Measurement Invariance in the CESD-8 and Assessment of Mood Correlates Between American and Mexican Community Studies on the Multisystemic Geriatric Depression Cycle

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MEASUREMENT INVARIANCE IN THE CESD-8 AND ASSESSMENT OF MOOD CORRELATES BETWEEN AMERICAN AND MEXICAN COMMUNITY STUDIES ON THE MULTISYSTEMIC GERIATRIC DEPRESSION CYCLE

By

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ABSTRACT

The Health and Retirement Study is a longitudinal study that is conducted every other year in the United States and has served as a precursor to similar studies across the world, such as the Mexican Health and Aging Study. The purpose of the current project was evaluating the relationship between depressive symptoms, inflammation and physical activity levels using data from the HRS and MHAS studies. The current project was divided in two main studies. The first study aimed at establishing measurement invariance in a brief questionnaire about depressive symptoms, the CES-D 8. The second study looked at various theories of depression including the inflammation theory and the economic theory of depression and other constructs in a larger, novel model called the Multisystemic Geriatric Depression Cycle. For the first study, MPlus was used to conduct measurement invariance analyses in 15,319 participants ages sixty-five and older from both datasets. For the second study, various regression, mediation and moderation analyses were conducted using variables like C-reactive protein, physical activity, loneliness, frequency of social contact and perceived social support. Because measurement invariance was not supported by results in Study 1, analyses and results in Study 2 were only interpreted for the HRS sample. Results from second study showed support for the inflammation theory of depression, partial support for the economic theory of depression with cross-sectional data, and the cross-sectional and longitudinal mediating effect of perceived negative social support in the relationship between loneliness and depression. This project highlights further need for improvement of measures administered cross-culturally to guarantee meaningful comparison of construct of depression among culturally diverse groups. This study adds to
the growing body of literature guiding harmonization efforts from the Program on Global Aging, Health and Policy.

*Keywords:* longitudinal study, older adult, HRS, MHAS, CESD-8, depression, measurement invariance, loneliness, physical activity, inflammation, perceived social support
“A mi madre y abuela Celida, mis protectoras; a Fita e Hilda, mis primeras educadoras; a mi padre y abuela Catalina, quienes me abrieron nuevos caminos y a mi tía Clara, mi luz acompañante.”

To my mother and grandmother Celida, my protectors; to Fita and Hilda, my first educators; to my father and grandmother Catalina, who opened new paths for me and to my aunt Clara, my accompanying light.
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CHAPTER ONE: BACKGROUND

Longitudinal Designs and Public Health Research

Longitudinal studies are an important source of information for public health and healthcare trajectories. Longitudinal data facilitates the study of healthcare trajectories, the impact of major developmental milestones, and the interactive interrelationships between variables over time, while controlling for the influence of demographic, comorbidity, and other factors (Solomon, Kirwin, Van Ness, O'Leary, & Fried, 2010; Stanziano, Whitehurst, Graham, & Roos, 2010). This is particularly important for older adults who are disproportionately subject to comorbid health conditions and, often, declining capacity for independence (Fries, 2005).

The Health and Retirement Study (HRS) is the largest biennial longitudinal study investigating work, aging and retirement in the United States that has been conducted since 1992, studied more than 43,000 individuals and resulted in nearly 4000 journal articles using numerous distinct variables so far (Fisher & Ryan, 2017). The Health and Retirement Study data is sponsored by the National Institute on Aging (grant number U01AG009740) and is conducted by the University of Michigan (Health and Retirement Study). The HRS is unique in that it was designed by an interdisciplinary team with the goal of studying the trajectories of older adults’ economics, health, psychological well-being and demographics. The basic sampling goals included community dwelling, non-institutionalized individuals, who are representative of the general population of older adults, and their spouses to gather data not only on the individual but also at the household level. One strength of the HRS is the use of a steady-state sample design, meaning that they add a new cohort of individuals every six years. These data have contributed
to a number of scientifically and socially impactful discoveries, such as quantifying the relationship between health and wealth among retired Americans (Willis, 1999), gauging the impact Social Security policy on retirement trends (Bound, Schoenbaum, Stinebrickner, & Waidmann, 1999; Gustman & Steinmeier, 2009), and characterizing the relationship between cognitive job complexity and cognitive functioning before and after retirement (Fisher et al., 2014).

The HRS is the model for a range of similar studies across the world, which are referred to as sister studies. Some of these similar longitudinal studies include the Mexican Health and Aging Study (MHAS); Survey of Health, Ageing and Retirement in Europe (SHARE); Korean Longitudinal Study of Aging (KLoSA); English Longitudinal Study of Ageing (ELSA); and many others. The goals of these studies are similar to that of the HRS. For example, the MHAS, conducted since 2001, was designed with the goal of examining ageing processes, disease and disability burden in a large sample of older Mexicans. The protocols of the sister studies are designed to be similar, making these studies a terrific resource for the study of aging trajectories across cultures. The MHAS has contributed to more than 100 peer-reviewed publications from different disciplines including but not limited to epidemiology, microeconomics, public health and many others (Wong, Michaels-Obregon, & Palloni, 2015). These longitudinal studies provide the opportunity to study human aging cross-culturally, cross-nationally and cross-language simultaneously (Arokiasamy, Bloom, Lee, Feeney, & Ozolins, 2012; Börsch-Supan et al., 2013; Kowal et al., 2012), and they also present researchers with the unique capacity to study mental health including depression within the context of aging trajectories (Aldwin, Spiro III, Levenson, & Cupertino, 2001; Hong, Hasche, & Bowland, 2009; Yang, 2007).
Theoretical background: Depression in Older Adults

Depression is a common but serious psychological disorder and illness that goes beyond mere sadness, and it affects feelings, thinking and behavior. The Diagnostic and Statistical Manual-5 (American Psychiatric Association, 2013) characterizes the symptoms of depression as pervasive sadness, anhedonia, loss of energy, dysregulation in eating behavior, sleep impairment, difficulty in concentration among many others. While symptoms listed in the DSM-5 are very similar to those listed in the International Classification of Diseases-11 (World Health Organization, 2018); the DSM-5 is a manual of culturally bound disorders. Depression symptoms manifest differently as a function of cultural factors. In addition to cultural factors, it has been well-established that many mental health disorders, depression included, present somewhat differently among older adults (Blazer, 2003). Depression in older adults presents with fewer cognitive-affective symptoms, such as dysphoria and worthlessness, than depression in younger adults (Fiske, Wetherell, & Gatz, 2009; Hegeman, Kok, Van der Mast, & Giltay, 2012). On the other hand, somatic symptoms and anhedonia are more common in depression in older adults than depression in younger populations (Kiosses & Marino, 2019). Cognitive impairment and executive dysfunction are also more prevalent in older adults than in younger adults who are depressed (Butters et al., 2004; Fiske et al., 2009).

Depression in older adults is common even though it is not a normal part of the aging process (National Institute of Aging, 2017; National Institute of Mental Health, 2013). Depression in adults 65 and older was among the most common chronic conditions affecting Medicare beneficiaries. The prevalence rate for depression in the United States was 13.6% in 2014, which was higher than that of cancer, HIV, osteoporosis, chronic obstructive pulmonary
disease and other conditions (Centers for Medicare and Medicaid Services, 2014). Prevalence rates vary widely depending on the country and selection criteria that are being used to diagnose depression. Depression in older adults is frequently comorbid with other physical and mental illnesses that are distinctive to the process of aging such as cardiovascular disease, diabetes and Alzheimer (Blazer, 2003). Thus, due to its particular clinical presentation and its comorbidity with other medical illnesses, depression in older adults often goes undiagnosed (Hybels, Blazer, Landerman, & Steffens, 2011; National Institute of Aging, 2017). Furthermore, depression is the sixth costliest health condition in the United States costing $71 billion in the span of thirteen years (Dieleman et al., 2016; Winerman, 2017).

