually involved with people who are not established romantic partners, such as acquaintances, friends, and former romantic partners (Manning, Giordano, & Longmore, 2006). In these situations—colloquially termed "hooking up" or "friends with benefits"—there are no clear expectations of emotional intimacy, exclusivity, or commitment (Manning, Longmore, & Giordano, 2005).

Approximately one third of sexually active teenagers engage in both romantic and nonromantic intercourse during adolescence (Manning et al., 2005). Within the scientific community, some scholars conceptualize adolescent sexual intercourse a "problem behavior" (e.g., Guilamo-Ramos, Jaccard, Dittus, Gonzales, & Bouros, 2008; Jessor & Jessor, 1977; McGue & Iacono, 2005), and others suggest it is an integral and normative part of development, enabling adolescents to feel pleasure and connectedness in relation to others (Fine, 1988; Tolman & McClelland, 2011; Vrangalova & Savin-Williams, 2011). As is the case with dating, there are clear epidemiological associations between engagement in sexual activity and symptoms of depression (e.g., Hallfors et al., 2005; Spriggs & Halpern, 2008). Yet understanding whether dating and sexual activity represent distinct risks for depression is somewhat complicated, in part because researchers have not distinguished between sexually active and nonsexually active dating relationships. One salient differentiation is that sexually active relationships confer risks for pregnancy and/or sexually transmitted infections (STIs), and such risks may be exacerbated in nonromantic sexual relationships.

Importantly, the direction of effect between dating/sexual activity and depression is not established, and bidirectional effects are plausible. Adolescents with high levels of dysphoria or stressful life experiences may seek out sexual or romantic partners, as a way of acquiring support or regulating negative emotions (Davila et al., 2009). Depressed teenagers may additionally be more likely to acquiesce to sexual propositions—even if such activity may not be desired—either to avoid awkward or painful conversations (Hershenberg & Davila, 2010) or because they are symptomatically apathetic. In fact, adolescents who have intercourse with nonromantic partners present with elevated levels of depression even prior to their first sexual experience (Grello et al., 2003; Monahan & Lee, 2008; Welsh, Grello, & Harper, 2003). More severe levels of depressive symptoms, moreover, have been shown to predict engagement in sexual risk-taking prospectively (Lehrer, Shrier, Gortmaker, & Buka, 2006).

A small, but growing, body of research suggests that hooking up may actually be a critical moderator of the association between adolescent sexual behavior and depression. Higher numbers of sexual partners correlate with severity of depressive symptoms (Kosunen, Kaltiala-Heino, Rimpela, & Liappala, 2003; Lehrer et al., 2006); conversely, adolescents are less likely to report negative emotions associated with sexual activity if that activity occurred in a relationship marked by good communication (Donald, Lucke, Dunne, & Raphael, 1995) and a minimum of 4 months of stable duration (Shulman, Walsh, Weisman, & Shelyer, 2009). Similar results have been found for educational achievement, substance use, and delinquency; sexual activity that occurs in the context of nonromantic relationships predicts higher rates of delinquency, substance use, and poorer grades, but no such associations are found when sexual activity occurs within a romantic partnership (McCarthy & Casey, 2008; McCarthy & Grodsky, 2011). Both gender and age may additionally be relevant when considering nonrelationship sexual activity. Within the National Longitudinal Study of Adolescent Health (Add Health, the data employed in the present study), two separate sets of findings suggest the relation between nonromantic sexual activity and depressive symptomatology is strongest for younger girls (Monahan & Lee, 2008). However, older adolescents, boys, and/or those involved in stable relationships do not necessarily exhibit comparable decrements in mood (Meier, 2007). As worry about pregnancy may be particularly prominent if girls are in an unstable sexual relationship, these circumstances might explain why gender could moderate hooking up with adolescent depression.

Lastly, nonromantic and romantic sexual experiences differ in critical ways besides the dichotomous nature of the relationship with the sexual partner. Adolescents are less likely to use condoms or any contraception method with nonromantic partners compared to romantic partners (Ford, Sohn, & Lepkowski, 2001; Manning, Longmore, & Giordano, 2005). Moreover, more than half of nonromantic sexual experiences among college students are preceded by alcohol or drug use (Grello, Welsh, & Harper, 2006). The health consequences of unprotected sex (or anxiety regarding potential health consequences), as well as the substance use coincident with nonromantic sex, may contribute to the association between nonromantic sex and depression.

Why Behavior Genetics?

The present article leverages a behavior genetics design to investigate associations of adolescent dating, sexual activity, and depression. The utility of this methodology hinges on two primary points: (a) general questions of causal inference, and (b) established genetic influences for both sexual behavior and depression. First, understanding the causal relations between predictors and outcomes has proved an ongoing conundrum for the field of developmental psychopathology, in large part due to the impossibility of random assignment to risk factors and the large number of potential confounding variables. Because assumptions of causal processes between risks and outcomes contribute to decisions regarding public policy and intervention targets, a primary concern is that such assumptions are accurate. Behavior genetics offers a method to model and evaluate whether observed associations are due to causal influences versus selection processes that would presumably still be operating even if the putative predictor were changed. This application of behavior genetics methodology to resolve etiological questions—rather than the more traditional partitioning of variance into genetic and environmental components—has been termed the "new look of behavior genetics" (Moffitt, 2005) and hailed as "a substantial advantage over cross-sectional correlational studies" and "a set of strategies that warrants much greater use" (Rutter, 2007, p. 391).

There are multiple permutations for using behavior genetics to evaluate causality, including the basic twin model (e.g., Johnson, Turkheimer, Gottesman, & Bouchard, 2009), children-of-twins design (D'Onofrio et al., 2003), the recently developed mother-daughter-aunt-niece design (Rodgers, Bard, Johnson, D'Onofrio, & Miller, 2008), and the sibling comparison design (e.g., Lahey & D'Onofrio, 2010) employed in the current study. All of these designs share a core logic: By comparing children who differ in a particular risk or circumstance but who share genes *and* the environmental background factors shared by family members, we can test whether an established association between two variables is attributable to selection factors that differ between families. In the case of the present study, the risks of interest are engagement in dating and sexual activity and the outcome is depression, but such designs have been applied to a broad range of predictors and outcomes. Although it might seem counterintuitive to use genetic relatedness as a means to understand the potentially causal effects of environmental experiences, a key issue is that of *gene-environment correlation*, or the extent to which genetically influenced traits predict an individual's likelihood of encountering a particular environmental circumstance. In the case of dating and depression, for example, traits such as social anxiety or timing of pubertal onset may influence an individual's likelihood of becoming involved in a dating relationship (with high social anxiety decreasing the likelihood and earlier pubertal timing increasing the likelihood). Both of these traits, disparate as they are, are correlated with depression during adolescence and strongly heritable (e.g., Mendle, Turkheimer, & Emery, 2007; Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004; Stein, Jang, & Livesley, 2002). Studies which do not account for the possibility that a correlational association may be attributable to shared genes for both risk and outcome will obtain results which mask the "true" mechanism. More simply, without genetic controls, genes may be a "third variable" confound.

