ORIGINAL ARTICLE

First Onset of Depressive or Anxiety Disorders Predicted by the Longitudinal Course of Internalizing Symptoms and Parent-Adolescent Disagreements

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Background: Growing evidence indicates that anxiety and depressive disorder onset may involve a prodromal buildup of symptoms. Also, stressful life events may precipitate gradual symptom increase, leading to the development of full-blown disorders. This study used prospective longitudinal data to examine the theory that, over time, stressful events, such as parent-adolescent disagreements, influence the longitudinal course of adolescents' internalizing symptoms, which in turn predict first onset of a depressive or anxiety disorder.

Methods: A community sample of 303 families with an adolescent aged 12 or 13 years in year 1 provided repeated measures of parent-adolescent disagreements and adolescents' internalizing symptoms over 3 and 4 years, respectively. At age 19 or 20 years, the adolescents were assessed for anxiety and depressive disorders using structured interviews based on *DSM-III-R* criteria. The hypothesized associations were estimated using latent growth curve modeling procedures.

Results: Year 1 parent-adolescent disagreements predicted year 1 adolescents' internalizing symptoms, and changes in disagreements from year 1 to year 3 predicted changes in internalizing symptoms from year 1 to year 4. Both the year 1 level and changes in symptoms predicted internalizing disorder onset in years 4 through 7, and both the year 1 level and changes in disagreements indirectly predicted disorder onset.

Conclusions: Among adolescents, persistent or escalating stressful events, such as disagreements with parents, indirectly increase the risk for internalizing disorder onset through their direct association with high or increasing symptom levels. Chronically high or increasing symptom levels directly increase risk for internalizing disorder.

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GROWING EVIDENCE suggests that onset of anxiety and depressive disorders involves a prodromal buildup of symptoms.[1-4] Specifically, evidence indicates that a clinical process spanning months or years often precedes disorder onset,[4,5] individuals with high yet subthreshold symptoms at one time point are the ones most likely to meet the criteria for disorder at a later time point,[1,3,4,6] and considerable variation in symptom levels occurs from one time point to another.[7] Other research implicates stressful events in the slow onset of, for example, a major depressive episode,[7] such that they precipitate a gradual increase in symptoms leading up to full-blown disorder.

Unfortunately, reports linking prodromal symptoms to the onset of anxiety or depressive disorders rely entirely on either one-time follow-up studies that cannot describe symptom course or retrospective recall, which is often unreliable.[8] Likewise, heavy reliance on cross-sectional or one-time follow-up designs has limited researchers and clinicians to the static knowledge that severity or presence of a stressful event predicts severity or presence of symptoms or disorder. Yet to be tested is how changes in stress over time relate to changes in symptoms over time and how changing symptoms relate to internalizing disorder onset. The present study undertook this test, focusing on parent-adolescent disagreements as the stressor of interest.

In designing this investigation, we attended to specific findings from earlier research. For example, we noted that prodromal symptoms of depression or anxiety may begin developing several years prior to disorder onset,[5,7] at least three fourths of prodromal symptoms precede disorder onset by less than 5 years,[5] and, among adolescents, the risk for first onset of depression increases sharply between ages 15 and 18 years.[9] Therefore, symptom reports were collected annually over 4 years, beginning when the adolescents were aged

PARTICIPANTS, MATERIALS, AND METHODS

SAMPLE

At year 1 (1989), the sample consisted of 451 white, primarily lower–middle- and middle-class families that included 2 biological parents, a grade 7 target adolescent (mean age, 12.7 years; SD, 0.54 years; 236 girls, 215 boys), and a male or female sibling who was within 4 years, either older or younger, of the target adolescent's age. Median family income for 1988 was $33 399.

Families were recruited from all 34 schools with a grade 7 class in selected communities in Iowa. They received a letter explaining the research project[10] and were subsequently contacted by telephone and asked to participate. About 78% of the eligible families joined the study. Each participating family member was paid approximately $10 per hour of participation.

Over 7 years, 29 families withdrew from the study, producing a retention rate of 93.6%. During years 1 through 3, however, 9 families experienced divorce. Because the present study required complete family data during these years, these families were dropped from the analyses. Sixty-two target adolescents (34 girls, 28 boys) reported disorder onset (depressive disorder, n = 15; anxiety disorder, n = 47) prior to year 4. They were dropped from the analyses because of this study's focus on first onset of an internalizing disorder during or after year 4. Missing data from 48 families resulted in a final sample of 303 families.