Depressive symptomatology among older adults relates to a variety of medical and demographic risk factors. Specifically, loss of independence resulting from motor impairment and medical burden, chronic pain, cognitive decline, involuntary employment exit (Hyde, Hanson, Chungkham, Leineweber, & Westerlund, 2015) or retirement (Olesen, Rod, Madsen, Bonde, & Rugulies, 2015), and social withdrawal (Dorfman et al., 1995; Nicholson, 2012; Teo et al., 2015). Other factors, such as socio-economic status, gender and ethnicity also play a role in rates of depression. Furthermore, medical comorbidity, cognitive impairment, disability, frailty, and infections can adversely direct the course of depression in older adults, and depression in turn can aggravate these medical and functional problems and confer additional impairment.

Onset and development of depressive symptoms is associated with a broad range of risk factors and correlates. These are described using different theoretical frameworks, such as the economic theory of depression, the inflammatory theory of depression, the vascular depression hypothesis, and others. The inflammatory theory of depression argues that active inflammation
and activation of the enzyme indoleamine-2,3-dioxygenase (IDO) in the liver results in degradation of tryptophan into higher concentrations of potentially neurotoxic kynurenine metabolites and reduction in the production of serotonin, which has been associated with neurodegenerative processes and depressive symptoms (Dantzer, O’Connor, Lawson, & Kelley, 2011; Réus et al., 2015). Thus, tryptophan degradation also would mean that it becomes unavailable for 5HT synthesis. Although the prevalence of individuals with depression due to inflammatory processes varies depending on clinical sample and medical comorbidities, prevalence of depression has been shown to be high in individuals with coronary heart disease (Dantzer, O’Connor, Freund, Johnson, & Kelley, 2008; G. E. Miller, Stetler, Carney, Freedland, & Banks, 2002; Nikkheslat et al., 2015), cancer (Lanser et al., 2020; Sforzini, Nettis, Mondelli, & Pariante, 2019) and inflammatory bowel disease (Kochar et al., 2018; Mikocka-Walus et al., 2007; Moulton et al., 2019) among many other conditions with well-known inflammatory responses in the body. The prevalence of low-grade inflammation in depression has been shown to be 27%. Furthermore, a quarter of individuals experiencing depression exhibit low-grade inflammation and over half appeared to have somewhat elevated CRP (Osimo, Baxter, Lewis, Jones, & Khandaker, 2019). C-reactive protein (CRP) is commonly used as a marker for inflammation; several studies have shown a positive association between CRP levels and depressive symptoms, and several studies agree that this relationship is attenuated when controlling for BMI (Au, Smith, Gariépy, & Schmitz, 2015; Howren, Lamkin, & Suls, 2009). Inflammation has been shown to be secondary to higher BMI with higher macronutrients in adipose tissues resulting in higher concentrations of inflammatory mediators and secretion of CRP by the liver (Ellulu, Patimah, Khaza’ai, Rahmat, & Abed, 2017). Furthermore, past
literature has shown that psychosocial stressors, poor diet, sedentary behavior and cigarette smoking can produce an inflammatory response (Berk et al., 2013; A. H. Miller & Raison, 2016). Inflammation is also a neuroprogressive pathway of depression meaning that individuals with depression are at risk for higher inflammation over time (Berk et al., 2013; Zunszain, Hepgul, & Pariante, 2012).

In turn, the health economics depression literature (Strulik, 2019) reports that depression reduces lifetime utility, meaning that depressed individuals have less incentive to behave in a healthy way resulting in that they invest less in their health, exercise less, consume more unhealthy goods, and save less for health-related expenses when compared to their healthy counterparts. In this model, lifetime utility is comparable to life satisfaction. According to this model, depression does not modify individual preferences for certain activities but merely reduces the capacity to experience pleasure from an activity that provides utility. Thus, the health economic theory argues that depression causes changes in health behavior and health outcomes, but not the other way around, so aging and longevity are endogenous variables in this model. Health deficits caused by depression accumulate over a lifetime increasing mortality risk among depressed individuals. (Strulik, 2019). The maxim argued by this theory that individuals with depression are more likely to engage in unhealthy behaviors such as smoking, using alcohol and other substances, sedentary behavior and poor diet is supported by past literature (Hallgren et al., 2016; NSDUH, 2004; Schulz, Drayer, & Rollman, 2002; Swendsen & Merikangas, 2000; Teychenne, Ball, & Salmon, 2010).
CHAPTER TWO: STUDY ONE

Abstract

Measurement invariance of various measures has become more commonplace especially in the field of cross-cultural research. The American Health and Retirement Study created a remarkable standard for public socioeconomic and healthcare research for retirement-aged populations in the United States, and similar projects called “sister studies” are being conducted in various countries. Comparability of data between these samples has not been thoroughly studied. The purpose of this first study was to establish measurement invariance in a brief mood measure, the CES-D 8, used in two longitudinal datasets, the HRS and the MHAS in two different countries, the United States and Mexico respectively. MPlus Editor Version 1.7 was used to conduct these analyses using CFA. Results showed that measurement invariance was not supported in a series of two steps proposed in prior literature for measurement invariance with dichotomous data. These findings implied that there were differences in ways of responding between HRS and MHAS participants at the conceptual level, and their responses might not be comparable. It also means that further analyses performed with MHAS participants in this study that make use of the CESD-8 variables cannot be meaningfully interpreted. Future studies should use measures of depressive symptoms that have been shown to be invariant. At the same time, cross-cultural research should include instruments with items that characterize the construct of depression in a particular population.
Introduction

Measurement Invariance

Considering differences between cultures, one question to consider is whether results obtained using data from harmonized longitudinal datasets like the Health and Retirement Study and its counterparts in other countries are truly comparable considering cultural differences, especially between individualistic and collectivistic cultures. For example, the construct of depression varies across cultures and samples whereas the expression of depression in Western individuals is more clearly explained by cognitive and affective symptoms, such as anhedonia and negative thoughts, while non Western individuals as well as some Hispanic groups tend to focus more on the somatic aspects of depression, such as sleep disturbances and appetite (Huang, Beshai, & Yu, 2016). These cultural variations have been found by using varied popular measures of depression, such as the CES-D and the BDI-II (Huang et al., 2016; Kanazawa, White, & Hampson, 2007). Cultural values influence cognitions and behaviors, and it is assumed that people in individualistic cultures have more private self-cognitions than those with a collectivistic background (Darwish & Huber, 2003). However, it is important to recognize that while Mexico is considered primarily collectivistic and the United States primarily individualistic, there are within-group differences to the level in which individuals in these countries assume the values that characterize their cultures.

Another issue when evaluating research across nationalities could be that of differences in language considering that measures created in one country might not necessarily translate well. For example, the CES-D has been translated in multiple languages including English and Spanish. The CES-D has been shown to be reliable and valid to measure depressive symptoms
for use with individuals with various clinical presentations in different clinical settings and with
different cultural background (Cosco, Prina, Stubbs, & Wu, 2017; Thombs et al., 2008; L.
Yang, Jia, & Qin, 2015); however, the great majority of the analyses in the literature measure
quality of this measure by using classical test theory (CTT), which focuses on true and error
scores of observed variables. Within the context of CTT, groups’ overall means are compared,
and reliability and validity are considered general psychometric attributes of the measure.
Furthermore, CTT is rooted in the assumption that the theoretical construct is the same across
nationalities, and that the items composing the test are interpreted similarly by different groups.
However, equivalency of measures across cultural, ethnic, and nationality groups is poorly
understood. Without first establishing that these measures are equivalent across groups, we
cannot confidently compare those groups. The uncertainty of measurement equivalence between
HRS sister studies is one barrier to the intended use of these complementary international
datasets. There are different methods in order to establish measurement equivalency including
confirmatory factor analyses (CFA) and item response theory (IRT) approaches, and
appropriateness of each depends on the type of analyses being conducted (Meade &
Lautenschlager, 2004). While CFA examines covariance between test items, IRT examine
participant item responses (Reise, Widaman, & Pugh, 1993). For the purposes of this study, CFA
was sufficient to study equivalency for CESD-8 use across cultures.

