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PTSD is associated with poor health behavior and greater Body Mass Index through depression, increasing cardiovascular disease and diabetes risk among U.S. veterans

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Highlights

- We examined a model of the association of PTSD with CVD and diabetes in veterans.
- PTSD without depression was associated with better physical activity and diet quality.
- Depression accounted for associations of PTSD with BMI and poor health behaviors.
- Health behaviors should be targeted in those with depression to improve health.

Abstract

Posttraumatic stress disorder (PTSD) is a risk factor for cardiovascular disease (CVD) and diabetes. Dedert and colleagues hypothesized a model whereby PTSD leads to poor health

behaviors, depression, and pre-clinical disease markers, and that these factors lead to CVD and diabetes (*Ann Behav Med*, 2010, 61–78). This study provides a preliminary test of that model. Using data from a mailed cross-sectional survey conducted 2012–2013, path analysis was conducted among N=657 with complete demographic data. We first analyzed the hypothesized model, followed by four alternatives, to identify the best-fitting model. The alternate model that specified pathways from depression to health behaviors had the best fit. Contrary to hypotheses, higher PTSD symptoms were associated with *better* physical activity and diet quality. Of the specific indirect pathways from PTSD to Body Mass Index (BMI), only the path through depression was significant. Higher depression symptoms were significantly associated with less physical activity, poorer diet, and greater likelihood of smoking. In addition, the specific indirect effect from depression to BMI through physical activity was significant. Current smoking and higher BMI were associated with greater likelihood of diabetes, and hypertension was associated with greater likelihood of CVD. PTSD symptoms may increase risk for CVD and diabetes through the negative impact of depression on health behaviors and BMI. With or without PTSD, depression may be an important target in interventions targeting cardiovascular and metabolic diseases among veterans.

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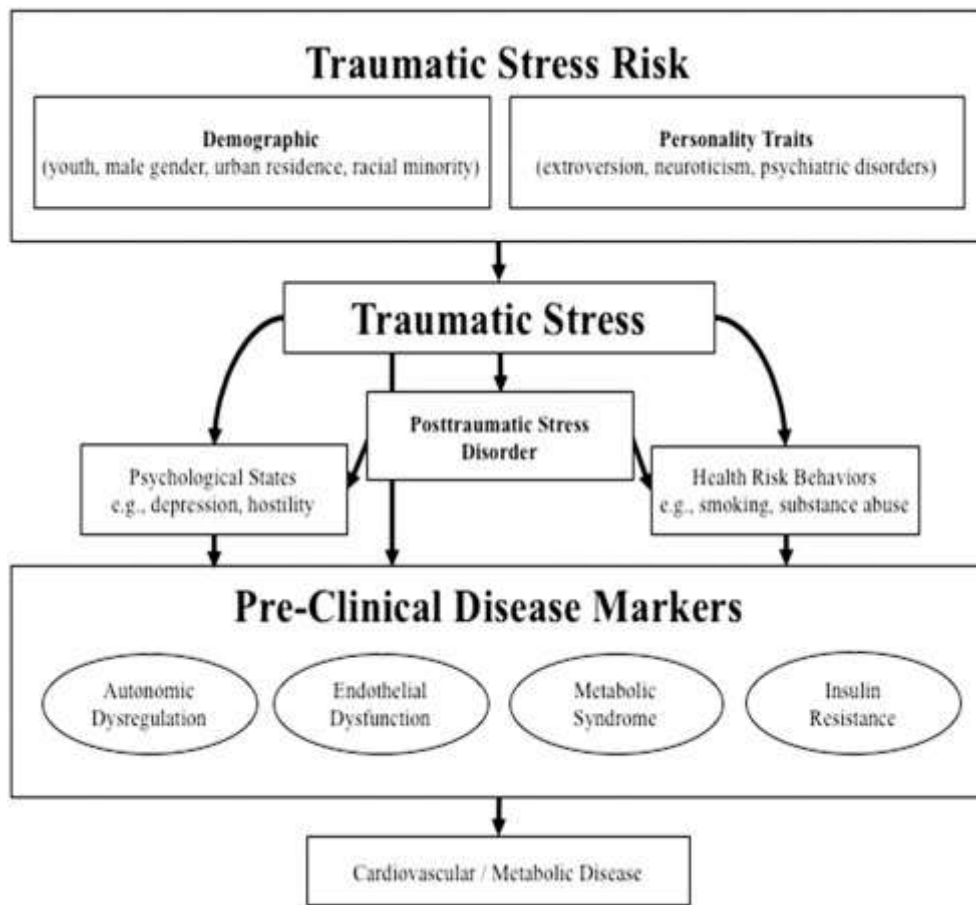
Keywords

