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Distinct trajectories of depression symptoms in early and middle adolescence: Preliminary evidence from longitudinal network analysis

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ABSTRACT

Adolescent depression is a clinically relevant concern that has major implications for mental and physical health. The trajectory of depressive symptoms among adolescents suggests that there is likely variability throughout this developmental period. The aim of the study was to assess the longitudinal relationship between individual symptoms of depression among early and middle adolescents to provide guidance for future research investigating targeted intervention efforts. Data were extracted from electronic medical records (2015–2017) from a pediatric primary care clinic in an urban setting. Cross-Lagged Panel Network analysis was used to evaluate symptoms of depression measured with the Patient Health Questionnaire (PHQ-9) measured twice over a 1-year period among early adolescents (ages 11–13 years; $n = 309$) and middle adolescents (ages 14–16 years; $n = 255$). The sample was predominantly Hispanic (90%) and 56% female. The analyses highlighted key differences and similarities between early and middle adolescence, largely focused on the role of suicidal ideation and tightly linked with feelings of failure and appetitive disturbance. In early adolescence suicidal ideation was highly likely to lead to other symptoms. In middle adolescence, however, suicidal ideation no longer had connections to other symptoms and instead the strongest connections were *toward* suicidal ideation. Interestingly, across both early and middle adolescence feelings of failure and appetitive disturbance were highly likely to lead to suicidal ideation. These exploratory findings highlight several longitudinal associations between early and middle adolescence that provide insight into differences and similarities regarding how symptoms might progress within those developmental periods. Taken together these results can provide direction for future research to evaluate brief, targeted interventions for adolescents.

Depression is a prevalent clinical concern among adolescents, with more than 1 in 10 experiencing a major depressive episode each year in the United States, which is associated with significant life impairment (Substance Abuse and Mental Health Services Administration, 2019). Adolescents experiencing elevated depression symptoms are sixteen times more likely to experience suicidal ideation (Glied and Pine, 2002) and 8.5% of adolescents attempt suicide (Grunbaum et al., 2004). Suicide continues to rank as the second leading cause of death during adolescence and beyond (Substance Abuse and Mental Health Services Administration, 2019). Adolescents experiencing depression are also at risk for additional health compromising conditions that further affect quality of life, like obesity (Alaie et al., 2019; Goodman and Whitaker, 2002). Thus, routine screening for major depressive disorder is accepted as a central feature of evidence-based preventative health intervention for children aged 12 years and older (Siu et al., 2016).

The developmental stages of early, middle, and late adolescence are characterized by an evolution from concrete thinking and egocentrism to improved impulse control and future-focused decision making. Simultaneously, pubertal changes further affect self-perception of mood and interpersonal relationships (Kaltiala-Heino et al., 2003). Alongside these major mental and physical changes, the percentage of children experiencing major depressive episodes more than triples from early to late adolescence (i.e., 12–17 years of age; Substance Abuse and Mental Health Services Administration, 2019). Earlier age at onset appears to be associated with a variety of negative outcomes later in life (Zisook et al., 2007). Previous longitudinal research has suggested that there is some

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variability in trajectories of adolescent depression (Ellis et al., 2017).

These findings underscore that adolescent depression is highly heterogeneous (Zimmerman et al., 2015); different symptom profiles may constitute meaningfully different presenting concerns, which can have implications for treatment strategies. Network analysis is an analytic methodology that has been used to understand fine-grained relationships between symptoms (McNally et al., 2015), and is particularly useful for conceptualizing mental health disorders, which are often composed of a variety of interrelated symptoms. Major depressive disorder, for example, is a single diagnosis that can involve hundreds of thousands of different symptom combinations (Fried and Nesse, 2015). It is also possible that comorbidities like obesity or an anxiety disorder may exert differential influences on the heterogeneous presentation of depressive symptoms, although has not always been taken into account in network analyses.