While measurement invariance (MI) analyses using CFA is well established in the
literature by now, the majority of studies evaluating MI to date use the modeling proposed by
Meredith and then reviewed by Vandenberg (Vandenberg & Lance, 2000; Wu & Estabrook,
2016). However, this method was mostly developed for continuous data, which has means,
variances and covariances. Categorical or dichotomous data lacks all these required features; thus, different modeling approaches need to be applied. The method identified by Millsap and Yun-Tein (Millsap & Yun-Tein, 2004) proposes thresholds, factor loadings, intercepts and unique variances as constraints that can be used in models using categorical data. Furthermore, different models were proposed by Wu and colleagues depending on whether the data was dichotomous or polytomous. Muthen and Muthen (L. Muthén & Muthén, 1998) also have specifications for modeling measurement invariance using categorical data. It is worth noting that the items in the CES-D as they appear in these community studies are dichotomous. Thus, past literature on measurement invariance as described above guided modeling decisions on this particular study. This is described in greater detail in the Method section below.

One final word pertains to scale characteristics that could affect measurement invariance analyses. It is worth noting that unlike the Geriatric Depression Scale, the original CES-D was not originally designed for older adults, but it was established with adults between the ages of 18 and 64. The original CES-D consists of 20 items, but different versions have been created including a 12, 10, and 8-item versions (Assari & Moazen-Zadeh, 2016; Baron, Davies, & Lund, 2017; Karim, Weisz, Bibi, & ur Rehman, 2015). It is quite known that test length as well as level of difficulty of test items, among many other factors, can affect reliability and validity of a test. Prior examination of measurement invariance across groups using the original CES-D (Canady, Stommel, & Holzman, 2009; Fried et al., 2016; Rivera-Medina, Caraballo, Rodríguez-Cordero, Bernal, & Dávila-Marrero, 2010), did not focus on older adults (Morin et al., 2011; Skriner & Chu, 2014; Wang et al., 2013), or were conducted in European (Karim et al., 2015; Van de Velde, Bracke, Levecque, & Meuleman, 2010; Van de Velde, Levecque, & Bracke, 2009) or
Asian countries (Wang et al., 2013; Zhang et al., 2011). However, to the best of our knowledge, no study has been conducted evaluating measurement invariance of the 8-item CES-D, Spanish version, in a longitudinal sample of older adults in a highly collectivistic country like Mexico.

The purpose of this study is to first evaluate measurement invariance in the 8-item, Spanish version of the CES-D as completed by a sample of older adults in Mexico across different time points in comparison to a sample of older adults in the United States; measurement invariance analyses will be conducted using a series of hierarchical nested hypotheses. Once measurement invariance has been demonstrated by equal thresholds, factor loadings, intercepts, and residual variances between groups, then further analyses will be conducted on the relationships between depression as measured by the CES-D and other variables of interest in Study 2.

**Hypothesis 1.** There will be invariant thresholds between the Mexican and American samples as determined during a baseline model establishing configural invariance.

It is worth noting that performing further MI analyses will only be necessary if null for hypothesis 1 is not supported. Hypotheses 2 is nested in the sense that previous hypotheses need to be supported before performing subsequent analyses.

**Hypothesis 2.** There will be invariant thresholds, loadings and intercepts as evaluated simultaneously for both groups in a second model.
Method

This study include two main groups based on country of residence. One of the groups as described above was the HRS, a cohort study on health and aging on adults who are 50 years of age and older. This study is being conducted by the University of Michigan with support from the National Institute of Aging and the Social Security Administration. The complete HRS data set includes approximately 20,000 participants, and data collection has been taken place since 1992, and starting in 1996, data has been collected in waves every two years. Last available follow-up dates back to 2016 at the time this study was proposed. The comparison group included participants from the MHAS, a cohort study on health and aging on adults who are 50 years of age and older living in Mexico. The MHAS is a collaborative effort among researchers from the University of Texas Medical Branch (UTMB), the Instituto Nacional de Estadística y Geografía (INEGI, Mexico), the University of Wisconsin, the Instituto Nacional de Geriatría (INGER, Mexico), the Instituto Nacional de Salud Pública (INSP, Mexico), and University of California Los Angeles (UCLA). The MHAS is partly supported by the National Institutes of Health/National Institute on Aging and the INEGI in Mexico. The complete MHAS data set includes approximately 15,000 participants since the beginning, and data collection has been taken place since 2001; the MHAS has been conducted in the span of five waves in the years 2001, 2003, 2012, 2015 and 2018 although data for last wave was not available at the time this study was started.

One exclusionary criterion used in the current project was being under the age of 65 at any of the studies’ waves that would be included in statistical analyses. Although there might be various arguments as to what age is the minimal age for older adulthood, the age of 65 is selected
considering that this is the typical retirement age in many countries although there is a trend around the world to increase this based on increasing life expectancy (Hagen, 2018). Another exclusionary criterion in an attempt to minimize the impact of historic bias was excluding data prior to 2001, which applied mainly to the HRS cohort, considering that there is no data available for the MHAS cohort prior to 2001.

MPlus Version 8.4 (B. Muthén, Muthén, Asparouhov, & Nguyen, 2021) was used to conduct measurement invariance analyses on the CESD-8 scale for both longitudinal samples including American and Mexican cohorts. Item responses were dichotomous where 0 represented a negative response to that item and 1 represented a positive response. For items 4 and 6 in the CESD-8 scale, which are reverse-coded, appropriate adjustments were made before calculating total CESD-8 scores. Samples size for measurement invariance analyses using the Health and Retirement Study was 10,931 while sample size for the Mexican Health and Aging Study was 4,388. Process of selection of participants can be found in consort tables in Figure 1 and Figure 2.

Categorical data, and in this case, binary data, needed to be treated differently from continuous data in measurement invariance analyses. Otherwise, serious problems can arise on parameters, model fit and cross-group comparisons if the categorical nature of variables is not taken into account (Svetina, Rutkowski, & Rutkowski, 2020). The approach to measurement invariance for categorical observed variables has been already demonstrated and established in prior literature (Muthén & Asparouhov, 2002; Muthen & Christoffersson, 1981). The common practice is usually to identify a baseline model and then impose increasing parameters restrictions typically testing for configural invariance, followed by testing with equal loadings,
followed by constraints in intercepts and thresholds. However, a more recent approach is concerned with testing for threshold invariance first while establishing configural invariance, and then use invariance testing of loadings as a second step (Svetina et al., 2020; Wu & Estabrook, 2016).

Figure 1. Consort table for HRS sample selection. HRS = Health and Retirement Study; CES-D 8 = Center for Epidemiological Studies Depression, Eight-Item Version; RAND = HRS Longitudinal File = Streamlined data product containing information from Core and Exit Interviews of the HRS.
For the purposes of the current analyses, a baseline model for binary data was identified using threshold invariance. Since for binary items, a linear transformation on the latent variable in one group matches thresholds of the other group, configural invariance is equivalent to a baseline model of invariant thresholds (Wu & Estabrook, 2016). The next model tested for three types of parameters being invariant conjointly including invariant thresholds, loadings and intercepts. An intermediate step testing for threshold and loading invariance was not identified or subsequently tested because such model with binary data would be equivalent to the baseline.
model and as a consequence could not be tested by itself (Wu & Estabrook, 2016). The steps for measurement invariance analyses as described above can be found in **Figure 3** while corresponding syntax that was used can be found in the **Appendix B** section. Partial invariance or other methods to deal with failure to achieve measurement invariance are beyond the scope of this study (Svetina et al., 2020).

