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ORIGINAL ARTICLE

The dynamic interdependence between family support and depressive symptoms among adolescents in Ghana

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Abstract
Objectives This study identified temporal sequencing in the associations between family support and depressive symptoms over the course of adolescence for youth in Ghana.
Methods Data derived from a longitudinal cohort study of 718 Ghanaian adolescents (58 % female) who were, on average, 13.84 years at Wave 1. Youth completed surveys at three time points separated by an 18-month time lag from early through late adolescence. Latent growth curve techniques were used to investigate the degree to which family support predicts changes in youth depressive symptoms and/or depressive symptoms precede changes in family support from early through late adolescence.
Results Youth in Ghana experience declines in family support and increases in depressive symptoms over the course of adolescence. The associations between lower family support and higher depressive symptoms are recursive or bidirectional over time.
Conclusions Study results suggest the value of promoting family support and reducing youth's depressive symptomology as a way of interrupting a recursive cycle of declining family support and increasing depressive symptomology from early through late adolescence.

Keywords Sub-Saharan Africa · Adolescent depressive symptoms · Family support · Latent growth curve models

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Introduction

Depression is the leading cause of illness and disability among adolescents worldwide (World Health Organization 2014). Adolescents with unipolar depressive disorders face increased risks of suicide, adult depressive disorder, and negative health and social outcomes (Naicker et al. 2013). Even at subclinical levels, youth depressive symptoms are associated with compromised functioning in school, work, family, and peer contexts, concurrently, and into adulthood (Aalto-Setälä et al. 2002; Jaycox et al. 2009). In sub-Saharan Africa, where youth comprise the single largest segment of the population (Blum 2007), little is known about the etiology of depressive symptoms (Hoven et al. 2008). Yet, surveillance data suggest a high prevalence of depressive symptoms among sub-Saharan African youth (Cortina et al. 2012). In Ghana, where the present study is situated, 38 % of senior high school students report feeling "so sad or hopeless almost every day for 2 weeks or more in a row" that they stopped doing usual activities during the past 12 months (World Health Organization 2008). Although family support is a well-established correlate of fewer adolescent depressive symptoms, the degree to which family support precedes changes in Ghanaian adolescents' depressive symptoms is unclear. The present study aims to identify temporal sequencing between family support and depressive symptoms throughout adolescence.

Support from family appears protective against adolescent depressive symptomology (Casey-Cannon et al. 2006). In the US, adolescents with more parental support—warm and affectionate parenting behaviors and parental trust and understanding (Armsden and Greenberg 1987)—report less depressive symptomology than do youth reporting lower levels of parental support (Kouros and Garber 2014). Family support, rather than parental support alone, may be especially salient to youth's mental health in countries such as Ghana, where adolescents commonly are the shared responsibility of parents and other "family", including aunts and uncles, grandparents, other relatives, and non-relatives (Adepoju and Mbugua 1997; Oppong 1997). In this regard, two features of sub-Saharan African families are particularly relevant. One is the greater importance of extended family relative to the nuclear family in sub-Saharan Africa. Many youth in sub-Saharan Africa reside with family units including aunts, uncles, grandparents, and other adult relatives in addition to biological parents (Annim et al. 2013). A second feature is the institution of child fosterage, in which a child from one family is sent to live in the household of an extended family member. Although being fostered can improve the young person's living conditions, she or he may become a low status member of the receiving household and be subject to emotional and physical maltreatment (Hampshire et al. 2014).

Knowledge about the impact of family process on adolescents in sub-Saharan Africa largely stems from studies examining youth's sexual risk behaviors. Greater parental monitoring and closer, more nurturing parent–youth relationships are associated with less self-reported sexual risk behavior among youth in Ghana and other sub-Saharan African countries (Biddlecom et al. 2009; Bingenheimer et al. 2015). As adolescent sexual risk often co-occurs with adolescent mental health, it is reasonable to consider how family processes may influence changes in depressive symptoms over the course of adolescence for youth in sub-Saharan Africa.

Family support and youth depressive symptomology are likely to operate in a bidirectional manner over the course of adolescence. Longitudinal research conducted in the US has shown that parental support precedes changes in adolescent depressive symptomology and that youth depressive symptoms lead to reduced warmth and connection in the parent–youth relationship (Needham 2008). The theoretical mechanism explaining protective effects of family support for reduced adolescent depressive symptomology may be youth's enhanced sense of security, belongingness, and positive self-image (McNeely and Barber 2010). In the event that depressive symptomology adversely impacts youth's support by, and connection to, adult family members, depressive symptoms may elicit negative responses from family caregivers and/or cause young people to withdraw from their family, leading to greater distance in the caregiver–child relationship (Boeninger et al. 2013). It is critical to identify the degree to which family support precedes adolescent depressive symptoms, or the reverse, in order to inform the timing and target of preventive interventions.

