Psychological Medicine, 2006, **36**, 913–922. © 2006 Cambridge University Press
doi:10.1017/S0033291706007586 First published online 2 May 2006 Printed in the United Kingdom

In severely suicidal young adults, hopelessness, depressive symptoms, and suicidal ideation constitute a single syndrome

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ABSTRACT

Background. Despite voluminous research on the role of hopelessness and depression in suicidality, a systematic examination of various causal models pertaining to these variables is conspicuous in its absence.

Method. The directions of relationships between the three variables were examined by means of a prospective-longitudinal, cross-lagged, three-wave design in a severely suicidal young adults.

Results. Structural Equation Modeling (SEM) analyses revealed synchronous, but not longitudinal, associations between hopelessness, depressive symptoms, and suicidal ideation.

Conclusions. In severely suicidal young adults, the three clinical constructs appear to constitute a single depressive syndrome.

INTRODUCTION

Since its inception, suicide research has identified depression and hopelessness as two causal agents. Approximately 90% of people dying by suicide have a diagnosable psychiatric disorder, mostly major depressive disorder, at the time of death (Henriksson *et al.* 1993; Cheng, 1995; Conwell *et al.* 1996; Vijayakumar & Rajkumar, 1999; Bertolote *et al.* 2003; see Joiner *et al.* 2005*a* for review). Moreover, depressive symptoms and depressed mood have been found to be a robust predictor of suicidal ideation and attempts (Hawton, 1987; Brent *et al.* 1988; Driessen *et al.* 1998; Kessler *et al.* 1999; Chioqueta & Stiles, 2003). As for hopelessness, defined as 'a system of cognitive schemas whose common denominator is negative expectations about the future' (Beck *et al.* 1974, p. 864), it, too, was reported as an important predictor of completed suicide, suicide attempts and suicide ideation in a wide range of populations (Hughes & Neimeyer, 1993; Cox *et al.* 2004; Wen-Hung *et al.* 2004; Hawton *et al.* 2005; Stewart *et al.* 2005). Indeed, in several studies hopelessness predicted found suicide ideation better than did depression (Silver *et al.* 1971; Beck *et al.* 1974; Wetzel, 1976; Wetzel *et al.* 1980; Chioqueta & Stiles, 2003). Nevertheless, reports of null results regarding the effect of hopelessness on suicidality also exist (Harris & Lennings, 1993; Metha *et al.* 1998).

Yet in considering the extant research on the associations between depression, hopelessness, and suicidality, several questions are raised. First, it is not at all clear that depression and hopelessness are two distinct entities. For instance, Beck (1963) suggested that hopelessness is the active ingredient in the relationships between depression and suicidality, and this conceptualization locates hopelessness as a key feature of depression, rather as a distinct entity

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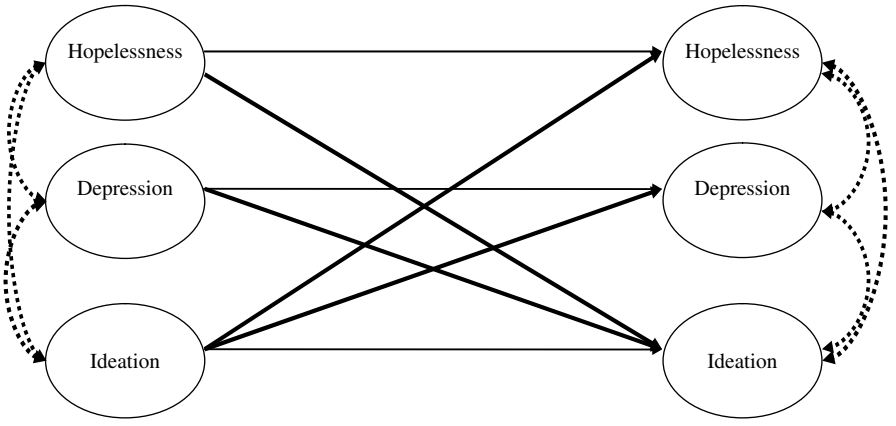

FIG. 1. Conceptual model of the relations between hopelessness, depressive symptoms, and suicidality. —, Cross-lagged effects; ·····, synchronous associations; —, stability effects.