![Flowchart](image)

**Figure 3.** Flowchart of analyses procedures to determine MI in CES-D 8 (English and Spanish).
Results

The final HRS sample included in statistical analyses following MI analyses included 10,931 participants. Mean age of participants varied depending on the wave, but mean age in Wave 6 or 2002 was 74.43 (7.21) while mean age in Wave 13 or 2016 was 83.64 (5.48). Minimum age across waves was 65 years old while maximum age was 109 years old occurring in two of the waves, wave 6 and 10. Females constituted 56.9% of the sample with the rest identifying as males. Regarding race and ethnicity, 83.4% of the sample was White and 92.6 identified as non-Hispanic (6 participants did not have race or ethnicity information). Mean education of the sample was 11.94 (3.37) years. The mean score for baseline depressive symptoms using the CESD-8 scale was 1.46 (SD = 1.89).

The final MHAS sample included in statistical analyses following measurement invariance analyses included 4388 participants. Mean age of participants varied depending on the wave, but mean age in Wave 1 or 2001 was 73.01 (6.73) while mean age in Wave 4 or 2015 was 84.58 (4.85). Minimum age across waves was 65 years old while maximum age was 113 years old in wave 13. Females constituted 53.3% of the sample with the rest identifying as males. There was no race or ethnicity information for the MHAS sample. Mean education of the sample was 4.58 (4.10) years. The mean score for baseline depressive symptoms using the CESD-8 scale was 3.35 (SD = 2.48). Demographic data for both samples can be found on Table 1.
Table 1. Demographic characteristics of HRS and MHAS samples.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD) or % (Unless indicated otherwise)</th>
<th>HRS (N = 10,931)</th>
<th>MHAS (N = 4388)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td>83.64 (5.48)</td>
<td>84.58 (4.85)</td>
</tr>
<tr>
<td>Sex (Female)</td>
<td></td>
<td>56.90%</td>
<td>53.30%</td>
</tr>
<tr>
<td>Race (White)</td>
<td></td>
<td>83.40%</td>
<td>-</td>
</tr>
<tr>
<td>Ethnicity (Non-Hispanic)</td>
<td></td>
<td>92.60%</td>
<td>-</td>
</tr>
<tr>
<td>Education Level</td>
<td></td>
<td>11.94 (3.37)</td>
<td>4.58 (4.10)</td>
</tr>
<tr>
<td>Marital Status (Married)</td>
<td></td>
<td>40.20%</td>
<td>33.10%</td>
</tr>
<tr>
<td>Employment Status (Retired)</td>
<td></td>
<td>87.20%</td>
<td>19.71%</td>
</tr>
<tr>
<td>CESD-8 Score</td>
<td></td>
<td>1.46 (1.89)</td>
<td>3.35 (2.49)</td>
</tr>
</tbody>
</table>

HRS = Health and Retirement Study; MHAS = Mexican Health Aging Study; LW = Last Wave; CESD-8 = Center for Epidemiological Studies Depression, Eight-Item Version.

The Weighted Least Square Mean and Variance Adjusted (WLSMV) estimator was used over other estimation methods because this is a robust estimator with categorical data and does not depend on normal distribution assumptions (Li, 2016). Examining fit indices for the baseline or configural model testing for threshold invariance showed adequate fit as shown by two out of four indices. The Chi-square index was not taken into account due to the large sample size of both groups. The CFI is .95; the TLI is .93; the RMSEA is .09, and the SRMR is .07. Threshold invariance implies that when observed scores are regressed on each factor, threshold are equal across groups. In spite of two indices showing poor fit (e.g. TLI and RMSEA), a decision was made to test the second, more restrictive model based on adequate fit shown by CFI and SRMR.
The DIFFTEST indicated that adding the loadings and intercepts constraints to the invariant threshold model significantly worsen the model fit when compared with the baseline model. For this second model, the CFI is .93; the TLI is .91; the RMSEA is .10, and the SRMR is .07 meaning that three out of four indicators showed poor fit possibly indicating that measurement invariance does not hold. These results appear in Table 2.

Table 2. Measurement Invariance CES-D 8

<table>
<thead>
<tr>
<th></th>
<th>Baseline Configural Model</th>
<th>Full Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(df = 40)</td>
<td>(df = 47)</td>
</tr>
<tr>
<td><strong>Fit Criteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ΔX^2</td>
<td>2629.10***</td>
<td>3911.236 (1282.133)***</td>
</tr>
<tr>
<td>Δ CFI</td>
<td>.95</td>
<td>.93 (-.02)</td>
</tr>
<tr>
<td>Δ TLI</td>
<td>.93</td>
<td>.91 (-.02)</td>
</tr>
<tr>
<td>Δ RMSEA</td>
<td>.09</td>
<td>.10 (.01)</td>
</tr>
<tr>
<td>Δ SRMR</td>
<td>.07</td>
<td>.07 (.00)</td>
</tr>
<tr>
<td>Δ McDonald's</td>
<td>.92</td>
<td>.881 (-.04)</td>
</tr>
<tr>
<td>Δ Gamma Hat</td>
<td>.96</td>
<td>.941 (-.02)</td>
</tr>
<tr>
<td><strong>Sample and Model</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total sample size</td>
<td>N = 15,319</td>
<td>N = 15,319</td>
</tr>
<tr>
<td>Group size</td>
<td>HRS = 10,931</td>
<td>HRS = 10,931</td>
</tr>
<tr>
<td></td>
<td>MHAS = 4388</td>
<td>MHAS = 4388</td>
</tr>
</tbody>
</table>

df = degrees of freedom; HRS = Health and Retirement Study; MHAS = Mexican Health and Aging Study. Only bold fit indices were considered for MI decisions. Baseline Configural Model = Invariant Thresholds; Full Model = Invariant Thresholds, Loadings and Intercepts.

When examining items’ regression on the factor in HRS output data, CESD-8 items 3 and 8 showed the least improvement in fit while items 1 and 7 produced the most improvement in fit.
When examining thresholds in HRS output data, items 2 and 8 produced the least improvement in fit while items 4 and 6 produced the most improvement. Similar findings were observed for the MHAS sample. These results for individual items appear in Table 3.

Table 3. Analyses of CES-D 8 Items examining factor and threshold coefficients.

<table>
<thead>
<tr>
<th>CES-D 8 Items</th>
<th>F1 by Y</th>
<th>Thresholds</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>b</td>
</tr>
<tr>
<td>(7) You felt sad</td>
<td>.87</td>
<td>.007</td>
<td>.71</td>
</tr>
<tr>
<td>(1) Depressed</td>
<td>.85</td>
<td>.008</td>
<td>.87</td>
</tr>
<tr>
<td>(6) You enjoyed life</td>
<td>-.79</td>
<td>.01</td>
<td>-1.3</td>
</tr>
<tr>
<td>(4) You were happy</td>
<td>-.77</td>
<td>.009</td>
<td>-1.1</td>
</tr>
<tr>
<td>(2) Everything you did was an effort</td>
<td>.67</td>
<td>.01</td>
<td>.48</td>
</tr>
<tr>
<td>(8) You could not get going</td>
<td>.62</td>
<td>.011</td>
<td>.48</td>
</tr>
<tr>
<td>(3) Your sleep was restless</td>
<td>.53</td>
<td>.012</td>
<td>.53</td>
</tr>
</tbody>
</table>

F1 = Factor; Y = Item, CESD-8 = Center for Epidemiological Studies Depression, Eight-Item Version.

Discussion

Primary findings of this study did not support MI between CESD-8 items as answered by participants in the HRS sample versus participants in the MHAS sample. Further examination of results, showed that items 2 (“You felt everything you did was an effort”), 3 (“Your sleep was restless”) and 8 (“You could not get going”) in the CESD-8 added the least to model fit while
items 1 (“You felt depressed”), 4 (“You were happy”), 6 (“You enjoyed life”) and 7 (“You felt sad”) produced the most improvement. When measurement invariance is not supported as it is the case in this study, comparing and interpreting results between groups is not meaningful (Finch, 2014; Milfont & Fischer, 2010; Van de Schoot, Lugtig, & Hox, 2012). Failure to reach full measurement invariance implies that any further analyses that make use of the CESD-8 as a measure of depressive symptoms can only be interpreted meaningfully in the HRS sample since the original measure was created for English speaking adults in the American community. It should be noted that these findings do not necessarily generalize to other English and Spanish speaking samples like ELSA or CRELES for example; thus, measurement invariance between other samples would need to be studied independently.