Adolescent depression is certainly influenced by genes. Twin studies yield higher concordance for depressive symptoms within monozygotic twins than dizygotic twins (Neale & Mayes, 1996), while molecular studies implicate polymorphisms in serotonergic genes in depression, particularly in response to stressful events (Caspi et al., 2003; Eley et al., 2004; Karg, Burmeister, Shedden, & Sen, 2011). In addition, although developmentalists tend to emphasize the environmental aspects of romantic behavior, adolescent sexuality is also genetically influenced, with heritability estimates for age of first intercourse ranging from 37% to 72% (Bricker et al., 2006; Dunne et al., 1997; Martin, Eaves, & Eysenck, 1997; Mustanski, Viken, Kaprio, Winter, & Rose, 2007; Rodgers et al., 2008). Interestingly, a few studies have implicated the short (*s*) allele of the serotonin transporter gene (5-HTTLPR; more commonly linked with vulnerability to depression) in sexual behavior, number of partners, and sexual desire (e.g., Hamer, 2002; Kogan et al., 2010).

In our previous work, we have found that many associations between dating, sexual behavior, and risk for psychopathology *disappear—or even switch direction*—once we account for common genetic influences in a genetically informed design (Harden, Mendle, Hill, Turkheimer, & Emery, 2008). Of particular interest for the current article is the finding that, when comparing siblings discordant for age at first sexual intercourse, an earlier age of sexual initiation was associated with *reduced* delinquency in early adulthood (Harden et al., 2008). Follow-up analyses suggested that this effect was evident only for teenagers who had sex in a romantic relationship; nonromantic sexual activity was associated with higher levels of delinquency, even after comparing within sibling pairs (Harden & Mendle, 2011). Such findings raise questions for the established wisdom regarding the origins and pathogenic effects of early sexual behavior. These questions are not purely academic, but also hold clinical, economic, and policy relevance.

Goals of the Current Study

In the current study, we employ a sibling comparison design to investigate the association between depressive symptoms and dating; sexual activity within a romantic relationship and sexual activity which occurs outside of a romantic relationship (from here on, referred to as *relationship context*). Analyses targeted four main research questions. First, do mean levels of depressive symptoms vary as a function of relationship context? Second, does this association also hold at clinically severe levels of depression? Third, does the association between relationship context and depressive symptoms persist after controlling for genetic and shared environmental familial confounds? If so, then fourth: Can we ascertain whether this association is attributable to genetic or to environmental mechanisms?

Method

Participants

Data are drawn from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 2003), a nationally representative study designed to evaluate adolescent health and risk behaviors. The Add Health Study targeted a stratified random sample of

U.S. high schools, 79% of which agreed to participate in the study (N = 134 schools). Of the participating schools, 96% allowed students (N = 90,118) to complete a confidential in-school survey during the 1994–1995 academic year. The rosters of participating schools were used to randomly select a subsample of 20,745 participants who completed a follow-up, 90-min in-home interview between April and December 1995 (Wave I interview; 10,480 female; 10,264 male). Participants in the full Add Health sample ranged in age from 11 to 21 at Wave I (M = 16 years, 25th-75th percentile = 14–17 years). Three additional waves of data collection were conducted; given the broad age range and focus of the current study, analyses in the current study were restricted to adolescents between 13 and 18 years old at Wave I.

The focus of the current analyses is a subsample of 1,551 sibling pairs: 233 MZ twin pairs, 208 DZ twin pairs, 651 full sibling (FS) pairs, 201 half sibling (HS) pairs, 87 Cousin (CO) pairs raised as siblings, and 171 nonbiologically related (NR) pairs (e.g., stepsiblings, adopted siblings). During the in-school interview, adolescents were asked whether they currently lived with another adolescent in the same household. The information was used to oversample adolescent sibling pairs who resided in the same home deliberately, even if one member of the pair did not attend a high school in the original probability sample. Twin zygosity was determined by 11 molecular genetic markers and responses to four questionnaire items concerning similarity of appearance and frequency of being mistaken for one's twin (Harris, Halpern, Smolen, & Haberstick, 2006). Similar questionnaires have been utilized widely in twin research and have been repeatedly cross-validated with zygosity determinations based on DNA (e.g., Spitz et al., 1996). Jacobson and Rowe (1999) compared the sociodemographic composition of sibling pairs to the full Add Health sample and found negligible differences. There have been three follow-up interviews with the Add Health participants: Wave II in 1996, Wave III in August, 2001−2002, and Wave IV in 2007–2008. Because the current study is most interested in associations of romantic activity with depression during adolescence, analyses were conducted only on data from the Wave 1 interviews; data from Wave IV, however, were utilized to confirm reports of sexual activity.

Measures

Depressive symptoms. A 19-item version of the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977) assessed level of depressive symptoms. The CESD is a self-report measure of cognitive, affective, and physiological symptoms of depression; in this sample, scores ranged from 0 to 54 (M = 11.64, SD = 7.53). Internal reliability was adequate (α = .86). A score of 18 on the CESD is typically used to demarcate clinical depression (Radloff & Locke, 1986); 19.5% of the sample reported a CESD score in the clinical range. Analyses were conducted using both continuous CESD scores (*depressive symptoms;* log-transformed to reduce positive skew) and a categorical variable representing whether or not the individual reported *clinical-range depression* (CESD score $\geq$ 18).

Dating and sexual activity. During the Wave I in-home interview, adolescents were asked whether they had a "special romantic relationship" with anyone in the past 18 months; if the participant answered "yes" they were classified as being in a dating relationship. Furthermore, if the adolescent denied having a special romantic relationship, but reported that he or she told another person (who was not a family member) that he or she "liked or loved them" and had held hands and kissed this person, then the adolescent was also classified as being in a dating relationship.[1] Overall, 62.6% of the sample reported being in a dating relationship. For each dating relationship in the last 18 months (up to three relationships), adolescents reported whether they had sexual intercourse in that relationship. If an adolescent reported intercourse in any dating relationship in the last 18 months, then they were classified as Romantic Sex = 1 (23.4% of the sample).

