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Impulsivity is an Independent Predictor of 15-Year Mortality Risk among Individuals Seeking Help for Alcohol-Related Problems

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Abstract

Background—Although past research has found impulsivity to be a significant predictor of mortality, no studies have tested this association in samples of individuals with alcohol-related problems or examined moderation of this effect via socio-contextual processes. The current study addressed these issues in a mixed-gender sample of individuals seeking help for alcohol-related problems.

Methods—Using Cox proportional hazard models, variables measured at baseline and Year 1 of a 16-year prospective study were used to predict the probability of death from Years 1 to 16 (i.e., 15-year mortality risk). There were 628 participants at baseline (47.1% women); 515 and 405 participated in the follow-up assessments at Years 1 and 16, respectively. Among Year 1 participants, 93 individuals were known to have died between Years 1 to 16.

Results—After controlling for age, gender, and marital status, higher impulsivity at baseline was associated with an increased risk of mortality from Years 1 to 16; however, this association was accounted for by the severity of alcohol use at baseline. In contrast, higher impulsivity at Year 1 was associated with an increased risk of mortality from Years 1 to 16, and remained significant when accounting for the severity of alcohol use, as well as physical health problems, emotional discharge coping, and interpersonal stress and support at Year 1. In addition, the association between Year 1 impulsivity and 15-year mortality risk was moderated by interpersonal support at Year 1, such that individuals high on impulsivity had a lower mortality risk when peer/friend support was high than when it was low.

Conclusions—The findings highlight impulsivity as a robust and independent predictor of mortality, and suggest the need to consider interactions between personality traits and socio-contextual processes in the prediction of health-related outcomes for individuals with alcohol use disorders.

Keywords

Impulsivity; Mortality; Alcohol Use Disorders; Socio-Contextual Processes

The deleterious effects of alcohol misuse on health and longevity have been well documented (Corrao et al., 2004). Moreover, numerous studies have identified demographic factors, drinking patterns and problems, indicators of physical health, coping styles, and socio-contextual processes that increase the risk of mortality among individuals with alcohol use disorders (AUDs; Finney and Moos, 1992; Holahan et al., 2010; Liskow et al., 2000; Mertens et al., 1996; Timko et al., 2006). In a separate line of research, personality traits

related to impulsivity (e.g., low conscientiousness) have been identified as significant predictors of poor health-related outcomes including mortality (Bogg and Roberts, 2004; Roberts et al., 2007). Although there is a well-established association between disinhibitory traits and AUDs (Labouvie and McGee, 1986; McGue et al., 1999; Sher et al., 2000), to our knowledge, no studies have tested these traits as predictors of mortality among individuals with alcohol-related problems or examined moderation of this effect via socio-contextual processes.

In this study, we examined whether individual differences in impulsivity – a dimension of normal personality and core risk factor for AUDs – independently predict mortality in a mixed-gender sample of individuals who, at baseline, initiated help-seeking for alcohol-related problems and were followed over 16 years. Building on prior research on predictors of mortality in this sample (Timko et al., 2006), we sought to bridge the alcohol and personality literatures by testing (1) whether impulsivity is associated with mortality risk among individuals with alcohol-related problems, (2) the extent to which this association is accounted for by other risk factors that have been linked to premature death in these samples, and (3) whether this effect is moderated by social-contextual processes of support and stress.

Predictors of Mortality Risk among Individuals with Alcohol Use Disorders

Relative to the general population, individuals with AUDs are more likely to die prematurely (Finney et al., 1999; Johnson et al., 2005; Valliant, 1996). Accordingly, several longitudinal studies have aimed to identify the most salient risk factors for mortality in this population (for a review, see Liskow et al., 2000). For example, being male, older, and unmarried increases the risk of premature death among individuals with AUDs, as do more frequent and heavier drinking patterns, drinking problems, and physical health problems (Finney and Moos, 1992; Finney et al., 1999; Greenfield et al., 2002; Holahan et al., 2010; Johnson et al., 2005; Lewis et al., 1995; Liskow et al., 2000; Moos et al., 1994; Smith et al., 1983; Timko et al., 2006; Vaillant, 1996). In addition, more reliance on avoidance coping, less social support, and more stress from interpersonal relationships increase the risk of mortality among individuals with AUDs (Finney and Moos, 1992; Holahan et al., 2010; Mertens et al., 1996; Moos et al., 1990).

Impulsivity and Risk for Mortality: Relevance for Individuals with Alcohol Use Disorders

Despite the litany of variables that have been examined as predictors of mortality among individuals with AUDs, tests of the significance of individual differences in personality are noticeably absent from this literature. In the clinical and health psychology literatures, however, personality traits have long been identified as possible risk factors for mortality (Friedman and Rosenman, 1959), with *low conscientiousness* emerging as one of the most consistent, trait-based predictors of poor health and reduced longevity (Kern and Friedman, 2008; Roberts et al., 2007). Conscientiousness is a broad domain of personality reflecting individual differences in the propensity to control one's impulses, be planful, and adhere to socially-prescribed norms (John et al., 2008). Impulsivity marks the low end of this dimension and reflects the tendency to engage in a pattern of behavior marked by risk-taking, poor self-control, and disregard for future consequences. In non-AUD samples, low conscientiousness is a significant predictor of mortality (Kern and Friedman, 2008; Roberts et al., 2007) – an effect that has been found to hold over seven decades (Friedman et al., 1993, 1995), and been replicated in diverse populations, including medical patients (Christensen et al., 2002; Weiss and Costa, 2005; Wilson et al., 2004) and epidemiological samples (Taylor et al., 2009; Terracciano et al., 2008).