Researchers have recently evaluated the trajectory of cross-sectional networks of child to early adolescent depression and anxiety over time, finding that the most important symptoms of depression were typically consistent; appetitive disturbance was the most common stable symptom, while feelings of worthlessness and fatigue were also stable across time (McElroy et al., 2018). In one study examining cognitive vulnerability for depression in adolescents (Marchetti et al., 2020) found that the networks were stable over a one-year time period. Although comparing cross-sectional networks at different time periods may provide some benefits over a single cross-sectional analysis, methods that leverage longitudinal methods can provide further insight into the interrelations between symptoms of depression across time. A longitudinal network analysis of youth in middle adolescence using cross-lagged relationships found that several specific symptoms (depressed mood, inattention, and worry) appeared to influence future symptoms over 18 months, after accounting for several covariates (Funkhouser et al., 2020).

The aim of this paper is to extend the existing longitudinal network investigations of adolescent depression by taking into consideration trajectories of depressive symptoms in different developmental periods, while accounting for other factors (e.g., obesity and biological sex) that may be implicated in those trajectories. Longitudinal network analysis distinguishing early and middle adolescent symptomatology can provide insight into how different developmental periods influence trajectories of depressive symptoms. Furthermore, network analysis may facilitate a better understanding of how individual screening items relate to future symptom burden. Using a naturalistic dataset from a primary care pediatric healthcare setting, we used recent developments in cross-lagged panel network analysis (Rhemtulla et al., 2021) to investigate interrelations among symptoms of depression over time, while controlling for all other symptoms and covariates.

Given that otherwise healthy adolescents typically only visit a primary care provider annually, utilizing depression screening instruments helps exploit the time-limited nature of preventative healthcare encounters. This approach is of particular clinical utility in the current context, since depression interventions delivered in early adolescence may be more effective than depression treatments delivered in later adolescence (Bennik et al., 2014; Braet et al., 2013). Moreover, early clinical contact plays a significant role in preventing the exacerbation of depression in adolescence (Neufeld et al., 2017). These exploratory analyses may inform future efforts aimed at implementing targeted intervention efforts in integrated care settings where treatment may be brief, but opportunities for clinical contact are higher for under-resourced youth.

1. Methods

1.1. Participants

The dataset comprised adolescents ages 11–17 years seen at a federally qualified pediatric primary care clinic who had completed the Patient Health Questionnaire (PHQ-9), as part of their which is typically administered at all annual well-child visits for children age 11 years and older, and also for individuals previously diagnosed with depression. Fully de-identified data from 2015 to 2017 extracted from the electronic medical record were provided to the first author with a strict data usage agreement prohibiting data sharing. The IRB at [removed for blinded peer review] classified the study as exempt from further review based on the complete de-identification of the data prior to it being provided to the first author.

1.2. Measures

Variables available were demographic information (age, sex, race, and ethnicity), a subset of possible physical and mental health diagnoses and the PHQ-9 item scores. The PHQ-9 is a 9-item self-report instrument that measures depression symptoms. Each item is rated on a 0–3 point scale: “not at all”, “several days”, “more than half the days”, “nearly every day” (Kroenke et al., 2001). The PHQ-9 is a widely used instrument that has been validated for the assessment of depression in primary care settings (Arroll et al., 2010) and among adolescents (Richardson et al., 2010).

1.3. Data preparation

The data set included 4,208 individuals. Primary analyses were conducted for adolescents with PHQ-9 scores at two timepoints at least 300 days apart. As the data spanned 2015–2017 most individuals only had one timepoint of PHQ-9 data. Two timepoints with PHQ-9 data were moderately available, but very few individuals had more than two timepoints available. For those with more than two visits we took the two that were approximately 1 year apart. The time between the two visits averaged 390 days (SD = 63 days). We then created two “Time 1 cohorts” – those ages 11–13 years ($n = 309$) and those 14–16 years ($n = 255$) reflecting early and middle adolescent periods. Finally, based on the strong positive skew of the PHQ-9 items, we discretized all values to reflect symptom endorsement (yes/no). See Table 1 for a summary of

Table 1
Demographic variables and depression symptom endorsement.

