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PATTERNS OF SYMPTOM ONSET AND REMISSION IN EPISODES OF HOPELESSNESS DEPRESSION

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Abstract

Background—Hopelessness depression (HD) is a subtype of depression postulated by the Hopelessness Theory of Depression to present as a constellation of symptoms occurring when an individual with a specific cognitive vulnerability (negative inferential style) experiences negative life events. In the current study, the course of HD episodes was evaluated prospectively and analyzed to explore patterns of symptom onset and remission.

Methods—In 169 HD episodes reported by 65 participants, survival analyses were conducted on the time to onset or remission for 29 individual symptoms. Survival analyses yielded probability density graphs for risk of onset and risk of offset that indicated whether the symptom tended to appear or remit early, late, or unpredictably during the episode.

Results—The symptom of hopelessness often appeared earliest in HD episodes, followed by self-blame, brooding/worry, decreased self-esteem, dependency, and decreased appetite. Hopelessness, decreased self-esteem, self-blame, brooding/worry, dependency, and increased appetite were typically the latest symptoms to remit.

Conclusions—The current study provided evidence for patterns of symptom onset and remission in HD episodes. Hopelessness and other symptoms predicted to appear according to the Hopelessness Theory were generally the earliest to appear, latest to remit, and appeared to form the core syndrome of these HD episodes. Identifying patterns of symptom onset and remission may provide a tool for subtyping depression episodes. Clinically, these results point to the utility of attending to patterns of symptom onset and remission in patients presenting with HD episodes, particularly for treatment planning and monitoring.

Keywords

depression; hopelessness; symptoms; psychology; psychiatry

INTRODUCTION

Depressed individuals vary in the number, duration, and severity of the depression episodes that they experience, as well as the specific symptoms of depression that arise during an episode. Historically, it has been suggested that depression is not a single disorder, but rather a group of disorders heterogeneous with respect to symptoms, cause, and course.^[1–5] Due to the heterogeneous presentation of depression across individuals, some have argued for the importance of identifying distinct subtypes of depression.^[6] Indeed, particular subtypes of depression have been proposed in the literature and they are differentiated in part based on the specific symptoms experienced (e.g., Hopelessness Depression [HD]^[7] and Endogenous Depression,^[8] and the Melancholic, Atypical, and Catatonic specifiers for major depressive disorder diagnosis described in the Diagnostic and Statistical Manual for Mental Disorders-Fourth Edition-Text Revision [DSM-IV-TR^[9]]). However, research into the symptomatic course (i.e., the longitudinal patterns of symptom onset and offset) of depression episodes is lacking, despite investigations into these patterns being potentially informative to psychopathology researchers and clinicians. The transition from asymptomatic to symptomatic (or vice versa) and the emergence of specific symptoms is presumably the result of underlying psychopathological processes. Elucidating patterns in the emergence or remission of symptoms could help illuminate these underlying mechanisms, differentiate depression subtypes, and provide valuable clinical information for treatment providers.

Some relatively recent research has focused on the longitudinal patterns of symptom onset and remission, and has primarily involved investigations aimed at identifying prodromal symptoms of depression episodes^[10–12] and similarities between prodromal and residual symptoms.^[13, 14] Iacoviello and colleagues^[14] have proposed a model of the emergence and remission of depression symptoms, in which prodromal symptoms form the core syndrome of the episode, typically remaining as the last (often residual) symptoms to remit. Secondary symptoms might stem from the core symptoms (e.g., prodromal insomnia might contribute to the later emergence of fatigue) or from other processes (e.g., cognitive vulnerabilities triggered by early vegetative and appetite symptoms may then give rise to symptoms such as persistent thoughts of worthlessness and helplessness, as well as dysphoria).

According to the model proposed by Iacoviello and colleagues,^[14] distinct subtypes of depression would be expected to display particular prodromal symptoms that emanate from and, thus, should reflect the particular pathological process underlying the disorder subtype. The consistency of these symptoms across phases of the episode (prodromal, acute, and residual) would indicate that they represent the core syndrome of the depression subtype. This model also hypothesizes that the core symptoms would remain as the last to remit, after any secondary symptoms have remitted. This process is congruent with the rollback phenomenon described by Detre and Jarecki^[15] and demonstrated in analyses of data from a large sample of depression episodes,^[14] as well as in studies of bipolar illness^[16] and panic

disorder.^[17] Thus, support for this model exists in the literature, not limited only to depression.