To our knowledge, no studies in this literature have tested impulsivity as an independent predictor of mortality in a sample of individuals with alcohol-related problems. This is a surprising omission, given that impulsivity is a well-established risk factor for alcohol misuse (Elkins et al., 2006; McGue et al., 1999; Sher et al., 2000) and therefore may be an especially potent predictor of mortality among individuals with AUDs. Furthermore, the role of impulsivity as an independent predictor of mortality risk among individuals with AUDs is relevant from the standpoint of the stage of the alcohol recovery process.

Thus, we sought to examine the impulsivity-mortality link at baseline and one year after participants had initiated help-seeking for their alcohol use problems. At baseline, participants were in a state of distress due to their problematic alcohol use, whereas at Year 1 most participants had obtained help for their alcohol-related problems and reduced their drinking (Finney and Moos, 1995). Given prior research on acute clinical states and self-report assessments of personality (e.g., Brown et al., 1991; Peselow et al., 1994; Reich et al., 1987), we hypothesized that individuals' self-reports of impulsivity at Year 1 would be less a reflection of their alcohol problems – and therefore more likely to be independently linked to mortality risk – than their reports at baseline, which may be more closely associated with concurrent alcohol use and problems (i.e., state effects).

Potential Covariates and Moderators of the Association between Impulsivity and Mortality

In non-AUD samples, some attempts have been made to identify covariates that account for the association between impulsivity and mortality. These efforts have primarily focused on health-risk behaviors such as substance use (e.g., alcohol, tobacco, illicit drugs) and indicators of physical health problems (e.g., obesity). Although impulsivity is a robust predictor of these health-related variables (Bogg and Roberts, 2004; Caspi et al., 1997), its relationship with mortality is largely independent of these indices (Friedman et al., 1995; Taylor et al., 2009; Terracciano et al., 2008; Wilson et al., 2004). However, past research on this issue has been limited in several ways. For example, the assessment of alcohol use has typically been limited to patterns of consumption (Friedman et al., 1995; Wilson et al., 2004) rather than indicators of alcohol severity (e.g., problems, dependence), which are known risk factors for mortality (Finney et al., 1999). Thus, a comprehensive assessment of alcohol use may be a more robust covariate of the impulsivity-mortality link.

Similarly, prior investigations of physical health indicators as covariates of this link have used single indicators (e.g., Body/Mass Index; Martin et al., 2007) rather than a broad composite that includes multiple indicators such as number of medical conditions and physical ailments and reports of distress from these conditions (Liskow et al., 2000). Finally, attempts to explain the impulsivity-mortality link in non-AUD samples have neglected to test either avoidant coping strategies marked by health-risk behaviors (e.g., emotional discharge coping) or measures of interpersonal stress and support – variables linked to premature death among individuals with AUDs (Finney and Moos, 1992).

Beyond the issue of potential covariates of the impulsivity-mortality link, few efforts have been made to explore potential moderators of this association. Among the aforementioned risk factors, interpersonal stress and support have the most theoretical support as moderators of the impulsivity-mortality link based on conceptual models that emphasize the importance of interpersonal contexts in associations between personality and health (Magnusson, 1999; Revenson, 1990). In terms of empirical research on the role of interpersonal contexts in the relationship between impulsivity and mortality, there is indirect support for moderation based on evidence that a proxy of high impulsivity (i.e., low emotion regulation; Hinshaw, 2003) predicts higher stress hormone levels only among individuals low on social support (Wirtz et al., 2006). Thus, we targeted interpersonal stress and support (i.e., from spouse/

partner and peers/friends) as potential moderators of the association between impulsivity and mortality.

Present Study

In a mixed-gender sample of individuals who initiated help-seeking for alcohol-related problems at study intake and were followed over 16 years, we investigated the following questions: (1) Is impulsivity associated with mortality risk among individuals with alcohol-related problems, and does the significance of this association vary based on the stage of the alcohol recovery process? (2) Is the association between impulsivity and mortality risk accounted for by other risk factors that have been linked to premature death among individuals with alcohol-related problems? (3) Is the impulsivity-mortality link moderated by the social context? After controlling for demographics, we tested the significance of impulsivity at baseline and Year 1 as predictors of 15-year mortality risk, examined the degree to which this association was explained by relevant covariates (i.e., drinking patterns and problems, physical health problems, emotional discharge coping, interpersonal stress and support), and tested the interpersonal variables as moderators of the impulsivity-mortality link. In previous work with this sample, Timko and colleagues (2006) examined the role of drinking outcomes at Year 1 and duration of help for drinking as predictors of 16-year mortality. The present study expands the focus of Timko et al. (2006) by examining impulsivity as a risk factor for mortality and testing potential covariates and moderators of this relationship.

Methods

Sample and Procedure

Participants included individuals with alcohol-related problems who, at baseline, had not previously received any professional treatment for their problematic use. All individuals had an initial contact with the alcohol treatment system through either an information and referral center or a detoxification program. For individuals who were sufficiently detoxified, informed consent was obtained on-site by staff at these programs in a manner compliant with the local Institutional Review Board. Participants who provided informed consent were then screened to verify their eligibility for the study. A total of 628 individuals were deemed eligible ($N_{\text{Men}} = 332$, $N_{\text{Women}} = 296$) based on the following criteria: (a) no prior history of professional substance use disorder treatment, and (b) an alcohol-related problem as indicated by one or more dependence symptoms, substance use problems, episodes of drinking to intoxication in the past month, and/or the perception that alcohol use is a significant problem in their life. At baseline, these individuals consumed an average of 13.1 ounces of ethanol ($SD = 11.2$) on a typical drinking day, were intoxicated an average of 13.7 days ($SD = 10.8$) in the past month, and reported an average of 3.9 ($SD = 6.8$) symptoms of physical dependence (e.g., 70.6% had blackouts, 70.2% had fevers, and 64.9% had shakes) and 3.8 drinking-related problems ($SD = 6.1$). These individuals were divided almost equally between men (52.9%) and women (47.1%), were primarily Caucasian (81.4%), unmarried (79.0% - i.e., never married, cohabiting, divorced, or widowed), and unemployed (59.6%), and were 34.7 years of age, on average ($SD = 9.4$), with 13.1 years of education ($SD = 2.3$) and an annual income of \$12,225.