PTSD; Depression; Cardiovascular disease; Diabetes; Health behavior

1. Introduction

Cardiovascular disease (CVD) and diabetes are prevalent, often preventable causes of premature mortality ([Aguilar et al., 2015](#); [Danaei et al., 2009](#); [Go et al., 2013](#); [Santulli, 2013](#)). Posttraumatic stress disorder (PTSD) has been found to be associated with increased risk for premature mortality, in part due to increased risk for CVD, diabetes, and related risk factors such as hypertension and obesity among individuals with PTSD ([Dedert et al., 2010](#)). Prior research suggests these associations are due to CVD and diabetes risk factors common among individuals with PTSD, such as over-activation of the hypothalamic-pituitary-adrenal (HPA) stress axis, inflammation, conditions such as obesity and hypertension, and poor health behaviors like tobacco use ([Boscarino, 2012](#); [Dedert et al., 2010](#)). Regarding the latter, evidence suggests tobacco use is elevated among individuals with PTSD both due to nicotine use for emotional regulation and genetic risk factors ([Boscarino, 2012](#)). [Dedert et al. \(2010\)](#) proposed a theoretical model to comprehensively explain and help guide study of the association of PTSD with these outcomes and to inform clinical practice (see [Fig. 1](#)). Based on existing literature, they hypothesized that

PTSD leads to depression and health risk behaviors (e.g., tobacco and alcohol use), which influence pre-clinical disease markers, and in turn CVD and metabolic diseases (Dedert et al., 2010).



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Fig. 1. Dedert and colleagues' hypothesized model.

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Evaluating Dedert et al.'s (2010) theoretical model would provide further data on whether and how PTSD is associated with CVD and diabetes and information regarding possible CVD and diabetes prevention targets to address the high disease burden among people with PTSD. For example, if tobacco use largely drives the association, this would identify a priority health behavior intervention target. The present study presents a preliminary test of Dedert et al.'s (2010) model relating PTSD symptoms, depression symptoms, health behaviors, pre-clinical disease markers, and CVD and diabetes among veterans. It is especially important to examine the roles of PTSD, depression, and health risk behaviors in CVD and diabetes among veterans given the high prevalence of these mental health and chronic health conditions in that population (Dohrenwend

et al., 2006; Hoerster et al., 2012; Hoge et al., 2004; Lehavot et al., 2012; Seal et al., 2007; Vasterling et al., 2006). Minor changes were made to the model to incorporate current empirical literature. Although not specified in Dedert and colleagues' model, emerging but inconclusive evidence suggests that PTSD may also be associated with inactivity and poor eating behaviors (Hall et al., 2015), which are additional risk factors for CVD, diabetes, and obesity (Danaei et al., 2009). Poorer activity and diet may be due to PTSD symptoms, such as hyperarousal (Rutter et al., 2013), sleep disturbance (Baron et al., 2013; Boutcher and Dunn, 2009; St-Onge, 2013), and social isolation (Brug, 2008; Charuvastra and Cloitre, 2008; Shaikh et al., 2008; Sherwood and Jeffery, 2000). Moreover, although Dedert et al. specified depression as a mediator only of the relationship between PTSD and health outcomes, we determined it important to examine the association of depression with health behaviors in alternative models, given that depression is highly co-morbid with PTSD (Dedert et al., 2010) and has been found to be associated with poor health behaviors (Bonnet et al., 2005; Grant et al., 2004; Lasser et al., 2000; Strohle, 2009), CVD, and diabetes (DeHert et al., 2011), with likely shared etiologic factors such as HPA axis disruption and inflammation (Marazziti et al., 2014).

2. Material and methods

2.1. Sample

Data were collected as part of a larger survey study focused on PTSD and health (Hoerster et al., 2015). In 2012, surveys were mailed to 1997 veterans. To ensure adequate sampling of veterans with PTSD, surveys were mailed to veterans who had attended at least one visit in primary care or PTSD clinics at a Northwest Veterans Affairs (VA) Medical Center in the year prior. Surveys were re-mailed to 1221 individuals who had not responded by March 2013. A total of 193 veterans did not respond due to an incorrect or out-of-state address or because they were deceased. Of the 1804 possible respondents, 717 veterans returned completed surveys (40% response rate). Path analyses were conducted using maximum likelihood estimation among the 657 participants with complete data on the demographic (exogenous) variables (Kline, 2010) included in the model: age, sex, and race/ethnicity. Veterans received \$10 in VA canteen coupons for returning the survey. This study received Institutional Review Board approval and a waiver of signed informed consent. Basic demographic characteristics of the sample are shown in Table 1.

Table 1. Bivariate correlations and descriptive statistics among a clinical sample of U.S. veterans, VA Puget Sound, Seattle 2012–2013 (N = 657).