The HD subtype provides an opportunity to investigate these hypotheses. The Hopelessness Theory of Depression^[7] represents a theory-based approach to the classification of a subset of depressive disorders and postulates the existence of the HD subtype. The HD subtype is hypothesized to cut across some of the existing recognized subtypes of depression, but is also hypothesized to differ from melancholic/endogenous depression. Hopelessness has also been conceptualized by Engel^[18] and Schmale^[19] in the Giving-Up model of psychiatric and physical illness, and operationalized within the Diagnostic Criteria for Psychosomatic Research construct of demoralization.^[20] However, research has demonstrated that depression and demoralization are distinct clinical phenomena,^[21] and the HD subtype extends beyond just the symptom of hopelessness. According to the Hopelessness Theory of Depression,^[7] depressogenic inferential styles, in combination with negative life events, increase the likelihood of hopelessness, which, in turn, leads to the development of the HD cluster of symptoms (motivational deficit, sad affect, suicidal ideation, low energy, apathy, psychomotor retardation, sleep disturbance, poor concentration, and mood-exacerbated negative cognitions). Thus, hopelessness could be considered a prodromal symptom in HD as it should appear earliest in the syndrome before the onset of the other HD symptoms, which also precede any secondary symptoms. Furthermore, hopelessness presumably emerges as a direct result of the underlying psychopathological processes: the interaction of specific cognitive vulnerabilities (inferential styles) and negative life events. The rollback phenomenon proposed by Detre and Jarecki^[15] and the model proposed by Iacoviello and colleagues^[14] would then predict that the HD symptoms, followed last by hopelessness, should be the latest symptoms to remit.

In support of the existence of the HD subtype, studies have found that negative cognitive styles, both alone and interacting with negative life events, are more strongly related to depressive symptoms hypothesized to be part of the HD symptom cluster than to symptoms not part of the HD symptom cluster^[22–26] or to symptoms of other forms of psychopathology.^[22] In addition, preliminary analyses based on the first 2.5 years of prospective follow-up in the Cognitive Vulnerability to Depression (CVD) project indicate that a negative cognitive style predicts first onsets and recurrences of HD, but not of DSM melancholic depression. Studies have also demonstrated that individuals who exhibit negative inferential styles are at greater risk for developing depression, and specifically the HD subtype, when exposed to stress or negative life events, compared to individuals who do not exhibit these negative inferential styles.^[27, 28] Panzarella and colleagues^[29] also provide support for the HD subtype by demonstrating that hopelessness mediates the relationship between negative inferential style $\times$ stressful events $\times$ inferential feedback interaction and prospective onset of depressive symptoms and diagnosable HD episodes. However, some studies have failed to find evidence of this vulnerability $\times$ stress interaction predicting depression symptoms or episodes^[30–32] and at least one study failed to find a mediating role of hopelessness between this interaction and the development of depression symptoms.^[33] Nonetheless, several studies have demonstrated that cognitive vulnerability and hopelessness prospectively predict HD symptoms better than non-HD symptoms.^[22, 23, 34, 35] Still, no research to date has investigated the longitudinal course of

HD symptom onset and remission. Thus, a prospective, longitudinal investigation into the symptomatic course of HD is warranted.

The current report presents results from a prospective, longitudinal investigation of the emergence and remission of symptoms in HD. Survival analyses were utilized to explore patterns in symptom onset and remission in HD episodes, with the hypothesis that hopelessness and the HD symptoms would be the earliest to appear and latest to remit.

METHODS

All study procedures complied with the Code of Ethics of the World Medical Association (Declaration of Helsinki) and were preapproved by the Institutional Review Boards of Temple University and University of Wisconsin-Madison, as well as the granting agency (National Institute of Mental Health).