Variable	Early Adolescence (n = 309)		Middle Adolescence (n = 255)	
	T1	T2	T1	T2
	Mean (SD)			
Age	12.04 (0.78)	13.03 (0.79)	14.90 (0.84)	15.88 (0.84)
PHQ-9 sum score	2.17 (3.30)	2.38 (3.82)	2.71 (3.62)	2.44 (3.24)
	No (%)			
Sex (female)	175 (56.6)		144 (56.5)	
Ethnicity (Hispanic)	283 (91.6)		228 (89.4)	
Race				
Hispanic	152 (49.1)		133 (52.2)	
Black or African American	8 (2.6)		12 (4.7)	
Asian	1 (0.3)		0 (0)	
White	72 (23.3)		66 (25.9	
Pacific Islander	2 (0.6)		0 (0)	
Unknown/not reported	67 (21.7)		44 (17.3)	
Asthma Diagnosis	15 (4.9)		20 (4.9)	
Obesity Diagnosis	32 (10.3)		21 (8.2)	
PHQ-9 endorsement				
Anhedonia	89 (28.8)	96 (31.0)	74 (29.0)	83 (32.5)
Depressed Mood	49 (15.9)	40 (31.1)	61 (23.9)	46 (18.0)
Sleep Disturbance	75 (24.2)	90 (29.1)	85 (33.3)	75 (29.4)
Low Energy	79 (25.6)	77 (24.9)	87 (34.1)	91 (35.7)
Appetitive Disturbance	69 (22.3)	55 (17.8)	64 (25.1)	55 (21.6)
Feeling of Failure	29 (9.4)	41 (13.3)	42 (16.5)	36 (14.1)
Impaired Concentration	50 (16.2)	61 (19.7)	54 (21.2)	48 (18.8)
Disturbed Movement	33 (10.7)	32 (10.3)	26 (10.2)	24 (9.4)
Suicidal Ideation	14 (4.5)	18 (5.8)	18 (7.1)	12 (4.7)

demographic and symptom variables.

1.4. Statistical analyses

Cross-lagged panel networks (CLPNs) were generated using the method developed by Rhemtulla et al. (2021) and implemented in R 4.0.1 (Computing, 2020). CLPNs are constructed by conducting regularized regressions for each dichotomous variable at time 1 and time 2 (autoregression) and for one variable at time 1 predicting a different variable at time 2 (cross-lagged). In the current context, autoregressive associations reflect the likelihood that if a symptom (e.g., appetite disturbance) is endorsed at time 1 then it will also be endorsed at time 2. Cross-lagged associations on the other hand, reflect the likelihood that if a symptom (e.g., appetite disturbance) is endorsed at time 1 then a different symptom (e.g., SI) will be endorsed at time 2. In order to incorporate key covariates (age at time 1, biological sex, asthma diagnosis and obesity diagnosis), we used the modified approach suggested by Funkhouser et al. (2020), in which covariates are included in the prediction of the outcomes but are not themselves predicted.

Both autoregressive and cross-lagged estimations account for all other variables. In this CLPN method we used 10-fold cross-validation with LASSO regularization, which shrinks all very small associations to zero. In the context of binomial regression, we exponentiated the values to reflect odds ratios, thus 1 is equivalent to no association. Additionally, we computed the in expected influence (in-EI) and out expected influence (out-EI), which is the sum for *directional* associations between a specific symptom and all other symptoms. We then tested the stability of each edge and node in/out EI using the *bootnet* package (Epskamp et al., 2018), by bootstrapping the networks 5,000 times (using nonparametric bootstrapping to test edge stability and case drop bootstrapping to test centrality stability). For edge stability we calculated the proportion of times each edge crossed or included 1 – due to shrinkage in regularization procedures, 95% confidence intervals do not reflect true intervals. Finally, we compared the edges for CLPNs between early and middle adolescence by evaluating (1) the correlation between edge lists, (2) the cumulative percentage of associations that overlapped between the networks, and (3) the Jacard Similarity Index, which is the proportion of edges present in both networks that also share the same edge features (direction and sign).