(see also Joiner *et al.* 2005*b*, for a similar perspective). Consistently, the hopelessness theory of depression (Abramson *et al.* 1989) describes *hopelessness depression* as a theoretically based subtype of depression with characteristic causes, symptom profile, course, treatment and prevention (see also Metalsky & Joiner, 1997). Discussing this theory, Joiner *et al.* (2001) observed: 'Technically, hopelessness is not framed as a symptom of hopelessness depression (Abramson *et al.* 1989); rather, it is viewed as the final common pathway to hopelessness depression' (Joiner *et al.* 2001, p. 526, footnote 2).

To complicate matters further, rather than being described as an outcome of hopelessness and depression, suicidality is frequently considered a component of these clinical conditions. To illustrate, suicidal ideation and intent is one of the nine components of hopelessness depression, and this component currently is included as one of the subscales of the only measure of this condition, i.e. the Hopelessness Depression Symptoms Questionnaires (HDSQ; Metalsky & Joiner, 1997). Similarly, items assessing suicidal ideation and intent are included in standard measures of depressive symptoms, such as the Beck Depression Inventory (BDI), as well as in the DSM-IV (APA, 1994) definition of major depression.

These considerations point out the need to compare two conceptualizations of the relationships between hopelessness, depression, and suicidality: a causality conceptualization, whereby hopelessness, depression, or both influence suicidality, and a synchronous association conceptualization, according to which the three constructs constitute interrelated components of a single clinical syndrome. What would be the best way to compare these two conceptualizations? We submit that this is best done by means of the cross-lagged design combined with Structural Equation Modeling (SEM; Marmor & Montemayor, 1977; Hays *et al.* 1994; Shahar & Davidson, 2003; Shahar *et al.* 2004).

The cross-lagged design includes the measurement of two or more variables (in our case, hopelessness, depressive symptoms, and suicidality), at two or more times. It yields *synchronous effects* (e.g. the cross-sectional association between the study constructs at each point in time), *stability effects* (i.e. the prediction of a construct by its previous levels), and, most importantly, *cross-lagged effects*. These latter effects refer to the prediction of one or more constructs by other variables that have been measured previously, controlling for the baseline level of the predicted variable (i.e. controlling for stability effects).

In Fig. 1 we present a cross-lagged design in which the aforementioned causality and synchronous association conceptualizations are compared with respect to hopelessness, depressive symptoms, and suicidality. Consistent

with the causality conceptualization, we would expect cross-lagged effects leading from hopelessness and/or depression to suicidality. Consistent with the synchronous association conceptualization, we would expect no such cross-lagged effects. Rather, strong cross-sectional relationships between the variables are expected.

Notice that Fig. 1 presents yet another interesting description of the relationships between the putative constructs, i.e. that suicidality predicts an increase in hopelessness and depression over time. This possibility, while not extensively investigated, is nevertheless supported by research showing elevated levels of distress and adjustment problems in suicidal individuals (Spirito *et al.* 1992; Pfeffer *et al.* 1993; Fergusson & Lynskey, 1995; Granboulan *et al.* 1995; Laurent *et al.* 1998; Spirito *et al.* 2000; Boergers & Spirito, 2003; Rosario *et al.* 2005). Note that this effect of suicidality on distress is consistent with two patterns. The first, labeled herein as a 'cause' pattern, refers to a situation whereby the presence of suicidal ideations, intent, or attempts, brings about depression and hopelessness by signaling to individuals that their clinical condition is worsening. The second, labeled here as a 'course' pattern, pertains to a situation whereby suicidality is an earlier indication of an emerging, serious, depressive disorder.