Virtually no systematic research has investigated the associations between family support and adolescent depressive symptoms for youth in sub-Saharan Africa. Longitudinal research in the US primarily has used cross-lagged regression methods to model changes in average scores for depressive symptoms as a function of prior scores for depressive symptoms, in addition to prior and concurrent scores for parenting variables (as an example, see: Rueger et al. 2010). Latent growth curve modeling techniques are uniquely valuable for understanding changes within and across individuals over time. In the present study, latent growth curves will be used to examine baseline levels and trajectories, of family support and youth depressive symptoms in Ghana. We will investigate the degree to which depressive symptoms precede changes in youth reports of family support and the extent to which the process of influence operates in the reverse. We anticipate that Ghanaian youth will experience increases in depressive symptomology and declines in family support during adolescence, as has been shown in the US (Kouros and Garber 2014; McGue et al. 2005). Finally, we anticipate that associations between parental support and youth depressive symptoms will be recursive: family support will predict changes in adolescent depressive symptoms and depressive symptoms will predict declines in support.

Methods

Data source and procedures

We use data from a longitudinal study of youth in two towns in southeastern Ghana, a region that is relatively more urban and educated than other parts of Ghana. Youth were surveyed approximately every 18 months from 2010 to 2013 for a total of three survey time points. Field teams from the Institute for Statistical, Social, and Economic Research at the University of Ghana enumerated all unmarried 13- to 14-year-olds, a younger cohort, and 18- to 19-year-olds, an older cohort, from two towns (National AIDS/STI Control Programme 2013). Among a simple random sample of 1594 youth, 75 % ($n = 1275$) participated at Wave 1. Response rates were 95 % at both Wave 2 and Wave 3. Due to our focus on transitions through adolescence, analyses are restricted to the 718 youth from the Wave 1 younger cohort. (A small number of 12- and 15-year-olds ended up being included in the younger

cohort.) Interviews, conducted in English and three Ghanaian languages (Twi, Dangbe, Ewe), covered a range of topics, including sociodemographics, family processes, and health knowledge, attitudes, and behaviors. The protocol was approved by Institutional Review Boards at a US university, as well as the University of Ghana.

Measures

Depressive symptoms

Five items from the internalizing subscale of the Child Behavior Check List (Achenbach 1991) were used to measure adolescent depressive symptoms. Loading on a single dimension in principal components analyses were statements about the degree to which youth feel that no one loves them; unhappy sad or depressed; lonely; a desire to be alone rather than with others; and worthless (1 = very true to 3 = not at all true; Achenbach 1991). Responses were reverse coded, summed, and then averaged. Higher scores indicated more depressive symptomology ($\alpha = 0.60$ Wave 1; $\alpha = 0.73$ Wave 2; $\alpha = 0.73$ Wave 3).

Family support

Seven items from the Inventory of Parent and Peer Attachment (Armsden and Greenberg 1987) and the Network of Relationships Inventory (Furman and Buhrmester 2009) were adapted to measure family support in a manner reflecting the importance of extended family kin in Ghana (Bingenheimer et al. 2015). Each item begins with the phrase, "There is an adult in your life who…", and end with: "comforts you", "gives you advice", "praises you", "gives you attention", "encourages you", "trusts you", and "understands you". For each item, the response options included 1 = not at all true, 2 = somewhat true, and 3 = very true. Responses were summed and averaged so that higher scores indicated greater support ($\alpha = 0.74$ at Wave 1; $\alpha = 0.78$ at Wave 2; $\alpha = 0.73$ at Wave 3).

Background variables

Youth's age (continuous in years) and biological sex (0 = female; 1 = male) are included because depressive symptoms are more prevalent among females and increase with youth's age and because family support is less prevalent among males and declines with youth's age (Kouros and Garber 2014). Adolescent school enrollment (dummy coded for "not in school" and "behind in school", with "in school and on time" as the referent group); household wealth (continuous score based on eight items indicating ownership of resources such as a television and refrigerator; Rutstein and Johnson 2004); and household composition (dummy coded for "single mother", "single father", "kin only", "no adult family member", with "two biological parents" as the referent group) are included due to associations between each of these factors and less adolescent depression (Conger et al. 2000; Hammen 1992). Finally, due to pernicious effects of the HIV epidemic in many parts of sub-Saharan Africa, analytic models account for the prevalence of HIV in the youth's community of residence (0 = less than 1 % prevalence; 1 = 10 % prevalence; Earls et al. 2008).