Included and not-included families and target adolescents did not significantly differ on income or any study variable. Included and not-included fathers did differ significantly ($P<.001$) on education (not-included fathers, 12.74 years; SD, 1.4 years; included fathers, 13.58 years; SD, 2.2 years).

PROCEDURES

Participants (2 parents and 2 children) received 2 home visits from a trained interviewer from 1989 (year 1) through 1992 and then again in 1994 and 1995 (year 7). During visit 1, participants completed informed consent forms and a set of questionnaires. In year 7, target adolescents completed the Composite International Diagnostic Interview (CIDI).[11] During visit 2, participants completed questionnaires and videotaped structured interaction tasks.

MEASURES

Parent-adolescent disagreements were measured in years 1, 2, and 3 using a parent-child disagreement checklist developed for the study. This 20-item checklist covered areas of disagreement such as household chores, transportation, and use of alcohol. Both parents and children completed the checklist, but to avoid artificially inflating associations between disagreements and target adolescents' symptoms,[12-15] target adolescents' reports were not included in the disagreements measure. For each checklist item, parents rated how often (0, never, to 4, always) they disagreed with or engaged in upsetting arguments with the target adolescent. The sibling rated disagreements between him/her and the parents. For each of the 3 years, the parents' and siblings' measures were added to create a single disagreements variable. These 3 variables served as indicators of the base level and slope for parent-adolescent disagreements. Internal consistency reliability for these indicators were 0.92 in each year. Other empirical studies provide support for the construct validity of this checklist.[16]

To assess target adolescents' internalizing symptoms in years 1 through 4, they completed the depression, anxiety, and hostility subscales of the Symptom Checklist-90-Revised (SCL-90-R).[17] Each year's responses were added to create single measures. Internal consistencies ranged from 0.92 in year 2 to 0.96 in year 4. The 4 annual measurements were used to indicate the base level and slope for internalizing symptoms.

In year 7 (mean age, 19.4 years; SD, 0.52 years), internalizing disorder onset was assessed using the University of Michigan's modified CIDI.[11] This fully structured diagnostic interview generates *DSM-III-R*[18] lifetime psychiatric disorder diagnoses for both adolescents and adults. Its primary modification brought all screening questions together into an introductory diagnostic screen. World Health Organization field trials show that the CIDI possesses good reliability and validity.[19] To further assure reliability, all interviewers underwent a 5-day training workshop, all interview materials were double-checked by

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12 or 13 years; we predicted first onset of internalizing disorder beginning at age 15 or 16 years. Also, the number and severity of parent-adolescent disagreements were measured annually across 3 years, starting at age 12 or 13 years. This design allowed us to test 2 hypotheses: The level and course of parent-adolescent disagreements predict the level and course of symptoms, which in turn predict first onset of internalizing disorder. The level and course of parent-adolescent disagreements indirectly predict first onset of internalizing disorder through their association with internalizing symptoms.

RESULTS

In support of the first study hypothesis, **Table 2** shows that, at each time point, the target adolescents with disorders reported higher levels of internalizing symptoms and their families experienced more parent-adolescent disagreement than adolescents and families in the nondisordered group. Logistic regression results, presented in **Table 3**, were also supportive, showing that, when considered separately, both disagreements and internalizing symptoms generally predicted disorder onset. Table 3 also presents results supporting the second hypothesis. These results show that, when considered simultaneously, only symptom levels directly predicted disorder at 2 of 3 time points, suggesting that internalizing symptoms most often mediated the effect of disagreements.

While useful as a first step, these cross-sectional analyses do not fully test our theoretical model. The model hypothesized a longitudinal relationship among disagreements, symptoms, and disorder onset. Using LGC analy-

research staff, and all interviews were audiotaped. Reliability tests using 10% of the audiotapes showed 100% agreement between CIDI diagnoses and diagnoses made by advanced clinical psychology graduate students blind to the CIDI diagnoses.

Forty target adolescents experienced first onset of an internalizing disorder in years 4 through 7 (**Table 1**). Those who reported first onset of a depressive disorder (major depressive episode or dysthymia) during or following year 4 were considered to have experienced depressive disorder onset (0, no depressive disorder; 1, depressive disorder onset during or after year 4). Those who experienced first onset of an anxiety disorder (simple phobia, social phobia, agoraphobia, or panic attack) during or after year 4 were considered to have experienced anxiety disorder onset (0, no anxiety disorder; 1, anxiety disorder onset during or after year 4). These 2 variables served as indicators of internalizing disorder onset.