In the present study we tested the causality, synchronous associations, and the cause/course conceptualizations utilizing data collected as part of a randomized clinical trial (RCT) comparing problem-solving therapy and treatment as usual for suicidal young adults (cf. Rudd *et al.* 1996). Assessments in this RCT were made pretreatment, 1 month into the treatment and after 6, 12, and 18 months. In the current study we will focus on the first three assessment times. The assessment protocol includes established measures of hopelessness, depressive symptoms, and suicidal ideation and intent (see below). Consistent with the recommendations of Hays *et al.* (1994), we (*a*) measured each construct as a latent variable assessed using multiple indicators, (*b*) focused on the three waves of measurement, and (*c*) employed SEM analysis to test the above described patterns corresponding to the three conceptualizations.

METHOD

Participants and procedure

The entire sample on which this study is based ($n=332$) consisted of individuals evaluated as they entered a study on the efficacy of time limited, problem-solving-based treatments for suicidal young adults (mean age = 22·32, S.D. = 2·72; range 18–31 years). Most participants (82%) were men. Most were Caucasian ($n=73$ or 64·6%); 18·6% were African-American; 12·4% were Hispanic; 1% were Native American; 1% were Asian or Pacific Islander; the remainder were classified as 'Other'. As reported by Rudd *et al.* (1996), 72% received a diagnosis of depressive disorder (e.g. major depression, dysthymia, bipolar disorder or atypical depression), with the remainder receiving a diagnosis of anxiety disorder. Co-morbidity in this sample was very high (over 90%, see Rudd *et al.* 1993, 1996). Diagnoses were made using the National Institute for Mental Health (NIMH) Diagnostic Interview Schedule (Rogier *et al.* 1984).

All participants were referred for severe suicidality (i.e. recent attempt, or ideation serious enough to warrant immediate evaluation for hospitalization) from two out-patient clinics, a 20-bed in-patient facility, and an emergency room, all affiliated with a major U.S. Army Medical Center. They provided full, informed, and written consent for research participation. All later received rigorous treatment [either a problem-solving treatment as described by Rudd *et al.* (1996) or treatment as usual (often a few days of in-patient psychiatry then out-patient antidepressant medicines plus supportive therapy)]. Assessments were conducted by trained clinical staff.

Following assessment at intake, participants received rigorous treatment in one of two forms. The first type of treatment was a form of intensive, structured group therapy emphasizing interpersonal skill development and adaptive coping. Groups consisted of 8–14 members, and treatment took place for 9 hours per day over a period of 10 days. Each afternoon was spent focused on six components of problem solving: problem orientation, problem identification/goal setting, generation of alternatives, evaluation of alternatives, implementation, and evaluation of initial efforts. This

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six-step approach to problem-solving was consistent with that detailed by Nezu *et al.* (1989). A second facet of treatment included eight 1-hour psychoeducational sessions taught in didactic format using detailed outlines of targeted issues (i.e. communication, impulsivity/anger control, emotion regulation, stress management/relaxation). A third aspect was experiential-affective, involving discussion of connection between symptoms and past and current experiences and feelings.

The second type of treatment was referred to as 'treatment as usual', and consisted of a combination of in-patient and out-patient services. In-patient stays averaged approximately 7 days. Out-patient services typically combined antidepressant medication with individual and/or group therapy, with the specific nature of the treatment left up to the discretion of the provider. Among poor problem solvers, the problem-solving treatment was slightly, but significantly, better than treatment as usual at retention.

Instruments

Beck Depression Inventory (*BDI; Beck et al. 1961*). The BDI is a 21-item self-report inventory of depressive symptoms. Each item is rated on a 0–3 scale; inventory scores thus may range from 0 to 63. The BDI is a reliable and well-validated measure of depressive symptomatology (Beck *et al.* 1988). In the present study we constructed a latent depression variable by computing two manifest indicators: a cognitive-affective symptoms indicator (e.g. sadness, anhedonia, sense of failure) and a physiological symptoms indicator (e.g. difficulty sleeping, weight loss; Beck *et al.* 1988). To prevent spurious correlations with the other constructs in this study, i.e. hopelessness and suicidality, we left out BDI items 2 (hopelessness) and 9 (suicidal ideation).