At baseline, eligible participants completed an inventory assessing their substance use, physical health, coping strategies, and psychosocial functioning (for more information about the initial data collection process, see Finney and Moos, 1995). At 1 and 16 years after the baseline assessment, participants were contacted by phone and asked to complete an inventory via mail that was largely identical to the baseline inventory. Of the 628 participants at baseline, 82% participated in the Year 1 follow-up assessment ($N = 515$). At

Year 16, 80% of the baseline sample who were not known to have died ($N = 405$) participated in the follow-up assessment at that time. Of the total sample at baseline ($N = 628$), 121 individuals were known to have died (four in the first year and 117 between Years 1 and 16). Further descriptive statistics on mortality in this sample and the procedures used to obtain death records is provided by Timko et al. (2006).

Measures

Impulsivity—Impulsivity was measured at baseline ($\alpha = .74$) and Year 1 ($\alpha = .73$) using the 10-item impulsivity scale from the Differential Personality Inventory (Jackson and Messick, 1971). Items were rated on a 4-point scale (1 = *strongly disagree*, 4 = *strongly agree*) that reflected respondents' level of agreement with statements regarding lack of planning (e.g., "I usually act upon the first thought that comes into my head") and impulsive behavior and risk-taking (e.g., "I believe I act more impulsively than do most people"). Higher scores denote greater self-reported impulsivity. The correlation between impulsivity at baseline and Year 1 was .53 ($p < .001$). The means and standard deviations of impulsivity at baseline ($M = 14.84$, $SD = 4.36$) and Year 1 ($M = 13.14$, $SD = 4.18$) indicate significant ($p < .01$) and modest mean-level change over this time period based on a repeated measures ANOVA ($d = -.38$; see Blonigen et al., 2009).

Alcohol variables—At baseline and Year 1, participants were asked about the *quantity* of alcohol (in ounces) they drank on typical drinking days in the past month, as well as their *pattern* of drinking in the past month, which was reported on a six-point scale (1 = *did not drink at all*, 6 = *occasional drinking binges*). *Drinking problems* at baseline ($\alpha = .80$) and Year 1 ($\alpha = .89$) were assessed with 9 items drawn from the Health and Daily Living Form (Moos et al., 1992) and rated on a 5-point scale (0 = *never*, 4 = *often*). These items were summed to index the frequency with which participants experienced problems due to drinking in the past 6 months (e.g., legal, financial, work). *Alcohol dependence severity* was assessed at baseline ($\alpha = .88$) and Year 1 ($\alpha = .92$) and comprised the sum of 11 items from the Alcohol Dependence Scale (Skinner and Allen, 1982) that measured physical symptoms as a result of drinking in the past 6 months (e.g., shakes when sobering up). These items were rated on a 5-point scale (0 = *never*, 4 = *often*). At both baseline and Year 1, these four alcohol variables were moderately to highly intercorrelated (Baseline: r range = .33 – .65; Year 1: r range = .45 – .78); thus, we constructed an *alcohol composite* at each time point, which represented the average of their standardized scores. Based on the means of the individual indices, a mean score on this composite at baseline corresponded to a pattern of "fairly heavy" drinking during the past month, consumption of approximately 13 ounces of ethanol on a typical drinking day during the past month, and approximately four drinking-related problems and four alcohol-related physical symptoms during the past 6 months. Principal components analyses revealed one large component accounting for 55.4% of the variance across these measures at baseline (range of loadings: .66 – .83) and 65.9% of the variance at Year 1 (range of loadings: .77 – .86).

Physical health variables—At baseline and Year 1, we assessed the number of 13 chronic medical conditions (e.g., cancer, diabetes, high blood pressure) diagnosed by a physician in the past year. In a similar fashion, participants at baseline and Year 1 reported on the number of 13 physical ailments they experienced in the past year (e.g., pain in the heart or tightness in the chest; trouble breathing or shortness of breath; an injury that caused problems). For each medical condition and physical ailment endorsed, participants also rated, on a 5-point scale (0 = *never*, 4 = *often*), the frequency with which they were distressed by these health problems. These variables (number of medical conditions, number of physical ailments, frequency of distress due to medical conditions, frequency of distress due to physical ailments) were moderately to highly intercorrelated (Baseline: r range = .50

– .94; Year 1: r range = .51 – .94); thus, we constructed a composite of *physical health problems* at each time point – i.e., the average of their standardized scores. Based on the means of the individual indices, a mean score on this composite at baseline corresponded to having one chronic medical condition, three physical ailments, and “sometimes” feeling distressed by these health problems. Principal components analyses revealed one large component accounting for 74.6% of the variance across these measures at both baseline and Year 1 (range of loadings: .84 – .88).

Emotional discharge coping—At baseline and Year 1, *emotional discharge coping* was assessed with a 5-item scale adapted from the Coping Responses Inventory (Moos, 1993). Items were rated on a 4-point scale (1 = *no*, 4 = *fairly often*) and reflected an avoidant coping style by which individuals tended to reduce tension (e.g., by smoking, taking tranquilizers, overeating). This scale was included because it is comprised largely of health-risk behaviors that have been linked to impulsivity (Bogg and Roberts, 2004). Although the internal consistency of this scale was lower than optimal (Baseline α = .54; Year 1 α = .56), we included it because of its conceptual importance and positive correlation with drinking problem severity (Finney and Moos, 1995).