Variable	Mean	Correlations among variables
	(SD) or	

Variable	% Mean (SD) or %	Correlations among variables											
		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Sex (female)	13.5%												
2. Race (white)	72.7%	0.02											
3. Age	61.1 (13.3)	-0.26 ***	-0.08 *										
4. PTSD severity	44.4 (20.5)	0.02	0.08*	-0.22 ***									
5. Depression severity	9.3 (7.1)	0.04	0.06	-0.20 ***	0.76* **								
6. Current smoking	21.5%	0.04	0.01	-0.19 ***	0.11* *	0.15* **							
7. Alcohol use	1.7 (2.3)	-0.08 *	0.02	-0.11 **	0.04	-0.02	0.08						
8. Weekly min physical activity	529.7 (611.2)	-0.07	0.02	-0.06	-0.07	-0.18 ***	-0.08	0.08					
9. Diet quality (higher = worse)	6.53 (2.7)	-0.05	-0.02	-0.01	0.05	0.14* *	0.20* **	0.01	-0.14 **				
10. Body Mass Index	29.1 (5.8)	0.06	0.06	-0.14 ***	0.14* *	0.15* **	-0.08 *	-0.05	-0.11 *	-0.02			
11. Has hypertension	62.4%	-0.11 **	0.10*	0.21* **	0.06	0.06	-0.02	0.01	-0.09 *	0.02	0.17 ***		
12. Has CVD	34.3%	-0.12 **	0.01	0.27* **	0.01	0.05	0.03	-0.03	-0.11 *	0.06	0.06	0.24 ***	
13. Has diabetes	28.4%	-0.07	0.11* *	0.15* **	0.05	0.06	-0.10 *	-0.17 ***	-0.18 ***	-0.11 **	0.32 ***	0.26 ***	0.18 ***

Note. Sex: 0 = male, 1 = female (shown); Race: 1 = non-Hispanic white (shown), 2 = non-Hispanic black (10.5%), 3 = other (16.8%);

*

$p < .05$.

**

 $p < .01$.

 $p < .001$.

2.2. Measures

The Dedert model ([Dedert et al., 2010](#)) guided variable selection, though the hypothesized model was modified slightly based on measures available in the parent study. Demographic variables were age, sex, and race/ethnicity (exogenous) ([Kline, 2010](#)). Mental health, health behavior, and outcome variables are described below (endogenous) ([Kline, 2010](#)). When possible, continuous measures were used to maximize power ([Altman and Royston, 2006](#)). Timeframe and possible score ranges are listed for all relevant measures.

2.2.1. Mental health symptoms

Continuous measures of PTSD and depression symptoms were included. Validated, established screeners were used to assess past-month PTSD (PTSD Checklist-Military Version for DSM-IV (PCL-M) ([Weathers and Ford, 1996](#))) and past-two-week depression (Patient Health Questionnaire-8 (PHQ-8) ([Kroenke et al., 2009](#))) symptoms. Possible scores ranged from 17 to 85 and 0 to 24, respectively, with higher scores indicating greater symptom severity.

2.2.2. Health behaviors

Aside from current smoking, health behavior indicators were continuous. The Alcohol Use Disorders Identification Test-Consumption (AUDIT-C) is a valid and widely used measure that assesses typical alcohol use severity ([Bush et al., 1998](#)). Possible scores ranged from 0 to 12, with higher scores indicating greater alcohol misuse severity. Because PTSD symptoms may also be associated with risk behaviors of poor diet and physical inactivity ([Hall et al., 2015](#)), we also included activity and diet measures. The widely-used International Physical Activity Questionnaire (IPAQ)--long form assessed total minutes of past-week physical activity ([Craig et al., 2003](#)). Diet quality in the “past few months” was assessed with an 8-item screener called Starting the Conversation ([Paxton et al., 2011](#)), with possible scores ranging from 0 to 16 and higher scores indicating poorer diet. Current smoking was assessed with “Do you now smoke cigarettes?” We combined “every day” and “some days” to create a dichotomous variable reflecting current smoking.