Beck Hopelessness Scale (*BHS; Beck et al. 1974*). The BHS is a 20-item inventory that assesses the degree to which an individual's cognitive schemata are characterized by pessimistic expectations. The scale uses a true–false response format. Scores can range from 0 to 20, with higher scores indicating a greater degree of hopelessness. The scale's reliability and validity have been supported (e.g. see Metalsky *et al.* 1993). Recent Varimax-rotated principal-components analysis of the BHS (Dyce, 1996) identified three components: Expectations of Success, Expectations of Failure, and Future Uncertainty. In the present study we used these three components as manifest indicators of a latent hopelessness variable.

Suicidality

We used two measures of suicidality, each of which served as a manifest indicator for a latent suicidality variable.

Modified Scale for Suicidal Ideation (*MSSI; Miller et al. 1986*). The MSSI is an 18-item semi-structured interview administered by professionals or paraprofessionals. Items cover such topics as wish to die; intensity, frequency, and duration of suicidal ideation; suicide plans; and writing about suicide. Interviewers were instructed to assess symptoms occurring over the past year, with emphasis (particularly at the follow-up assessment) on recent symptoms. Each MSSI item was rated on a 0–3 scale; overall scale scores thus may range from 0 to 54, with a score of $\geqslant$11 indicating clinical significance. Miller *et al.* (1986) have reported reliability coefficients [e.g. coefficient (α) = 0·94] and construct validity data (see also Clum & Yang, 1995; Joiner *et al.* 1997).

Suicide Probability Scale (*SPS; Cull & Gill, 1989*). The SPS is a 36-item, self-report measure of suicidal and related symptoms. The scale provides a total score, as well as scores on four subscales: hopelessness, negative self-evaluation, hostility, and suicidal ideation. In the present study, we used the suicidal ideation subscale. Items cover such topics as wish for death, thoughts of suicide, and not wanting to go on. The time frame for the SPS is unspecified, but the context in which participants completed the scale (e.g. in crisis) implies a focus on relatively recent symptoms. The scale has accrued adequate reliability and validity data (see, e.g. Cull & Gill, 1989).

Data analysis

Analysis was conducted in two phases. In Phase 1 we established the measurement model of the manifest indicators by employing Confirmatory Factor Analysis (CFA). In Table 1 we present

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Table 1. *Latent variables and their respective manifest indicators*

	Latent variables		
	Hopelessness	Depression	Suicidal ideation
Factor 1	Expectations of Success	Cognitive-affective	MSSI
Factor 2	Expectations of Failure	Physiological	SPS_SI
Factor 3	Future Uncertainty		

MSSI, Modified Scale for Suicidal Ideation; SPS_SI, Suicide Ideation Subscale of the Suicide Probability Scale.

the latent variables and respective manifest indicators that were included in the CFA. The analyses were conducted on the three latent constructs – hopelessness, depression, and suicidal ideation, at Time 1 (pre treatment), time 2 (1 month into the treatment), and time 3 (6 months into the treatment).

Correlations were specified between these latent constructs. Autocorrelations were specified between the error terms of the manifest variables across time. For instance, the error term of the hopelessness manifest variables at Time 1 (i.e. the part of the variance in each manifest variable that is unrelated to the hopelessness latent construct) were correlated with the same error terms at Time 2 and Time 3, and error terms at Times 2 and Time 3 were also correlated. The same practice was employed with respect to manifest indicators for depression and ideation. Finally, to ensure measurement in variance over time, loadings of the manifest variables on their respective latent constructs were constrained to equality over time (cf. Hoyle & Smith, 1994). We expected an adequate model fit and strong loadings of the manifest variables on their respective latent constructs, which would establish the measurement model and will allow testing structural (i.e. directional) relationships between the latent constructs (Anderson & Gerbing, 1988).