PARTICIPANTS

This study analyzed data from the Temple-Wisconsin CVD project,^[36] a prospective study of cognitive and psychosocial factors in the development of depressive disorders among college freshmen at high (HR) and low cognitive risk (LR) for depression. Details of the selection procedures are described by Alloy and colleagues.^[37] Informed consent was obtained for all subjects after the nature of the procedures was explained. In Phase I, the Cognitive Style Questionnaire (CSQ)^[37] and Dysfunctional Attitudes Scale (DAS)^[38] were given to 5,378 freshmen. Those who scored in the highest or lowest quartiles on *both* the CSQ composite for negative events and the DAS were considered the HR and LR groups, respectively. In Phase II, a random subset of participants who met the Phase I criteria for the HR or LR groups were given an expanded Schedule for Affective Disorders and Schizophrenia-Lifetime diagnostic interview (SADS-L)^[39] by interviewers who were blind to risk status. Based on DSM-III-R^[40] and Research Diagnostic Criteria (RDC),^[41] participants were excluded if they exhibited any current Axis I disorder, psychotic symptoms, or any serious medical illness. Participants were retained if they met diagnostic criteria for a past depressive disorder but had remitted for at least 2 months (to insure that any depression onsets during the prospective phase were new episodes and not relapses). On average, the most recent past episode of depression was 2.31 years ($SD = 2.44$ years) before Phase I. The final CVD sample included 172 HR and 175 LR participants (see Alloy et al.^[37] for the sample demographics and representativeness).

The present study analyzed data from the 65 CVD project participants who experienced at least one depressive episode that met criteria for a HD episode during the first 2.5 years of prospective follow-up. This resulted in 169 HD episodes being included in these analyses: 10 participants had only one HD episode during the follow-up period, 23 experienced two HD episodes, 19 experienced three, nine experienced four, and four experienced five HD episodes. There were 12 participants who were mid-episode at the end of the follow-up period, so symptom offset dates for those episodes were estimated conservatively as the date the follow-up period ended. In the CVD study overall, 4.2% of the sample received psychotherapy or medication for their depression. Thus, 4.2% may have remitted from an

episode due to treatment; the remainder remitted naturally without treatment. Table 1 presents the demographic characteristics and cognitive risk status of this sample.

DIAGNOSIS OF THE HD SUBTYPE

All DSM-III-R MDD episodes were assessed for whether they met criteria for the hopelessness subtype.^[7] Diagnoses of HD were based on the criteria set forth by Alloy and colleagues^[27] for the CVD project. These criteria include (1) hopelessness present for at least 1 week, for 6 out of 7 days of each week; (2) at least four criterion symptoms present, overlapping 6 out of 7 days of each week for at least 1 week. The criterion symptoms of HD are sadness, retarded initiation of voluntary responses, suicidal ideation, sleep disturbance (initial insomnia), low energy, self-blame, difficulty in concentration, psychomotor retardation, brooding/worrying, lowered self-esteem, and dependency. All symptoms were assessed using the SADS-L and Change versions.

MEASURES

The SADS-L version^[39] is a widely used structured diagnostic interview that assesses current and past psychopathology according to the RDC. The SADS-L was used in this study as part of the Phase II screening procedure (described above). The SADS has demonstrated high inter-rater reliability across interview sessions and high test-retest reliability.^[39]

For the purposes of the CVD project, the SADS was modified and expanded in several ways.^[36] First, additional questions were included to allow DSM-III-R diagnoses to be made. Second, a more precise set of initial “probes” was included to assess the persistence of depressed mood. Last, questions were included to assess two cognitive subtypes of depression according to the Hopelessness Theory^[7] and Beck’s^[11] Theory. The expanded version of the SADS-L, like the original version, has demonstrated high levels of inter-rater reliability, with κ s for all diagnoses = 0.90.^[37]

To assess the emergence of symptoms and episodes an expanded SADS-Change (SADS-C) interview was conducted. The SADS-L and SADS-C differ in that the “L” version was administered to assess current and past depressive experiences, whereas the “C” version was given every 6 weeks throughout the first 2.5 years of follow-up. When an item was endorsed on the SADS-C strict dating of symptoms (onset and offset) was recorded. In the CVD project, inter-rater reliability of the expanded SADS-C was high (κ s = 0.90) for all diagnoses.^[27] Test-retest reliability, in which two interviewers blindly interviewed the same participant within 2 days of each other, obtained κ s = 0.90 for all diagnoses as well.^[27]

For the current study, the following 29 depressive symptoms were assessed via the SADS-L and SADS-C and included in the analyses: sad mood, decreased appetite, weight loss, increased appetite, weight gain, initial insomnia, middle insomnia, early waking, hypersomnia, decreased energy, decreased interest or pleasure, self-blame, difficulty concentrating, indecision, suicidal ideation, psychomotor agitation, psychomotor retardation, crying more, inability to cry, hopelessness, brooding/worry, decreased self-esteem,

irritability, dependency, self-pity, somatic complaints, decreased effectiveness, helplessness, and decreased initiation of voluntary responses.