STATISTICAL ANALYSIS

Initial hypothesis tests used logistic regressions with an outcome variable coded 0 for no disorder and 1 for internalizing disorder. Further hypothesis tests were performed using latent growth curve (LGC) modeling procedures.[20] An LGC model simultaneously estimates differences between individuals (eg, differences between numerous individuals' base symptom levels) and differences within individuals (eg, each individual's symptom changes over time). It extends random regression models[21] to allow covariation of slopes. For example, for a repeated measure assessed at 3 time points, each individual's base level score and change over time (slope) are estimated as latent constructs indicated by the 3 repeated measurements. Mean base levels and mean slopes are estimated and their variations are used as correlates or predictors of other variables. The correlated and predicted variables could include the base level and/or slope of another repeated measure or a time-invariant variable.

This study's LGC model was analyzed using maximum likelihood estimation procedures.[22] It included 2 repeated-measures variables (parent-adolescent disagreements and target adolescents' internalizing symptoms) and 1 time-invariant variable (internalizing disorder onset). The model specified that base-level (year 1) target adolescents' internalizing symptoms be regressed on base level (year 1) parent-adolescent disagreements. Simultaneously, the slope for symptoms, estimated using annual measurements from years 1 through 4, was regressed on the slope for disagreements from years 1 through 3, and disorder onset was regressed on both the base level and slope for symptoms. The model also specified correlations between the base level and slopes for both disagreements and symptoms and estimated the indirect effect of disagreements on disorder onset. For all model parameters, a t value of 1.96 or higher was considered statistically significant at the .05 level.

In general, this study used continuous, normally distributed[23] variables (skewness ranged from 0.12-1.71, kurtosis from 0.40-3.58). The disorder measures, however, were dichotomous, and although they combined with the other study variables to produce an acceptable multivariate distribution (kurtosis, 2.39), we chose to cautiously specify and interpret the LGC analyses. For example, Satorra[24] instructs that under conditions of nonnormality, maximum likelihood estimates of model parameters are reliable and accurate, except for covariances among the residual errors of variable indicators. Therefore, our model did not specify correlations among indicator residual errors. The model fit indices supported this cautious approach. Nonnormal data can also produce an inflated χ^2 statistic, increasing the chances of rejecting an acceptable model (type I error).[25] Therefore, we report multiple fit indices and acknowledge that these LGC analyses may be more conservative than those performed with more normally distributed data.

Preliminary tests using analysis of variance and LGC analyses failed to reveal significant subsets within the sample. For example, target adolescents reporting early disorder onset (in year 4 or 5) did not differ from target adolescents with later disorder onset (in year 6 or 7) on disagreement and symptom levels. Also, the disordered and nondisordered groups showed no sex differences except that girls with no disorders reported higher symptoms than boys with no disorders. Finally, although small sample sizes reduced the number of significant relationships, LGC models estimated separately for target adolescents with only anxiety and only depressive disorders produced similar results. Therefore, the reported analyses used the full sample.

ses, we were able to examine these longitudinal associations (Table 2).

LGC ANALYSES

Initial Findings

Table 4 presents the variable means, SDs, and the correlation matrix used to perform the LGC analyses. **Table 5** presents the LGC results. For univariate characteristics, the mean base level of symptoms (mean, 13.22) is the mean symptom level target adolescents reported in year 1. The statistically significant variance in base-level symptoms indicates that, around this mean, target adolescents reported substantial differences in base-level symptoms. The mean slope for symptoms (–0.71) was estimated by first calculating each target adolescent's slope for symptoms over time. All 303 slopes were then averaged to create the mean slope. The negative coefficient indicates that, on average, target adolescents' internalizing symptoms declined over time. Table 2 shows an overall symptom decline reported by target adolescents with no disorders. However, the statistically significant variance in the slope suggests substantial individual differences in symptom course. Some target adolescents seem to show more or less symptom decline than average, and others' symptoms may have increased over time.