This was done in Phase 2. We employed a cross-lagged, SEM analysis consistent with the guidelines of Hays *et al.* (1994; see also Shahar & Davidson, 2003). Specifically, we estimated the aforementioned synchronous, stability, and cross-lagged effects involving hopelessness, depression, and suicidal ideation at Times 1–3. Consistent with the synchronous association conceptualization, we would expect strong synchronous effects between hopelessness, depression, and suicidal ideation, but no statistically significant cross-lagged effects. Consistent with the hopelessness-as-cause conceptualization, we would expect indirect effects leading from Time 1 hopelessness to Time 3 suicidal ideation via Time 2 depression. Finally, consistent with the cause/course conceptualization, we would expect cross-lagged effects leading from Time 1 suicidal ideation to Time 2 hopelessness and depression, and the same effects involving Time 2 suicidal ideation and Time 3 hopelessness and depression.

All CFA and SEM analyses were conducted with the AMOS 5.00 software (Swall Waters Corp., Chicago, IL, USA) using the Maximum Likelihood (ML) iteration procedure. Model fit was assessed using the following indices: The χ^2/df index, the Non-Normed Fit Index [NNFI; Bentler & Bonnet, 1980; labeled the Tucker–Lewis Index (TLI) in AMOS 5.00], the Comparative Fit Index (CFI; Bentler, 1990), and the Root Mean Square of Approximation (RMSEA). In all analyses, we arrived at the most parsimonious model by omitting statistically non-significant parameters (Bentler & Moojaart, 1989). Conventionally, an NNFI and CFI $>0{\cdot}90$, and a RMSEA $<0{\cdot}08$, suggests an adequate model fit.

Of the 326 participants who had complete data at Time 1, 77 dropped out during the period between Time 1 and Time 2 (23·61% attrition). Joiner & Rudd (2000) already reported attrition analysis regarding this group, noting that those dropping out differed from the remaining participants only with respect to MSSI scores, with the former group evincing higher scores. Attrition during the Time 1–Time 3 period was much more substantial, in that only 154 participants had at least some data pertaining to one of the study variables (52·76% attrition). Nevertheless, attrition analysis revealed no statistically significant differences between those dropping out and those remaining with respect to the study variables.

We handled missing values using full information maximum likelihood (FIML) estimates (Anderson, 1957) that are enabled by AMOS 5.0. In comparison with other methods for handling missing values (e.g. listwise deletion,

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Table 2. *Factor loadings of the Confirmatory Factor Analysis*

Factor	DP 0	DP 1	DP 6	HS 0	HS 1	HS 6	SU 0	SU 1	SU 6
BDICA 0	0·982								
BDIPhys 0	0·742								
BDICA 1		0·955							
BDIPhys 1		0·740							
BDICA 6			0·947						
BDIPhys 6			0·772						
BHSUNC 0				0·859					
BHSFAIL 0				0·851					
BHSSUCC 0				0·897					
BHSUNC 1					0·746				
BHSFAIL 1					0·850				
BHSSUCC 1					0·866				
BHSUNC 6						0·744			
BHSFAIL 6						0·895			
BHSSUCC 6						0·891			
SPS 0							0·848		
MSSI 0							0·670		
SPS 1								0·881	
MSSI 1								0·635	
SPS 6									0·960
MSSI 6									0·752

DP, Depression – composed out of two indicators: a cognitive-affective symptoms indicator (BDICA) and a physiological symptoms (BDIPhys); HS, Latent Hopelessness construction composed out of three indicators: Expectations of Success (BHSSUCC), Expectations of Failure (BHSFAIL), and Future Uncertainty (BHSUNC); SU, Suicidality – composed out of two indicators: the Modified Scale for Suicidal Ideation (MSSI) and the suicide ideation subscale of the Suicide Probability Scale (SPS).

pairwise deletion, means imputation), FIML produces the least biased estimates of missing values (Muthen *et al.* 1987). Thus, our SEM analysis are based on a sample of $n = 326$.