2. Results

The key results can be seen in the graphical models in Fig. 1. Table 2 provides the odds ratios as well as confidence for the presence of the association based on the bootstrapped replication analyses.

In the early adolescent CLPN, the strongest association was Suicidal Ideation (SI; "Thoughts that you would be better off dead, or thoughts of hurting yourself in some way?") → Movement Dysregulation ("Moving or speaking so slowly that other people could have noticed? Or so fidgety or restless that you have been moving a lot more than usual?"), OR = 8.535. Meaning that if an early adolescent endorsed SI at time 1, they were eight times more likely to endorse movement dysregulation at time 2. The other two strongest associations were Feelings of Failure ("Feeling bad about yourself — or that you are a failure or have let yourself or your family down?) → SI (OR = 3.675) and Appetitive Disturbance ("Poor appetite or overeating?) → SI (OR = 3.153). In the middle adolescent CLPN, the strongest association was from Feelings of Failure → SI (OR = 6.023). The other two strongest were Feelings of Failure → Movement Dysregulation (OR = 2.798) and Appetitive Disturbance → SI (OR = 2.684).

In comparing the two networks, the coefficient of similarity was 0.10, which is quite low – suggesting that the associations in the two networks differed substantially and only 59.5% of the edges in the early adolescent network were present in the middle adolescent network. Moreover, the Jaccard Similarity Index was 0.379, suggesting that most edges present in both networks did not have the same direction. Autoregressive edges (a symptom at time 1 leading to that same symptom at time 2) were not on the whole very different in early and middle