The CSQ^[37] was utilized, in addition to the DAS, to determine cognitive risk status (HR or LR for depression onset). The CSQ was developed from the original and revised versions of the Attributional Style Questionnaire (ASQ)^[42, 43] to assess depressogenic inferences for positive and negative events, although only the composite score for negative events was used in the CVD project. The CSQ consists of hypothetical situations representing positive and negative interpersonal and achievement events. Participants are asked to identify the cause of an event, assess its degree of importance, and make attributions as to the internality, stability, and globality of the cause, as well as inferences concerning characteristics about the self and event consequences, using a 1–7 rating scale. The CSQ composite for negative events consists of the total stability, globality, consequences, and self-ratings for the 12 negative hypothetical events. Within the CVD project, internal consistency of the CSQ was good for both negative and positive events ($\alpha = 0.88$ and 0.86 , respectively).^[37] The 1-year test–retest reliability was also good ($r = 0.80$ for both negative and positive events).^[37] With respect to validity, various studies^[27, 37, 44] have shown that the CSQ in combination with the DAS significantly predicted depressive episodes and suicidality.

The DAS^[38] is a 40-item self-report measure of depressogenic attitudes on a 7-point scale ranging from “totally agree” to “totally disagree.” The DAS assesses perfectionistic expectations of performance, concerns about disapproval, pessimism, and causal attributions. In the CVD project, 24 additional achievement- and interpersonally oriented items were included. Internal consistency for the expanded DAS was high ($\alpha = 0.90$) and 1-year test–retest reliability was good ($r = 0.79$).^[37] Regarding validity, Weissman and Beck^[38] found the correlation of the DAS with the BDI to range from 0.48 to 0.55 in a college sample.

PROCEDURES

Participants in the CVD project provided informed consent and were randomly assigned to interviewers after the Phase II assessment using the SADS-L. They were interviewed every 6 weeks for the first 2.5 years of the study and every 4 months for the remaining 3 years. Each of these subsequent interviews included the SADS-C, among other assessments. Interviews were conducted in person, when possible; otherwise, interviews were conducted via telephone. Rohde, Lewinsohn, and Seeley^[45] have shown excellent comparability of phone and face-to-face interviews in assessing Axis I disorders with semistructured diagnostic interviews. Moreover, all CVD project interviews were conducted face to face with all participants for at least the first 2 years of follow-up. After that, some participants moved out of state or even to another country and interviews were then conducted with them by telephone, rather than lose them from the study. Less than 10% of the CVD study interviews were conducted by phone. CVD study participants were interviewed in person consistently by the same interviewers for multiple-year periods. By the time any phone interviews were conducted the participant had already had multiple in-person interviews with the interviewer, resulting in great familiarity with the interview and rapport with the interviewer, both contributing to the reliability and validity of the subsequent phone

interviews. In addition, no systematic differences in the data obtained from phone versus in-person interviews were observed.

DATA ANALYTIC STRATEGY

For the current study, SADS-C data from the CVD project were utilized to diagnose HD episodes and to ascertain the onset and offset dates of depressive symptoms experienced in relation to a depressive episode. To test the hypotheses that specific symptoms would be the first to appear and last to remit in HDs, the symptoms present throughout episodes of depression meeting criteria for HD were subjected to survival analyses. Survival analyses^[46] of symptoms have been employed in previous studies of the order of symptom onset in depressive episodes of Seasonal Affective Disorder.^[47] A survival analysis for each symptom provides an estimate of the symptom's probability density (risk of onset) over time. Graphs are generated that plot the probability density over time throughout the study. The slope of the line indicates whether the symptom is equally likely to appear at any given time (indicated by a flat slope) or more likely to appear earlier in the course of the episode (indicated by a negative slope; high initial risk of onset, dropping off as time goes on).