Adolescents also reported at Wave I whether they had ever had a "sexual relationship" with anyone, "not counting the people you described as romantic relationships." Adolescents who reported sexual activity in the context of a nonromantic relationship were classified as Nonromantic Sex = 1 (23.7% of the sample). This coding scheme had the advantage of creating relationship contexts that were not mutually exclusive categories: for example, adolescents who reported sexual activity both in and out of romantic relationship were scored as Dating = 1, Romantic Sex = 1, Nonromantic Sex = 1 (13.3% of the sample). Adolescents who reported that they were virgins were classified as not having sex in either romantic or nonromantic relationships [Dating = 1 or 0, depending on report; Romantic Sex = 0; Nonromantic Sex = 0; 66.6% of the sample].[2]

Within-pair correlations for dating and sexual behaviors according to level of genetic relatedness may be found in Table 1. On the whole, MZ twins were consistently more similar with regards to romantic and sexual behaviors than DZ twins and full sibling pairs. Most of the correlations for pair types with minimal genetic relatedness (half-siblings, cousins, and nonbiologically related siblings) were not significantly different than zero. For interested readers, a complete biometric decomposition of sexual intercourse within romantic and nonromantic relationship contexts may be found in Harden and Mendle (2011).

[1] This classification scheme is consistent with the original design of the Add Health Wave I interview and with previous analyses of the Add Health data (e.g., Cavanagh, 2007; Harden & Mendle, 2011; Joyner & Udry, 2000). Notably, Joyner and Udry (2000) found that adolescents who reported a "liked" relationship did not significantly differ from adolescents who reported a "special romantic relationship" with regards to depressive symptoms. In the sibling pairs sub-sample used here, younger adolescents were more likely to report a "liked" relationship ($p < .01$), but there were no gender differences in likelihood of reporting a "liked" relationship ($p = .52$). More importantly, as with Joyner and Udry's findings for the full sample, adolescents in this subsample who reported being in a "liked" relationship did not report significantly different levels of depressive symptoms from adolescents in a "special romantic relationship" ($p = .26$).

[2] To ensure that the experiences of sexual activity reported at Wave 1 were consensual, we compared adolescents' reports on this item to reports of age of first sexual intercourse and more detailed reports of prior episodes of nonconsensual intercourse obtained at Wave IV (age 24–32). A follow-up sensitivity analysis excluded any participants who endorsed nonconsensual sex prior to the Wave I interview (N = 145 participants (4.7%). Importantly, even after excluding these individuals, the pattern of significant results was unchanged for both models of depressive symptoms and of clinical-level depression. Results of sensitivity analyses may be obtained by request.

Table 1
Sibling Pair Correlations for Dating, Romantic Sex, and Nonromantic Sex

Sibling pair type	Dating	Romantic sex	Nonromantic sex
MZ twins	**.45***	**.61***	**.42***
DZ twins	**.31***	**.25***	**.18***
Full siblings	**.23***	**.19***	**.20***
Half siblings	.13	.06	−.13
Cousins	**.37***	−.09	.13
Nonbiologically Related siblings	.09	.16	**.29***

* Significantly different than zero at $p < .05$.

Analyses

The present study employs a sibling comparison design. When using siblings to evaluate causality, there are two comparisons of interest: between-family and within-family. In a traditional correlational design, participants who differ in a particular risk or circumstance are compared to each other (e.g., adolescents who date to adolescents who do not date). In these studies, the participants come from a variety of different family backgrounds and, therefore, all associations of interest are inherently *between-family*. Thus, the design does not account for genetic differences among participants in the sample, as mentioned above. It is also worth noting that the design is further limited in its ability to account for many major environmental differences which may exist among participants raised in different families. Most previous research has dealt with the problem of selection factors using a combination of longitudinal designs and exhaustive statistical covariates (e.g., income, parental education, family structure, parental monitoring, and so on). However, it is simply impossible to control for all potentially relevant covariates, as it depends on which variables are available in the data; whether or not a given researcher includes the relevant variables in a model; and how well those variables represent the constructs they are measuring.

The sibling comparison is a *within-family* comparison and, therefore, inherently takes into account differences in environmental and genetic third-variables which may vary among participants who grow up in different families. In other words, to the extent that siblings are genetically similar, comparing siblings to each other automatically controls for genetic differences between families, as well as environmental factors shared by siblings, such as family income or parental monitoring—without having to specify such factors explicitly in a model (Dick, Johnson, Viken, & Rose, 2000; Lahey & D'Onofrio, 2010). If the magnitude of the within-family effect is attenuated compared to the between-family effect, this suggests that genetic and/or shared environmental factors in the same family account for the behavioral differences. These factors may include the sort of potentially heritable interpersonal qualities (e.g., neuroticism) highlighted by the individual differences model. Alternatively, if the effect is not attenuated, findings support an environmental mechanism for depressive symptoms being influenced by adolescent dating and sexual experiences (consistent, for example, with the precipitating mechanisms posited in the *attention impairment* model).

Although this logic broadly describes the underlying principles of all sibling comparison designs, there is one important moderating factor: the genetic relatedness of the siblings. Siblings who are more genetically similar to each other are assumed to be more similar to each other in terms of psychosocial outcomes, such as dating or age of first sexual intercourse, than siblings who are less genetically similar. This principle is most often employed in comparisons of twins reared together in the same household: when monozygotic twins (who share 100% of their genetic code) are more similar with regard to a particular outcome than dizygotic twins (who share 50% of their genetic code), the greater *phenotypic* similarity of MZ pairs relative to DZ pairs is assumed to be attributable to genetic variance. However, although most commonly used with twins, the sibling comparison can be applied to nontwin sibling dyads of any differing levels of genetic relatedness, as long as siblings who are more genetically similar to each other are compared to siblings who are less genetically similar.

In the present study, we conducted a series of models which estimate both between- and within-family effects. As a preliminary step, we estimated the basic phenotypic associations between dating, sexual activity with a romantic and nonromantic partner, and depressive symptoms. These associations are comparable to the effects obtained in traditional epidemiological studies. Participants were divided into groups based on dating and sexual history, and mean CESD scores and frequencies of clinical-range depression were calculated. Because this analysis does not take into account the nesting of siblings within families, it should be considered entirely descriptive: What is the "raw" difference in depressive symptoms between adolescents who date and/or have sex, versus those who do not?