RESULTS

Phase 1: Measurement model

A CFA conducted based on this table yielded an excellent model fit [$\chi^2(140) = 221{\cdot}64$, $p < 0{\cdot}001$; $\chi^2/\mathrm{df} = 1{\cdot}58$; NNFI = 0·96; CFI = 0·98; RMSEA = 0·04]. All the loadings of the manifest variables on their respective latent constructs were strong (ranging from 0·635 to 0·982) and statistically significant ($p < 0{\cdot}001$). The loadings appear in Table 2 and the zero-order correlations between the latent constructs are presented in Table 3.

Table 3. *Correlations among the latent variables*

Factor	DP 1	DP 6	HS 0	HS 1	HS 6	SU 0	SU 1	SU 6
DP 0	0·347	0·244	0·821	0·242	0·227	0·727	0·303	0·260
DP 1		0·472	0·285	0·801	0·311	0·255	0·705	0·322
DP 6			0·225	0·367	0·780	0·235	0·515	0·726
HS 0				0·295	0·280	0·774	0·294	0·238
HS 1					0·486	0·214	0·696	0·232
HS 6						0·225	0·427	0·685
SU 0							0·532	0·388
SU 1								0·582
SU 6								

DP, Depression – composed out of two indicators: a cognitive-affective symptoms indicator (BDICA) and a physiological symptoms (BDIPhys); HS, Latent Hopelessness construction composed out of three indicators: Expectations of Success (BHSSUCC), Expectations of Failure (BHSFAIL), and Future Uncertainty (BHSUNC); SU, Suicidality – composed out of two indicators: the Modified Scale for Suicidal Ideation (MSSI) and the suicide ideation subscale of the Suicide Probability Scale (SPS).

Phase 2: Structural model

The structural, cross-lagged model evinced a very good fit to the data [$\chi^2(140) = 221{\cdot}64$, $p < 0{\cdot}001$; $\chi^2/\mathrm{df} = 1{\cdot}586$; NNFI = 0·95; CFI = 0·98; RMSEA = 0·042]. Nevertheless, many of the parameters were statistically non-significant. To arrive at the most parsimonious model, we omitted these non-significant parameters, culminating in a model that also fit the data well [$\chi^2(161) = 245{\cdot}65$, $p < 0{\cdot}001$; $\chi^2/\mathrm{df} = 1{\cdot}526$; NNFI = 0·93; CFI = 0·98; RMSEA = 0·04]. In this most parsimonious model, we did not omit correlations between the error terms of the manifest variables, even when those were non-significant, so as to maintain continuity with the measurement model. The parameters linking the latent constructs in this most parsimonious model are presented in Fig. 2.

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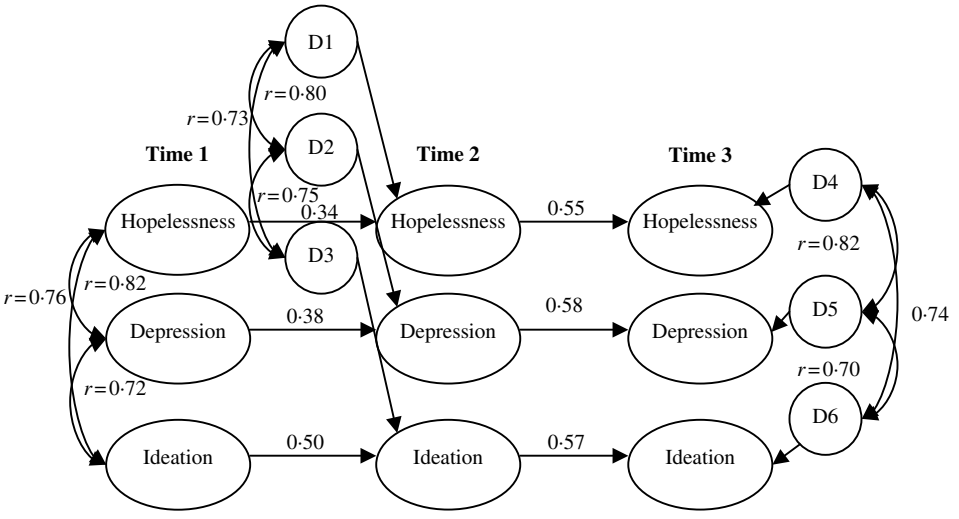

