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High Social Coping Self-Efficacy Associated with Lower Sweat Interleukin-6 in Older Adults with Chronic Illness

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Abstract

Inflammation, particularly Interleukin-6 (IL-6), is associated with chronic disease in older adults, but not all older adults have the same progression of poor health outcomes. Self-efficacy may play a role in buffering the inflammatory burden in chronic disease. To evaluate associations between self-efficacy and IL-6, 159 community dwelling older adults (N=159, M_{age}= 82 years, SD= 6.3) with one or more chronic illnesses were recruited for this cross-sectional study. Sweat IL-6 was collected using a non-invasive sweat patch worn for 72 hours. Multiple linear regression with bootstrapping showed a significant association between Social Coping Self-Efficacy and IL-6 (β= −0.534, p=0.010) after adjustment for age, sex, race, body mass index, financial strain, chronic conditions and social support. Although preliminary, this study creates a rationale to explore the self-efficacy inflammatory biomarker association further. Enhancing self-efficacy might be a viable non-pharmacological treatment to lower or slow the inflammatory burden in older adults.

1. Introduction

Sixty percent of U.S. adults report at least one chronic disease (Buttorff et al., 2017). Older adults are disproportionally affected by chronic disease. As the number of adults over 65 doubles to 71 million in the coming years (Anderson, 2010), we can also expect an increase in chronic disease burden. Inflammation is strongly associated with aging (Singh & Newman, 2011; Walston et al., 2009) and chronic diseases including: heart disease (Everett et al., 2013; Yudkin et al., 2000), diabetes (Lukic et al., 2014), frailty (Puzianowska-Kuźnicka et al., 2016) and arthritis (McGeer & McGeer, 2004). An important part of the inflammation pathway is cytokines, which are signaling proteins that modulate immune function (Guzmán et al., 2009). Of particular interest is Interleukin-6 (IL-6), which in a recent prospective cohort study of 6,755 older persons, best predicted all-cause mortality compared to most other cytokines (Varadhan et al., 2014).

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Importantly, not all older adults with chronic disease experience the same disease progression. Of the many factors influencing this, psychological states such as self-efficacy influence both subjective and objective health outcome measures. Self-efficacy is the confidence to successfully perform behaviors or influence life domains (Albert Bandura, 1997). It is a dynamic concept highly influenced by social context, personal perception and physiology. Coping self-efficacy involves cognitively, emotionally and outwardly engaging one's resources to respond best to a perceived burden. It includes the domains of problem solving (i.e. evaluating a challenge in multiple ways to make the solution easier to achieve), emotional regulation (i.e. stop or redirect unpleasant emotions or thoughts) and social coping (i.e. reaching out to one's social network for help). It is particularly relevant to older adults with chronic disease as the psychological stress of aging and chronic disease negatively affects health outcomes (Gaffey et al., 2016; Prior et al., 2016).

Self-efficacy affects the immune system and cytokines through the stress response network. If someone appraises a situation as beyond their control and perceived abilities, it manifests as stressful and activates the stress response network, including cytokines within the immune system(Lazarus & Folkman, 1987; Maddux, 1995). This direct effect of self-efficacy on physiology was examined in small interventional studies that showed an attenuation of the proinflammatory cytokine Interleukin-2 once mastery over a stressful situation was obtained(Wiedenfeld et al., 1990). Other studies have shown similar decreases in heart rate and catecholamine secretion with acquisition of higher coping self-efficacy(A Bandura et al., 1985; Albert Bandura et al., 1988). Mausbach and colleagues (2011) showed an interaction effect between coping self-efficacy and role stress on IL-6 in a cross-sectional study of Alzheimer's caregivers (N= 62) concluding that high coping self-efficacy may be protective against increases in IL-6 and chronic disease progression. However, all of these studies enrolled small numbers of healthy subjects and, Bandura's work was all performed on middle aged persons.