For the purposes of the current study, the probability densities for the onset and offset of the 29 SADS-C depression symptoms were ascertained for 3-day periods beginning with the appearance of the first (prodromal) symptom and ending with the offset of the last symptom to remit. For prodromal symptoms, the onset of the first symptom served as time 0 for each episode, and the onset of subsequent symptoms was coded in subsequent 3-day intervals. Probability density graphs were evaluated for the initial risk of onset and slope of the line. Symptoms with an initial risk of onset ≥ 0.25 and demonstrating a rapidly declining (then relatively flat) probability density over time were identified as those most likely to appear earliest in the course of the HD episode.

For the remission of symptoms, the date of offset of the last symptom to remit served as time 0 for each individual, and the offset of preceding symptoms was coded in preceding 3-day intervals corresponding to the number of days the symptom remitted before the last symptom. Such a reverse-coding procedure was used to standardize the endpoint of the remission period across participants, to address the possibility that periods of residual symptoms can vary across individuals. As with symptom onset graphs, graphs with initially high (risk of onset ≥ 0.25) and rapidly declining probability density functions represent symptoms that are most likely to remit at the end of the remission phase (there was less time between the symptom remission and the end of the remission phase). For symptoms that did not remit while the participant was in the study (12 participants were mid-episode at the end of the follow-up period), the latest date the participant was evaluated in the study was used as a conservative estimate of the symptom offset date for all remaining symptoms.

Statistical tests of the differences between probability density graphs were not conducted because there did not appear to be a clear method of doing so. Thus, the survival analyses employed to test these hypotheses were descriptive rather than inferential statistics. Other survival techniques (e.g., Cox regression) make assumptions that are not applicable to the data involved in the current study, as the curves in question did not represent different participant groups, but different symptoms within the same participants. Indeed, previous

studies of the pattern of depressive symptom onset^[47] did not statistically test for differences in rates for the same reasons.

RESULTS

One hundred sixty-nine episodes of HD were included in the analyses. Table 2 summarizes the probability density scores for early onset and late remission for all 29 symptoms assessed. Five symptoms had initial risk of onset 0.25 that rapidly decreased over time. The symptom of hopelessness had the highest initial and most drastically declining probability density function of all the symptoms assessed. Brooding/worry, decreased self-esteem, dependency, and decreased appetite also had initially high (0.25) and generally declining probability densities as well. Figure 1 presents the probability density functions for the onset of each of these symptoms. Relatively linear probability density rates were observed for the other symptoms.

Survival analyses for each symptom experienced during a HD also yielded probability densities for each 3-day period from the symptom offset to the end of remission, which were graphed and analyzed. Six symptoms were observed to have probability densities that rapidly increased to 0.25 at the end of the episode: hopelessness, decreased self-esteem, self-blame, brooding/worry, dependency, and increased appetite. Figure 2 presents the probability density functions for the offset of each of these symptoms. The other symptoms had generally linear and relatively constant probability density graphs.

To address the concern that a prior history of depression may influence symptom presentation, the above analyses were also conducted on the group of participants with no prior history of depression ($n = 27, 99$ episodes). The results were essentially identical to those reported above: the same HD symptoms that were observed to appear early and remit late (beyond the 0.25 cutoff). Given the smaller sample size, the probability density function graphs had significantly more noise, however. Coupled with the fact that the results were so similar to the primary analyses, these secondary analyses are not reported in depth.

DISCUSSION

Episodes of HD were subjected to analyses to elucidate patterns of symptom onset and offset. It was hypothesized that hopelessness would be the first symptom to emerge in HD episodes, followed by HD criterion symptoms and any secondary symptoms. Secondary symptoms were predicted to appear after the core HD symptoms. Survival analyses of the time to onset for each of the 29 symptoms assessed yielded probability density functions, which display the probability of the symptom presenting over time. Consistent with the hypothesis, hopelessness exhibited a clear pattern of being the earliest symptom to emerge. Moreover, the HD criterion symptoms of brooding/worry, decreased self-esteem and dependency, and the non-HD (secondary) symptom of decreased appetite, were also most likely to onset at or near the beginning of the episode. The other symptoms did not have a particular pattern to their onset. According to the model proposed by Iacoviello and colleagues,^[14] the core syndrome of these episodes comprised the predominantly HD criterion symptoms appearing at or near the beginning of the episode. The model predicts

these symptoms would be the latest to remit, and indeed, probability density functions for symptom remission indicated that hopelessness and decreased self-esteem were typically the last symptoms to remit, preceded by self-blame, brooding/worry, dependency, and increased appetite. Thus, there was significant overlap between the earliest symptoms to appear and latest symptoms to remit in HD episodes; and, these core symptoms were predominantly HD criterion symptoms.

