RESEARCH ARTICLE

International Journal of Geriatric Psychiatry

The relationship between moderate alcohol consumption, depressive symptomatology, and C-reactive protein: the Health and Retirement Study

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Objective: Moderate alcohol use has been broadly associated with health benefits among older adults, including improved mood. Aims of this study were to evaluate the relationship of moderate alcohol use and depressive symptomatology over a period of eight years, and to examine inflammation, indicated by C-reactive protein (CRP), as one mechanism by which this relationship functions.

Methods: The study included 3177 community-dwelling participants over the age of 65 in 2008 drawn from the Health and Retirement Study. Data from the 2006, 2008, 2012, and 2014 waves were used. Alcohol use was measured via self-report and was dichotomized as abstinent (0 drinks per week) and moderate (1–14 drinks per week). Inflammation was measured using CRP, which was collected using an enzyme-linked immunosorbent assay and provided in units of μg/mL. Control variables included gender, age, body mass index (BMI), and medical burden.

Results: A latent growth curve model with full information maximum likelihood was used, with results revealing that moderate drinkers endorsed fewer depressive symptoms at baseline and a steeper rate of change over time. Abstinent respondents' depressive symptomatology was characterized by a more linear change rate. Further, moderate drinkers had lower CRP levels suggesting that inflammation partially mediates the relationship between moderate alcohol use and depressive symptomatology.

Conclusions: Moderate alcohol use predicts fewer depressive symptoms among older adults. This relationship is partially moderated by CRP and is eroded by the passage of time. Future research should identify additional mechanisms relating alcohol to positive health outcomes and less depressive symptomatology. Copyright © 2017 John Wiley & Sons, Ltd.

Key words: depression; mood; later life; inflammation; longitudinal
History: Received 21 November 2016; Accepted 02 May 2017; Published online 14 June 2017 in Wiley Online Library (wileyonlinelibrary.com)
DOI: 10.1002/gps.4746

Introduction

Community studies estimate the base rate of major depressive disorder at 4% among older adults, and up to 16% of older adults experience clinically significant depressive symptomatology (Blazer, 2003). Depression in older adults is associated with negative prognostic, health, and functional implications (Lichtenberg *et al.*, 1993; Bruce *et al.*, 1994; Yochim *et al.*, 2003; Paulson *et al.*, 2013). Given the burgeoning population of older adults in the United States (Colby and Ortman, 2015), identifying and understanding risk and protective factors of depression are crucial to informing both prevention and intervention efforts. While past findings have linked excessive alcohol to mood disorders (Grant and Harford, 1995; Devanand, 2002; Kirchner *et al.*, 2007), moderate alcohol use has been associated with reduced depressive symptomatology (Goldberg *et al.*, 1999; Lang *et al.*, 2007). Mechanisms by which moderate alcohol use relates to reduced depressive symptomatology remain unknown; however,

regulation of inflammation has been suggested as one possible mechanism for other health benefits associated with moderate alcohol use (Mukamal *et al.,* 2004; Puts *et al.,* 2005; Shah and Paulson, 2016).

Alcohol and depression

Studies employing heterogeneous methods converge on findings that moderate alcohol use relates to improved subjective well-being and psychosocial functioning, mood enhancement, stress reduction, sociability, and mental health broadly defined (Peele and Brodsky, 2000). Similar to the relationships between alcohol and frailty (Strawbridge *et al.,* 1998; Woods *et al.,* 2005) and alcohol and cognition (Huang *et al.,* 2002; Mukamal *et al.,* 2003; Heuberger, 2009; Moussa *et al.,* 2014), the relationship between alcohol use and depressive symptomatology is characterized as J- or U-shaped (Lang *et al.,* 2007) and can be methodologically identified as a quadratic relationship. For example, in a sample of older Australian men (Coulson *et al.,* 2014), the odds ratio for major depressive disorder was greater for those who abstained from alcohol and for those who drank three or more drinks per day who consumed less than two drinks per day had the lowest odds ratio for major depressive disorder (Coulson *et al.,* 2014). A recent study (Brennan *et al.,* 2016) using HRS data reported that baseline alcohol use predicted membership in depression symptomatology classes over 10 years. These authors suggested medical and social history variables as drivers of class inclusion but did not examine the role of concurrent biomarkers.