In the context of health and illness, higher self-efficacy is associated with better health behavior and outcomes. For example, increases in self-efficacy are associated with decreased blood glucose levels in diabetic patients (Walker J et al., 2014; Zulman et al., 2012), increased cardiac function (Sarkar et al., 2009), decreased dyspnea symptoms in patients with COPD (Jackson E et al., 2014), and decreased pain associated with arthritis (Lorig et al., 1989). Self-efficacy has been deemed important in chronic disease management in an older adult population as well (Motl & McAuley, 2010).

Using a psychoneuroimmunological conceptual approach along with self-efficacy theory, we hypothesize that higher self-efficacy mechanistically interacts with the physiological stress response networks acting as a buffer to the higher psychological stress burden of chronic illness in older adults(Maddux, 1995; Mccain et al., 2005).Therefore, the purpose of this study was to evaluate the direct association between coping self-efficacy and IL-6 in an older adult population with chronic illness. This adds to the current literature by evaluating the direct effect of coping self-efficacy on IL-6 in older adults, rather than as a moderator of stressor effects or in middle aged adults (both described above).

To accomplish these objectives, we used sweat as our biospecimen source. Sweat is a biospecimen rich in many of the same proteins found in blood (Saga, 2002; Sonner et al., 2015; Wilke et al., 2007). Cytokines, including IL-6, have been successfully isolated from sweat (Cizza et al., 2008; M. Hladek et al., 2020). There is also preliminary research that sweat cytokine values correlate with serum findings (Marques-Deak et al., 2006). Sweat is collected with the sweat patch, a non-invasive, band-aid like sweat collection device that traps electrolytes and proteins in an absorbent pad while allowing water to evaporate through its semi-permeable membrane. This method of collection was shown to be feasible in older adults (M.D. Hladek et al., 2018). Due to these findings, feasibility and ease of collection, sweat science continues to garner much interest (Heikenfeld, 2016; Mena-Bravo & Luque de Castro, 2014).

We also hypothesized that depression mediates the association between coping self-efficacy and inflammation. The self-efficacy theory of depression states that low appraisals of one's abilities leads to decreased motivation, lower expectations and goals and, feelings of worthlessness and inadequacy(Maddux, 1995), which all contribute to depressive symptoms. This relationship is likely bidirectional too with depressive symptoms contributing to someone's sense of what they can successfully accomplish.

2. Methods

This cross-sectional study was conducted over two study visits. The first visit included screening for eligibility, consent, applying the sweat patch, taking blood pressure and heart rate measurements, and reviewing the questionnaire. Written consent included all required elements and participants were given opportunities to ask questions. In between visits, the participant completed the questionnaire. The second visit took place 72 hours later and consisted of patch removal and questionnaire collection. Each participant received a $25 gift card for participation.

2.1. Participants:

Participants were primarily recruited using convenience sampling from an active older adult retirement community using flyers and in person recruitment tables. A mutually agreeable time was found to meet in person at each participant's home for the study visits. See Table 1 for inclusion and exclusion criteria.

2.2. Measures and Data Collection

IL-6 Levels.—A sweat patch was used for IL-6 collection. It is a non-occlusive, hypoallergenic and FDA-cleared collection device, like a small adhesive bandage, that captures sweat as its specimen source (PharmChem, Fort Worth, TX). Patches were placed on the participant's abdomen at visit 1 and removed 72 hours later (+/− 90 minutes) at visit 2. The abdomen was chosen because of its predominance of eccrine sweat glands (as opposed to apocrine glands) and its decreased sympathetic nervous system (SNS) innervation relative to other areas of the body to avoid sweat capture due to SNS activation (Wilke et al., 2007). Participants could shower and perform daily activities but were asked to avoid vigorous exercise in order to capture insensible sweat loss and avoid exercise-

induced sweat loss. The sweat patch procedures, sweat extraction and analysis protocols are previously described in Hladek et.al. (2018).