Interpersonal stress and support—Selected items, adapted from the Life Stressors and Social Resources (LISRES) Inventory (Moos and Moos, 1994), were used to assess interpersonal stress and support at baseline and Year 1. Stress from spouse/partner (α = .81 at baseline and Year 1) was the sum of 5 items (e.g., spouse disagrees on important issues), and stress from peers/friends (Baseline α = .73; Year 1 α = .67) was the sum of 4 items (e.g., friends get angry or lose their temper with you), each rated on a 5-point scale (0 = *never*, 4 = *often*). Support from spouse/partner (Baseline α = .91; Year 1 α = .92) was the sum of 10 items (e.g., can count on spouse to help you), and support from peers/friends (Baseline α = .88; Year 1 α = .86) was the sum of 6 items (e.g., can confide in your friends), rated on the same 5-point scale. The means (SDs) for these variables at baseline and Year 1, respectively, were as follows: spouse/partner stress [9.75, 8.09 (4.27, 4.08)], peer/friend stress [5.91, 5.41 (2.66, 2.22)], spouse/partner support [27.78, 30.22 (8.71, 8.31)], peer/friend support [16.88, 17.80 (5.03, 4.48)].

Statistical Analyses

To examine predictors of mortality risk, we employed a series of continuous-time survival analyses (i.e., Cox proportional hazard regressions) in *SPSS 17.0*. These analyses consider the length of time to an event and estimate the probability that this event will occur at any given time across the study period. The time interval for the analyses was based on number of months from Year 1 until death. The hazard rates obtained from each time interval, which represent the risk of death during that time interval, given the risk through all prior time intervals, were combined to estimate the hazard function for the sample over the 15-year period. Hazard functions can be interpreted in terms of hazard ratios (HRs), which reflect the change in the probability of an event as a function of a one-unit change in a given predictor. To facilitate comparisons of the HRs, raw scores on continuous predictors (e.g., impulsivity) were transformed to standardized (z) scores so that they were on a common metric. Impulsivity at both baseline and Year 1 met the proportionality assumptions of Cox regressions.

Given our hypothesis about the significance of impulsivity as an independent risk factor at Year 1, the event predicted by our hazard models was mortality from Years 1 to 16 (i.e., the subsequent 15-year mortality risk). Of note, the pattern of results for the hazard model using baseline predictors was essentially the same when the event to be predicted was mortality risk from baseline to Year 16. Thus, the hazard model for the baseline variables was based

on prediction of mortality risk from Years 1 to 16 so that the outcome was comparable to the hazard model for the Year 1 variables. Out of the 117 individuals who were known to have died from Years 1 to 16, 93 participated in the follow-up assessment at Year 1 and could be used to evaluate the significance of the predictors at that time. Information on date of death, gathered via death certificates (see Timko et al., 2006), was available for 90 of these cases. Across the Cox regression models, the number of censored cases (i.e., those who were not yet deceased by the end of the study period [Year 16] and who participated in the follow-up assessment at that time) ranged from 345 – 351, and the number of “missing” cases (i.e., those who were not known to have died by Year 16, but did not return for the Year 16 assessment) ranged from 74 – 82.

To test for bias due to attrition, we examined whether participants who were missing in the hazard models differed from individuals who were included in these analyses on any of the baseline or Year 1 variables. Compared to those who were included in the analyses, missing participants at Year 16 were more likely to be male [$\chi^2(1, 628) = 3.86, p = .05$] and to have slightly greater involvement with alcohol ($d = .23$) and more stress from peers/friends at baseline ($d = .29$), but did not differ significantly on any other baseline variables (range $d = .04 - .18$). For the Year 1 variables, all differences between missing participants at Year 16 and those included in the hazard models were nonsignificant (range $d = -.18 - .12$).

In the first step of our analyses, we examined correlations between impulsivity and other risk factors for mortality at baseline and Year 1 (i.e., alcohol composite, physical health problems, emotional discharge coping, interpersonal stress and support), and partial correlations at these time points between each predictor variable and mortality from Years 1 to 16 [controlling for baseline demographics of age, gender (1=male), marital status (1=unmarried)]. Next, we constructed separate hazard models to examine whether impulsivity at baseline and Year 1 was associated with risk of mortality from Years 1 to 16, after controlling for baseline demographics and other potential covariates. Stress and support from spouse/partner were analyzed in supplemental analyses, given that including these variables substantially reduced the sample size for the hazard models (i.e., only individuals who reported being in a serious romantic relationship provided data on these variables). Finally, we examined whether stress and support from either spouse/partner or peers/friends moderated the association between impulsivity and mortality at either baseline or Year 1.

Among the predictors examined in this study, the interpersonal variables were targeted as potential moderators based on their theoretical support in the personality-disease literature (Revenson, 1990). At baseline and Year 1, the four interaction terms between the interpersonal variables and impulsivity were tested after controlling for significant baseline demographics and the main effects of impulsivity and the interpersonal variable. Significant interactions were followed by tests of conditional moderation in which separate hazard models were run for individuals at high (+ 1 SD above the mean) and low levels (–1 SD below the mean) of the moderator (Holmbeck, 2002).

Results

Cause of Death between Years 1 to 16 and Relationship with Impulsivity

Out of the 90 individuals at Year 1 with information on date of death, information on cause of death was available for 83 cases. Cause of death was related to alcohol use in 46 cases (Timko et al., 2006). Individuals who died from alcohol-related versus non-alcohol-related causes did not differ on impulsivity at either baseline [$F(1, 82) = 0.56, p = .46$] or Year 1 [$F(1, 82) = 0.35, p = .56$]. In 11 cases, cause of death was related to violent or accidental means (e.g., assault, suicide, car accident). Scores on impulsivity at baseline [$F(1, 82) =$

1.04, $p = .31$] and Year 1 [$F(1, 82) = 0.44, p = .51$] were not significantly higher for these 11 individuals than for individuals who died from other causes.