Table 5 also presents correlations between the base level and slope for disagreements and symptoms. The negative correlation shown for target adolescents' internalizing symptoms (–0.30) indicates that target adolescents reporting the highest base-level symptoms usually showed the slowest increase, or possibly a decline

Table 1. Incidence of Disorder Among Target Adolescents Experiencing Disorder Onset From Year 4 (Age 15 Years) to Year 7 (Age 19 Years)

Diagnosis	No. (%) of Cases*		
	Girls (n = 160)	Boys (n = 143)	Total (N = 303)
Major depressive episode	19 (11.9)	2 (1.4)	21 (6.9)
Dysthymia	3 (1.9)	0 (0.0)	3 (1.0)
Agoraphobia	2 (1.2)	3 (2.1)	5 (1.7)
Social phobia	9 (5.6)	6 (4.2)	15 (5.0)
Simple phobia	6 (3.8)	2 (1.4)	8 (2.6)
Panic attack	3 (1.9)	1 (0.7)	4 (1.3)
Depressive disorder only	13 (8.1)	1 (0.7)	14 (4.6)
Anxiety disorder only	11 (6.9)	8 (5.6)	19 (6.3)
Depressive and anxiety disorder	6 (3.8)	1 (0.7)	7 (2.3)
Total	30 (18.8)	10 (7.0)	40 (13.2)

*Because of comorbidity, the numbers of cases exceed the totals.

Table 2. Mean Levels of Parent-Adolescent Disagreement and Internalizing Symptoms Among Target Adolescents With (n = 40) and Without (n = 263) Disorders*

Study Year	Parent-Adolescent Disagreements		Internalizing Symptoms	
	Target Adolescents With Disorders	Target Adolescents With No Disorders	Target Adolescents With Disorders	Target Adolescents With No Disorders
1	93.24 (5.3)	80.53 (1.2)	17.53 (2.8)	14.35 (0.75)
2	87.61 (5.2)	81.08 (1.2)	19.18 (2.5)	11.15 (0.65)
3	92.15 (5.2)	80.27 (1.3)	21.41 (2.5)	10.56 (0.59)
4	. . .	. . .	20.90 (3.5)	11.74 (0.75)

*Values are mean (SE). Ellipses indicate that no measurement of disagreement was performed in study year 4.

Table 3. Unstandardized Results of the Logistic Regression of Disorder Onset on Parent-Adolescent Disagreements, Internalizing Symptoms of Target Adolescents, and Disagreements and Symptoms at Each Time Point

Study Year	Independent Variable	β (SE)	P
	Parent-Adolescent Disagreements		
1	Disagreements	0.02 (0.01)	.01
2	Disagreements	0.02 (0.01)	.10
3	Disagreements	0.02 (0.01)	.01
	Target Adolescents' Internalizing Symptoms		
1	Symptoms	0.01 (0.01)	.52
2	Symptoms	0.04 (0.01)	.01
3	Symptoms	0.03 (0.01)	.01
4	Symptoms	0.03 (0.01)	.00
	Parent-Adolescent Disagreements and Target Adolescents' Internalizing Symptoms		
1	Disagreements	0.02 (0.01)	.01
	Symptoms	0.00 (0.01)	.92
2	Disagreements	0.01 (0.01)	.52
	Symptoms	0.03 (0.01)	.01
3	Disagreements	0.02 (0.01)	.06
	Symptoms	0.03 (0.01)	.01

in symptoms over time, relative to target adolescents initially reporting few symptoms. This result could also be an artifact of a ceiling effect or of the regression to the mean phenomenon.

Hypothesis Testing

The LGC model fit indices in Table 5 indicate how well the associations proposed in the theoretical model correspond to the experiences of the families and target adolescents in the study.[26] In each case, the fit indices support the theoretical model.

The path coefficients presented in Table 5 also support the theoretical model. Base-level parent-adolescent disagreements predicted base-level target adolescents' internalizing symptoms ($R^2 = 0.13$, $P < .001$), the slope for disagreements predicted the slope for internalizing symptoms ($R^2 = 0.09$, $P < .001$), and both the base level and slope for symptoms predicted disorder onset ($R^2 = 0.23$, $P < .001$). Finally, both the base level and slope for disagreements indirectly predicted disorder onset, although the statistical significance of the slope's effect was marginal (Table 5).