Per Rissin and colleagues(2011), we used a single molecule enzyme-linked immunoarray (Simoa, Quanterix Corporation, Lexington, MA) for analysis. This immunoarray increases the measurement sensitivity by 200- to 500-fold compared to a traditional ELISA. All assays were run in duplicate. The manufacturer's lower limit of quantification was 0.011pg/mL for IL-6 (Quanterix Corporation, 2016). Inter-assay coefficient of variance (CV) was less than 10%. IL-6 values were included in analyses if the CVs were less than 20% according to acceptable practice with SIMOA technology (Olivera et al., 2015). This resulted in the exclusion of 20 samples for IL-6 analysis. However, for IL-6 values below the lower limit of quantification with CVs above 20%, the manufacturer's lower limit of quantification was used as that sample's value (Croghan & Egeghy, n.d.; EPA Quality Staff, 2006). The high CVs in the presence of low detection limits were likely the result of very low sample values. If we excluded these values completely from the analysis, we would introduce bias towards higher IL-6 levels in our sample. 25 samples for IL-6 were included using this method. The average CV for IL-6 was 9.2%. All data was de-identified.

Self-Efficacy.—The Coping Self-Efficacy Scale was used, which consisted of 13-items on a 10-point Likert type scale with labels at 1= "not at all confident", 5="moderately confident" and 10="totally confident"(Chesney et al., 2006). It contained 3 subscales for problem-solving, emotional regulation and social coping with Cronbach alpha's for each subscale of 0.91, 0.91, and 0.80 respectively. Coping self-efficacy and subscales were dichotomized into low and high self-efficacy based on the value of five on the 10-point Likert-type Coping Self-Efficacy scale. Five was chosen as the cut off because it was labeled and interpretable on the scale with a description as "moderately confident". Each question started with: "When things aren't going well for you, or when you're having problems, how confident or certain are you that you can do the following:" The questions would end with statements such as: "Make a plan of action and follow it when confronted with a problem". Of particular interest for our findings, the 3-item social coping self-efficacy (SCSE) subscale statements were: 1. "get emotional support from friends and family", 2. "get friends to help you with the things you need" and 3. "make new friends".

Co-Variates.

Social Support.: The ENRICHD Social Support Instrument was used, which consisted of 7-items, 6 on a 5-point Likert-type scale and one yes/no question asking about living alone (Vaglio et al., 2004). It asks 4 questions that begin with: "Is there someone available to you to…" and asks about giving emotional support, advice, and help with daily chores. Its scoring ranged from 6-30 with a Cronbach's alpha of 0.88.

Perceived Stress.: The perceived stress scale was used to measure cognitive appraisal and perceptions of stress over the last month using 10 items on a 5-point Likert scale (Cohen et al., 1983; Ezzati et al., 2014).

Life Events.: The Homes Rahe Social Readjustment Scale created a cumulative score based on weighted answers to 43 life events occurring within the last year (Holmes & Rahe, 1967; Holt et al., 2012). The higher the measure, the greater likelihood of illness in the near future.

Financial Strain.: The Financial Strain Instrument consists of a 3-item, 4-point Likert-type scale assessing difficulty paying bills, buying food and affording medication care (Cornoni-Huntley et al., 1993). Financial strain was dichotomized so that if someone indicated any financial strain on any of the 3 items, they were characterized as having financial strain.

Medical History and Socio-Demographics.—Participants were asked about chronic conditions using the Charlson Co-morbidity Index (Rozzini et al., 2004). Socio-demographics, blood pressure, heart rate, and self-disclosed weight, height and smoking status were recorded.

Depressive Symptoms.—The 8-item patient health questionnaire (PHQ8) was used to address depressive symptoms, a potential mediator of the self-efficacy-inflammation association as a sensitivity analysis. It asks questions like: ‘Over the past two weeks were you bothered by feeling down, depressed or hopeless” with answer options of ‘not at all’, ‘several days’, ‘more than half the day’ or ‘nearly every day’(Kroenke et al., 2009). Internal consistency for this sample was 0.79.