There are many possible interpretations for the lack of MI between American and Mexican samples. The following propositions do not represent an exclusive list of these reasons, but they are based on considerations described above such as CESD-8 scale characteristics as well as cultural considerations. For example, the CESD-8 only contains 8 items, and most of these fall within the cognitive or affective domain. Somatic complaints, which have been more related to the construct of depression as interpreted and expressed by Hispanics (Cabassa, Hansen, Palinkas, & Ell, 2008; Lewis-Fernandez, Das, Alfonso, Weissman, & Olfson, 2005), is not well-represented in this brief scale. Hispanics might conceptualize depression as the direct result from psychosocial stressors rather than as a syndrome with a possible genetic or biological etiology (Lewis-Fernandez et al., 2005), which is important because it might influence perceived locus of control in Hispanics and willingness to disclose certain symptoms. Also, somatic symptoms might be less stigmatized than affective symptoms in Hispanic culture (Lara-
Cinisomo, Akinbode, & Wood, 2020); thus, somatization might be the primary mode through which Hispanic communicate experiencing depression.

In addition to psychosocial and demographic distinctions between the US and Mexico, there are psychometric considerations that may further elucidate these findings. The CESD-8 was originally created in English, and most prior studies evaluating validity of Spanish version have been conducted using CTT, which as stated above, might not fully capture differences at the item level like measurement invariance does. Additionally, dichotomous data is more restrictive than continuous data (e.g. no means, variances or covariances), which in addition to a limited pool of items might impact comparability of CESD-8 across these two different cultures. Another issue relevant to MI with categorical data is that fit indices for invariance testing using categorical data have not been well established. By contrast, most fit indices that are currently used have been extensively evaluated with continuous data (e.g. CFI, TLI, RMSEA) while their effectiveness identifying model fit with categorical data is less clear (Bovaird & Koziol, 2012; Bowen & Masa, 2015). Thus, it might be possible that the fit indices that were used in analyzed models might not capture true invariance between the studied samples.

Primary limitations of this study are that this study did not account for bilingualism or acculturation factors, which might represent a source of error. For example, some of the participants in MHAS reported traveling to the United States to work; however, this variable was not controlled in this study. In addition, part of the American sample was composed by Hispanics, and it is unclear whether these have been participants born in the United States or who had immigrated at some point. Socioeconomic status (SES) is another factor that might determine between and within sample differences in answering a brief depression scale;
however, SES was not considered in our analyses. Furthermore, the CESD-8 scale itself, although a useful screener, might not fully capture something as complex as depressive symptoms. For example, the CESD-8 presented to participants in both, the HRS and MHAS, asked about symptoms during the past week. Considering that DSM-V criteria asks for symptoms of depression for the past two weeks, answers to the CESD-8 as it appears in these epidemiological surveys might relate merely to fleeting feelings and not necessarily to depressive symptoms from a major depressive episode. The cyclical nature of depression is also not captured by questions in the CESD-8. Another issue that might impact comparability of results and attempts at harmonization might be characteristics of the samples themselves. For example, participants in the HRS sample proved to be more highly educated in general than those in the MHAS sample with about eight years of education difference on average. There might also be strong gender differences across cultures in these samples responding to CESD and other mood measures. The concept of “machismo” in some Latin-American countries is well known, and this might affect how male participants in these countries in particular respond to mood measures by suppressing or limiting the extent to which they might be willing to share their experience of depression (Fuentes & Aranda, 2019; Vega, Rodriguez, & Ang, 2010). Although current globalization trends might mitigate stark cultural idiosyncrasies in younger generations, this might still be a sample issue to consider in current community studies with cohorts that were born prior to the widespread advent of the internet.

Directions for future research suggested by these findings include the development or use of measures that might better capture depressive symptoms in the Mexican population. It might be argued that a measure can be created that includes equally weighted affective, cognitive and
somatic items loaded in different factors, so that people from different cultures can interpret questions asking about depressive symptoms from their own perspective; thus ensuring that the construct in question is being truly measured. Another approach for future waves of the HRS and MHAS as well as sister studies include using measures that have already been validated within a specific culture. The question then becomes how to establish MI when participants complete different scales measuring the same construct or the same scale answered differently depending on culture. This is a question that is worth posing for future cross-cultural psychometric studies.

The current study examined for the first time measurement invariance in the CESD-8 in community dwelling adults in Mexico using MHAS data. A CFA approach was used to examine factor structure and psychometric properties of this brief scale in Spanish, and at the same time compare it to the English version used with the HRS sample. Although a few studies have looked at MI in the CESD administered to Spanish speaking populations, they have looked at other versions of the CESD or used populations that might have acculturated to the dominant culture, such as Mexican Americans. Considering that this project is the first to examine measurement invariance in the brief CESD-8 in Spanish with participants living in their respective country, Mexico, this project becomes particularly informative for harmonization efforts between MHAS and HRS developmental projects.
CHAPTER 3: STUDY TWO

Abstract

Several past studies have established loneliness and physical activity levels as correlates of depression. The purpose of this second study was to examine these two variables within the scope of the inflammation theory of depression and the economic theory of depression integrated in a conceptual model called the Multisystemic Geriatric Depression Cycle. Two longitudinal datasets from community studies of older adults in the United States and Mexico were used to examine various hypotheses looking at the role of CRP in the maintenance of depression and vice versa, the possible moderating effect of physical activity in this relationship, the role of physical activity in reducing physical activity, and the mediating effect of positive and negative perceived social support in the relationship between loneliness and depression. Results showed support for the inflammation theory of depression, partial support for the economic theory of depression when using cross-sectional data and a continuous variable for physical exercise, and the mediating effect of negative (but not positive) perceived social support in the relationship between loneliness and depression on various waves. This study evidence the need to mitigate inflammation in older adults through diet, exercise and other venues to reduce chances of experiencing depressive symptoms since CRP seems to be a risk factor as posited by the inflammation theory of depression. Also, it is important to continue to support and develop programs aimed at improving the quality of social relationships in older adults considering that perceived negative social support might be a mechanism through which lonely individuals might experience depression.
Introduction

Loneliness and Social Isolation

Social isolation is described as lacking social contact objectively while loneliness refers to the psychological experience of social isolation, which is associated with distress. Loneliness arises as a discrepancy between objective and preferred level of social contact that is preferred (Ong, Uchino, & Wethington, 2016). An individual could still endorse feelings of loneliness regardless of whether he or she experiences social isolation (Cacioppo, Fowler, & Christakis, 2009; Kobayashi & Steptoe, 2018). The prevalence of loneliness in older adulthood varies depending on the type of measure that was used, age group, and other considerations. Across studies, prevalence rates are high enough to warrant clinical intervention ranging from 19.3% of community dwelling older adults over the age of 65 in the United States (Theeke, 2009) to 29% in individuals who are 75 years or older (Hughes, Waite, Hawkley, & Cacioppo, 2004; Ong et al., 2016). Furthermore, loneliness is a major risk factor in old age for mental and physical decline in older adults as well as mortality (Hagan, Manktelow, Taylor, & Mallett, 2014), and in turn declining physical and mental health places older adults at risk for social isolation (Ong et al., 2016). Among older adults, loneliness was a predictor of cognitive decline independent of depressive symptomatology in a longitudinal study over the course of twelve years. Notably, this relationship was not found to be bidirectional in that cognitive decline did not predict increased loneliness (Donovan et al., 2017). Furthermore, loneliness but not social isolation has been found to be a predictor of dementia onset (Holwerda et al., 2014). The relationship between loneliness and depression is mediated by social support, suggesting that this may be one mechanism by which this relationship exists (Liu, Gou, & Zuo, 2016).
At least two types of loneliness have been identified (Weiss, 1973), emotional loneliness, which is the perceived absence of a significant other in someone’s life, is better predicted by marital status, and social loneliness, which is the perceived absence of meaningful connections with family and friends, is better predicted by frequency of contact. These dimensions of loneliness and its corresponding predictors have demonstrated to be consistent across different cultural groups (Cacioppo & Cacioppo, 2012). It has been proposed that for those individuals with small social networks, loneliness is associated with depression, which in turn relates to lack of social support. By contrast, for those individuals with larger social networks, loneliness is not associated with depression, but instead relates to marital status. Other factors that are likely to influence these relationships are gender and age of the participant as well as geographic living area; thus, these are variables that need to be controlled for when performing analyses involving loneliness (Domènech-Abella et al., 2017). Finally, when evaluating loneliness between individualistic and collectivistic cultures, findings show that lack of social interaction with family better predicts loneliness in collectivistic societies whereas lack of social interaction with friends better predicts loneliness in individualistic societies (Lykes & Kemmelmeier, 2014).