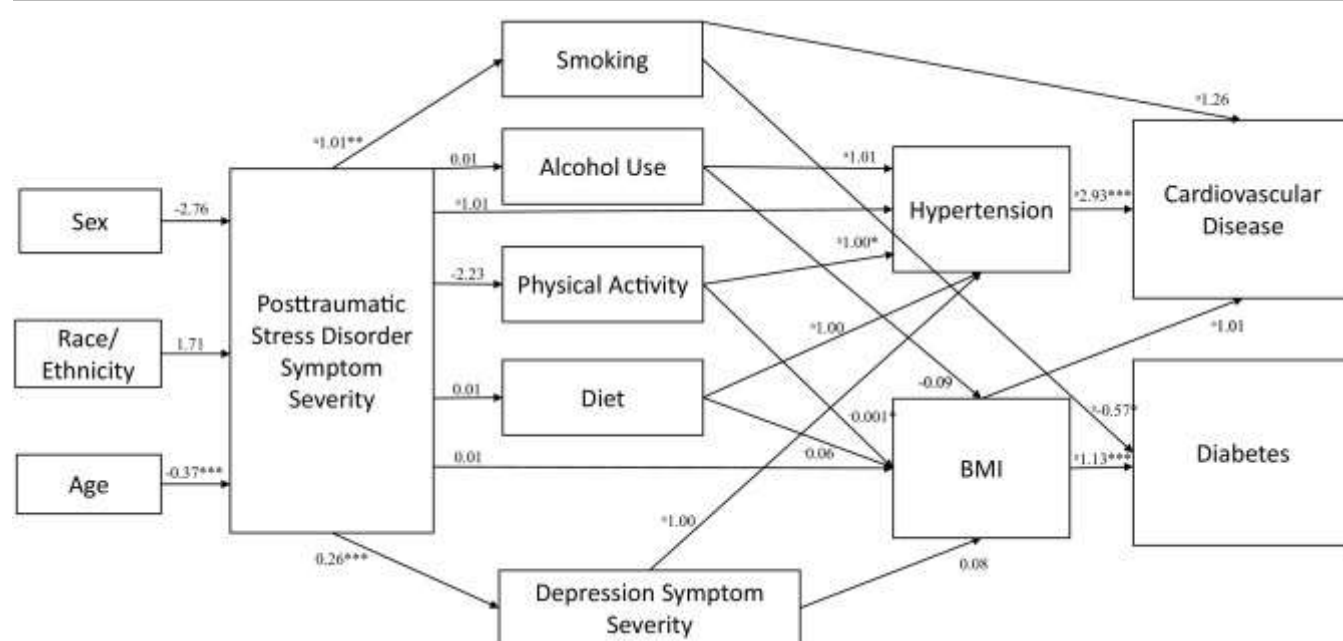
2.2.3. Pre-clinical disease markers, and CVD and diabetes outcomes

Self-reported height and weight were used to calculate a continuous measure of Body Mass Index (BMI). Self-reported lifetime diagnosis on the Seattle Index of Comorbidity (SIC) ([Fan et al., 2002](#))

was used to create categorical variables reflecting presence or absence of hypertension, and diabetes and CVD outcomes. Participants who indicated they had been told by a doctor or nurse that they had congestive heart failure, coronary artery disease, heart attack, stroke, and/or that their chest pain was due to heart problems (i.e., angina) were deemed to have CVD. Self-report is a valid method for capturing CVD, diabetes, and related outcomes (Kriegsman et al., 1996; Robinson et al., 1997; Tisnado et al., 2006), and the SIC is one valid self-report measure for capturing medical diagnoses (Fan et al., 2002).

2.3. Statistical analysis

Path analysis was conducted using Mplus Version 8. In addition to testing all paths depicted in Fig. 2, we examined novel indirect paths (i.e., whether psychiatric conditions lead to pre-clinical indicators and outcomes by way of health behavior and whether PTSD symptoms lead to pre-clinical indicators by way of depression symptoms). Although not specified in Dedert's model, we examined the association of depression with health behaviors starting with Model 2 because prior research indicates depression is co-morbid with PTSD (Dedert et al., 2010) and associated with poor health behaviors (Bonnet et al., 2005; Grant et al., 2004; Lasser et al., 2000; Strohle, 2009), CVD, and diabetes (DeHert et al., 2011), indicating these might be important explanatory pathways. We did not test other indirect paths because the links between health behaviors, pre-clinical disease markers, and CVD and diabetes are well-established (Danaei et al., 2009). All health behaviors but smoking were modeled as direct effects on both pre-clinical disease markers. Current smoking was instead modeled to be associated directly with CVD and diabetes based on prior research (American Heart Association, 2017; Dare et al., 2015). Hypertension was modeled to be associated only with CVD, while BMI was modeled to be associated with both outcomes, due to CVD and diabetes' distinct risk pathways.