Alcohol and inflammation

Little is known about the mechanisms of these effects, although inflammation, typically measured using C-reactive protein (CRP), has emerged as one possible mechanism by which health benefits are conferred (Albert, 2003; Mukamal *et al.,* 2004; Shah and Paulson, 2016). C-reactive protein is an acute phase reactive protein that is implicated in multiple diseases associated with disability and mortality in older adults (Harris *et al.,* 1999; Kiecolt-Glaser *et al.,* 2002). C-reactive protein has been identified as a sensitive biomarker of inflammation, as it is generally resistant to changes conferred by most pathologies and medications (Hirschfield and Pepys, 2003).

Several studies have reported a relationship between alcohol consumption and inflammation as measured by CRP. Results of a population-based survey were that alcohol consumption among adults is associated with lower levels of CRP (Stewart *et al.,* 2002). By comparison to abstention, moderate alcohol consumption remained associated with lower CRP levels even after controlling for health and lifestyle variables including smoking, diabetes, cholesterol, and sex (Albert, 2003). Additionally, in a study of adults between the ages of 70 and 79 years, a J-shaped association was found between weekly alcohol use and CRP levels (Volpato *et al.,* 2004), suggesting that those who consumed alcohol moderately had the lowest levels of CRP as compared to those who abstained or drank in excess. Other work has examined the directionality of this relationship. By employing a cross-over design with randomized assignment and a wash-out period to minimize carry-over effects, Sierksma *et al.* (2002) found that moderate alcohol consumption, as compared to abstention, significantly decreased CRP levels among middle-aged participants. Collectively, these findings suggest an anti-inflammatory mechanism through which moderate alcohol consumption may confer beneficial health outcomes.

Inflammation and depression

Research on the relationship between depression and inflammation has produced variable findings (Kuo *et al.,* 2005). Utilizing the National Health and Nutrition Examination Survey data from 2005 to 2010 and a sample size of over 12,000 men and women 18 years or older, Liu *et al.* (2014) found that the odds ratio of depression increased as CRP levels increased for men. For women, however, this association diminished when controlling for body mass index (BMI). Study using a specified sample of US Army personnel, depressive symptomatology was weakly correlated with CRP levels, and the significance of effect was diminished when controlling for BMI (Douglas *et al.,* 2004). These authors identified both the distinct sample and heavily skewed range of depressive symptomology scores (Mean = 3, SD = 2 on a range of 0–26 on the Patient Health Questionnaire-9) as limitations to the validity of their results. A recent meta-analysis found a significant relationship between depression and CRP among both community and clinical samples (Howren *et al.,* 2009). In addition, when adjusting for BMI, the relationships were weakened but remained significant; this suggests that BMI is an important control variable.

The primary goals of this study were to (a) examine the relationship between moderate alcohol use and

depressive symptoms over a period of 8 years, and (b) to examine the hypothesis that CRP partially mediates the relationships between moderate alcohol use and both endorsement of depressive symptoms at baseline and the rate of change in endorsement of depressive symptoms over time. This study utilizes a large, demographically representative sample, and a longitudinal design that is robust to mortality- and variables individually predicted.

Method

Participants

This study utilized the Health and Retirement Study (HRS)—a cohort study on health and aging on adults 50 and older conducted by the University of Michigan with support from the National Institute of Aging. Information on HRS design and collection methods can be found in published reports (Hauser and Willis, 2004).

The complete HRS data set includes 37,319 participants; this study uses HRS data from the 2006, 2008, 2010, 2012, and 2014 waves. The first exclusionary criterion, being below the age of 65 at the 2006 wave, reduced the sample size to 11,349 participants. The following additional exclusionary criteria were applied sequentially to the remaining participants: (a) reported drinking more than four drinks in a sitting, so as to exclude those who, through occasional binge-drinking may be mistaken for moderate drinkers based on their average daily consumption of alcohol; (b) missing CRP values at the 2006 wave or CRP values above 10 μg/mL as CRP values exceeding 10 μg/mL suggest a possibility of an acute phase response (Ridker, 2003); and (c) identified as heavy drinkers (i.e., those consuming more than 14 drinks per week). Alcohol use was dichotomized as abstinent (0 drinks per week) and moderate (1–14 drinks per week). This method allowed us to better elucidate the mechanism by which moderate alcohol has a protective effect by comparing moderate drinkers to non-drinkers. The final sample consisted of 3177 participants.