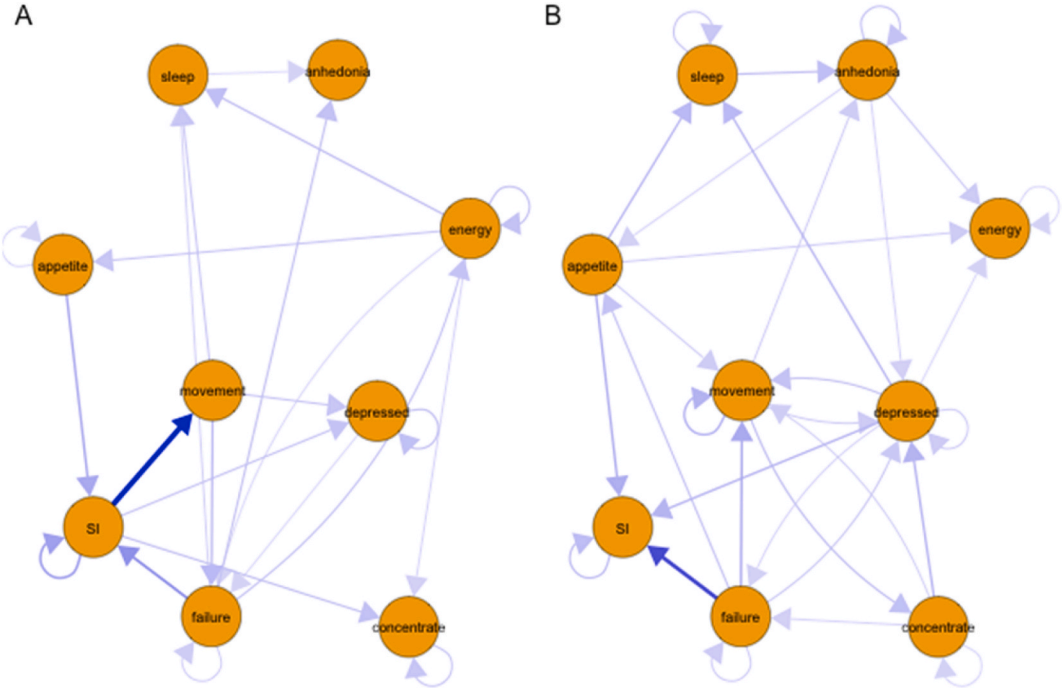

Fig. 1. Cross-Lagged Panel Networks of relationship between symptoms of depression in (A) early (ages 11–13 at time 1) and (B) middle adolescence (ages 14–16 at time 1). Width of the arrow reflects the strength of the association. A threshold was manually set to OR = 1.35 to make the figures more interpretable. Each arrow between nodes (symptoms) reflects a cross-lagged association where the symptomA → symptomB means that symptomA at time 1 predicts symptomB time 2, controlling for all other associations and covariates (age at time 1, biological sex, asthma diagnosis and obesity diagnosis, not included in the figure). Each curved arrow 'loop' reflects an autoregressive association (a symptom at time 1 predicting the presence of that symptom at time 2).

Table 2
Odds Ratio and Confidence for each cross-lagged network association.

Early Adolescent Edges				Middle Adolescent Edges			
Out Node	In Node	Odds Ratio	Confidence	Out Node	In Node	Odds Ratio	Confidence
SI	movement	8.535064	0.9918	failure	SI	6.0230099	0.8694
failure	SI	3.674514	0.9330	failure	movement	2.7982805	0.8398
appetite	SI	2.844684	0.9260	appetite	SI	2.6849474	0.8320
movement	failure	2.296373	0.9132	concentrate	depressed	2.4622466	0.9510
energy	sleep	2.118878	0.9862	depressed	SI	2.4010515	0.7414
SI	concentrate	2.014391	0.8264	appetite	sleep	2.2987504	0.9798
failure	anhedonia	1.983634	0.8196	depressed	sleep	2.2940670	0.9730
failure	energy	1.894185	0.8316	sleep	anhedonia	2.0880369	0.9748
SI	depressed	1.892445	0.7952	movement	concentrate	2.0373255	0.8896
energy	appetite	1.867852	0.8922	depressed	movement	1.9419751	0.7776
movement	depressed	1.860521	0.8684	failure	appetite	1.9295178	0.8846
movement	sleep	1.834801	0.9040	failure	depressed	1.7528325	0.8208
failure	sleep	1.579375	0.7482	anhedonia	energy	1.7218559	0.9354
energy	concentrate	1.503422	0.8660	movement	depressed	1.6612578	0.7902
energy	failure	1.381765	0.7360	appetite	energy	1.6489054	0.9018
sleep	anhedonia	1.379500	0.7154	appetite	movement	1.6099382	0.7594
depressed	failure	1.359801	0.6434	movement	anhedonia	1.5964339	0.7580
movement	concentrate	1.337547	0.7158	concentrate	movement	1.5528974	0.7382
SI	anhedonia	1.333619	0.5398	concentrate	failure	1.5211921	0.7512
appetite	failure	1.313198	0.6766	anhedonia	depressed	1.5180419	0.7426
sleep	SI	1.277288	0.5924	anhedonia	appetite	1.4832883	0.8480
sleep	appetite	1.223639	0.5598	depressed	failure	1.4794775	0.7602
failure	depressed	1.215395	0.6640	depressed	energy	1.3879119	0.8010
energy	depressed	1.205234	0.6790	sleep	appetite	1.3072578	0.7892
concentrate	energy	1.177174	0.6138	appetite	concentrate	1.2965624	0.7212
appetite	depressed	1.133650	0.5972	movement	failure	1.1944981	0.6158
depressed	SI	1.127233	0.5882	failure	anhedonia	1.1807867	0.5008
depressed	energy	1.125680	0.5390	energy	appetite	1.1205473	0.6548
movement	energy	1.093705	0.5382	depressed	concentrate	1.1053367	0.5728
energy	anhedonia	1.078207	0.4482	movement	energy	1.0981142	0.5980
failure	appetite	1.054192	0.4014	sleep	movement	1.0971849	0.5500
sleep	concentrate	1.014873	0.5332	concentrate	energy	1.0886388	0.5968
movement	SI	1.007654	0.5824	concentrate	anhedonia	1.0652991	0.5100
movement	appetite	1.004769	0.4324	sleep	depressed	1.0442672	0.5396
				failure	sleep	1.0327451	0.5088
				SI	sleep	1.0182848	0.5350
				movement	SI	1.0069713	0.5284
				concentrate	SI	1.0013112	0.5990
				energy	movement	0.8876993	0.5220
				anhedonia	movement	0.7041362	0.6028
				energy	depressed	0.6438071	0.6368
				energy	SI	0.3686559	0.7314
				SI	movement	0.3507708	0.6910