Intercorrelations among Predictor Variables at Baseline and Year 1, and Partial Correlations with Mortality from Years 1 to 16

Table 1 provides intercorrelations among the predictor variables at baseline and Year 1, as well as point-biserial correlations between each of these continuous predictors and the dichotomous outcome of mortality (correlations at baseline and Year 1 are presented above and below the diagonal, respectively). Correlations with mortality represent partial correlations after controlling for baseline demographics of age, gender, and marital status. Impulsivity was significantly correlated with all predictor variables at baseline and Year 1. Impulsivity at baseline and impulsivity at Year 1 were both significantly correlated with mortality at Year 16. Most of the intercorrelations among the other predictor variables at baseline and Year 1 were significant and modest in magnitude, with the exception of moderate to large correlations between the alcohol composite and emotional discharge coping, and between spouse/partner variables of stress and support at each time point. All other predictor variables at baseline and Year 1 were significantly related to mortality except for spouse/partner support at baseline and peer/friend stress and support at Year 1.

15-Year Mortality Risk from Impulsivity at Baseline, Controlling for Demographics and Other Predictors at Baseline

Table 2 provides the results of a hazard model predicting 15-year mortality risk from impulsivity at baseline, controlling for baseline demographics, and examining the degree to which other predictors at baseline (i.e., alcohol composite, physical health problems, emotional discharge coping, peer/friend stress and support) can account for this relationship. In Block 1 of the model, being older, male, unmarried, and high on impulsivity independently predicted a higher risk of mortality (HR range = 1.38 to 3.36, $ps < .01$). Regarding impulsivity, for every 1 SD increase in this variable at baseline, there was a 38% increase in the risk of mortality across the 15-year period. However, after entering the additional predictor variables into the model in Block 2, the effect of baseline impulsivity was reduced to non-significance and was largely accounted for by the significant effect of the alcohol composite. With the exception of the baseline demographics, no other baseline predictors were significant in this block of the model. The finding of the alcohol composite accounting for the effect of baseline impulsivity on mortality was confirmed in a subsidiary analysis in which this composite, when entered by itself in Block 2 of this model, was significant (HR = 1.69, $p < .01$), whereas baseline impulsivity was not significant (HR = 1.13, $p = .33$).

In a supplemental analysis, we tested the preceding hazard model (i.e., controlling for baseline demographics, alcohol composite, physical health problems, emotional discharge coping, peer/friend support and stress) and included variables of stress and support from spouse/partner at baseline. Among individuals who reported being in a serious romantic relationship at baseline and provided data on these variables ($n = 280$), neither stress (HR = 1.27, $p = .30$) nor support (HR = 1.16, $p = .49$) from a spouse/partner was a significant predictor of 15-year mortality risk. Moreover, the pattern of results was the same as the model in Table 2 – i.e., all baseline demographics (HR range = 2.34 to 2.93, $ps < .01$) as well as the alcohol composite were significant in Block 2 (HR = 1.73, $p < .05$); baseline impulsivity was nonsignificant after accounting for the baseline alcohol composite (HR = 1.10, $p = .56$).

15-Year Mortality Risk from Impulsivity at Year 1, Controlling for Baseline Demographics and Other Predictors at Year 1

Table 3 provides the results of a model predicting 15-year mortality risk from impulsivity at Year 1, and the degree to which the other predictors of mortality risk at Year 1 (i.e., alcohol composite, physical health problems, emotional discharge coping, peer/friend stress and support) can account for this relationship. For this model, impulsivity at Year 1 and the baseline demographics of age, gender, and marital status were entered into the first block, followed by entry of the potential covariates in the second block. In Block 1, all demographics were significant predictors of 15-year mortality risk, as was impulsivity at Year 1. Specifically, for every 1 SD increase in impulsivity at Year 1, there was a 42% increase in the risk of mortality across the subsequent 15 years. In Block 2, after entering the other predictor variables into the model, the effect of impulsivity remained significant and largely unchanged. Among the other variables entered at this block, the alcohol composite was the only significant predictor of mortality risk.

As a supplemental analysis, we tested the preceding hazard model (i.e., controlling for baseline demographics, alcohol composite, physical health problems, emotional discharge coping, peer/friend support and stress) and included variables of stress and support from spouse/partner at Year 1. Among individuals who reported being in a serious romantic relationship at Year 1 and provided data on these variables ($n = 260$), spouse/partner stress was not a significant predictor of mortality risk ($HR = 0.97, p = .85$); however, spouse/partner support was significant ($HR = 0.61, p < .01$). The remaining pattern of results was comparable to the model presented in Table 3 – i.e., Year 1 impulsivity was a significant predictor of mortality risk in both the first block of the model, which controlled for baseline demographics ($HR = 1.81, p < .001$), as well as the second block, which controlled for baseline demographics and the other Year 1 predictor variables ($HR = 1.73, p < .01$).

To address the possibility of bias in the Year 1 predictors, we compared non-participants and participants at Year 1 on their baseline scores for these predictor variables. Non-participants and participants at Year 1 did not differ significantly on baseline scores for impulsivity, the physical health composite, or emotional discharge coping (range $d = .06 - .19$). These two groups did differ significantly on the alcohol composite and the stress and support variables for spouse/partner ($ps < .05$), with non-participants demonstrating greater severity on these variables at baseline (range $d = .26 - .38$). However, when baseline scores for these particular variables were substituted for Year 1 scores in the hazard model in Table 3, Year 1 impulsivity remained significant (HR range = $1.32 - 1.37, ps < .05$).