Measures

Alcohol use. Alcohol use, measured by average number of drinks per week, was collected via self-report. Consistent with the Dietary Guidelines for Americans 2010 (US Department of Agriculture and US Department of Health and Human Services, 2010), moderate drinking was characterized by 1–14 drinks per week. Respondents who reported 0 drinks per week were identified as abstinent.

C-reactive protein (CRP). C-reactive protein was collected through an enzyme-linked immunosorbent assay (ELISA) using dried blood spot (DBS) (Crimmins *et al.*, 2013). The assays were done at the University of Vermont and provided in units of μg/mL. The within-assay imprecision is 8.1%, and between-assay imprecision is 11.0% (Crimmins *et al.*, 2013).

Outcome variable

Depressive symptomatology. Depressive symptomatology was measured using the abridged 8-item Center for Epidemiological Studies-Depression (CES-D) measure from the HRS data (Radloff, 1977). Participants answered "yes" or "no" to each item-statement with respect to how they were feeling "much of the time" in the past week. Six of the statements were worded negatively ("felt depressed, felt that everything he/she did was an effort, sleep was restless, could not get going, felt lonely, and felt sad"), and two of the statements were worded positively ("enjoyed life and was happy"). Scores range on a scale from 0 to 8, with higher scores suggesting higher levels of depression.

Control variables

Participant's *gender*, *age*, and *body mass index* were controlled for. Participant's *medical burden* was controlled for as these diseases increase levels of stress and inflammation, which thereby increase levels of CRP (Kiecolt-Glaser *et al.*, 2002). Medical burden was assessed by summing the number of endorsed comorbidities: hypertension, diabetes, cardiac disease, arthritis, pulmonary disorder, and cancer.

Auxiliary variables

Auxiliary variables can be included in models such as slope-intercept models (described later) to improve parameter estimates despite missing data. Activities of daily living (ADLs) were assessed by asking participants whether they required any degree of assistance with the following: walking across a room, getting in and out of bed, dressing, bathing, and eating. Scores on this ADL measure ranged from 0 to 5. Instrumental activities of daily living (IADLs) employed a similar methodology, and items assessed

need for assistance using the telephone, taking medication, and handling money. Scores on this IADL measure ranged from 0 to 3. Cumulatively, these variables accurately identified 66% of the attrited participants, and all three variables individually predicted attrition ($p \leq .001$).

Statistical methods

Like other longitudinal community samples of older adults, the HRS data have a relatively high rate of attrition, which largely reflects mortality and morbidity, both of which are characteristic of aging demographic groups. Listwise deletion of attrited participants results in systematic underrepresentation of those with the greatest disease burden and, typically, with the fewest resources (Laughton *et al.,* 1958; Adler *et al.,* 1994). Thus, the present study employed a latent growth curve (LGC) modeling approach specifically designed to account for missing data (Enders, 2010). This strategy was developed using past work employing maximum likelihood estimation (McArdle and Hamagami, 1991) and auxiliary variables for parameter estimation (Graham, 2003). Specifically, primary analyses in this study were completed using the full information maximum likelihood (FIML; Arbuckle, 1996) framework with informative covariates. Auxiliary variables—ADLs, IADLs, and self-rated health change—that strongly predicted attrition were selected so as to improve parameter estimation (Little and Rubin, 2002; Graham, 2003). Thus, data from all 3177 participants with complete data on exogenous variables were used, regardless of number of repeated measures of depression provided. To handle deviations from normality in the dependent variable, robust maximum likelihood method of estimation was used for all LGC models described in this article (Muthen and Muthen, 2007b). Data were prepared in SPSS V23 (IBM Corp, 2015), and latent growth models were prepared using the *MPlus* (Muthen and Muthen, 2007a).