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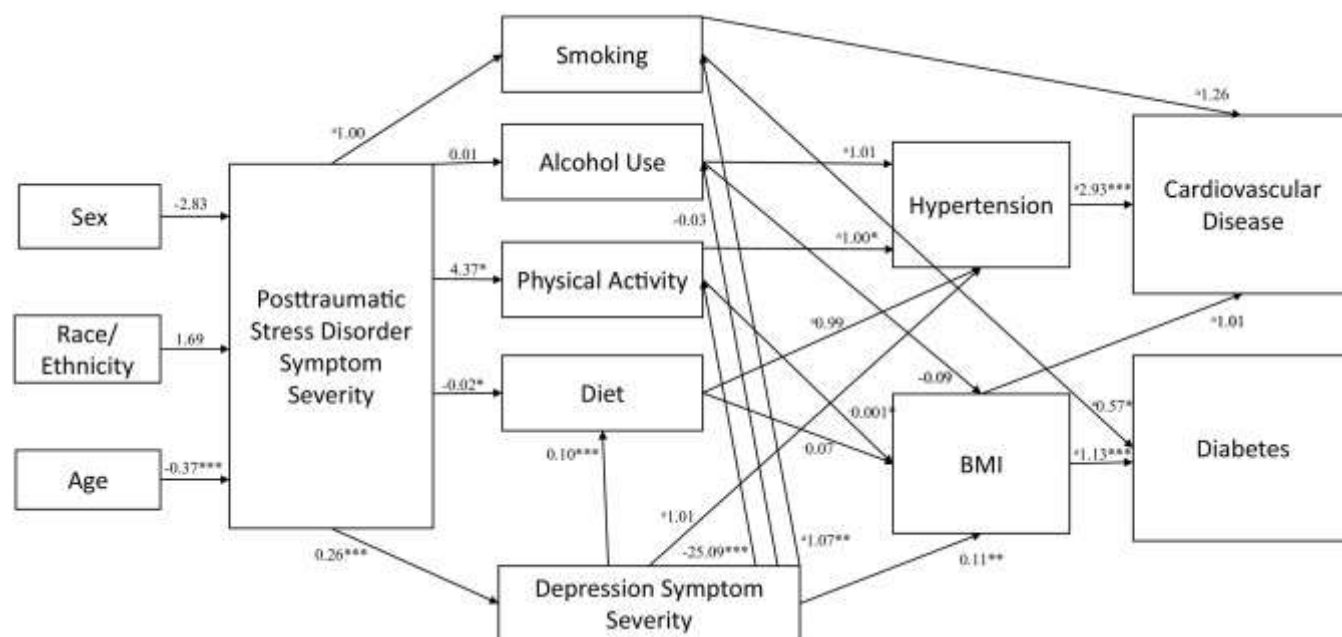
Fig. 2. Hypothesized model (Model 1) with unstandardized path coefficients, VA Puget Sound, Seattle 2012–2013 (N = 657).

^aOdds ratios are reported for dichotomous outcomes. Sex, smoking, hypertension, cardiovascular disease, and diabetes were coded as dichotomous variables in the model. * $p < .05$; ** $p < .01$; *** $p < .001$.

Model 1 (hypothesized) fit indices	Akaike information criterion	Sample-size adjusted Bayesian information criterion
	29526.46	29577.66

Note. Compare fit indices for alternative models in Fig. 3.

Because several variables were non-continuous, model fit statistics typically used to evaluate path models (e.g., chi-square test of model fit, TLI, RMSEA; Kline, 2010) are not available, and covariances between variables cannot be tested. Alternatively, nested path models can be evaluated by comparing their Akaike Information Criterion (AIC) and sample-size adjusted Bayesian Information Criterion (BIC), where the model with the lowest AIC and BIC is the best-fitting model (Kline, 2010). Using this approach, we first tested the hypothesized model (see Fig. 2), followed by four alternative models that removed or added hypothesized pathways (see Fig. 3). We compared the five models' AIC and BIC to determine the best-fitting model.¹



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Fig. 3. Best fitting model (Model 4) with unstandardized path coefficients, VA Puget Sound, Seattle 2012–2013 (N=657).

^aOdds ratios are reported for dichotomous outcomes. Sex, smoking, hypertension, cardiovascular disease, and diabetes were coded as dichotomous variables in the model. * $p < .05$; ** $p < .01$; *** $p < .001$.

Model fit indices for alternative models.

	AIC	BIC
Model 2: paths added from depression symptom severity to health behaviors	29492.65	29549.10
Model 3: from Model 2, paths removed from PTSD symptom severity to health behaviors	29499.33	29550.52
Model 4 (shown): from Model 2, paths removed from PTSD symptom severity to BMI and HTN	29490.20	29544.02
Model 5: from Model 2, paths removed from depression symptom severity to BMI and HTN	29490.31	29544.13

Note. AIC=Akaike information criterion; BIC=sample-size adjusted Bayesian information criterion. Bolded model (model 4) has the lowest AIC and BIC fit indices and is therefore the best-fitting model (shown).