Second, we constructed pair-level averages for each of the three romantic behaviors (dating, sexual activity with a romantic partner, sexual activity with a nonromantic partner) by averaging the siblings' scores in each pair, resulting in pairwise variables with values of 0 (neither sibling was involved in that behavior), 0.5 (one sibling was involved), or 1 (both siblings were involved). We also constructed individual-level deviation scores by subtracting the pairwise average from each individual's score, resulting in individual-level variables with values of −0.5 (e.g., the individual had not dated, but his or her sibling had), 0 (e.g., the individual and his or her sibling were concordant for dating or not dating), or 0.5 (e.g., the individual had dated, but his or her sibling had not).

The effect of the pairwise average is the *between-family effect*: Do families in which at least one sibling has dated or had sex show, on average, higher depressive symptoms? Notably, this association is confounded by genetic and environmental selection factors that vary between families. The effect of the individual-level deviation is the *within-family effect*: Do siblings who differ in their romantic or sexual experience also significantly differ in their depressive symptoms? Unlike the between-family effect, the within-family effect controls for genetic and environmental "third variables" that are shared by siblings raised in the same home, and thus, constitutes a stronger test of the causal relationship between romantic and sexual behavior and depressive symptoms.

Lastly, using the pairwise average and individual-deviation scores for each of the three romantic behaviors, we fit a series of mixed-effects models in the statistical program SAS (PROC MIXED), as described below. Although means comparisons can be an informative initial analytic step, mixed effects models are designed to account for nested data (in this case, individuals nested within siblings pairs; Raudenbush & Bryk, 2002), and are well-

suited for analyses of complex family designs. For all models, age and gender were included as covariates.

Results

Are Dating and Sexual Activity Correlated With Increased Depression?

As shown in Figure 1 and Figure 2, adolescents who were sexually active showed higher levels of depressive symptoms and had higher rates of clinical-level depression. Adolescents who reported dating but who were not yet sexually active reported roughly comparable levels of depressive symptoms ($M = 11.5$ for age 16–18; 11.0 for age 13–15) as adolescents with no experience with dating or sex ($M = 10.8$ for age 16–18, 10.2 for age 13–15). The rates of clinical-level depression were also similar for adolescents who were dating but not sexually active (18% for age 16–18, 15% for age 13–15) as for adolescents who were neither dating nor sexually active (17% for age 16–18, 15% for age 13–15). Consistent with previous research, the highest levels of depressive symptoms were among adolescents who had had sex with a nonromantic partner. Moreover, there was some initial evidence for age differences, with younger adolescents (age 13–15) who were involved in both romantic and nonromantic sex showing more depressive symptoms ($M = 14.9$) and higher rates of clinical-level depression (34%) than older adolescents (age 16–18; M CESD score = 13.4, rate of clinical-level depression = 29%).

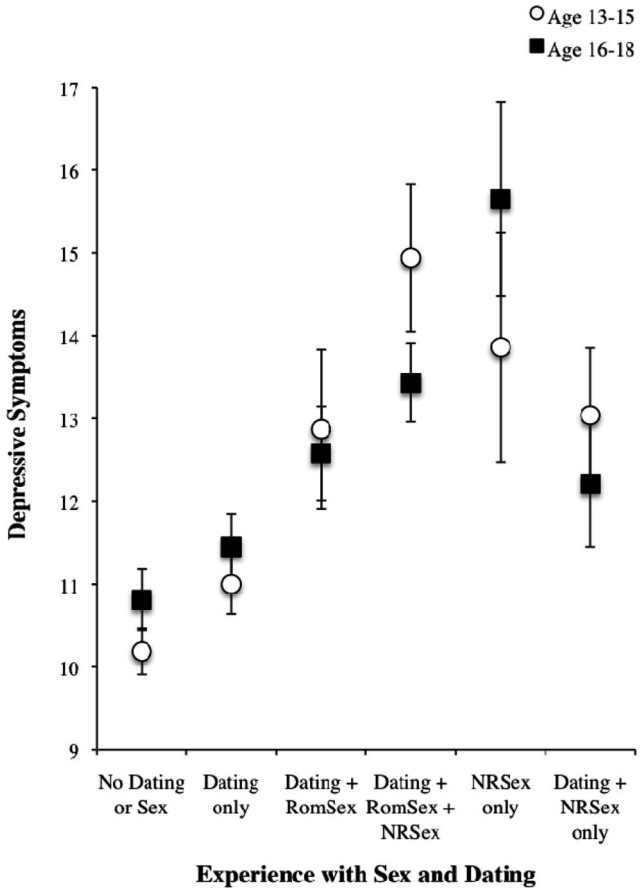

Figure 1. Mean numbers of depressive symptoms by experiences with sex and dating and by age group. *Note*. "RomSex" = sex in a romantic relationship. "NRSex" = sex in a nonromantic relationship.

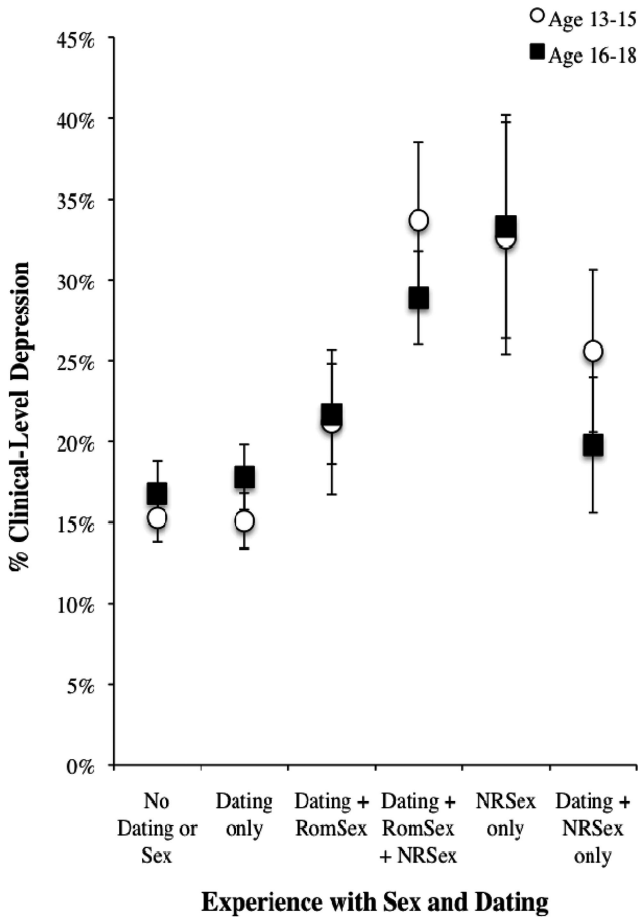

Figure 2. Rate of clinical-level depression by experiences with sex and dating and by age group. *Note.* "RomSex" = sex in a romantic relationship. "NRSex" = sex in a nonromantic relationship. Clinical-level depression classified as a CESD score ≥ 18.