2.3. Data Analysis:

G*Power version 3.1.9.2 general power analysis software tool was used to perform a power analysis for multiple linear regression (fixed model, R2 increase) with an alpha of 0.05, power of 0.80 and the calculated effect size of 0.13, which yielded a total sample size of 135 study participants.

Means, standard deviations and proportions were used to describe variable distributions. All cytokines were log transformed to reduce the positive skewness in their distributions. Simple linear regressions examined the unadjusted relationships between each of the potential 13 covariates (age, sex, race/ethnicity, education, life events, Charlson co-morbidity, BMI, heart rate, blood pressure, perceived stress, financial strain and social support, self-efficacy and subscales) and our primary outcome variable, IL-6. Results with 2-tailed $p<0.05$ were considered significant. SCSE and financial strain showed significant unadjusted associations with IL-6. Simple linear regression also examined the relationships between our main independent variable, self-efficacy, and each of the other co-variates. Multi-collinearity was evaluated using variance inflation factors, which were all below 1.5. The assumptions of this parametric analysis were also checked with residual analyses, assessment of heteroscedasticity and influential points. One high leverage point was discovered whose sample characteristics did not differ in any meaningful way from the rest of the sample. Therefore, the point was included in the final analysis but results without inclusion of this outlier are also discussed.

Selection of covariates for multiple linear regression modeling was guided by the literature with input, recognizing limitations to covariate inclusion due to our sample size, from our simple linear regression analyses. Age, sex, race and co-morbidities were included

because of their associations with IL-6 in our literature review (Singh & Newman, 2011). Financial strain was added based on its association with IL-6. Social support was added based on its association with SCSE. The final model included age, sex, race, co-morbidities, SCSE, social support, and financial strain. Reporting includes simple linear regressions and bootstrapped (1000 reps) multiple linear regression results to enhance standard error validity. Statistical analysis was performed using STATA version 13.1 (StataCorp, College Station, TX).

3.0. Results

3.1. Participants

The 159 participants were 82 years old on average (SD +/− 6.3 years), and predominantly female (72.9%), white (92.8%) and highly educated (mean education 17 years +/− 3.1 years). This group was also relatively healthy with an average BMI of 27.2 (SD= +/− 5.3), average blood pressure and heart rate of 133/72 and 67.8, respectively, and average Charlson score of 1.19 (SD= +/− 1.45). See Table 2 for more detail on participant characteristics.

3.2. Psychosocial Factors

Social Coping Self-Efficacy (SCSE), a subscale of the Coping Self-Efficacy Scale, was significantly associated with the log of sweat IL-6, the main outcome variable (β= −0.544, *p*-value= 0.006) indicating that high SCSE was associated with lower log IL-6. (See Table 3) Spearman correlation of the 3 item SCSE subscale was 0.86 for this sample.

Differences in the distribution of characteristics between high and low SCSE were analyzed using T-tests (Table 2) with social support being the only variable significantly associated with SCSE (β=0.28; 95% CI, 0.015-0.041, p <0.0001).

The final bootstrapped regression model explained 13.6% of the log IL-6 variance. The final adjusted model included age, race, sex, SCSE, co-morbidities, social support and financial strain. Within these measures, only two were significant in the final model. Particularly, higher log IL-6 was associated with low SCSE (p=0.010; 95% CI, −0.937 through −0.132) and high financial strain (p=0.036; 95% CI, 0.031 through 0.910). See Table 3 for more detail. Importantly, when a high leverage point was removed from the final model, SCSE only approached significance (β=−0.38; CI −0.79-0.028, p=0.068) and the association with financial strain strengthened (β=0.517; CI 0.220-0.814, p=0.001). Financial strain remained significant in all models (See Table 3).

3.3 Stratified Analysis

To further distinguish the role of depressive symptoms in our model, we conducted a stratified analysis of our bootstrapped adjusted model for those below the clinical threshold for depression (PHQ-8 < 10). The beta coefficient for SCSE changed only slightly from −0.534 (SE=0.26, CI= −1.05 −0.018) in the main model to −0.532 (SE=0.31, CI= −1.13 0.07) in the sensitivity analysis (N=123) but the p-value was no longer significant (p-value=0.043 vs p-value= 0.082). Stratified analysis of those with depressive symptoms (PHQ-8) ≥ 10 was not possible due to the small sample size.