**Physical Activity and Psychomotor Functioning**

The World Health Organization defines physical activity (PA) as movement produced by contracting skeletal muscles resulting in an increase in energy expenditure. Overall, older adults’ level of PA are typically less intense than younger adults, though significant benefits of maintaining physically active for older adults is well established. Cardiovascular exercise in particular, reduces the risk of developing chronic degenerative diseases associated with older age, especially in those older adults who are considered to be highly active (Chodzko-Zajko et
Vigorous PA has been shown to increase gait speed and improve balance and performance in activities of daily living (ADLs) in frail older adults (Chou, Hwang, & Wu, 2012). Several meta-analyses have concluded that routine PA reduces symptoms of depression (Josefsson, Lindwall, & Archer, 2014; Schuch et al., 2016) and improves cognitive functioning and psychological well-being in frail older adults (Langlois et al., 2013). In a longitudinal study, a higher level of PA per day was associated with reduced risk of onset and development of Alzheimer disease (Buchman et al., 2012). The inverse relationship has also been reported. Another study demonstrated that depression results in psychomotor retardation as a cognitive task becomes more difficult, and depressive symptoms interact with age, which is another predictor of psychomotor retardation (Beheydt et al., 2015). Thus, the relationship between depressive symptoms and levels of PA is likely to be bidirectional whereas one has the potential to affect intensity and frequency of the other.

Interestingly, vigorous PA produces a short-term inflammatory response; however, the effect of PA is anti-inflammatory when evaluated over a long period (Kasapis & Thompson, 2005). Social isolation and loneliness have been associated with decreased gait speed, and this relationship seems to be stronger in older adults with low socioeconomic status. Furthermore, loneliness but not social isolation has been associated with impairment in ADLs (Shankar, McMunn, Demakakos, Hamer, & Steptoe, 2017). Other studies support the relationship between loneliness and social isolation and decreased PA and increased mortality (Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015; Ong et al., 2016).
Theoretical Synthesis

The Multisystemic Geriatric Depression Cycle, summarized in Figure 4, is an effort to integrate a broad range of diverse studies addressing complementary aspects of depressive symptomatology, clinical correlates, risk factors, and prognostic implications. By contrast to processes such as grief and transient low mood that resolve in time (Jacobsen, Zhang, Block, Maciejewski, & Prigerson, 2010; Rosenzweig, Prigerson, Miller, & Reynolds III, 1997), depression as a clinical construct tends to be cyclical, recurrent, and impairing (Hardeveld, Spijker, De Graaf, Nolen, & Beekman, 2010; D. A. Solomon et al., 2000). Prior endorsement of depressive symptoms is a robust predictor of future depressive symptom endorsement (Hardeveld, Spijker, De Graaf, Nolen, & Beekman, 2013; Vemur, 2004). By conceptualizing depressive symptom endorsement in the broader context of risk factors, correlates, and prognostic implications, we may better understand the long-term stability of depressed mood and thus refine interventions to better address late-life mood disorders and mitigate the myriad associated health risks. Depression and C-reactive protein (CRP) have a bidirectional relationship; depression is associated with immunological dysregulation marked by elevated global inflammation (Stewart, Rand, Muldoon, & Kamarck, 2009), and elevated inflammation is a risk factor for later depression (Berk et al., 2013; Dantzer et al., 2011). Based on the benefits of PA described above, it is expected that the effect of depression on increased inflammation will be attenuated by PA. Reflecting prior work summarized in the economic model of depression, depressive symptoms will predict higher risk for adverse health outcomes over time (Strulik, 2019). This integration model also reflects past work suggesting that depression predicts loneliness, and that
cultural and socialization variables impact the magnitude of this relationship (Domènech-Abella et al., 2017; Lykes & Kemmelmeier, 2014). The cycle perpetuates as loneliness interacts with poor social support amplifying subsequent depression risk (L. Liu et al., 2016). This model broadly reflects past work, but also posits testable hypotheses regarding mechanistic relationships between variables and complex, longitudinal interrelationships between depression and associated phenomena. Some of those hypotheses are described below.

Figure 4. Multisystemic Geriatric Depression Cycle.

More specifically, the proposed model will integrate the inflammation theory of depression using CRP, the economic theory of depression, and the Weiss’ model of loneliness
with variables like exercise, social support and socialization practices using longitudinal data from the United States and Mexico. The hypotheses below are proposed,

**Hypothesis 3.** Consistent with the inflammation theory of depression, higher CRP levels at baseline will predict higher depressive symptomatology in both samples. It is also predicted that controlling for BMI will attenuate this relationship.

**Hypothesis 4.** The relationship between CRP and depressive symptomatology will be moderated by physical activity whereas those with high CRP levels and higher levels of physical activity will endorse fewer depressive symptoms compared to those with high CRP levels and less physical activity. These benefits of physical activity will be apparent in both samples.

**Hypothesis 5.** According to the economic theory of depression, depressive symptomatology at baseline will predict lower level of physical activity in subsequent waves. It is also hypothesized that higher depressive symptomatology will predict higher CRP levels whereas physical activity will moderate this relationship.

**Hypothesis 6.** Socialization practices and culture will moderate the relationship between depressive symptomatology and loneliness whereas higher frequency of contact with friends will mitigate feelings of loneliness in individuals with depressive symptoms in the HRS sample. By contrast, higher frequency of contact with family members will mitigate feelings of loneliness in individuals with depressive symptoms in the MHAS sample.
**Hypothesis 7.** Loneliness will predict higher depressive symptomatology, and this relationship will be mediated by perceived social support whereas those who feel lonely but endorse feeling more supported will experience less depressive symptomatology.

Comparisons between both samples were dependent on proven MI between both versions of the 8 item CES-D used in the HRS and MHAS samples. Considering that MI was not supported, analyses were still conducted in both samples although results could not be compared meaningfully, and in the case of the MHAS sample, results could not be meaningfully interpreted. Results for the MHAS sample have been included for reader’s reference only.

**Method**

**Sample**

Considering that CRP and depression are being used as predictors for other variables based on the inflammation theory and economic theory of depression respectively, participants with missing CES-D values or CRP values at the first wave of data collection of anthropometric measures, or who presented CRP values above 10 μg/mL as CRP values exceeding 10 μg/mL suggesting the possibility of an acute phase response (Ridker, 2003) were excluded. Final sample size depended on the type of analysis being conducted, and this was specified in each section for different analyses below. The same pool of participants used for measurement invariance analyses was used initially to conduct analyses in Study 2. Please refer to Table 1.
Measures

**C-Reactive Protein.** This biomarker in the HRS was collected through an enzyme-linked immunosorbent assay using dried blood spot at the University of Vermont. Within- and between-assay imprecision are 8.1% and 11.0%, respectively (Crimmins et al., 2013). This biomarker in the MHAS was collected by MHAS in collaboration with the Instituto Nacional de Salud Pública (INSP, Mexico). Precision was determined over five days with two runs and four replicates of each control per day (Wong et al., 2015).