adolescence, see Supplemental Fig. 2, with some exceptions: whereas in early adolescence suicidal ideation was the strongest autoregressive edge (OR = 3.153), in middle adolescence movement disturbance was the strongest (OR = 2.509). Additionally, anhedonia (loss of interest or pleasure in things) in middle but not early adolescence, was predictive of itself (middle OR = 1.904; early OR = 1.071).

Results from the stability analyses for in/out EI centrality indicated that they were not stable, all were below 0.25, the minimum threshold for reliability: for early adolescence the in EI centrality = 0.107 and the out EI centrality = 0.220; for middle adolescence the in EI centrality = 0.051 and the out EI centrality = 0.165. Therefore, we do not report any findings for the centrality indices in either network, although they are provided in the supplementary materials.

3. Discussion

We conducted a cross-lagged panel network analysis of the progression of symptoms of depression in early and middle adolescence in a diverse primary care setting. Results from these analyses highlight consistent key symptom indicators that predict the presence of suicidal ideation as well as key differences in depression symptoms within these developmental periods. Across both early and middle adolescence, feelings of failure and appetitive disturbance were predictive of SI. Yet, in early adolescence, SI may more strongly motivate other symptoms of depression, particularly movement dysregulation. In contrast, in middle adolescence SI appeared to be solely a consequence of other symptoms of depression. Thus, there might be consistency of some symptoms that perpetuate existing SI, but the influence of suicidal ideation on *other* symptoms of depression appeared to be attenuated in middle adolescence.

Suicidal ideation represents a widely acknowledged critical screening item for youth in primary care contexts (Horowitz et al., 2009; Wintersteen, 2010) and suicide is a leading cause of adolescent mortality (Substance Abuse and Mental Health Services Administration, 2019). General trends in reporting of SI appear to suggest around 18% prevalence in adolescents (Kann et al., 2018); however, research has suggested that in primary care settings, endorsement appears to be closer to 6% (Etter et al., 2018), which is similar to the rate reported in the current sample - approximately 5%. Prior longitudinal network analysis has not included SI, which highlights the relevance of this contribution. However, a recent cross-sectional network analysis specifically investigated symptoms of depression in relation to suicidal ideation and found that loneliness was most strongly linked to greater SI. While cross-sectional networks provide limited insight into the predictive role of symptoms over time, the PHQ-9 does not assess loneliness which may be worth considering in future research.

Critically, our analyses identified two symptoms, feelings of failure and appetitive disturbance, that appear to consistently predict the

presence of SI in both early and middle adolescence. Although further validation of these findings is needed, our results suggest that these two symptoms may serve as additional indicators of potentially concerning depression-related pathology in the absence of SI endorsement or globally elevated symptoms. Feelings of failure could emerge from different sources depending on the adolescent's developmental stage (e.g., authority figures like caregivers and teachers in early adolescence, versus peers in middle adolescence). Decreased appetite has been previously linked to a 5-fold increase in the likelihood of experiencing SI, in a large cross-sectional sample of adolescents (Kitagawa et al., 2017). Moreover, in prior network research, appetite disturbance was both the most prevalent and stable feature of depression across time (McElroy et al., 2018). Appetitive disturbance is a potentially important link to SI, although it is important to note that the PHQ-9 assesses loss of appetite *or* increased appetite, limiting the potential interpretation of our findings. Professionals in a primary care setting might probe adolescents about sources of stress linked to feelings of failure as well as more closely consider changes in appetite in order to identify potentially actionable targets during a brief encounter.