Planned analyses employed Level 1, Level 2 latent growth models, and an adapted mediation analysis in which latent factors reflecting rate of change serve as outcome variables. First, an unconditional latent growth model (Raykov and Marcoulides, 2008) was estimated. This Level 1 model was employed to establish a defensible strategy for modeling change in endorsement of depressive symptomatology over time and included no predictor variables. The initial model included parameters reflecting intercept and linear slope terms. This model was compared against a similar model including a quadratic term, which enables modeling of non-linear slope in depressive symptomatology over time.

A conditional Level 2 model was then tested. As with the prior model, primary endogenous variables included

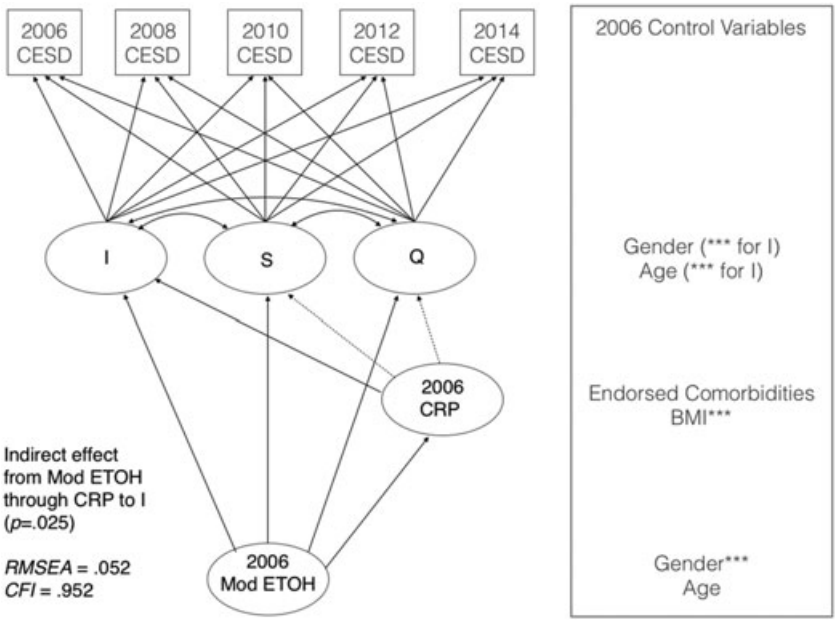

Figure 1 Results of the structural equation model used to examine longitudinal relationship between moderate alcohol use, CRP, and depressive symptomatology. *Solid and broken arrows indicate statistically significant ($p < .05$) and non-significant pathway coefficients, respectively (see* Table 2*). CESD = Center for Epidemiological Studies-Depression; CRP = C-reactive protein; Mod ETOH = moderate alcohol use; I = intercept; S = linear slope; Q = quadratic slope.*

the three latent variables representing depressive symptomatology at baseline, and the linear slope in depressive symptomatology, and the quadratic slope in depressive symptomatology over 8 years. Direct effects were estimated for exogenous variables including moderate user status (dichotomous – moderate drinker versus abstinent), CRP level (continuous), gender (dichotomous), and age (continuous). Finally, mediation was examined by adapting the classic mediation model (Baron and Kenny, 1986) in which significant partial mediation is indicated by (a) significant pathway coefficients relating primary predictors of interest (moderate alcohol use and CRP level) with the criterion variable(s) (intercept, linear slope, quadratic slope latent variables) in the hypothesized directions; (b) a significant pathway coefficient relating the upstream predictor (moderate alcohol use) with the mediating variable (CRP) in the hypothesized direction; and (c) a significant indirect effect relating the upstream predictor (moderate alcohol use) to the criterion variable (depressive symptomatology intercept, linear slope, and quadratic slope) through the mediating variable (CRP level).