Included and excluded participants did not significantly differ on any variable. Data were missing on diabetes (11.0%) and CVD (12.3%). Those with and without missing data on study outcomes did not differ significantly by study demographic characteristics. Maximum likelihood estimation was used to account for missing values on mental health, health behavior, and outcome (endogenous) variables (Kline, 2010). To maximize power (Altman and Royston, 2006), and consistent with research demonstrating that subclinical symptoms of PTSD are associated with significant impairment (Bergman et al., 2017), we used continuous measures of PTSD and depressive symptom constructs.

3. Results

Table 1 provides descriptive statistics and bivariate correlations among study variables, including demographic characteristics. See model fit statistics for the hypothesized (Fig. 2) and four alternative models (Fig. 3). Because the AIC and BIC decreased substantially from Model 1 to Model 2, subsequent models used Model 2 as their base model. AIC and BIC fit indices indicate that Model 4 provided the best fit to the data. We thus present path coefficients for the hypothesized (Model 1, Fig. 2) and best-fitting (Model 4, Fig. 3) models, respectively. Fig. 3 details addition and removal of paths for each subsequent model.

Moving from left to right in the figure, in the hypothesized model (Model 1), PTSD symptoms were not significantly associated with physical activity, diet or alcohol use but were positively associated with current smoking and depression symptoms. Depression symptoms were not significantly associated with BMI, nor hypertension. Physical activity was positively associated with HTN, which was positively associated with CVD. Physical activity was negatively associated with BMI, which was positively associated with diabetes. Smoking was negatively associated with diabetes. In Model 1, none of the sum of indirect effects or specific indirect effects were significant.

In contrast, in the final model (Model 4), PTSD symptoms were unexpectedly associated with higher physical activity and better diet, and were not associated with smoking or alcohol use. PTSD symptoms were associated with higher depression symptoms, which were associated with lower physical activity, worse diet, and greater likelihood of smoking. Lower physical activity and higher depression symptoms were associated with higher BMI. Surprisingly, more physical activity was associated with greater likelihood of hypertension. Finally, current smoking and higher BMI were associated with greater likelihood of diabetes, and hypertension was associated with greater likelihood of CVD.

In contrast to Model 1, in Model 4, the sum of indirect effects from PTSD symptoms to BMI was significant (0.02, *S.E.* = 0.01, *p* = .01), as was the specific indirect effect through depression symptoms (0.03, *S.E.* = 0.01, *p* = .001). The sum of the indirect effects from depression symptoms to BMI was not significant. However, the specific indirect effect from depression symptoms to BMI

through physical activity was significant (0.02, *S.E.* = 0.01, *p* = .04). Neither the sum of, nor the specific, indirect effects to hypertension from PTSD or depression symptoms were significant.

4. Discussion

This study conducted a preliminary test of Dedert's model regarding how PTSD symptoms lead to CVD and diabetes (Dedert et al., 2010) using cross-sectional data from a large sample of veterans. Contrary to expectations, the hypothesized model did not provide the best fit to the data. As hypothesized, PTSD symptoms were associated with depression. However, in a model accounting for depressive symptoms and depression symptoms' associations with health behaviors, PTSD symptoms were associated with better diet quality and physical activity, although the association with physical activity was non-significant in the exploratory model using a dichotomized PTSD variable (data not shown). This study suggests that veterans with higher PTSD, but lower depressive, symptoms may maintain healthy eating and activity patterns, behavioral patterns that could be capitalized upon to improve health. While contrary to hypotheses, the present study's findings are consistent with one recent rigorously conducted longitudinal analysis of the association of PTSD with prospective weight change trajectories (LeardMann et al., 2015). In that study, consistent with prior literature, PTSD was associated with prospective weight gain. Surprisingly, PTSD also increased risk for a weight *loss* trajectory, suggesting that subgroups of individuals with PTSD may have differing health behavior and CVD risk factor trajectories (LeardMann et al., 2015).

Although a recent literature review (Hall et al., 2015) and meta-analysis (van den Berk-Clark et al., 2018) noted that in general PTSD appears to be associated with poorer activity and diet in several studies, evidence is mixed and few studies looking at these associations have examined the role of depression, used rigorous objective measurement procedures, or applied longitudinal designs (Hall et al., 2015). Findings regarding associations with eating behaviors are especially limited and inconsistent (Theal et al., 2018), though some studies have found positive associations between PTSD and dietary intake (van den Berk-Clark et al., 2018). In addition to a need for more rigorous research on the association between PTSD and dietary behaviors, it is important to distinguish between dietary behavior types and whether some differentially affect the association of PTSD with CVD risk factors. In the present study, an overall measure of diet quality was used to measure eating behavior, but binge eating was not assessed. Two recent studies found that binge eating (Cronce et al., 2017) and disordered eating (Mitchell et al., 2016) explain the relationship between PTSD and Body Mass Index. Future studies should examine associations with different types of physical activity, given that a recent study found that hypervigilance interferes with vigorous physical activity, but not other physical activity levels (Harte et al., 2015). While it is possible that PTSD is a risk factor for poor diet and activity, it is likely that this relationship is more complex and requires further study.