While these analyses support the study hypotheses, alternative hypotheses must be considered. For example, base-level parent-adolescent disagreements could potentially predict changes in the target adolescents' internalizing symptoms. The LGC model testing this alternative hypothesis included the path from base-level disagreements to slope for symptoms. A second alternative model tested the hypothesis that parent-adolescent disagreements directly influence disorder onset. This alternative model included direct paths from the base level and slope for disagreements to disorder onset. Third, we tested curvilinear relationships among the study variables by adding the quadratic forms of the slope for both disagreements and symptoms to a third alternative LGC model. The fourth alternative model tested the mediating effect of conduct disorder on the relationship between disagreements and internalizing symptoms. This model included direct paths from the base level and slope for disagreements to an observed conduct disorder factor and from conduct disorder to the base level and slope for symptoms. In all 4 cases, the alternative model χ^2 values did not significantly differ from the theoretical model's χ^2 value. Also, none of the additional paths produced statistically significant coefficients. Taken together, these results indicate that, compared with the alternative models, the theoretical model provided the best, most parsimonious representation of the sample's experiences.

One final alternative hypothesis remained. Considering evidence showing that emotionally distressed individuals may induce conflict among family members,[27,28] this hypothesis proposed an influence from the slope for target adolescents' internalizing symptoms to the slope for parent-adolescent disagreements. To test this bidirectional hypothesis, we developed an autoregressive crosslagged structural equation model. This form of statistical model estimates change over time by simulta-

Table 4. Correlations, Means, and SDs for the Study Variables

Variables	Study Variables 1	2	3	4	5	6	7	8	9	Mean (SD)
Parent-adolescent disagreements										
1. 1989	1.00									81.08 (21.62)
2. 1990	0.70*	1.00								80.96 (20.03)
3. 1991	0.55*	0.66*	1.00							80.58 (21.34)
Adolescents' internalizing symptoms										
4. 1989	0.27*	0.22*	0.17*	1.00						14.69 (13.61)
5. 1990	0.19*	0.21*	0.17*	0.54*	1.00					11.57 (11.08)
6. 1991	0.19*	0.22*	0.24*	0.40*	0.59*	1.00				11.66 (11.57)
7. 1992	0.17*	0.15*	0.23*	0.32*	0.51*	0.61*	1.00			11.79 (14.22)
Internalizing disorder onset										
8. Depressive disorder	0.12†	–0.01	0.09	0.04	0.18*	0.23*	0.21*	1.00		0.07 (0.21)
9. Anxiety disorder	0.06	0.05	0.07	0.08	0.15*	0.25*	0.18*	0.35†	1.00	0.09 (0.22)

*P<.01.
†P<.05.

neously controlling a variable's earlier level and estimating the contribution of a covariate to rank order changes in the variable. The model estimated the interrelationships among 1989 disagreements and 1989 internalizing symptoms and 1991 disagreements and symptoms. It produced statistically significant effects only from disagreements to symptoms (β = .15; b, 0.25; 95% CI, 0.03-0.50; t = 1.98), supporting the study hypothesis and not the alternative hypothesis.

COMMENT

This study's results provide a rare demonstration of the longitudinal links among stressful events, symptom course, and disorder onset. In doing so, the findings both replicate earlier research and extend what we know about the link between stress and disorder and the prodromal period preceding disorder onset. Of course, the findings must be discussed within certain limits. Specifically, this study investigated processes leading to first onset of internalizing disorder among adolescents. Findings from other reports do indicate that interpersonal disagreements among adults also lead to emotional distress[29,30] and that a prodromal buildup of symptoms may precede adult onset[3,4] and recurrence[6,31] of internalizing disorder. However, until supported by a comprehensive test, this study's findings should not be generalized to adults or to the recurrence of internalizing disorder. Also, the experiences of this rural Midwestern US sample may not be generalizable to individuals from different backgrounds. Finally, the study employed retrospective recall of the disorder onset date, and many question the accuracy and precision of such reports.[32] To alleviate some concerns, this study required recall to within a period of years rather than to a specific time point. That is, if a target mistakenly reported disorder onset in year 6 when it actually occurred in year 5, the study's results would not change. Nevertheless, the results require confirmation from tests using prospective disorder onset reports.

Within these limitations, this study's findings are significant in a number of respects. First, they replicate earlier findings of a positive association between stress, in the form of interpersonal disagreements, and internalizing symptoms.[27,33,34] They extend earlier work by demonstrating a positive link between changes in disagreements and changes in symptoms. Specifically, we found that families reporting intensifying parent-adolescent disagreements over time usually included an adolescent who reported increasing internalizing symptoms over that same period. Conversely, diminishing disagreements predicted symptom decline. No other study directly demonstrates this longitudinal association between changes in stress and changes in prodromal symptoms.