FIG. 2. Final Structural Equation Model. Numbers pertain to statistically significant parameters. **Hopelessness** – Latent construction composed out of three indicators: Expectations of Success (BHSSUCC), Expectations of Failure (BHSFAIL), and Future Uncertainty (BHSUNC); **Depression** – Latent construction composed out of two indicators: a cognitive-affective symptoms indicator (BDICA) and a physiological symptoms (BDIPhys); **Ideation** – Latent construction of suicidal ideation composed out of two indicators: the Modified Scale for Suicidal Ideation (MSSI) and the suicide ideation subscale of the Suicide Probability Scale (SPS); D1–D6: each 'D' stands for a 'Disturbance', which is a technical term representing the part of the variance of a latent variable that is unexplained by specified causes (e.g. incoming arrows). To illustrate, D1 represents the part of the variance of Time 2, a set of unspecified causes of hopelessness which is unexplained by Time 1 Hopelessness, Depression, and Suicidal Ideation.

As shown in Fig. 2, the only statistically significant associations between the latent variables were stability effects and synchronous associations. Indeed, the pattern of results was consistent with the synchronous association conceptualization, in that the synchronous associations were strong, statistically significant, and equivalent in magnitude across the three time waves (all significant at $p < 0{\cdot}001$).

Time 1: $r_{\text{h-d}} = 0{\cdot}82$, $r_{\text{h-i}} = 0{\cdot}76$, $r_{\text{d-i}} = 0{\cdot}72$;
Time 2: $r_{\text{dh-dd}} = 0{\cdot}80$, $r_{\text{dh-di}} = 0{\cdot}73$, $r_{\text{dd-di}} = 0{\cdot}75$;
Time 3: $r_{\text{dh-dd}} = 0{\cdot}82$, $r_{\text{dh-di}} = 0{\cdot}74$, $r_{\text{dd-di}} = 0{\cdot}70$;

where h-d = hopelessness-depression; h-i = hopelessness-ideation; d-i = depression-ideation; dh-dd = disturbance of hopelessness-disturbance of depression; dh-di = disturbance of hopelessness-disturbance of ideation; dd-di = disturbance of depression-disturbance of ideation.

DISCUSSION

Using data collected as part of a RCT on severely suicidal young adults, we found that hopelessness, depression, and suicidal ideation evinced strong synchronous associations, but not prospective longitudinal ones. These findings are inconsistent with *a causal conceptualization*, which posits that hopelessness and depression bring about suicidality. Nor are they consistent with *a cause/course* conceptualization, which suggests that suicidality brings about hopelessness and depression.

How do we reconcile these findings, which provide support only for the synchronous association conceptualization, with the aforementioned previous findings as to the predictive effects of hopelessness and depression on suicidality? First, we are reminded that, at least in the case of hopelessness, reports of null findings also exist (Harris & Lennings, 1993; Metha *et al.* 1998). Second, we would like to emphasize that, from a logical-methodological point of view, an assumption regarding synchronous association is much more conservative than that of causal relationship. This is because synchronous associations are 'nested' within a causal relationship: for one entity to cause the other, they both have to be *related*. The implication of this reasoning is twofold. First, considerably more conditions need to be met when establishing the causal relationships between depression,

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hopelessness, and suicidality (Davis, 1985; Pearl, 2000). Second, any finding that is consistent with a synchronous association but not with a causal interpretation carries a lot of weight, because of its greater conservative nature.