Are Dating and Sexual Activity Associated With Increased Depression Within Families?

Effect size calculations. For depressive symptoms, we calculated the "phenotypic" effect sizes for experiences with sex and dating as Cohen's *d* (i.e., the mean difference in depressive symptoms between adolescents who did and did not report each behavior, divided by the pooled standard deviation.) Similarly, we calculated with "within-family" effect size, also expressed as Cohen's *d*, by taking the mean difference between siblings who were discordant for each behavior. These results are illustrated in Figure 3. As shown in the gray bars, there were small to moderate phenotypic effects of romantic behaviors on depressive symptoms, with the biggest mean difference evident for sexual activity with a nonromantic partner. However, these effects were all attenuated when comparing within discordant sibling pairs (the black bars of

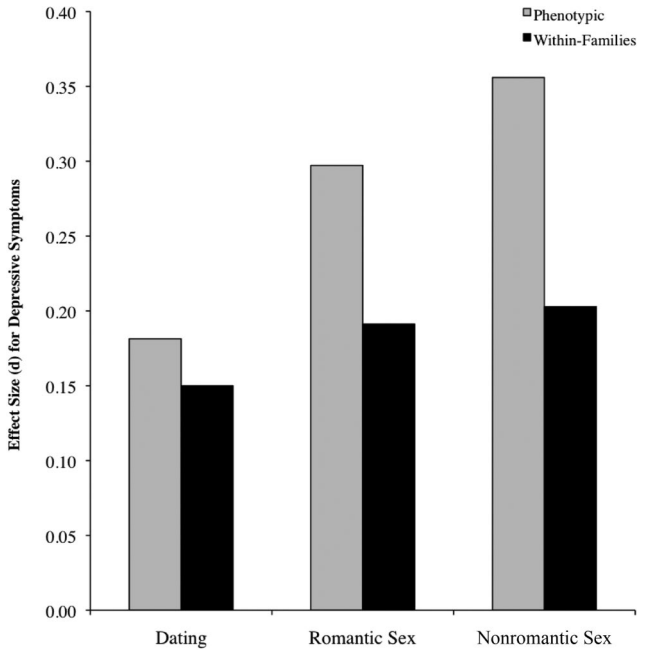

Figure 3. Effect sizes for dating, sex in a romantic relationship, and sex in a nonromantic relationship on depressive symptoms. *Note*. Effect sizes represented as Cohen's *d*. "Phenotypic" effect sizes represent mean differences between individuals who did versus did not report each behavior. "Within-family" effect sizes represent mean differences between siblings discordant for each behavior. If the magnitude of the within-family effect is attenuated compared to the phenotypic effect, this suggests that genetic and/or shared environmental factors in the same family account for the behavioral differences. If the effect is not attenuated, findings support an environmental mechanism for depressive symptoms being influenced by adolescent dating and sexual experiences.

Figure 2). In the case of sex with a nonromantic partner, the within-family effect size was approximately half of the phenotypic effect size. This pattern of results suggests that, while involvement in nonromantic sexual activity may be the strongest predictor of depressive symptoms, at least part of this association is due to between-family differences in environmental background and/or genes.

Similarly, we calculated the phenotypic and within-family effect sizes for clinical-range depression, expressed as odds ratios (ORs). That is, what is the likelihood that a participant will report a clinically severe level of depression given his or her involvement in dating, relationship sex, or nonromantic relationship sex? These results are illustrated in Figure 4, with the phenotypic ORs represented in gray and the within-family ORs represented in black. Phenotypically, the odds of reporting clinical-level depression were 1.25 times higher among individuals who were dating, 1.72 times higher among individuals who had sex in a romantic relationship, and 2.01 times higher among individuals who had sex in a nonromantic relationship. When comparing within discordant siblings, the ORs were commensurate or only slightly attenuated compared to the phenotypic effects. Again, the largest effect was evident for sexual activity in a nonromantic relationship.

Models of depressive symptoms. To evaluate the statistical significance of these results, as well as consider the potential moderating roles of gender and age, we first fit a series of mixed effects models of depressive symptoms, using SAS PROC MIXED (see Table 2). Each model estimated both random effects, representing the residual variance in depression attributable to the two levels of clustering in the design (between- and within-sibling pairs), and fixed effects, representing the regressions of depressive symptoms on the three romantic behavior variables, including an intercept. Because depressive symptom scores were log-transformed to reduce positive skew, regression coefficients are exponentiated in the text for interpretability.

Model 1 was a phenotypic model, which by definition does not include sibling or genetic information, and therefore mimics the results of a simple regression that would be obtained in epidemiological research. In Model 1, the three types of relationship behaviors—dating, sexual activity in a relationship, and sexual activity outside of a relationship—were modeled as fixed effects. We additionally included interactions between nonromantic sex and both dating and romantic sex. Although dating, in and of itself, was not significantly related to depressive symptoms, engaging in sexual activity in a romantic relationship was significantly associated with greater depressive symptoms ($\exp(B) = 1.13$), as was sexual activity with a nonromantic partner ($\exp(B) = 1.40$, $p < .05$). That is, adolescents who had sex outside of the context of a