Analysis plan

Prior to analyzing data, we addressed missing data due to item non-response (8 %) and survey non-response (5 % Time 2; 5 % Time 3). Data were multiply imputed 20 times using a fully conditional specification for the iterative Monte Carlo Markov Chain method, a best-practice modern missing data imputation methods (Enders 2010). We used the 20 fully imputed data sets (to allow maximum recoverability from missing data) with restricted maximal likelihood estimation for multivariate analyses in M*plus* 7.11 (Muthen and Muthen 2013). Analyses conducted on complete-case data or that do not use a best-practice modern missing data imputation method are known to produce biased results (Enders 2010).

Latent growth curve (LGC) modeling was used to model developmental trajectories of youth depressive symptoms and perceived family support over time. LGC uses a structural equation modeling framework to estimate growth factors approximating observed measures (Little 2013). We obtain estimates for individuals' (a) initial level of depressive symptoms and family support; (b) rates of change in depressive symptoms and family support over the three time points, and (c) associations between baseline scores and rates of change for both constructs. LGC models also indicate the degree to which there is significant individual variation in the initials levels of, and changes in, depressive symptoms and family support. For LGC models, we fix intercept loadings at a value of one in order to interpret the intercept as the baseline level for depressive symptoms or family support. Slope loadings are fixed at 0, 1 and 2 at the three respective time points in order to assess linear growth in outcomes over time.

We began with unconditional (excluding background variables) models in order to assess how well linear models of change for youth's depressive symptoms and family support fit the data. Next, we compared results from the combined, or parallel process, LGC models illustrated in

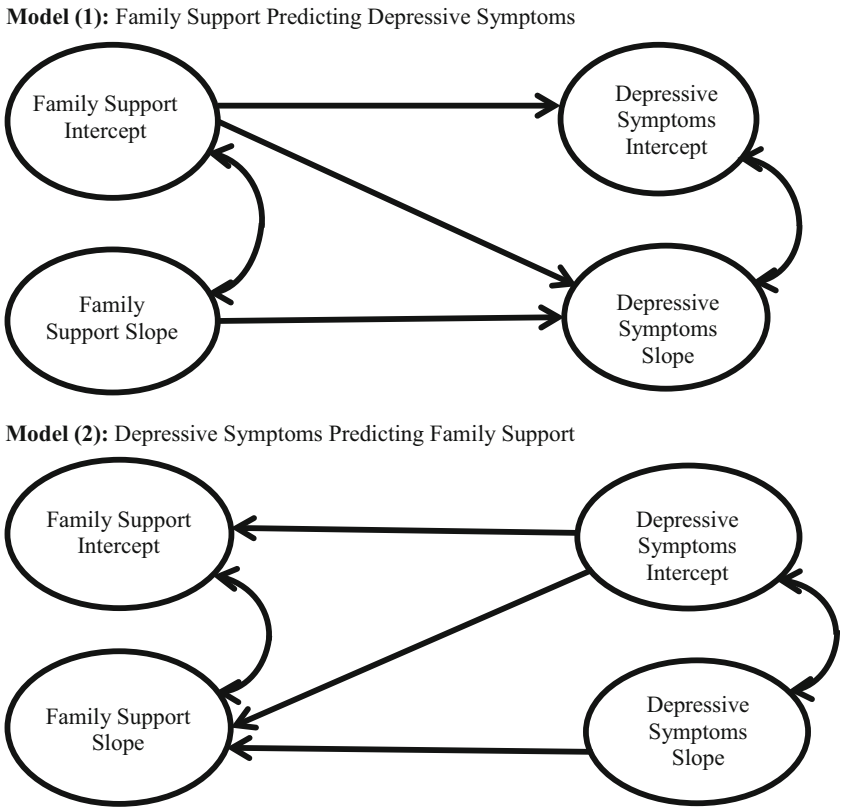

Fig. 1 Parallel process latent growth curve models examining temporal sequencing for associations between family support and youth's depressive symptoms, Ghana, 2010–2013

Fig. 1 (Kofler et al. 2011). Parallel process models facilitate examining the interrelatedness of trajectories for both family support and adolescent depressive symptoms simultaneously. Model 1 examines how initial levels and changes in family support predict changes in depressive symptoms, as well as how the initial level of family support predicts initial levels of depressive symptoms. Model 2, examining the reverse, investigates how initial levels and change in depressive symptoms predict changes in family support and how initial levels of depressive symptoms predict initial levels of family support. Next, we ran conditional models, whereby intercepts and slopes for both support and depressive symptoms were regressed on study background variables. As recommended by Hu and Bentler (1999), criteria used to determine acceptable model fit were RMSEA < 0.08 and CFI > 0.90, and criteria indicating an excellent model fit were RMSEA < 0.05 and CFI > 0.95.