Results

The final sample included 3177 older adults described in Table 1. The average age was 74.3 years (SD = 7.0 years), and the average years of education was 12.3 (SD = 2.1 years). The sample was predominantly female (57.3%) and White/Caucasian ethnic identity (86.9%). Abstinent and moderately drinking respondent groups were similar in age (t = 1.8, $p > .05$). By comparison to abstinent participants, moderate drinkers were less likely to be female (χ^2 = 13.0, $p < .001$), more likely to be of White/Caucasian ethnicity (χ^2 = 39.6, $p < .001$), have a lower BMI (t = 5.6, $p < 001$), fewer endorsed comorbidities (t = 8.8, $p < .001$), and have lower CRP levels (t = 4.2, $p < .001$). Moderate drinkers endorsed less depressive symptomatology than did abstinent respondents across all five waves ($p \leq .001$).

Level 1 model

The unconditional growth model was fitted to the data including five repeated assessments of depressive symptomatology and indices indicated very adequate fit (χ^2 = 39.69, df = 7, $p < 001$; RMSEA = 0.038, CFI = .992). Given past research suggesting nonlinear change in depressive symptomatology throughout later life (Teachman, 2006), a quadratic term was included in the model. The revised model including the quadratic term also produced acceptable fit indices (χ^2 = 21.83, df = 4 $p < 001$; RMSEA = 0.037, CFI = .996), and the associated reduction in χ^2 suggested a statistically significant improvement in model fit (df = 3, $p < .05$). Results are that the rate of change in endorsement of depressive

Table 1 Descriptive statistics and basic comparisons of abstinent and moderately drinking respondents

	Total sample N = 3177	Abstinent n = 2257	Moderate drinkers n = 920	Comparison t or (χ2)
	Mean (SD) or %			
Age	74.3 (7.0)	74.5 (7.1)	74.0 (6.9)	1.8
Years of education	12.3 (2.1)	11.9 (3.2)	13.4 (2.7)	13.0***
% female	57.3%	62.9%	46.4%	73.1***
Ethnicity				39.6***
White/Caucasian	86.9%	84.3%	92.6%	
Black/African American	10.5%	12.5%	6.1%	
Other	2.6%	3.2%	1.3%	
Alcohol use pattern				
Drinks per occasion	1.14 (2.2)	—	1.5 (0.7)	107.6***
Occasions per week	0.49 (0.9)	—	3.5 (2.4)	70.2***
BMI	27.3 (5.0)	27.6 (5.2)	26.6 (4.4)	5.6***
Endorsed comorbidities	2.1 (1.2)	2.2 (1.2)	1.9 (1.2)	8.8***
C-reactive protein	2.5 (2.2)	2.6 (2.3)	2.2 (2.0)	4.2***
2006 CES-D	1.2 (1.8)	1.5 (1.9)	.93 (1.5)	8.8***
2008 CES-D	1.3 (1.8)	1.5 (1.9)	1.0 (1.5)	6.5***
2010 CES-D	1.3 (1.8)	1.4 (1.9)	1.1 (1.5)	5.5***
2012 CES-D	1.3 (1.8)	1.4 (1.9)	1.2 (1.6)	3.4**
2014 CES-D	1.3 (1.8)	1.5 (1.9)	1.0 (1.6)	5.5***

***Indicates that significant differences (p<.001) between abstinent and moderate drinkers.

symptomatology over 8 years is variable, becomes steeper over time, and so is best modeled using non-linear terms. Thus, this three-factor (intercept, linear slope, and quadratic slope) model was retained for Level-2 analyses.

Level-2 and conditional latent growth models

As a follow-up to the unconditional growth model which was used to examine group-level characteristics, a Level-2 model examined variability in intercept values and rates of change over time associated with individual differences. Variance in initial depressive symptomatology was estimated at 2.20 (SE = 0.13, $p < .001$), suggesting significant individual differences at baseline. Similarly, significant variance in both linear slope ($B = 0.46$, SE = .10, $p < .001$) and quadratic slope (estimate = .02, SE = .01, $p < .001$) terms suggests significant between-subject variability. Significant bidirectional pathway coefficients related intercept and linear slope ($B = -0.41$, SE = .11, $p < .001$), intercept and quadratic slope ($B = .07$, SE = .02, $p = .001$), and linear and quadratic slope ($B = -.09$, SE = .02, $p < .001$). This finding indicates that endorsement of fewer depressive symptoms at baseline related to a higher rate of change in symptom endorsement over time.