Taken together, the results of this study are consistent with the rollback phenomenon described by Detre and Jarecki,^[15] as well as the model for the onset and remission of depression episodes proposed by Iacoviello and colleagues.^[14] Specifically, the symptoms that tended to appear earliest in the HD episodes were predominantly HD criterion symptoms and represented a majority of the symptoms observed to remit latest. This suggested a set of core symptoms appearing earliest and remaining throughout the episodes. That these core symptoms were predominantly HD criterion symptoms could speak to the validity of the HD subtype.^[7] However, there were also HD criterion symptoms that did not demonstrate a pattern of early onset and late remission and did not appear to constitute core symptoms of the HD episodes followed in this study. This may call into question some of the HD criterion symptoms predicted by the Hopelessness Theory.^[7] Or, this may indicate that some of the HD criterion symptoms are more proximal to the cognitive vulnerabilities underlying HD episodes, and other HD criterion symptoms are more distal, stemming from similar and/or different processes further “downstream.” The early symptoms of hopelessness, decreased self-esteem, self-blame, and brooding/worry might contribute to the later onset of other HD criterion symptoms such as sad mood, initial insomnia, decreased energy, difficulties concentrating, psychomotor retardation, decreased initiation of voluntary responses, and suicidality. In addition, appetite and weight symptoms appeared to demonstrate a pattern of onset and offset that was not predicted by Hopelessness Theory or the Iacoviello model: decreased appetite was observed to appear near the beginning of episodes but not remain throughout, and increased appetite was observed to be a later symptom to remit when it was present in an episode. These symptom patterns could be an artifact of small sample size in the analyses and not representative of a true pattern in the population. Or, this might indicate that a reconsideration of the HD symptom criteria is warranted, to include early appetite reduction and later increased appetite.

The results of this study have clinical as well as research implications. When confronted with a patient experiencing an episode of HD, knowledge of how core HD symptoms may be expected to occur and remit could benefit clinicians greatly in treatment planning and monitoring of treatment progress. The results of this study suggest that the core HD symptoms might be the most efficient treatment targets. That would mean focusing first on the hopelessness, decreased self-esteem, brooding, self-blame, and dependency that patients exhibit. It might also suggest that psychotherapeutic interventions may be preferred in cases of HD, as these core symptoms appear to stem proximally from underlying cognitive vulnerabilities and may be more effectively treated with talk therapy (particularly cognitive therapy) than medications. In terms of monitoring treatment progress, clinicians could expect that secondary symptoms would begin to remit first, followed by the core HD symptoms. This could provide a roadmap for clinicians to assess the degree to which the

episode has been treated or has remitted, as well as provide insight into the risk of relapse if some of these core symptoms do not fully remit.

This study has several strengths. First, the prospective design enabled the onset and remission of depression symptoms to be monitored in regular, short intervals, and not assessed through longer term retrospective report. The prospective design and frequent (every 6 weeks) assessments increased the reliability of the semistructured interview measures of depression symptoms. The broad range of symptoms assessed and the sensitive nature of the measures allowed for the study of many potential depressive symptoms at subclinical levels. These are all improvements over the methodologies of previous studies of depressive prodromes. Moreover, it is important to note that the semistructured diagnostic interviews did not bias to diagnosing HD in those patients who exhibited early onset of hopelessness or the other core HD symptoms. All 29 symptoms were assessed for each participant at each time point and the diagnosis of HD was made post hoc after data were collected.