**Depressive symptomatology.** Depressive symptomatology was measured using the abbreviated 8-item Center for Epidemiological Studies-Depression (CES-D) measure (Radloff, 1977). Participants answered “yes” or “no” to each 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. Internal reliability of the eight items CES-D can ranges from 0.72 to 0.88 (Bracke, Levecque, & Van de Velde, 2008). This measure has shown high nomological validity with related variables (Karim et al., 2015) and construct validity for both men and women (Van de Velde et al., 2009).
**Physical activity.** Level of PA in both samples was captured by questions asking about frequency of physical activity. A first PA variable was created by combining several other variables that asked about frequency of various activities such as maintenance or gardening, playing sports/exercise and walking for twenty minutes. The values for this first PA variable, which was continuous, ranged from 0 to 90. In addition, participants in the HRS sample were asked questions like “How often do you take part in sports or activities that are vigorous, such as running or jogging, swimming, cycling, aerobics or gym workout, tennis, or digging with a spade or shovel?” Three variables reflecting three different levels of physical activity including mild, moderate and vigorous were obtained. Each of these variables had five different levels with zero being the minimum and thirty being the maximum value. For both PA values, higher values indicated higher levels of PA. On the other hand, participants in the MHAS sample answered the question “On average during the last two years, have you exercised or done hard physical work three or more times a week?” This resulted in a dichotomous variable that differentiated those who exercised three or more times a week versus those who did not.
**Loneliness and socialization.** Both, the HRS and the MHAS, include a three-item loneliness scale that ask participants whether they felt lack of companionship, left out, or isolated from others. Higher values on this scale denote more loneliness. Internal reliability from this scale ranges from .73 to .81 (Chen & Feeley, 2014; Luo, Hawkley, Waite, & Cacioppo, 2012). Socialization practices was measured by frequency of social contact per week with family members and friends which is asked in both questionnaires. Additionally, perceived social support is assessed by questions like “How much can you rely on [spouse/partner, children, family members, friends] if you have a serious problem?” in the HRS and “Do you have neighbors or friends you can count on for daily activities, such as bringing food if you are sick, or bringing you something from the store?” or “How much do they listen if you need to talk about your worries” in the MHAS.

**Statistical analysis and procedures**

Analyses described below were performed using SPSS Version 23 (IBM Corp., 2015) and the MPlus (B. Muthén & Muthén, 1998-2017; B. Muthén et al., 2021) software program. Hypotheses were tested using a moderated path model, which included autoregressive pathways to demonstrate the effect of measurement of one variable (e.g. loneliness) at an earlier time point in the same variable later in time. Also, a moderated path model included cross-lagged pathways that permitted representation of hypothesized causal effects of predictor variables in outcome variables established in hypotheses above (Scott & Paulson, 2017). Further analyses also included a moderation model with depressive
symptoms as the IV, loneliness as the DV, and socialization practices as moderator of this relationship. Finally, among the most common longitudinal mediation models are cross-lagged panel models (CLPM) and latent growth curve models (LGCM). A LGCM allows for more complex analyses than the CLPM because it accounts not only for individual differences but also changes among constructs over time (O'Laughlin, Martin, & Ferrer, 2018). A slope-intercept model was used to analyze the possible mediating effect of social support on the relationship between loneliness as a predictor and depressive symptomatology at various time waves. Analyses described below were conducted separately on each sample, and as such, they are reported separately on the Results section to maintain consistency with analyses procedures and facilitate readers’ understanding.

Results

Health and Retirement Study Analyses

Hypothesis 3. Multiple Regression Analyses with depressive symptoms as outcome variable.

Hypothesis 3, which relates to the inflammation theory of depression, argued that higher CRP levels at baseline would predict higher depressive symptomatology. Based on prior studies that posit BMI as a possible attenuating variable, this and other possibly influencing variables like baseline depression scores, gender, age, ethnicity and health status were included in the regression analyses as covariates. A stepwise method was used for multiple regression analyses for a sample size of 5602 participants. The correlation between
CRP and depression within the same wave was .11 ($p < .0001$) while the relationship between CRP on wave 8 and depression on wave 13 was .05 ($p = .05$). Results show that CRP did not predict depressive symptoms at a later wave when including covariates; the CRP variable for removed from final model ($p = .99$). However, depressive symptoms at a later wave were predicted by baseline depressive symptoms ($\beta = 0.38$, $SE = 0.03$, $p < .0001$), health status ($\beta = 0.24$, $SE = 0.05$, $p < .0001$), and ethnicity ($\beta = 0.49$, $SE = 0.19$, $p = .01$). CRP predicted depressive symptoms within the same wave ($\beta = 0.01$, $SE = 0.04$, $p = .048$). Thus, the inflammation theory of depression was supported in cross-sectional analyses. Results of the stepwise multiple regression appears below in Table 4.

Table 4. Stepwise longitudinal multiple regression with CRP predicting depressive symptoms.

<table>
<thead>
<tr>
<th>Model</th>
<th>b</th>
<th>SE</th>
<th>B</th>
<th>t</th>
<th>p</th>
<th>F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline model</td>
<td>1.40</td>
<td>.04</td>
<td>32.48</td>
<td>&lt;.0001</td>
<td>26.94</td>
<td>2435</td>
<td></td>
</tr>
<tr>
<td>CRP (B)</td>
<td>.02</td>
<td>.00</td>
<td>.11</td>
<td>5.19</td>
<td>&lt;.0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Final model (5)</td>
<td>-2.72</td>
<td>.50</td>
<td>-5.44</td>
<td>&lt;.0001</td>
<td>92.10</td>
<td>2435</td>
<td></td>
</tr>
<tr>
<td>CRP (B)</td>
<td>.01</td>
<td>.004</td>
<td>.04</td>
<td>1.98</td>
<td>.048</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Health status (B)</td>
<td>.67</td>
<td>.03</td>
<td>.39</td>
<td>20.51</td>
<td>&lt;.0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.40</td>
<td>.07</td>
<td>.10</td>
<td>5.63</td>
<td>&lt;.0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>.02</td>
<td>.01</td>
<td>.04</td>
<td>2.23</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td>.28</td>
<td>.14</td>
<td>.04</td>
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$B =$ Baseline; $CRP =$ C-Reactive Protein; $BMI =$ Body Mass Index; $CESD-8 =$ Center for Epidemiological Studies Depression, Eight-Item Version. There were five models in this stepwise multiple regression analysis. C-Reactive Protein and age were excluded throughout the stepwise multiple regression analysis due to non-significant findings.
**Hypothesis 4. Moderation analysis with CRP as predictor, depressive symptomatology as outcome variable and physical activity as the moderator.**

Hypothesis 4 stated that the relationship between CRP and depressive symptomatology would be moderated by physical activity whereas those with high CRP levels and higher levels of physical activity would endorse fewer depressive symptoms compared to those with high CRP levels and less physical activity. Process (Hayes, 2012) was used to conduct a moderation analysis using Model 1. Bootstrapping was performed with 5000 iterations using the bias corrected method and 95% confidence intervals. Two variables for PA were used as described above. The second variable for PA had three different levels, mild, moderate and vigorous PA while the first PA variable was a continuous variable. Covariates included in both moderation analyses with these PA variables were baseline depression for longitudinal analyses only, age, gender, BMI, ethnicity, health status and job type.