In the context of PTSD symptoms, co-morbid depressive symptoms may contribute to CVD and diabetes through their negative impact on health behaviors. Although [Dedert et al.'s \(2010\)](#) model did not specify direct paths from depression to health risk behaviors, these were important explanatory pathways in our final model. After accounting for PTSD, consistent with prior research ([Bonnet et al., 2005](#); [Grant et al., 2004](#); [Lasser et al., 2000](#); [Strohle, 2009](#)), depression was associated with greater likelihood of smoking, poor diet, and less physical activity. Although PTSD has been associated with tobacco use and hazardous alcohol use in prior research ([Buckley et al., 2004](#); [Debell et al., 2014](#); [Fu et al., 2007](#)), PTSD was not directly associated with either health risk behavior in the present study. This aligns with findings from a prospective study among 800 US Army soldiers, in which neither alcohol use nor smoking mediated the relationship between PTSD and somatic functioning ([Vasterling et al., 2008](#)). In addition, a recent study found that alcohol use did not mediate the relationship between PTSD and BMI ([Cronce et al., 2017](#)). Importantly, in the present study depression was associated with current smoking, which was associated with increased likelihood of diabetes, underscoring the need to address tobacco use among veterans with depression. Finally, contrary to the hypothesized model, PTSD was not indirectly associated with pre-clinical outcomes that increase CVD and diabetes risk when not accounting for depression symptoms and their associations with health behaviors. However, as noted by [Dedert et al. \(2010\)](#), there is limited research indicating an association of PTSD with diabetes, including a recent longitudinal study that found no association in adjusted models ([Boyko et al., 2013](#)). Moreover, they also note that too few studies account for the role of depression in their models, and that future work should incorporate depression. Thus, the present study lends support to their suggestion that future research account for the possible role of co-morbid depression in CVD and diabetes outcomes. Results suggest that co-occurring depression is likely a critical target when addressing cardiovascular and metabolic diseases, and is likely to be commonly occurring, given high rates of comorbidity with PTSD, as seen in this sample. Individuals with depression—with or without PTSD—should be prioritized for receiving comprehensive health behavior interventions.

The treatment engagement of the sample may provide some explanation for why PTSD symptoms were not directly associated with deleterious health behaviors and outcomes. It may be that the VA healthcare system is effectively promoting healthy lifestyles among Veterans with PTSD symptoms, given the increased emphasis on doing so in VA. For example, tobacco cessation services were effectively integrated into PTSD care with superior cessation outcomes ([McFall et al., 2010](#)), and emerging evidence indicates that weight loss ([Johannessen and Berntsen, 2013](#)) and physical activity reduce and prevent PTSD symptoms ([Goldstein et al., 2018](#); [LeardMann et al., 2011](#); [Smith et al., 2011](#)), consistent with literature showing mood enhancing and anxiolytic effects of exercise ([Barbour et al., 2007](#); [Strohle, 2009](#)). The notion that veterans with PTSD may use healthy lifestyles as a way of coping is aligned with a prior study purporting that as PTSD symptoms start to diminish, individuals engage in fewer negative *and* positive health behaviors they may have been using to cope ([Shipherd et al., 2014](#)).

Although the bivariate relationship between PTSD and BMI was significant and prior research has demonstrated an association ([Buckley et al., 2004](#)), the final model failed to find evidence for this relationship. Instead, PTSD predicted BMI through depression, and depression predicted BMI through reduced activity. As such, the present study's findings indicate that the relationship between PTSD and weight is likely complicated, in need of further study, and potentially driven by depression. Thus, targeting behavioral weight management programs to individuals with depression—with or without PTSD—would likely be beneficial. Standard weight management programs may require modification to address unique barriers to weight loss among individuals with depression and other mental health conditions ([Hoerster et al., 2014a](#)). Such tailored efforts may help address the reduced effectiveness of VA's national weight management program among veterans with psychiatric conditions ([Hoerster et al., 2014b](#)). Importantly, behavioral weight management programs may also improve depression and PTSD symptoms ([Barbour et al., 2007](#); [Hall et al., 2015](#); [Strohle, 2009](#)).