Results

Descriptives

Wave 1 characteristics of participants and the means and standard deviations for depressive symptoms and family support are shown in Table 1.

Unconditional latent growth curves

Results from unconditional models provide information about the average initial values, or intercepts, and linear rates of change, or slopes, for depressive symptoms and family support. Fit statistics were good for the model of depressive symptoms, RMSEA = 0.067, 95 % CI 0.011, 0.138; CFI = 0.954, and the model of family support, RMSEA = 0.063, 95 % CI 0.020, 0.112; CFI = 0.932. The mean value on the depressive symptoms scale at baseline was 1.26, and scores increased an average of 0.09 ($p < 0.001$) from one survey time point to the next, spaced roughly 18 months apart. A statistically significant correlation between the intercept and slope for depressive symptoms ($r = -0.39$, $p < 0.05$) indicated that youth with higher levels of depressive symptoms tended to experience less steep increases in depressive symptoms ($r = -0.39$, $p < 0.05$). The unconditional model for family support indicated that youth, on average, reported a baseline score of 2.85 for family support and significant declines in support, with an average rate of change of -0.02 ($p < 0.01$). The significant correlation between the intercept and slope for family support ($r = -0.54$, $p < 0.001$) indicated that youth with greater family support at baseline experienced stronger declines in support over time.

Table 1 Descriptive statistics for participants: depressive symptoms and family support at Waves 1, 2 and 3 and control variables at Wave 1, $N = 718$, Ghana 2010–2013

Variable	Number (%) or mean (±SD)	Range of scores
Youth gender		
Female	420 (58.5 %)	
Male	298 (41.5 %)	
Youth age	13.84 (0.59)	12.31–15.09
Youth's school status		
In school and on-time	463 (64.5 %)	
In school and behind	214 (29.8 %)	
Not in school	41 (5.7 %)	
Household wealth	1.47 (0.22)	1–2
Household composition		
Two biological parents	217 (69.8 %)	
Single mother	187 (26.0 %)	
Single father	52 (7.2 %)	
Kin only	207 (28.8 %)	
No adults	55 (7.7 %)	
Community of residence		
Low HIV prevalence town	370 (51.5 %)	
High HIV prevalence town	348 (48.5 %)	
Youth depressive symptoms		1–3
Wave 1	1.23 (0.35)	
Wave 2	1.26 (0.45)	
Wave 3	1.39 (0.46)	
Family support		1.44–3.00
Wave 1	2.85 (0.25)	
Wave 2	2.83 (0.28)	
Wave 3	2.82 (0.30)	

Parallel process latent growth curves

The two unconditional (without covariates) parallel process models indicate the degree to which (a) family support predicted depressive symptoms (Model 1) and (b) depressive symptoms predicted family support (Model 2). For Model 1 ("support to depressive"), the intercept for depressive symptoms was regressed on the intercept for support and the slope of depressive symptoms was regressed on the intercept and slope of support. For Model 2 ("depressive to support"), associations between support and depressive symptoms were modeled in the reverse order as for Model 1. There was an acceptable model fit for Model 1 (RMSEA = 0.032, 95 % CI 0.016, 0.047; CFI = 0.93) and Model 2 (RMSEA = 0.032, 95 % CI 0.015, 0.046; CFI = 0.93). Due to space constraints and the similarity in findings between unconditional and conditional models, we provide results from conditional models in which control variables are included (Table 2).