Results of the primary analysis were that the hypothesized model (Figure 1) fit well overall (RMSEA = .052, CFI = .952). Specific model results (Table 2) were that moderate drinkers endorsed fewer depressive symptoms at baseline ($B = -.49$, SE = .07, $p < .001$), but the rate of change in depressive symptomatology over time was greater ($B = .15$, SE = .06, $p = .013$). A significant and negative main effect of moderate drinking on the depressive symptomatology quadratic slope ($B = -.03$, SE = .02, $p = .045$) indicated that the baseline difference in depressive symptomatology between moderate drinkers and abstinent respondents narrowed more quickly through the first years of follow-up and slowed as it approached levels reported by abstinent respondents. By comparison, abstinent drinkers had a somewhat more linear rate of change in depressive symptomatology over time. In combination, these results suggest that benefits associated with moderate drinking at baseline were eroded by the passage of time. Endorsement of depressive symptomatology at baseline was also positively associated with CRP level, female gender, and age, although none of these variables related to rate of change over time.

Table 2 Results of slope-intercept model predicting depressive symptomatology intercept, linear slope, and quadratic slope

	Estimate	SE	Est/SE	p
Intercept				
Moderate use	−0.493	0.070	−7.046	<.001
CRP	0.054	0.014	3.797	<.001
Gender	0.280	0.064	4.345	<.001
Age	0.018	0.004	4.007	<.001
Linear slope				
Moderate use	0.151	0.061	2.472	0.013
CRP	0.000	0.013	−0.019	0.985
Gender	−0.031	0.057	−0.546	0.585
Age	0.003	0.004	0.706	0.480
Quadratic				
Moderate use	−0.030	0.015	−2.007	0.045
CRP	0.000	0.003	0.125	0.901
Gender	0.006	0.014	0.443	0.658
Age	0.000	0.001	−0.020	0.984

CRP, C-reactive protein.

By comparison to abstinent respondents, moderate drinkers had lower CRP levels ($B = -.24$, SE = .09, $p = .005$). C-reactive protein was also positively associated with BMI ($B = .09$, SE = .01, $p < .001$), suggesting that proportionally heavier respondents had higher levels of global inflammation. The indirect effect of moderate alcohol use on depressive symptoms at intercept through CRP was statistically significant ($B = -.013$, SE = .006, $t = -2.242$, $p = .025$). However, the indirect effects from moderate alcohol use through CRP to the linear slope ($B = 0.00$, SE = .003, $t = 0.019$, $p = .985$) and quadratic slope ($B = 0.00$, SE = 0.001, $t = -0.125$, $p = .901$) were not statistically significant. These findings partially support the hypothesis that inflammation, measured in this study by CRP level, mediates the relationship between moderate alcohol use and depressive symptomatology.

Four additional models were completed which examined the hypotheses that CRP as measured in 2006 mediates the relationship between moderate alcohol use in 2006 and endorsement of depressive symptomatology in 2008, 2010, 2012, and 2014, respectively. Overall, these models demonstrated variable fit, although the indirect relationship between moderate alcohol use and depressive symptomatology through CRP was statistically significant in 2008, 2010, and 2012. In the interests of thoroughly examining relationships between primary variables of interest, a final auxiliary model was tested in which directionality of primary hypothesized relationships was reversed. Although fit indices did suggest adequate fit, the hypothesized model fit better than did the reverse model.

Discussion

Primary findings of this study were that, in a large, demographically representative sample of community-dwelling older adults, moderate alcohol consumption related fewer depressive symptoms at baseline, although the gap in depressive symptom endorsement related to moderate alcohol use narrowed over time. In other words, benefits with respect to depressive symptomatology associated with alcohol consumption at baseline were slowly eroded over the course of the study. The second finding is that the relationship between moderate alcohol use and depressive symptomatology was partially mediated by CRP. This later finding suggests that inflammation is one mechanism by which moderate alcohol use confers benefits with respect to depressive symptomatology. Significant mediation of this effect was identified in auxiliary models examining outcomes by year. Absence of an indirect effect of baseline moderate alcohol use on depressive symptomatology through CRP at the final wave, 8 years after baseline, is consistent with the erosion of moderate-drinking related benefits.