4.1. Limitations

Findings should be considered in the context of several limitations. PTSD and depression are highly comorbid conditions and several symptoms of PTSD overlap with diagnostic criteria of depression ([Pietrzak et al., 2011](#)), making it difficult to distinguish between PTSD and depressive symptoms' unique contributions. This study's primary models used continuous measures of PTSD and depression symptoms, rather than dichotomous measures that reflect diagnostic categories. Indeed, in our sample, 12% exceeded cutoffs for probable PTSD (≥ 50 on the PCL-M ([Weathers and Ford, 1996](#)) but not depression, 12.2% for probable depression (≥ 10 on the PHQ-8 ([Kroenke et al., 2009](#)) but not PTSD, 32.5% for both disorders, and 43.4% for neither diagnosis. Still, findings were comparable when we used dichotomous variables in exploratory analyses. Moreover, descriptive data suggest that those with PTSD but not depression had better diet and higher levels of physical activity (diet: $M=5.97$, $SD=2.59$; physical activity: $M=675.85$, $SD=674.07$) than those with depression but not PTSD (diet: $M=7.28$, $SD=2.90$; physical activity: $M=384.03$, $SD=527.46$) and those with both diagnoses (diet: $M=6.77$, $SD=2.99$; physical activity: $M=445.37$, $SD=555.59$). These exploratory findings suggest qualitatively distinct health profiles for each group and that PTSD and depression symptoms can be considered unique but related constructs in the models.

Cross-sectional data limit the ability to interpret associations in the model as sequential or causal, although cross-sectional mediation models are suggested in support of theory or model development ([Hayes and Rockwood, 2017](#)). Moreover, measures of PTSD, depression, alcohol use, smoking, physical activity, and diet used different timeframes, further challenging ability to establish directionality. Relatedly, this meant we were unable to assess the temporal associations of symptoms and health behaviors, which may have contributed to null associations. For example, it may be that high levels of symptoms one day lead to high levels of physical activity on that day for coping, contributing to lower levels of symptoms on that or the next day. Ecological

momentary assessment or daily diary methods would be ideally suited to investigate such possibilities in future research, and have been successfully used with veterans with PTSD previously ([Campbell et al., 2017a](#); [Campbell et al., 2017b](#)).

The physical activity measure may also respond differently among veterans with PTSD symptoms, and reports of objective measures of activity among veterans with PTSD are not available to compare to the present study. Respondents may represent a self-selected group, not representative of the veteran population. The parent study oversampled veterans with PTSD. However, the impact of this on findings appears minimal ([Hoerster et al., 2015](#)).

This study slightly modified Dedert and colleagues' model because the parent study did not assess two of the proposed PTSD-antecedent constructs (i.e., personality traits and traumatic event history), nor proposed pre-clinical disease markers (e.g., autonomic dysregulation). Another factor not specified in Dedert's model—and not assessed independently from PTSD symptoms in the present study—is sleep disturbance, which affects physical activity and BMI ([Baron et al., 2013](#); [Boutcher and Dunn, 2009](#)) and healthy diet ([St-Onge, 2013](#)). This limited the extent to which a truly comprehensive model could be tested. Notwithstanding these limitations, no published work has yet tested the Dedert model; thus, this study's findings offer alternative comprehensive models for testing with longitudinal data.

5. Conclusions

This study provides an initial empirical test of Dedert's 2010 proposed model and offers an alternative model suggesting that depressive symptoms in the context of PTSD symptoms may play a role in contributing to CVD and diabetes. Indeed, Dedert and colleagues called for more evaluation of depression as a co-morbid condition in examinations of PTSD, health behaviors, and CVD and diabetes ([Dedert et al., 2010](#)). This study suggests that comprehensively addressing the health behaviors of veterans with depression symptoms, and among those with co-occurring depression and PTSD, may be an important clinical target. Further research on the subset of veterans with PTSD symptoms who appear to engage in positive health behaviors would advance intervention development. Replication of these findings is needed and future research would benefit from addressing the current study's limitations, particularly the cross-sectional nature of the data. Future studies examining the association of PTSD with health risk behaviors, pre-clinical disease markers, and CVD/diabetes should account for depression, and examine differential effects of PTSD symptom clusters. Interventions that address smoking, physical inactivity, and diet in the context of co-occurring depression and PTSD may be especially beneficial for addressing the disproportionate CVD and metabolic disease burden among individuals with depression ([DeHert et al., 2011](#)) and PTSD ([Dedert et al., 2010](#)) symptoms.

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Declaration of Competing Interest

The authors declare they have no conflicts of interest.

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¹ We also ran exploratory models using dichotomous versions of PTSD and depression variables representing diagnostic thresholds: ≥ 50 on the PCL-M as a conservative cut-point for probable PTSD ([Weathers and Ford, 1996](#)) and ≥ 10 for probable depression on the PHQ-8 ([Kroenke et al., 2009](#)). Model 4 remained the best-fitting model. However, PTSD was no longer significantly associated with physical activity, and depression was no longer significantly associated with smoking. Other paths remained consistent with models reported using continuous variables. Data not shown.

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