As a supplemental analysis, we examined whether change in impulsivity from baseline to Year 1 was associated with mortality risk from Years 1 to 16. Change scores were computed by subtracting scores at Year 1 from baseline ($\text{Impulsivity}_{\text{Baseline}} - \text{Impulsivity}_{\text{Year1}}$) such that high scores indicated a greater decline in impulsivity over this time period. For observational studies such as the present, change scores are preferable to an ANCOVA, given that the latter measures change after removing variability in impulsivity scores at baseline that occurred naturally due to selection factors (Fitzmaurice et al., 2004; Miller & Chapman, 2001). Controlling for age, gender, and marital status, decreases in impulsivity were not significantly related to 15-year mortality risk ($HR = 0.98, ns$).

Interactions between Impulsivity and Interpersonal Stress and Support at Baseline and Year 1

Four interaction terms were constructed from the standardized variables to examine, separately at baseline [spouse/partner variables ($n = 280$), peer/friend variables ($n = 426$)] and Year 1 [spouse/partner variables ($n = 260$), peer/friend variables ($n = 429$)], whether any

of the interpersonal variables of stress and support moderated the impact of impulsivity on 15-year mortality risk. At baseline, none of the four interaction terms were significant. At Year 1, only the interaction between impulsivity and peer/friend support attained significance ($HR = 0.82, p < .01$). This interaction did not appear to be spurious as it remained significant in a supplemental analysis in which the peer/friend support variable was log-transformed to increase normality ($skew = -.85$).

Consistent with our conceptualization of impulsivity as the focal predictor and peer/friend support as the moderator, we conducted separate hazard regressions using conditional moderators for individuals “high” (+1 SD above the mean) and “low” (−1 SD below the mean) on peer/friend support at Year 1. Using this approach, a significant increase in mortality risk was observed for individuals who were low ($HR = 1.74, p < .001$) but not high ($HR = 1.18, p = .25$) on peer/friend support. In other words, the deleterious effect of Year 1 impulsivity was reduced (or buffered) as peer/friend support increased. To facilitate interpretation of this effect, we plotted the effect of Year 1 impulsivity separately for those high (+1SD) and low (−1SD) on this variable, with the effect for those high on impulsivity plotted separately at high (+1SD) and low (−1SD) levels of peer/friend support. As shown in Figure 1, among individuals high on impulsivity, those high on peer/friend support ($n = 15$) exhibited a better 15-year survival probability than individuals low on peer/friend support ($n = 22$).

Discussion

The current investigation used a mixed-gender sample of individuals with alcohol-related problems who initiated help-seeking at the start of the study and were followed over 16 years, to examine the impulsivity-mortality link at different stages in the alcohol recovery process, the extent to which this relationship was accounted for by other known predictors of mortality in AUD samples, and whether this relationship was moderated by the social context. After controlling for demographic factors, impulsivity at baseline was a significant predictor of mortality risk from Years 1 to 16; however, this effect was accounted for by the severity of alcohol use at baseline. In contrast, impulsivity at Year 1 was associated with an increased risk of mortality over the subsequent 15 years and this association was not explained by either baseline demographics or other risk factors at Year 1 that have been linked to premature death in AUD samples. In addition, a significant interaction was observed between impulsivity and peer/friend support at Year 1, which suggested that, among individuals high on impulsivity, the mortality risk may be reduced for those high on support from peers/friends. Collectively, these findings highlight impulsivity as an independent risk factor for mortality in AUD samples, and set the stage for more in-depth, theory-driven research on personality variables as predictors of mortality among individuals with AUDs, and the potential moderation of these effects via the social context.

Implications for Individuals Seeking Help for Alcohol Use Disorders

The present study is unique in that it consisted of individuals who had just initiated help-seeking for their alcohol problems and followed these individuals over time, which allowed us to investigate predictors of mortality at two points in the alcohol recovery process. The findings suggest that knowledge of individuals’ impulsive tendencies when they are in a state of crisis and seeking help for drinking may not independently predict their risk for mortality after accounting for their severity of alcohol use at that time. In contrast, knowledge of individuals’ impulsive tendencies during other phases of the alcohol treatment system may foreshadow their risk for mortality over the next 15 years, independent of their alcohol use, physical health status, coping styles, and interpersonal resources at that time.

The amount of variability in impulsivity at baseline and Year 1 was comparable and thus cannot explain the differences in the significance of the impulsivity-mortality link over this timeframe. One possible explanation of this difference is that, at baseline (a time when all participants were seeking help for their alcohol problems), issues with drinking may have been such a potent predictor of mortality risk such that any additional risk from other factors (after accounting for severity of alcohol use) was negligible. In contrast, at Year 1 the majority of participants had reduced their drinking (Finney and Moos, 1995; Timko et al., 2000); thus, the significance of other risk factors of mortality was able to emerge. It is also conceivable that, given participants were in a state of crisis at baseline, their reports of their impulsive tendencies at that time partly captured “state” effects (e.g., psychiatric distress from concurrent substance use; withdrawal symptoms) and therefore were less an indication of their typical or “characterological” pattern of impulsivity, independent of alcohol use. However, at Year 1, most participants had reduced their drinking and were not in a state of crisis; thus, their reports at that time may have been a better reflection of their “trait-like” pattern of impulsivity, which in turn may be a more robust independent predictor of long-term outcomes such as mortality. Accordingly, future studies that seek to test impulsivity as an independent predictor of mortality among individuals with AUDs should consider the stage of the alcohol recovery process.

What Accounts for the Association between Impulsivity and Mortality among Individuals with Alcohol Use Disorders?