4.0. Discussion

The purpose of this study was to evaluate the association between coping self-efficacy and IL-6 as well as evaluations of potential important co-variates. Our main finding was that high social coping self-efficacy (SCSE), a subscale for coping self-efficacy, was significantly related to lower sweat IL-6 and remained significant after adjustment. These results are consistent with those of Mausbach et.al. (2011) which found that higher coping self-efficacy significantly attenuated the relationship between caregiver role stress and serum IL-6. They hypothesized that self-efficacy was potentially protective against the effects of psychological stress on increasing inflammation. Likewise, these results build upon Bandura's work showing that higher coping self-efficacy attenuated catecholamine release in participant's experiencing a stressor (1985). Catecholamines are an integral part of the sympathetic nervous system, which is bidirectionally associated with the immune system and inflammation, including IL-6. Wiedenfeld and colleagues (1990) were able to show longitudinally that increases in self-efficacy led to increases in lymphocytes and helper T cells, which are also associated with IL-6.

It is not surprising that evidence emerged to support an association with the social coping subscale. The concept of self-efficacy fundamentally relates to social support in that 2 of the 4 mechanisms by which Bandura hypothesizes acquiring self-efficacy involve one's social environment, namely verbal persuasion (positive coaching) and vicarious experience (modelling of thoughts and actions). More recently, Brembo and colleagues (2017) found that both social support and self-efficacy were significant predictors of total hip replacement recovery. Tovar and colleagues (2015) found that both self-efficacy and social support were mediators of adherence among participants with both diabetes and depression.

Although the association between social support and SCSE is strong in our study, it is interesting to note that social support itself was not a significant predictor of IL-6. One reason for this might be that SCSE specifically assesses someone's ability to *ask* their social network for help. The confidence to ask for help is distinct from *having* social support available. Asking for help can illicit feelings of inadequacy or fear of rejection and, therefore, asking for help can feel daunting.(Bohns & Flynn, 2010; Mackenzie et al., 2019) Our social support instrument (ENRICHD) captured social support using questions like "is there someone available to you…". The instrument did not capture the agentic capacity needed to activate that network into practical help. This confidence in the ability to activate one's social network (i.e. SCSE) may be an important distinction between SCSE and social support to explore in future research.

We also hypothesized that depressive symptoms partially mediated the relationship between SCSE and IL-6. Our stratified analysis for those without depression revealed a minimal change in the association between the full model and the stratified model lending evidence to the partial mediation hypothesis of depressive symptoms in our model but also clearly showing a direct effect of SCSE on IL-6, apart from depressive symptoms. SCSE in the stratified model was no longer significantly associated with IL-6 and, we hypothesize that this change in significance is due to the decreased sample size and increased standard error in the bootstrapped stratified analysis and not due to a dramatic change in our findings.

We found that high financial strain was associated with high IL-6. Twenty percent of people with low SCSE indicated financial strain compared to 10% with high SCSE. This is not surprising given that financial strain can be thought of as a proxy of chronic stress, especially in older adults with fixed incomes. Lazzarino and colleagues(2016) recently operationalized chronic stress using financial strain and found that fibrinogen (another inflammatory marker) was associated with a 5-fold increase in cardiac troponin, a heart disease marker, only in those indicating financial strain. The negative effect of financial strain did not suppress the positive effect of SCSE on IL-6 in the final model.

Our study is not without limitations. This study was cross-sectional allowing for no causal inferences. This study used a convenience sample of highly educated, mainly female white older adults. Therefore, there was less sociodemographic variation than is found in the general population. The participants were also healthy with an average BMI of 27, average blood pressure of 133/72, and mean heart rate of 67- all of which make generalizability challenging and may also reduce our ability to detect associations, making it difficult to interpret negative findings.