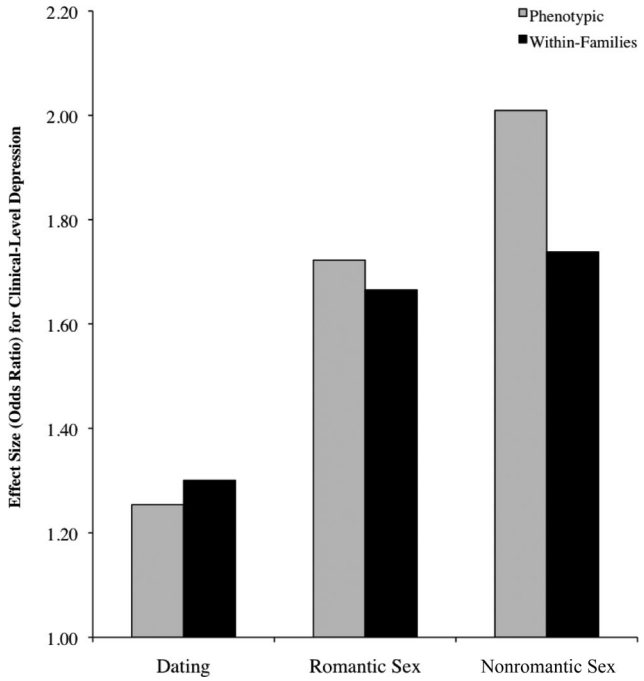

Figure 4. Effect sizes for dating, sex in a romantic relationship, and sex in a nonromantic relationship on clinical-level depression. *Note*. Effect sizes represented as odds ratios (ORs). "Phenotypic" effect sizes represent differences between individuals who did versus did not report each behavior. "Within-family" effect sizes represent differences between siblings discordant for each behavior. If the magnitude of the within-family effect is attenuated compared to the phenotypic effect, this suggests that genetic and/or shared environmental factors in the same family account for the behavioral differences. If the effect is not attenuated, findings support an environmental mechanism for depressive symptoms being influenced by adolescent dating and sexual experiences.

Table 2
Mixed Effects Models of Dating, Sexual Activity, and Depressive Symptoms

Parameter	Model 1	Model 2	Model 3**	Model 4
Random effects				
Twin pair	.12	.12	.12	.12
Individual residual	.36	.36	.36	.36
Fixed effects				
Intercept	**2.17* (.03)**	**2.23* (.03)**	**2.16* (.03)**	**2.24* (.04)**
Female	**.15* (.03)**	.08 (.05)	**.16* (.03)**	**.16* (.03)**
Age (centered at age 16)	.02 (.01)	**.04 (.02)***	**.03* (.01)**	**.03* (.01)**
Genetic relatedness				**−.14* (.05)**
Dating				
Dating (0 or 1)	.05 (.03)	−.02 (.04)		
Female × Dating		.09 (.06)		
Age × Dating		−.02 (.02)		
Within-family effect			.02 (.04)	−.04 (.09)
Between-family effect			.02 (.04)	.02 (.04)
Relatedness × Within-family effect				−.05 (.20)
Romantic sex				
RomSex (0 or 1)	**.12* (.05)**	.08 (.05)		
Female × RomSex		−.005 (.08)		
Age × RomSex		.02 (.03)		
Within-family effect			.03 (.04)	.06 (.10)
Between-family effect			**.11* (.06)**	**.10† (.06)**
Relatedness × Within-family effect				.13 (.16)
Nonromantic sex				
NRSex (0 or 1)	**.34* (.08)**	**.15* (.05)**		
Female × NRSex		.08 (.07)		
Age × NRSex		**−.06* (.03)**		
Within-family effect			**.12* (.05)**	.003 (.09)
Between-family effect			**.25* (.05)**	**.23* (.05)**
Age × Within-family effect			−.03 (.04)	−.04 (.04)
Age × Between-family effect			**−.07* (.03)**	**−.07* (.03)**
Relatedness × Within-family effect				.27 (.17)
Interactions between sex & dating behaviors				
NRSex × RomSex	−.08 (.08)			
NRSex × Dating	−.16 (.09)			

Note. Standard errors in parentheses.
* Significant at $p < .05$. † Marginally significant at $p = .06$. ** Model accepted as the best representation of the data.

romantic relationship were predicted to have 1.40 times greater depressive symptoms. Finally, nonromantic sex did not significantly interact with either dating experience or romantic sex, indicating that the effects of nonromantic sex did not depend on an adolescent's other sexual/romantic behaviors. Consequently, the interactions among behaviors were dropped in subsequent models.

Model 2 incorporated age and gender as potential moderators of the phenotypic association, to assess whether the association differed for boys versus girls, or for younger adolescents compared to older ones. There was a significant interaction between nonromantic sex and age ($b = -.05$, $P = .13$). This interaction is illustrated in Figure 5, which shows the association between age and depressive symptoms separately by whether or not the adolescent reported engaging in nonromantic sex. At all ages, adolescents who reported nonromantic sex also reported greater depressive symptoms, but this effect was greater for younger adolescents than for older adolescents. That is, the association between nonromantic sex and depressive symptoms decreased with age. The remaining age interactions and all gender interactions did not reach significance and were, therefore, eliminated from subsequent models.

Model 3 separated effects into between- and within-family components. First, there were significant between-family effects for sexual activity in a romantic relationship ($\exp(B) = 1.12$) and sexual activity in a nonromantic relationship ($\exp(B) = 1.28$). These between-family effects are confounded by genetic and environmental differences between families. In contrast, the within-family effect for sex in a romantic relationship was not significant, indicating that the elevated depressive symptoms seen among adolescents who have sex with romantic partners can be attributed entirely to genetic and environmental background factors that differ between families. However, there was a significant within-family effect for sex with a nonromantic partner ($\exp(B) = 1.13$): In a pair of siblings discordant for nonromantic sex, the sibling who had sex outside the context of a romantic relationship reported 1.13 times greater depressive symptoms, indicating an independent effect of nonrelationship sexual activity. Finally, the interaction between age and nonromantic sex continued to be significant between families, but not within-families. This result indicates that nonromantic sex is less strongly associated with depressive symptoms in older adolescents compared to younger adolescents. This is likely not because the "causal" effect of nonromantic sex differs with age, but rather because nonromantic sex is less of a marker for family environmental and genetic "third variables" (e.g., income, ethnicity, genes, family education, etc.) among older adolescents.