These findings are consistent with past work suggesting a relationship between moderate alcohol use and depressive symptomatology (Goldberg *et al.,* 1999; Coulson *et al.,* 2014; Brennan *et al.,* 2016). Other studies have found that the present research examines these relationships among adults over age 65, who may be at greater risk for adverse clinical trajectories associated with depression (Lichtenberg *et al.,* 1993; Bruce *et al.,* 1994; Yochim *et al.,* 2003; Paulson *et al.,* 2013). These results are consistent with other recent work (Shah and Paulson, 2016) identifying reduced inflammation as one mechanism by which moderate alcohol use may confer health benefits. Depressive symptomatology, like frailty, is determined by numerous factors, and a small effect size was consistent with expectations. Erosion of alcohol-use related benefits, and attenuation of the mediating role of CRP over time, may reflect accumulation of other health burden. The compression of morbidity hypothesis (Fries, 2003) posits that later-life is associated with the accumulation of comorbidities, many of which may be associated with increased inflammation, thus concealing the relationship between depressive symptomatology and upstream physiological predictors or determinants.

It is probable that future research will identify alternate mechanisms by which alcohol relates to positive health outcomes in general, and less depressive symptomatology in particular. For instance, recent work (Maraldi *et al.,* 2009) found that those who drink moderately may also appropriately moderate other health behaviors, including engagement in regular physical activity. Future research should also examine patterns of socialization around alcohol use, particularly among older adults. Isolation is a well-known risk factor for and result of depression across the lifespan (Schoevers *et al.,* 2000; Hackett *et al.,* 2012). It may be that older moderate drinkers may drink within the context of regular social interaction, and that such a behavior pattern could relate to improved affect regulation. Additionally, cognitive factors may be implicated in alcohol-related health outcomes. For instance, those with family or personal histories of alcohol abuse may adaptively avoid alcohol use citing significant perception of psychological, social, or other related health risks. It is also likely that alcohol benefits may be moderated by unidentified genetic characteristics, and future research using large datasets such as that employed here should examine these hypotheses.

Definitions of moderate drinking are numerous, and warnings about health risks associated with alcohol use are sundry. In conjunction with related research on alcohol use and health, these findings are one contribution to better understand the overall relationship between alcohol use and health outcomes. These results may facilitate future research and, subsequently, streamline evidence-based drinking guidelines for older adults. Present findings are inadequate to form a basis for recommending alcohol use as a clinical intervention, and future research should continue to examine mechanisms by which use relates to health benefits. One possible outcome of such work might involve identification of positive health behaviors (e.g. frequent socialization, dietary factors) to reduce morbidity in later life. Additionally, the current research may contribute to medical interventions that make use of biological mechanisms affected by alcohol use, such as reduced inflammation as indicated by CRP levels, that are associated with positive health outcomes.

The primary limitation of this study is reliance on subjective alcohol use data. Nonetheless, this practice is consistent with the vast majority of past work examining alcohol-related health outcomes, and laboratory measures of alcohol use are impractical for the study of alcohol use in large, community panel studies such as the HRS. Another limitation of this study is the use of cross-sectional alcohol use and CRP data. Both of these variables are subject to change over time, and it would be very informative to

examine how CRP changes in response to alcohol use. Given the data limitations in the HRS (CRP was measured at only one wave for each respondent), the authors were unable to examine these relationships longitudinally. An important direction for future research involves the assessment of the bidirectional relationship between CRP and depressive symptomatology among older adults, and how lifestyle behaviors such as alcohol use relate to this constellation of variables. Finally, the HRS data do not provide information on how alcohol use relates to patterns of socialization, and past work suggests this as one critical direction for future research on alcohol use and depressive symptomatology.

Conflict of interest

None declared.

Key points

- Moderate alcohol use relates to fewer depressive symptoms among older adults.
- The benefits of moderate alcohol use on depressive symptoms erode with increasing age.
- The relationship between moderate alcohol use and depressive symptoms is partially mediated by CRP.
- Findings support past research suggesting that reduced inflammation may be one mechanism by which moderate alcohol use confers health benefits.

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