A key question raised by the present findings is how an impulsive disposition increases risk for mortality among individuals with AUDs. Health-risk behaviors, such as alcohol consumption, have been posited as a potential factor in this relationship (Bogg and Roberts, 2004; Friedman et al., 1993; Friedman, 2000); however, these behaviors have failed to account for the impulsivity-mortality link in prior research with non-AUD samples (Friedman et al., 1995; Taylor et al., 2009; Terracciano et al., 2008; Weiss and Costa, 2005). One advantage of investigating this issue in the present sample was the availability of multiple indicators of alcohol use, as well as information on alcohol-related causes of death. Although there was a significant association between the alcohol composite and mortality at both baseline and Year 1, this composite was only able to account for the impulsivity-mortality link at baseline, and impulsivity scores at either time point did not differ between individuals who died from alcohol- versus non-alcohol-related causes.

These findings notwithstanding, the current design cannot identify causal mechanisms in the link between impulsivity and mortality, given that the covariates were measured contemporaneously with impulsivity. Nonetheless, it is worth speculating about potential mechanisms that should be explored in future designs with multiple assessments that can disentangle the pathway from impulsivity to mortality in AUD samples. Notably, impulsivity is linked to a wide range of health-risk behaviors beyond excessive alcohol use (e.g., violent crime, risky driving and sexual practices, illicit drug abuse; Caspi et al., 1997; Chalmers et al., 1990). Among individuals with AUDs, illicit drug abuse may be a strong candidate as an explanation of the association between impulsivity and mortality (but see Liskow et al., 2000; Moos et al., 1994). Alcohol and drug abuse are highly comorbid and may be conceptualized as different manifestations of a broad externalizing vulnerability (Krueger et al., 2002). Moreover, McGue et al. (1999) reported that, relative to individuals without a substance use disorder, elevated levels of behavioral disinhibition among individuals with an AUD are largely attributable to a subset of individuals who abuse other drugs.

Beyond health-risk behaviors, we examined several other risk factors that have been linked to mortality among individuals with AUDs (i.e., physical health problems, emotional discharge coping, interpersonal stress and support). In particular, testing the coping and

interpersonal variables as potential covariates of the impulsivity-mortality link is a unique contribution of the present study that has not been explored in prior research. Among these additional risk factors, only spouse/partner support at Year 1 was a significant predictor of 15-year mortality risk, and it accounted for only minimal variance in the association between impulsivity and mortality. Nevertheless, we encourage future research with AUD samples to explore these risk factors as potential mechanisms in the pathway from impulsivity to mortality.

Moderation of the Impulsivity-Mortality Link via the Social Context

The results of the moderator analyses suggest that the effects of impulsivity on mortality may become manifest through interactions between traits and socio-contextual process (Friedman, 2000). That is, the dire effects of impulsivity on risk for mortality may not reach fruition for individuals who are able to maintain a strong peer support network. Conceivably, by virtue of their strong bond with a high-risk individual, such peers may have sufficient leverage to discourage expression of the individual's impulsive tendencies and encourage consideration of the long-term consequences of his/her actions. Such a perspective is consistent with evidence from the AUD treatment-outcome literature that social support networks are a key mechanism by which Alcoholics Anonymous (AA) and other psychosocial treatments can improve long-term drinking-related outcomes (Humphreys and Noke, 1997; Kaskutas et al., 2002).

Furthermore, from the standpoint of treatment, the present findings suggest that interventions for AUDs may benefit from an ecological perspective that considers the contexts in which dispositional tendencies, such as impulsivity, become expressed in individuals' everyday lives. Notably, based on prior work with this sample, longer duration in AA and alcohol treatment was associated with a decline in impulsivity (Blonigen et al., 2009). In combination with the present findings, it appears that formal and informal help for AUDs may include "active ingredients" that can help curtail expression of impulsive tendencies (e.g., social integration, peer bonding; Moos, 2007, 2008) and buffer the otherwise deleterious impact of such tendencies on health and longevity. These issues aside, the interaction between impulsivity and peer/friend support in the prediction of mortality risk should be interpreted with caution until the effect can be replicated in an independent sample.

Limitations and Future Directions

Some limitations of the present work should be acknowledged. First, our assessment of health-risk behaviors, although comprehensive with regard to alcohol use, provided minimal assessment of other key health-risk behaviors. For example, the assessment of smoking, which is an independent predictor of mortality among individuals with AUDs (Hurt et al., 1996), was limited to a single item on the emotional discharge coping scale. Moreover, smoking has been found to partially account for the conscientiousness-mortality link in prior work with non-AUD samples (Friedman et al., 1995; Taylor et al., 2009; Terracciano et al., 2008). These issues underscore the need to assess multiple substances and consider both patterns of consumption and indicators of abuse/dependence to properly evaluate the significance of health-risk behaviors as explanatory mechanisms in the association between impulsivity and mortality.

Second, our assessment of risk factors was based exclusively on self-reports. A multi-method approach including experience sampling (e.g., ecological momentary assessments) and objective laboratory measures and biomarker data, as well as reports from peers and family members, may provide a better estimate of the extent to which diverse risk factors account for the relationship between impulsivity and mortality. In particular, reports from

other members of individuals' social networks may help clarify the role of psychosocial processes in the link between impulsivity and mortality, and the degree to which such factors work in concert with health-risk behaviors to increase risk for premature death among individuals with AUDs.