This study also adds to our understanding of the prodromal phase of anxiety and depressive disorders. The findings agree with earlier research[1,3,4,6] showing that individuals reporting high internalizing symptoms at one time point are at increased risk for later internalizing disorder onset and extend that research to include the first onset of an internalizing disorder during adolescence. Moreover, we found a clear relationship between the longitudinal course of symptoms and internalizing disorder onset. According to these data, increasing symptoms levels over a 4-year period predict disorder onset.

The analyses reveal 2 possible prodromal routes to the first onset of adolescents' internalizing disorder. The first route is usually characterized by chronically high prodromal symptom levels. That is, the negative association found between base-level symptoms and symptom slope indicates that target adolescents reporting high base-level symptoms often reported only slowly increasing or stable symptom levels over time. Symptom decline was unlikely since the probability of disorder onset decreased as symptoms declined. Future research should, however, test the possibility that high, stable symptom reports were caused by a ceiling effect. Second, among targets with disorders who reported relatively lower base-level symptoms, the positive relationship between symptom slope and disorder onset suggests a prodromal period characterized by increasing symptoms over time.

Predicting the first onset of a depressive or anxiety disorder is an enduring public health concern, and the identification of adolescents at risk for internalizing dis-

Table 5. Latent Growth Curve Results From the Maximum Likelihood Estimation of the Hypothesized Relationships*

Univariate Characteristics

Variables	Base Level		Slope	
	Mean	Variance	Mean	Variance
Parent-adolescent disagreements	81.12 (t = 66.12)	350.27 (t = 8.18)	–0.25 (t = –0.42)	38.80 (t = 2.26)
Adolescents' internalizing symptoms	13.22 (t = 19.34)	87.13 (t = 7.12)	–0.71 (t = 2.39)	10.51 (t = 4.13)

Correlations

Variables	Correlation	Covariance (95% CI)	t
Parent-adolescent disagreements			
Base level/slope	–0.41	–50.09 (–88.66 to –11.52)	–2.55
Adolescents' internalizing symptoms			
Base level/slope	–0.30	–9.14 (–17.85 to –0.43)	–2.06

Model Fit Indices

χ^2	SRMR	RMSEA	GFI	CFI	Critical N
44.04 (df = 28, P = .03)	0.03	0.04	0.98	0.98	332

Path Coefficients

Independent Variable	Dependent Variable	Standardized Coefficient	Unstandardized Coefficient (95% CI)	t
Base-level disagreements	Base-level symptoms	0.36	0.18 (0.12 to 0.24)	5.46
Slope for disagreements	Slope for symptoms	0.31	0.16 (0.02 to 0.30)	2.19
Base-level symptoms	Disorder onset	0.37	0.01 (0.003 to 0.013)	3.75
Slope for symptoms	Disorder onset	0.46	0.02 (0.01 to 0.03)	3.45

Indirect Effects

Independent Variable	Dependent Variable	Standardized Coefficient	Unstandardized Coefficient (95% CI)	t
Base-level disagreements	Disorder onset	0.13	0.001 (0.00 to 0.001)	3.10
Slope for disagreements	Disorder onset	0.14	0.002 (–0.000 to 0.005)	1.88

**CI indicates confidence interval; SRMR, standardized root mean residual; RMSEA, root mean square error of approximation; GFI, goodness of fit index; and CFI, comparative fit index.*

order is made all the more important by research indicating that adolescent onset of either an anxiety or depressive disorder substantially increases the risk of adulthood disorder.[35] In addressing this concern, much research has focused on the association between stressful events and internalizing symptoms. However, the bulk of this research uses a one-time follow-up design that limits the examination of the relationship between stress and disorder. This study's design used longitudinal data and an analytical strategy capable of specifying an indirect relationship between stressful events and disorder. We found that adolescents at risk for internalizing disorder onset likely experienced persistent or intensifying stress (parent-adolescent disagreements) throughout early to middle adolescence. In response to these disagreements, they likely reported chronically high or increasing internalizing symptoms over several years preceding disorder onset. The prodromal symptoms directly related to disorder onset, while stressful events indirectly predicted onset through their relationship to internalizing symptoms.

Of course, this model tests only one limited set of hypotheses. Other, more fully specified models must include factors such as parental disorder and additional stressful events. Also, data containing shorter and more numerous assessment intervals could examine the effect of various stressor and symptom courses on disorder onset. However, this study helps to clarify the longitudinal role of stressful events and prodromal symptoms in disorder onset and, in doing so, refines the definition of those most at risk for first onset of an internalizing disorder during adolescence.

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