In the context of risk factors, further mental health screening and intervention are critical, especially considering the evidence that youth, and particularly those from racial/ethnic minority groups, often do not receive much needed mental health services. These results highlight several symptoms that may be important to consider for future research. Broadly, interventions for adolescent depression *are* effective. However, given the complexity and heterogeneity of depression, as well as constraints on intervention accessibility (time, travel, etc.), more efficient and targeted interventions offer one way to enhance outcomes among adolescents experiencing symptoms of depression.

Integrated healthcare offers a promising and potentially effective way to provide intervention related to depression (Sawchuk et al., 2018) in populations not adequately resourced. However, there is room to improve the integration of evidence-based mental health practices for addressing depression in adolescents. There is increasing work to develop evidence-based "waiting room" interventions that can be widely disseminated without the need for direct clinical intervention or as an intermediate step in intervention efforts (Cohen et al., 2017; Naeem et al., 2019). One potential avenue of investigation is the use of computer-based attentional interventions (Hayes et al., 2018). Additionally, training pediatricians in motivational interviewing, an empirically supported transdiagnostic intervention modality (Lundahl et al., 2010), has had some success in engaging adolescents in an internet-based intervention (Hoek et al., 2011) and could be potentially adapted to address specific symptoms of depression identified in the current study. In the current sample the majority of participants identified as Hispanic and there is a large body of research examining how cultural features like familism may influence parental and adolescent interactions to promote health among Hispanics (Campos et al., 2014). Thus, the results from this research may serve as an empirical indicator to guide future educational conversations with general pediatric practitioners regarding the presentation of depression, its assessment, and the importance of early intervention. Future research is needed to examine whether our findings generalize to samples with different ethnic compositions and to test whether brief, targeted interventions addressing the specific symptoms of depression identified here serves to enhance clinical outcomes among adolescents.

3.1. Limitations

There are several important limitations to acknowledge. First, the sample sizes were fairly small. Second, in comparing trajectories of "early" and "middle" adolescence, we compared two different samples of individuals and only used age to separate the two samples (as opposed to sexual maturity rating, for instance). This limits the extent to which we can truly characterize comparisons between the networks as reflecting developmental stage as opposed to sample differences unrelated to developmental stage. Additionally, SI was endorsed relatively infrequently, which has the potential to have skewed the results despite efforts to provide robust checks on estimation procedures. It is worth noting that the different samples offer one strength in respect to the sparsity of suicidal ideation – similar findings between distinct samples provide additional support for its importance. Although we controlled for several important biological variables implicated in depression (sex, obesity, and asthma), other variables known to influence depression e.g., peer victimization, anxiety, substance use, or sexual trauma (Diamond et al., 2017) may differentially influence depressive symptoms as adolescents transition from early to middle adolescence.

4. Conclusions

It is of significant public health interest to address the overwhelming burden of depression on adolescents. Effects of depression often persists into adulthood and can also lead to or exacerbate physical and/or other mental health concerns. Our findings highlight specific differences in the progression of depression symptoms in early compared to middle adolescence, with an important focus on the role of suicidal ideation and symptoms associated with SI. Moreover, our findings related to a predominantly Hispanic sample of adolescents living in an urban setting. This is particularly important in the sense that it reflects an underserved population that receives fewer psychological services relative to non-Hispanic Whites, despite similar depression prevalence rates (Isasi et al., 2016; Substance Abuse and Mental Health Services Administration, 2019).

The data-driven approach offers a hypothesis generating framework for examining symptom-specific relationships across time that may inform treatment development efforts through identification of specific symptoms that could be targeted by evidence-based intervention approaches developed for adolescents in primary care settings.

Contributors

M.R. and A.B. conceptualized the secondary analysis. J.G. supervised access to the data. M.R. conducted the formal analyses and the visualization. S.P. consulted on the formal analyses and aided in interpreting the results. M.R. and A.B. wrote the first draft. J.S., S.P., J.G. and M.T. were involved in writing – review and editing. All authors contributed to the final manuscript.

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Declaration of competing interest

The authors have no conflicts of interest to report.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2021.07.053.

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