81% of adults aged 65 and older live with more than 2 chronic conditions (Buttorff et al., 2017). Our sample, however, had an average Charlson co-morbidity score of 1.19. This may account for why the Charlson co-morbidity variable was not significantly positively associated with IL-6 in our findings, which is a common research finding. It may be that this group has maintained a high level of disease control and therefore, has not suffered the same long-term sequelae associated with chronic conditions. Also, inflammation is usually associated with aging; yet, again, in our sample this was not observed. The hypothesis for why inflammation occurs with aging is proposed to result from cumulative inflammatory damage over the lifespan (Franceschi & Campisi, 2014). Therefore, given the overall health of the population, age may not play as important of a role in inflammation progression.

Additionally, this study used sweat as its biological specimen source. Individual sweat rates were not accounted for in this study. We were careful to instruct participants to avoid vigorous exercise in order to collect only insensible sweat loss and avoid the anti-inflammatory qualities of IL-6 post-exercise. The mechanism by which inflammatory proteins are secreted into the sweat lumen is also unknown, making it more challenging to interpret these results clinically. Past research with this same extraction method, however, has successfully shown an increase in cytokine protein amounts in older adults compared to younger adults (M.D. Hladek et al., 2018). It has also been shown to correlate with serum levels of inflammatory proteins, including IL-6, in one small study (Cizza et al., 2008; Marques-Deak et al., 2006). We recognize, though, that sweat science is still emerging as a viable specimen source in research and measurement error remains a concern.

Lastly, our results vary based on the leverage of one sample. This finding demonstrates the need for further research with larger sample sizes to more conclusively evaluate any adjusted association between IL-6 and SCSE.

Still, there is potential that an important relationship exists between SCSE and IL-6. SCSE could be an important intervention target for older adults because it is malleable, making

it an excellent target for intervention. (1) Exploring the barriers and fears older adults face in asking for help, (2) brain-storming and problem-solving around those barriers/fears, and (3) action-planning around specific goals for activating one's social network may be helpful techniques to increase SCSE.

In summary, this study offers preliminary insight into self-efficacy and inflammation in older adults with chronic illness. We found that high SCSE was associated with lower IL-6 and, high financial strain was associated with higher IL-6. It may be that SCSE is acting as a source of physiologic resilience. We also recognize that caution must be used in interpreting these findings given the study limitations. The potential implications of this study necessitate further longitudinal research looking at inflammation and self-efficacy using larger sample sizes, other biological specimen sources and longitudinal data.

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Table 1.

Inclusion & Exclusion Criteria

Inclusion:
(1) Age ≥ 65 years;
(2) Presence of at least one chronic condition *;
(3) Ability to speak, read and understand English
Exclusion:
(1) ≤ 2 Mini-Cog score,
(2) Presence of active cancer treatment or terminal illness;
(3) Presence of degenerative neurological condition **;
(4) Use of specific anti-inflammatory drags including IL-6 inhibitors and TNF-α inhibitors ***

* Chronic conditions include: diabetes, arthritis, high cholesterol, hypertension or cerebrovascular, cardiovascular, renal, chronic lung or liver disease.

** Degenerative neurological conditions including: Alzheimer's Disease, Parkinson's Disease, Huntington's Chorea, Guillain-Barre Syndrome, Amyotrophic Lateral Sclerosis

*** Drugs in this category include: infliximab (Remicade), adalimumab (Humira), certolizumab pegol (Cimzia), etanercept (Enbrel), tocilizumab (Actemra), atlizumab (RoActemra), sirukumab (Sylvant)

Table 2.