Finally, Model 4 introduced a measure of genetic relatedness, in order to test whether the level of genetic relatedness between siblings

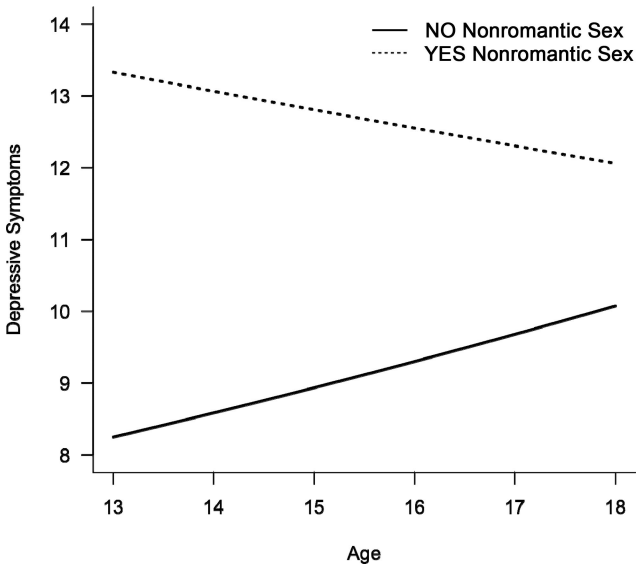

Figure 5. Interaction of nonromantic sexual activity by age.

moderated these findings. Genetic relatedness was coded according to additive genetic theory: 1 in MZ twins, .5 in DZ twins and full siblings, .25 in half siblings, .125 in cousins, and 0 in nonbiologically related pairs. If the relevant between-family confounds were genetic in origin, then we would expect that the magnitude of the within-family effect would decrease with increasing genetic relatedness of the siblings. All relatedness interactions were nonsignificant, and two out of three were in the opposite direction than would be predicted from a genetic model, suggesting that the relevant confounds are environmental, rather than genetic, in origin. These estimates, however, should be interpreted with caution; despite the reasonably large sample size, this analysis is still relatively underpowered to detect relatedness interactions. Because the relatedness interactions were not significantly different than zero, a model in which they are dropped (Model 3) is accepted as the best representation of the data.[3]

Models of clinical-range depression. We fit the same series of models using clinical-range depression (a binary variable) as the outcome (see Table 3). Because mixed effects models in SAS PROC MIXED are not available for binary outcomes, we instead accounted for nesting of siblings within families using generalized estimating equations (GEE) in SAS PROC GENMOD. Clinical-level depression was modeled using logistic regression, and effects are presented as ORs.

In Model 1, the phenotypic model, the three types of relationship behaviors—dating, sexual activity in a relationship, and sexual activity outside of a relationship—were introduced, along with age and gender. Notably, only sexual activity outside of a nonromantic relationship was associated with nearly three times the odds of clinical-level depression (OR = 2.77). In addition, females had more than twice the odds of clinical-level depression than males (OR = 2.13), consistent with a very large body of previous epidemiological evidence. As was the case with the models of depressive symptoms, neither romantic sex nor dating significantly moderated the effect of nonromantic sex on clinical-level depression; consequently, these interactions were dropped in all subsequent analyses.

Model 2 incorporated age and gender as potential moderators of the phenotypic associations. Unlike results for the continuous measure of depressive symptoms, there were no significant interactions between age and dating and sexual involvement. There were also no significant interactions with gender. Consequently, gender and age interactions were dropped from subsequent models.

Model 3 separated effects into between- and within-family components. There was a significant between-family effect for sexual activity in a nonromantic relationship. As described above, such between-family effects are confounded by genetic and shared environmental differences between unrelated individuals. However, the within-family effect for sex in a nonromantic relationship was also significant: In a comparison of discordant siblings, the adolescent who experienced sexual activity in a nonromantic relationship had 1.85 times greater odds of experiencing clinically severe depression than his or her sibling.

Finally, Model 4 tested whether the level of genetic relatedness between siblings moderated the within-family associations. As in the models of continuous depressive symptoms, the relatedness interactions were not significant, suggesting that the relevant between-family confounds are environmental rather than genetic in origin.

Discussion

A growing body of psychological research indicates that adolescent dating and sexual activity are robustly correlated with level and severity of depressive symptoms. Yet these associations covary with genetic and environmental influences for both depression and sexual behavior, limiting a straightforward interpretation. In the present article, we employed a family based design to clarify relations of dating and sexual activity with depression during adolescence.

In contrast to other studies, we obtained only marginal associations of dating with either depressive symptoms or clinically severe depression. This inconsistency may be due, in part, to the fact that not all previous research has distinguished between sexually active and nonsexually active dating relationships: Teenagers who date are more likely to be involved in an array of sexual behaviors, both in the context of their dating relationships and outside of them. Yet, as indicated by the difference of the between- and within-family effect sizes, much of the epidemiological asso-

[3] To examine more thoroughly varying genetic relationship between different types of sibling pairs, we conducted a set of follow-up analyses restricted to MZ twin pairs and nonbiologically related sibling pairs. Specifically, we estimated additional mixed effects models of depressive symptoms, similar to the models presented in Table 2, separately for MZ twin families and nonbiological sibling families. Because of the comparatively small number of MZ twin pairs and nonbiological sibling pairs, these follow-up models did not estimate any age interactions and were not adequately powered to detect statistically significant effects. In fact, the between-family effects for MZ pairs were consistently larger than the between-family effects for NR pairs; however, the within-family effects were also larger in MZ pairs. Given the small size of each group and the resulting unreliability of estimates, we think that these follow-up analyses suggest that the associations between romantic/sexual behavior and depressive symptoms could potentially be confounded by genetic factors. However, answering this question would require future research in a data set with a larger number of MZ twins. Results of these follow-up analyses are available upon request.

Table 3
GEE Models of Dating, Sexual Activity, and Clinical-Range Depression

Parameter	Model 1	Model 2	Model 3**	Model 4
Female	**2.13 (.11)***	**1.85 (.18)***	**2.18 (.11)***	
Age (centered at age 16)	1.04 (.04)	1.08 (.06)	1.05 (.04)	**1.05 (.04)**
Genetic relatedness				
Dating				
Dating (0 or 1)	1.01 (.14)	.91 (.12)		
Female × Dating		1.12 (.25)		
Age × Dating		.96 (.21)		
Within-family effect			1.08 (.19)	.84 (.32)
Between-family effect			0.84 (.14)	.84 (.14)
Relatedness × Within-family effect				1.69 (.69)
Romantic sex				
RomSex (0 or 1)	1.41 (.25)	1.31 (.20)		
Female × RomSex		.84 (.29)		
Age × RomSex		.97 (.29)		
Within-family effect			1.33 (.27)	1.42 (.60)
Between-family effect			1.22 (.25)	1.22 (.25)
Relatedness × Within-family effect				−.12 (.84)
Nonromantic sex				
NRSex (0 or 1)	**2.77 (.66)***	**1.82 (.24)***		
Female × NRSex		1.48 (.26)		
Age × NRSex		.91 (.25)		
Within-family effect			**1.50 (.28)***	1.48 (.51)
Between-family effect			**2.29 (.41)***	**2.29 (.41)***
Relatedness × Within-family effect				1.04 (.71)
Interactions between sex & dating behaviors				
NRSex × RomSex	.92 (.28)			
NRSex × Dating	.61 (.31)			

Note. Standard errors in parentheses.
* Significant at $p < .05$. ** Model accepted as the best representation of the data.

ciations between adolescent sexual behavior and depressive symptoms seems attributable to broad between-family differences in genes and/or shared environmental factors which are notoriously difficult to control for in standard correlational research. When accounting for these potential confounds, only sexual activity in a nonromantic relationship remained as a consistent predictor of both continuous levels of depressive symptoms and of clinical-range depression.