Family support predicting adolescent depressive symptoms

In the conditional parallel process model (Table 2), the initial level of family support was associated with a lower initial level of depressive symptoms ($B = -0.32$, $p < 0.01$). Adolescents who perceived more family support at baseline reported significantly less depressive symptomology at this time. Because depressive symptoms for the overall sample increased over time, the negative coefficient for the family support intercept predicting the depressive symptoms slope is interpreted to mean that increases in depressive symptoms were less steep for youth reporting more support at the initial time point ($B = -0.42$,

Table 2 Standardized regression weights for model predictions, Ghana, 2010–2013

Family support predicting depressive symptoms			Depressive symptoms predicting family support			Model comparison
Variable	*B*	95 % CI	Variable	*B*	95 % CI	
Support intercept			Depressive intercept			
Depressive intercept	−0.32**	−0.52, −0.13	Support intercept	−0.48***	−0.52, −0.13	Dep = Supp
Depressive slope	−0.42*	−0.76, −0.09	Support slope	0.34, ns	−0.56, 0.83	Dep = Supp
Support slope			Depressive slope			
Depressive slope	−0.69**	−1.09, −0.29	Support slope	−0.73**	−1.16, −0.32	Dep = Supp
Covariances/correlations	*r*		Covariances/correlations	*r*		
Depressive intercept and slope	−0.47*		Depressive intercept and slope	−0.14		
Support intercept and slope	−0.55***		Support intercept and slope	−0.64		

Following Kofler et al., the model comparison is determined by analyzing confidence intervals (CI) of standardized regression weight magnitudes

Supp family support predicting depressive symptoms model, *Dep* depressive symptoms predicting family support model

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

$p < 0.05$). There also was a significant coefficient for the slope of family support predicting the slope of depressive symptoms ($B = -0.69$, $p < 0.01$). This is interpreted to indicate that youth reporting less steep declines in family support over time experienced significantly less steep increases in depressive symptoms during adolescence. The significant covariance between the intercept and slope for depressive symptoms indicated that higher levels of depressive symptoms at baseline were associated with a less steep increase in depressive symptoms over time ($r = -0.47$, $p < 05$). Similarly, a higher level of family support at baseline (intercept) was associated with stronger declines (slope) in family support over time ($r = -0.55$, $p < 0.001$). Overall, 41 % of the variance for youth's change in depressive symptoms over time was explained by variables in the conditional model.

Adolescent depressive symptoms predicting family support

Results from Model 2 (Table 2) indicated that youth's higher initial level of depressive symptoms predicted a lower initial level of family support ($B = -0.48$, $p < 0.001$). Further, increases in depressive symptoms over the course of adolescence were associated with stronger declines in family support over time ($B = -0.73$, $p < 0.01$). There was no statistically significant association between youth's initial level of depressive symptomology and the rate of change in family support. Further, unlike in Model 1, baseline levels of depressive symptoms were not associated with changes in depressive symptoms, and baseline levels of family support were not associated with changes in family support. Two-thirds (66 %) of the variance in family support change over time was explained by variables in the conditional model.

Effects of background variables

The intercepts and slopes for family support and depressive symptoms were regressed on all background variables (detailed results are available from the authors). Results indicate that residing in a community with a high HIV prevalence was associated with a significantly higher initial level of depressive symptoms. In addition, in Model 1 (support predicting depressive symptoms), initial levels of family support were lower for youth living with a father only, kin only, or no adult family member, as compared to living with two biological parents. Declines in family support were slower among youth who were older at baseline and decreases in support were greater for youth not enrolled in school at baseline. There were no gender differences in baseline levels or trajectories of family support and depressive symptoms.

Comparision of models

In order to assess the degree to which the model of family support predicting depressive symptoms (Model 1) or the model of depressive symptoms predicting family support (Model 2) provided a better representation of observed data, we compared fit statistics using the CFI and BIC. In Model 1, the CFI was 0.931 and the BIC was 9991.94, neither of which was significantly different from the CFI of 0.993 and BIC of 9990.57 in Model 2. Thus, there was no support to suggest that family support predicting depressive symptoms was a better or worse fit of the data than the reverse. Next, we considered the extent to which the strength of individual coefficients from each model differed. Following procedures described by Kofler et al. (2011) and Cumming and Finch (2005), we examined confidence intervals for standardized regression paths to determine the degree to which the strength of coefficients from Model 1 (support predicting depressive) were significantly different from those of Model 2 (depressive predicting support). The proportion of confidence interval overlap was more than 0.50 for all regression weights, indicating no statistically significant differences in regression weights from Models 1 and 2.

Shared method variance

As youth reported on both their depressive symptomology and family support, significant results from our models may have been inflated due to shared method variance. We examine shared method variance by examining the correlations of unique error variances for constructs measured by the same method (Conway 2004). Results (available from the authors) indicated modest correlations, ranging from 0.15 to 0.22, suggesting little inflation due to shared method bias.