Third, and much more importantly, we believe that the unique nature of our sample holds the key to reconciliation of present and past findings. Specifically, we have investigated severely disturbed, already suicidal, young adults. Regardless of the onset of their suicidality, which might very well conform to the causal conceptualization whereby hopelessness and depression bring about suicidality, the chronic and persistent, and in many cases treatment- resistant, nature of these individuals' psychopathology might have changed the nature of the associations between hopelessness, depression, and suicidality. In the context of such persistent psychopathology, hopelessness, depression, and suicidality might converge into a single, very serious and difficult to treat, psychiatric syndrome, which might be labeled as suicidal depression.

Further support for the notion of suicidal depression is obtained from a very recent, and particularly pertinent, report by Chioqueta & Stiles (2005). These authors investigate the cross-sectional associations between personality traits [as measured by the revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992)], and BHS hopelessness, depressive symptoms [as measured by the Hopkins Symptom Checklist (HSCL-25), which is a shortened version of the Symptom Checklist (SCL-90; Derogatis *et al.* 1973)], and suicide ideations (as measured by item 14 of the HSCL-25). The authors found that of the five personality factors of the NEO-PI-R [i.e. Neuroticism (N), Extraversion (E), Agreeableness (A), Conscientiousness (C), and Openness to Experience (O)], only Neuroticism predicted the three clinical constructs, namely, hopelessness, depression, and suicide ideation. Further analyses focusing on specific personality facets revealed that of the six Neuroticism facets (i.e. anxiety, anger hostility, depression, self-consciousness, impulsiveness, and vulnerability), only depression predicted the three clinical constructs. These findings confirm the role of negative affectivity, as captured by the depression facet of Neuroticism, in symptomatic depression, hopelessness, and suicidal ideation. Our findings extend this study by suggesting that the strong associations between hopelessness, depressive symptoms, and suicidality might be particularly pronounced among individuals with a trait depressive vulnerability.

To the extent that suicidal depression indeed constitutes a *bona fide* subtype of major affective disorders is unclear. Voluminous research established the diagnostic stability of other subtypes of depression, such as psychotic, agitated-retarded, and endogenous (Nelson & Charney, 1981; Helms & Smith, 1983; Winokur, 1985; Young *et al.* 1990; Coryell *et al.* 1994; Nierenberg *et al.* 1996; but see Oquendo *et al.* 2004, for evidence of instability).

Our findings underscore several clinical implications. From the point of view of clinical assessment, they elaborate on previous conceptualizations (Jobes, 1995; Jobes & Drozd, 2004) in alerting practitioners to the complexity of symptoms presented by suicidal patients. More specifically, heterogeneity and diagnostic co-morbidity are often the norm among these patients (Rudd *et al.* 1993), requiring the identification of a broad spectrum of symptoms. Relatedly, from the point of view of treatment, clinicians need to be aware that because of this complexity, remission in one symptom area may not lower risk since there are other serious symptoms that co-occur in the presentation of suicidal patients. This realization is consistent with recent integrative approaches to the treatment of suicidality (Rudd *et al.* 2000).

Limitations of the present study should be noted. In addition to the aforementioned distinct nature of our sample, we need to mention that it consisted of chronically suicidal, mostly male, patients, and this restricts the generalizability of our findings. Moreover, this study, while longitudinal, only captures a 6-month interval. Different findings might be obtained with different time intervals. Yet despite these important limitations, our findings are, to the best of our knowledge, the first to pit various models of the relationships between depression, hopelessness, and suicidality, and to point out that among chronically suicidal young adults, these three clinical constructs make up an overarching unipolar depressive presentation.

ACKNOWLEDGMENTS

Golan Shahar acknowledges the National Alliance for Schizophrenia and Depression (NARSAD) Young Investigator Award, which supports his work on this manuscript. He also acknowledges the Department of Psychiatry at Yale University, with which he is affiliated as Visiting Associate Professor, for its support and encouragement in the course of this study.

DECLARATION OF INTEREST

None.

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