A limitation of the current study is the inclusion of some participants who had a prior history of depression. Prior history of depression could conceivably have an influence on patterns of symptom onset and remission, as could gender, SES, and psychiatric comorbidities. However, the analytic strategy employed in this study precluded the ability to investigate the potential effects of these variables. In addition, some participants contributed more episodes to the analyses than others, introducing a possible cluster effect in the analysis. Analyses were conducted to assess the cluster effect of these participants, and similar results were found as when all episodes were included in one analysis, which is what we report here. However, future studies should account for such factors in addition to those mentioned above. The current study was also limited in its ability to make inferences regarding the pattern of symptom onset and offset. Inferences relied on the visual comparison of probability density graphs to identify symptoms earliest to appear and latest to remit. There are also limitations in terms of the study sample, which consisted entirely of college students, a majority of whom (86.15%) were Caucasian, and whom were selected for the CVD study based on their HR or LR for developing depression. This may limit the generalizability of these findings. Lastly, there may be limitations associated with the use of phone interviews to assess symptoms, although this has been shown to be comparable to face-to-face interviews when using a semistructured interview for Axis I symptoms.^[45]

CONCLUSION

The current study provided further support for a conceptual model of the development and remission of depressive symptoms in episodes of depression.^[14] Probability density graphs indicated that hopelessness was typically the earliest symptom to emerge in HD episodes, followed by other HD criterion symptoms. These were also generally the latest symptoms to remit, with hopelessness and decreased self-esteem typically remitting near the very end of these episodes. These symptoms that present earliest and remit latest are important insofar as they might be thought to comprise the core syndrome of the depressive episode, could have applicability in identifying subtypes of depression, and could be informative for clinicians planning and monitoring treatment for patients with depression.

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Figure 1.
Probability density functions for symptom onset.

Figure 2.
Probability density functions for symptom remission.

TABLE 1

Demographic and clinical characteristics of the sample

	High cognitive risk (HR)	Low cognitive risk (LR)	Comparison
Sample size	49	16	
Age(years)	18.51 (1.02)	19.07 (1.71)	$t(63) = 1.59, P = .12$
Ethnicity	85.70% Caucasian 10.02% African-American 4.1% "Other"	75.0% Caucasian 18.75% African-American 6.25% "Other"	$\chi^2(63) = 1.84, P = .14$
Gender	63.27% female	62.5% female	$\chi^2(63) = 0.09, P = .95$
Prior history of depression	60.7%	18.75%	
CSQ-N item score	5.05 (0.41)	2.82 (0.42)	$t(63) = 18.78, P < .001$
DAS item score	4.41 (0.60)	2.25 (0.38)	$t(63) = 13.5, P < .001$
Number of depression episodes experienced during study	3.1 (1.97)	2.2 (1.66)	$t(63) = 1.64, P = .11$
Mean episode duration (days)	31.69 (16.98)	25.99 (19.96)	$t(63) = 1.12, P = .27$
Mean remission duration between episodes (days)	80.45 (38.88)	97.27 (41.23)	$t(63) = 1.48, P = .14$

Note: Standard deviations in parentheses. DAS, Dysfunctional Attitudes Scale; CSQ-N, Cognitive Style Questionnaire-Negative item composite.

TABLE 2

Summary of probability density scores for early symptom onset and late remission

	Onset—high risk of early presentation	Remission—high risk of late remission
Hopelessness symptoms		
Hopelessness	0.66	0.60
Sad mood	0.20	0.21
Initial insomnia	0.11	0.11
Decreased energy	0.15	0.18
Self-blame	0.23	0.28
Difficulty concentrating	0.18	0.20
Suicidality	0.19	0.12
Psychomotor retardation	0.24	0.0
Brooding/Worry	0.27	0.30
Decreased self-esteem	0.56	0.57
Dependency	0.28	0.27
Decreased initiation of voluntary responses	0.20	0.17
Other symptoms		
Decreased appetite	0.29	0.24
Weight loss	0.24	0.09
Increased appetite	0.20	0.33
Weight gain	0.21	0.24
Middle insomnia	0.15	0.04
Early waking	0.12	0.13
Hypersomnia	0.12	0.08
Decreased interest or pleasure	0.14	0.19
Indecision	0.24	0.10
Psychomotor agitation	0.12	0.19
Crying	0.12	0.09
Inability to cry	0.0	0.0
Irritability	0.16	0.15
Self-pity	0.20	0.16
Somatic complaints	0.17	0.15
Decreased effectiveness	0.17	0.18
Helplessness	0.17	0.13

Note: Bold indicates scores that cross the 0.25 probability density cutoff.