Discussion

Although rarely the focus of global public health research, adolescent depression represents the primary cause of poor health among adolescent males and females worldwide (World Health Organization 2014; Naicker et al. 2013). In the present study, we address the critical need for research that can inform prevention efforts mitigating adolescent depression in a sub-Saharan African context (Kieling et al. 2011; Patel 2013). In this regard, we were motivated by theory and empirical evidence supporting the primacy of family, particularly in sub-Saharan Africa, for healthy adolescent development (Adepoju and Mbugua 1997). Our findings were consistent with expectations. Ghanaian youth experience significant declines in family support and

increases in depressive symptomology over the course of adolescence, both of which likely reflect age-related changes in family relationships and youth's mental health. Further, declines in family support are associated with increasing depressive symptomology in a recursive manner. There were no significant gender differences in levels of, or changes, in depressive symptoms and family support.

Parallel process latent growth curve modeling techniques represent a unique strength of this study, enabling us to investigate temporal sequencing in the associations between family support and adolescent depressive symptomology. Ghanaian youth with more family support early on experience less depressive symptomology during early adolescence as well as slower increases in depressive symptoms into late adolescence. In addition, youth whose level of family support declines more slowly over the course of adolescence experience less increase in depressive symptomology over time. The proposition that family support predicts youth depressive symptoms appears to be equally as tenable as the idea that depressive symptoms predict family support. Interdependent associations between support and depressive symptoms hold even when accounting for effects of youth, household, and community characteristics on levels and trajectories of support and depressive symptomology.

Our results suggest that depressive symptoms may emerge from a recursive cycle of declining family support and increased depressive symptomology over the course of adolescence. Informed by child-effects research, we surmise that adolescent depressive symptoms elicit negative reactions from adult family members and/or cause youth to withdraw from adult family members (Boeninger et al. 2013). Consistent with the family effects literature, youth's lack of close, trusting, and understanding relationships with adult family members may reduce youth's positive self-image and sense of security important to emotional well-being (McNeely and Barber 2010). The dynamics of child and family effects may build upon one another, resulting in an intertwined process of mutual influence.

Several findings for sociodemographic factors suggest areas ripe for future research and interventions targeting families in Ghana. The fact that living in a community with a high prevalence of HIV is tied to more adolescent depressive symptoms suggests the need for future research identifying community stressors correlated with HIV but independent from family support and household composition and wealth. Given that youth living with both biological parents reported higher levels of family support and that youth enrolled in school reported less decline in support over time, policies and programs ensuring the stability of households and youth's education may help promote family processes protective for youth's mental health.

Some limitations of this study should be noted. The most important limitation is the absence of a measure of parental depression, a critical risk factor for youth depressive symptoms and strong correlate of low parental support (Cummings et al. 2013). An additional limitation is the reliance on youth self-reports. Not only may youth under-report depressive symptoms due to social desirability, but shared method variance may lead to overestimates for associations between support and depressive symptoms. We are reassured, however, by results suggesting limited problems of inflated estimation due to shared method bias (Conway 2004). Statistically speaking, our findings are generalizable only to youth from the two towns in which this study was conducted. Yet, there is no particular reason to believe that these towns are radically different from market towns in the southern third of Ghana, or from peri-urban communities on the outskirts of large Ghanaian cities. More caution should be exercised, however, in generalizing results to rural Ghana, less-developed areas in the north, or other settings in sub-Saharan Africa.

The present study represents one of the first attempts at identifying the importance of family process to depressive symptoms during adolescence in sub-Saharan Africa. Public health programs and interventions seeking to mitigate depressive symptomology in countries such as Ghana may be most effective when targeting youth's mental health and family support. Just as interventions promoting supportive family relationships may slow down cascading risks leading to youth depressive symptoms and further declines in support, interventions reducing depressive symptoms may interrupt the vicious cycle of declining support and increased depressive symptomology. In order to be effective in sub-Saharan Africa, existing family support interventions may need to be adapted to reflect the salient roles of extended family adults and consequences of family instability for adolescents in Ghana. Unexpected gender similarities in levels of depressive symptoms and family support shown in this study suggest that mental health programs and interventions in Ghana should be tailored to meet the needs of adolescent males to the same degree as females. Future research examining Ghanian adolescent males' depressive symptoms may be worthwhile given the lack of evidence in this study for a protective gender effect for males. Finally, as evidenced by the reciprocal influence of support and depressive symptomology throughout adolescence, the window of opportunity for reducing risks for depression persists well beyond childhood and early adolescence.

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