Third, the assessment of impulsivity was based on a relatively brief self-report scale from an established inventory rather than an omnibus measure based on structural models of personality (John et al., 2008). Future work on personality and mortality in AUD samples would benefit from use of contemporary measures of personality (e.g., Costa and McCrae, 1992), as well as examination of other trait-based risk factors of mortality (e.g., neuroticism; Roberts et al., 2007). Furthermore, impulsivity (i.e., self-control) is only one facet of the broader personality domain of conscientiousness. The findings cannot speak to whether other facets in this domain (e.g., responsibility, traditionalism) also predict mortality risk among individuals with AUDs.

The present study helps to bridge the alcohol and personality literatures by extending previous findings on the relationship between impulsivity and mortality to a large sample of individuals who initiated help-seeking for alcohol-related problems. The findings highlight impulsivity as a robust and independent predictor of mortality in this high-risk population, and suggest that the significance of this association may vary based on the stage of the alcohol recovery process. Ultimately, identifying causal mechanisms of the link between impulsivity and mortality will require that potential risk factors be measured at multiple points over time in order to understand the process that unfolds in the course of recovery from alcohol abuse. Furthermore, no single mechanism or process is likely to fully account for the impulsivity-mortality link. Thus, disentangling this relationship calls for measurement of the diverse array of health-risk behaviors associated with impulsivity (Caspi et al., 1997), and theory-driven studies of interactions between dispositional tendencies and socio-contextual processes that may moderate the risk of mortality among individuals with AUDs.

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Figure 1.
Predicted Probability of Survival by Levels of Impulsivity and Peer/Friend Support

Table 1

Intercorrelations among Predictor Variables at Baseline and Year 1, and Partial Correlations with Mortality from Years 1 to 16.

	1	2	3	4	5	6	7	8	9
1. Impulsivity	--	.37***	.18***	.40***	.21***	-.19***	.15*	-.21***	.14**
2. Alcohol Composite	.28***	--	.34***	.45***	.25***	-.26***	.32***	-.30***	.22***
3. Physical Health Problems	.17***	.25***	--	.28***	.17***	-.07	.20***	-.18**	.10*
4. Emotional Discharge Coping	.31***	.50***	.29***	--	.30***	-.18***	.26***	-.28***	.16***
5. Peer/Friend Stress	.15**	.23***	.09†	.27***	--	-.32***	.23***	-.15**	.15**
6. Peer/Friend Support	-.26***	-.31***	-.15***	-.28***	-.34***	--	-.12*	.19**	-.09†
7. Spouse/Partner Stress	.17**	.16**	.16**	.25***	.22***	-.18**	--	-.68***	.13*
8. Spouse/Partner Support	-.17***	-.12*	-.07	-.22***	-.17**	.38***	-.63***	--	-.09
9. Mortality	.15**	.20***	.13**	.14**	.08	-.04	.13*	-.18**	--

Notes. Above the diagonal are correlations at baseline ($Ns = 280 - 426$); below the diagonal are correlations at Year 1 ($Ns = 260 - 429$). Correlations with mortality represent partial correlations controlling for baseline demographics of age, gender, and marital status. Mortality is a dichotomous variable from Year 1 to Year 16 (1 = died).

†
 $p < .10$,

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$.

Table 2

Cox Proportional Hazard Model Predicting 15-Year Mortality Risk from Impulsivity at Baseline, Controlling for Demographics and Other Predictors at Baseline.

Predictor	Beta (SE)	Hazard Ratios (95% CI)
Block 1:		
Age	0.84 (0.10) ***	2.31 (1.89–2.82)
Gender (1=male)	0.70 (0.23) **	2.01 (1.28–3.16)
Marital Status (1=unmarried)	1.21 (0.36) ***	3.36 (1.66–6.80)
Impulsivity (Baseline)	0.32 (0.12) **	1.38 (1.10–1.74)
Block 2 (controlling for Block 1 demographics):		
Impulsivity (Baseline)	0.15 (0.13)	1.16 (0.90–1.50)
Alcohol Composite	0.39 (0.18) *	1.48 (1.03–2.12)
Physical Health Problems	−0.07 (0.13)	0.93 (0.72–1.20)
Emotional Discharge Coping	0.24 (0.15)	1.27 (0.94–1.70)
Peer/Friend Stress	0.17 (0.13)	1.19 (0.92–1.53)
Peer/Friend Support	0.11 (0.13)	1.11 (0.87–1.43)

Notes. $N = 426$,

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$.

Beta = standardized regression coefficients and standard errors (SE) from the Cox regression models. All predictors were entered together in each block of the model. All variables were measured at baseline.

Table 3

Cox Proportional Hazard Models Predicting 15-Year Mortality Risk from Impulsivity at Year 1, Controlling for Baseline Demographics and Other Predictors at Year 1.

Predictor	Beta (SE)	Hazard Ratios (95% CI)
Block 1:		
Age	0.86 (0.10) ***	2.35 (1.93–2.87)
Gender (1=male)	0.66 (0.22) **	1.93 (1.24–3.01)
Marital Status (1=unmarried)	1.30 (0.36) ***	3.67 (1.82–7.40)
Impulsivity (Year 1)	0.35 (0.11) **	1.42 (1.14–1.78)
Block 2 (controlling for Block 1 demographics):		
Impulsivity (Year 1)	0.30 (0.12) *	1.35 (1.06–1.72)
Alcohol Composite	0.29 (0.14) *	1.34 (1.02–1.77)
Physical Health Problems	0.16 (0.10)	1.17 (0.96–1.43)
Emotional Discharge Coping	0.03 (0.13)	1.03 (0.81–1.32)
Peer/Friend Stress	0.21 (0.13)	1.23 (0.96–1.59)
Peer/Friend Support	0.09 (0.11)	1.10 (0.88–1.37)

Notes. $N = 429$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Beta = standardized regression coefficients and standard errors (SE) from the Cox regression models. All predictors were entered together in each block of the model. Demographics were measured at baseline. All other variables were measured at Year 1.