Overall, these findings are consistent with the perspective that the developmental salience of adolescent sexual activity depends on the relationship context in which it occurs. They are moreover congruent with our previous genetically informed studies regarding delinquency and adolescent sexual activity (Harden & Mendle, 2011; Harden et al., 2008). Together, this work provides converging evidence that understanding the psychological correlates of adolescent sexual behavior is more complex than a simple dichotomous consideration of virginity status might indicate. In the case of the present results, since genetic and shared environmental confounds cannot explain the association between nonrelationship sexual activity and depression, our findings suggest that it is the social experiences linked with this activity which play a key role. These experiences are likely as varied and individualized as the circumstances which underlie each "hook up." They may include gossip or rumors about a teenager's sexual behavior within his or her social community; the discomfort of watching previous sexual partners devote attention to others; or intense feelings or conflicts in the aftermath of a hook-up. Given that adolescents are most likely to engage in nonrelationship sex with past boyfriends or girlfriends or with individuals with whom they would *like* to have a relationship (Manning et al., 2006), it is logical that such situations would be emotionally fraught. Thus, even though adolescents engage in nonrelationship sexual activity consensually and voluntarily, they may find the incongruities between sexual and emotional intimacy difficult; hold unrealized expectations for sexual intimacy; or utilize these encounters as a signal of or attempt to remedy preexisting distress. Yet even if these interactions may be tumultuous or vexing to the individuals invested in them, the capacity and interest for such liaisons is not necessarily a sign of pathology and may in fact index normal developmental and relational desires. Indeed, given the prevalence of such activity within our sample, it is likely this is the case.

Consistent with previous research (e.g., Monahan & Lee, 2008), our findings suggest an interaction of age with nonrelationship sexual activity and depression. At all ages, adolescents who reported nonromantic sex also reported greater depressive symptoms, but this effect was greater for younger adolescents than for older ones. One possible explanation is that nonrelationship sexual activity is simply a rarer—and thus, more aberrant—behavior among the younger adolescents in our sample. Second, as shown in Figure 5, the older adolescents in our sample tended to display greater levels of depressive symptoms, which may have diminished the independent associations of nonromantic sexual activity.

Surprisingly, our analyses obtained no evidence of gender as a moderator of sexual behavior and depressive symptoms. Both media attention (e.g., Flanagan, 2008) and previous academic scholarship (e.g., Cotton et al., 2004; Joyner & Udry, 2000) have

suggested romantic activity may be more salient and potentially distressing for girls than for boys, given concerns of pregnancy, stigmatization of sexually active women, and females' greater tendency toward rumination and depression. Yet it may be the case that although being depressed is more common among adolescent girls than boys, the mechanisms by which sexual experiences relate to depressive symptoms are nevertheless similar across gender. This could occur if these mechanisms are reliant on situations which are broadly characteristic of adolescence as a developmental stage, regardless of gender—such as high emotionality, immature prefrontal development, and increased sensitivity to social interactions.

Limitations and Future Directions

Although the present analyses do implicate a significant association between nonromantic sexual behavior and psychological distress, it should be clearly noted that these analyses are cross-sectional and, therefore, formally ambiguous regarding the temporal sequencing of such an association. This is particularly important given that previous, nongenetically informative analyses of the Add Health data have suggested that high levels of depressive symptoms longitudinally precede onset of hooking up and risky sexual behaviors (Lehrer et al., 2006; Monahan & Lee, 2008). Our data, in addition, is not informative about either the quality of participants' dating relationships or the identity of their nonrelationship sexual partners. Of particular benefit for future analyses will be a more detailed awareness of the interplay between these conditions and levels of psychological distress. It seems probable that adolescent sexual activity is not referencing the sexual act, per se, as much as serving as a proxy for intricate emotions, desires, attitudes, insecurities, and so on. As this depth cannot be captured in a binary "sex or no sex" coding scheme, research which incorporates additional specific details of partners will likely be of benefit in developing and testing more specific hypotheses for the mechanisms by which adolescent sexual behavior relates to psychological distress. Lastly, although sibling comparisons provide a powerful control for factors which are shared among children growing up in the same family, the comparison—by definition—does not control for the variables which differ between members of a sibling dyad. The design attributes significant differences among sibling dyads to an effect of the environmental experience of interest—here, romantic and dating behaviors. Because it does not control for nonfamilial confounds, it would be plausible for the more depressed member of a sibling dyad both to engage in nonrelationship sexual activity and be exposed to a third variable confound (to which their sibling was not exposed) which might explain the association between nonrelationship sexual activity and depressive symptoms. For example, certain stressful life events (including rape, trauma, or abuse) might both be nonshared experiences across members of sibling dyad and increase likelihood of both depressive symptoms and nonrelationship sexual activity.

Conclusion

Although many historical studies consider an earlier age of first intercourse a measure of problematic and socially deviant behavior, divergent opinions on the nature and meaning of adolescent sexuality have emerged in recent years (see Tolman & McLelland, 2011 for a review). Our results suggest this heterogeneity is merited, as adolescent sexual intercourse may index different underlying facets of development and emotion across different individuals and situations. The current study used a genetically informed sample of adolescent sibling dyads to investigate the relationship between adolescent dating, sexual activity, and depressive symptoms. Only sexual activity with a noncommitted partner proved to be a unique environmental risk factor for depression after controlling for between-family confounding factors. Continued exploration of how the transition to sexual maturity may be moderated by contextual factors can help clarify the particular developmental challenges and stressors of adolescence.

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