Participant Demographics and Health Characteristics by High and Low Social Coping Self-Efficacy

Participant Characteristics	Total (n=159)	Low SE (n=31)	High SE (n=128)
Age, mean (SD)	82.0 (6.3)	81.7 (7.5)	82.1 (5.9)
Sex, n female (% female)	116 (72.9%)	23 (74.2%)	93 (72.7%)
Race, n (% white)	142 (92.8%)	29 (96.7%)	113 (91.9%)
Education, mean # of years (SD)	17.0 (3.1)	16.8 (2.9)	17.1 (3.2)
Charlson Co-Morbidities, mean (SD)	1.19 (1.45)	1.23 (1.63)	1.19 (1.41)
Body Mass Index, mean (SD)	27.2 (5.3)	27.2 (5.9)	27.2 (5.2)
Tobacco, n (% former or current)	75 (47.7%)	12 (41.4%)	60 (49.18%)
Blood Pressure, mean (SD)	133(15.2) / 72 (7.4)	133 (14.4) / 73 (7.6)	133 (15.4) / 71.8 (7.4)
Heart Rate, mean (SD)	67.8 (7.8)	67.5 (8.5)	67.9 (7.6)
Social Support, mean (SD)	24.3 (4.5)	21.3 (5.2)	24.9 (4.0)*
Life Events, mean (SD)	90.48 (71.4)	109.0 (76.0)	86.0 (69.8)
Perceived Stress, mean (SD)	2.35 (0.29)	2.36 (0.34)	2.34 (0.28)
Financial Strain, n (% yes)	19 (12.0%))	6 (20.0%)	13 (10.1%)

*Significant Difference between High and Low Social Coping Self-Efficacy Groups at $p<0.0001$

Table 3.

Unadjusted and Adjusted Regression Models for Coping Self-Efficacy and Subscales Predicting Log of Sweat IL-6

Characteristics	***Unadjusted Model β (CI)***	**Final Adjusted Models β (CI)**			
		Coping Self-Efficacy	**Social Coping Subscale**	**Problem Solving Subscale**	**Emotional Regulation Subscale**
Coping SE	−0.131 (−0.595 0.334)	−0.009 (−0.418 0.400)			
Social Coping SE	−0.544 (−0.933 0.155)[a]		−0.534 (−1.03 −0.035)[b]		
Problem-Solving SE	−0.357 (−0.832 0.118)			−0.229 (−0.626 0.169)	
Emotional Regulation SE	−0.181 (−0.556 0.193)				−0.196 (−0.642 0.250)
Age	−0.022 (−0.045 0.002)	−0.017 (−0.041 0.006)	−0.014 (−0.039 0.011)	−0.019 (−0.042 0.005)	−0.019 (−0.043 0.004)
Sex	0.189 (−0.146 0.523)	0.142 (−0.281 0.566)	0.190 (−0.215 0.595)	0.137 (−0.288 0.563)	0.128 (−0.301 0.557)
Race	0.123 (−0.450 0.695)	−0.49 (−0.633 0.535)	0.021 (−0.581 0.624)	−0.018 (−0.601 0.572)	−0.008 (−0.600 0.584)
School	0.008 (−0.041 0.057)		--		
BMI	0.002 (−0.026 0.029)		--		
Co-Morbidities	−0.076 (−0.180 0.029)	−0.073 (−0.191 0.045)	−0.083 (−0.193 0.026)	−0.069 (−0.187 0.048)	−0.070 (−0.187 0.047)
Blood Pressure	0.260 (−0.416 0.935)		--		
Heart Rate	−0.003 (−0.023 0.018)		--		
Tobacco use	0.004 (−0.300 0.308)		--		
Social Support	−0.012 (−0.047 0.023)	−0.001 (−0.038 0.036)	0.010 (−0.025 0.045)	0.0002 (−0.036 0.037)	0.0001 (−0.036 0.036)
Life Events	0.001 (−0.001 0.003)		--		
Perceived Stress	0.125 (−0.398 0.698)		--		
Financial Strain	0.534 (0.101 0.970)[b]	0.517[a] (0.241 0.793)	0.470[a] (0.155 0.785)	0.474[a] (0.193 0.756)	0.485[a] (0.183 0.788)

[*] Final Models all Bootstrapped with 1000 reps, N=134

SE = self-efficacy; CI = Confidence Interval

a: p-value < 0.01;

b: p-value<0.05