Moderation analyses using continuous PA variable, CRP values from wave 9 and depressive symptoms within the same wave were performed with 1281 participants. Results were that PA as measured by the continuous PA variable was not a moderator in the relationship between CRP and depressive symptomatology on the same wave ($\beta = -0.001, SE = 0.0004, p = .10$). Covariates that were significant in this cross-sectional moderation analyses were gender ($\beta = 0.32, SE = 0.09, p = .0003$), ethnicity ($\beta = 0.53, SE = 0.17, p = .002$), health status ($\beta = 0.66, SE = 0.05, p < .0001$), and type of job ($\beta = 0.10, SE = 0.05, p = .02$). Similarly, when using the continuous PA variable in moderation analyses predicting depressive symptoms on wave 13 for a sample size of 940 participants, physical activity did not moderate the relationship between CRP
and depressive symptomatology ($\beta = 0.001, SE = 0.0004, p = .14$). Significant covariates in this longitudinal moderation analysis were baseline depression ($\beta = 0.32, SE = 0.03, p < .0001$), gender ($\beta = 0.45, SE = 0.10, p < .001$), and health status ($\beta = 0.33, SE = 0.06, p < .0001$). Thus, only gender and health status were significant predictors of depressive symptoms within and across waves.

When using the categorical PA variable, CRP values from wave 9 and depressive symptoms within the same wave, analyses were completed with 1337 participants. Results for mild PA showed that this variable did not moderate the relationship between CRP and depressive symptomatology ($\beta = 0.0001, SE = 0.001, p = .95$). Similarly to cross-sectional analyses using the continuous PA variable, significant covariates in this cross-sectional moderation analyses were gender ($\beta = 0.31, SE = 0.09, p = .0006$), ethnicity ($\beta = 0.50, SE = 0.16, p = .002$), health status ($\beta = 0.64, SE = 0.05, p < .0001$), and type of job ($\beta = 0.11, SE = 0.05, p = .01$). When evaluating moderate PA, results showed that PA did not moderate the association between CRP and depressive symptomatology ($\beta = -0.001, SE = 0.001, p = .54$). Also, covariates that were significant in this cross-sectional moderation analyses using moderate PA were gender ($\beta = 0.29, SE = 0.09, p = .001$), ethnicity ($\beta = 0.50, SE = 0.16, p = .002$), health status ($\beta = 0.63, SE = 0.05, p < .0001$), and type of job ($\beta = 0.11, SE = 0.04, p = .01$). Finally, results when using the vigorous PA variable evidenced that PA was not a significant moderator ($\beta = -0.001, SE = 0.001, p = .46$). Like stated before, covariates that were significant in this cross-sectional moderation analyses with vigorous PA variable were gender ($\beta = 0.29, SE = 0.09, p = .001$), ethnicity ($\beta = 0.50, SE = 0.16, p = .002$), health status ($\beta = 0.64, SE = 0.05, p < .0001$), and type of job ($\beta = 0.11, SE = 0.04, p = .01$). It is worth noting that sample size for cross-sectional moderation analyses.

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analysis using vigorous PA variable were performed in 1336 rather than 1337 participants due to missing data on that variable for one of the participants.

Moderation analyses using the categorical PA variable, CRP values from wave 9 and depressive symptoms for wave 13 were performed with 982 participants. Results for mild PA showed that PA did not moderate the relationship between CRP and depressive symptomatology ($\beta = 0.002$, $SE = 0.001$, $p = .13$). Significant covariates in this longitudinal moderation analyses were baseline depression ($\beta = 0.32$, $SE = 0.03$, $p < .0001$), gender ($\beta = 0.42$, $SE = 0.10$, $p < .0001$), health status ($\beta = 0.38$, $SE = 0.06$, $p < .0001$), and type of job ($\beta = 0.11$, $SE = 0.05$, $p = .02$). When evaluating moderate PA, results showed that PA did not moderate the relationship between CRP and depressive symptomatology ($\beta = 0.002$, $SE = 0.001$, $p = .10$). Covariates that showed to be significant in the longitudinal moderation analyses with moderate PA were baseline depression ($\beta = 0.32$, $SE = 0.03$, $p < .0001$), gender ($\beta = 0.45$, $SE = 0.10$, $p < .0001$), health status ($\beta = 0.37$, $SE = 0.06$, $p < .0001$), and type of job ($\beta = 0.11$, $SE = 0.05$, $p = .02$). Results from analysis with vigorous PA were that this variable did not moderate the relationship between CRP and depressive symptoms ($\beta = 0.000$, $SE = 0.002$, $p = .99$). Similarly with mild and moderate levels, covariates that were significant were baseline depression ($\beta = 0.32$, $SE = 0.03$, $p < .0001$), gender ($\beta = 0.45$, $SE = 0.10$, $p < .0001$), health status ($\beta = 0.36$, $SE = 0.06$, $p < .0001$), and type of job ($\beta = 0.11$, $SE = 0.05$, $p = .02$). In sum, gender and health status were significant covariates for both, cross-sectional and longitudinal moderation analyses, using both PA variables. Hypothesis 4 was not supported in any of the analyses.
Hypothesis 5. Moderation analysis with depression as a predictor, CRP as outcome variable and physical activity as the moderator.

The first part of Hypothesis 5 was that higher depressive symptomatology at baseline would predict lower level of PA in subsequent waves according with the economic theory of depression. For these analyses, two variables for PA were also used like prior analyses. A multivariate regression analysis was conducted on 553 participants using continuous PA variable while an ordinal regression analysis was conducted on 299 participants for cross-sectional analyses and on 214 participants for longitudinal analyses (212 in the case of moderate PA) using categorical PA variable. Covariates included in moderation analyses were baseline depression for longitudinal analyses only, age, gender, BMI, ethnicity, health status and job type.

Covariates that were included in the multivariate regression analyses predicting frequency of PA on wave 13 included baseline PA scores from wave 9, gender, age, BMI from wave 9, ethnicity and health status from wave 9. A stepwise method was used for regression analyses. The correlation between depression and PA within the same wave was -0.15 (p < .0001) while the relationship between baseline depression and later PA frequency on wave 13 was -0.07 (p = .07), and it was no longer significant. Results showed that depressive symptoms did not significantly predict frequency of PA at a later wave when including covariates; the depressive symptoms variable for removed from final model (p = .15). However, PA at a later wave was predicted by baseline PA (β = 0.38, SE = 0.04, p < .0001), baseline BMI (β = -0.52, SE = 0.18, p = .003), and ethnicity (β = 7.76, SE = 3.6, p = .03). Depression did not predict PA at wave 13 either when covariates were not included in regression analyses (β = -0.77, SE = 0.53, p = .14).
However, baseline depressive symptoms predicted PA within the same wave and all covariates remained significant including health status ($\beta = -3.52$, $SE = 0.45$, $p < .0001$), BMI ($\beta = -0.58$, $SE = 0.09$, $p < .0001$), age ($\beta = -0.35$, $SE = 0.07$, $p < .0001$), ethnicity ($\beta = 7.24$, $SE = 1.75$, $p < .0001$), gender ($\beta = -3.28$, $SE = 0.92$, $p < .0001$) and depressive symptoms ($\beta = -0.78$, $SE = 0.26$, $p = .003$). Thus, the economic theory of depression was partially supported in cross-sectional analyses only, but it was not supported across waves.

An ordinal logistic regression analysis was used to determine whether depressive symptoms would predict the categorical PA variable at its various levels, mild, moderate and vigorous, in cross-sectional and longitudinal analyses. When regression analyses were conducted within the same wave, depressive symptoms did not significantly predict mild PA ($\beta = 0.01$, $SE = 0.07$, $p = .91$). Covariates that remained significant were gender ($\beta = -0.46$, $SE = 0.22$, $p = .04$), age ($\beta = -0.06$, $SE = 0.03$, $p = .03$), and health status ($\beta = -0.32$, $SE = 0.12$, $p = .01$). Similarly, depressive symptoms did not significantly predict moderate PA within the same wave ($\beta = -0.06$, $SE = 0.07$, $p = .43$). In this case, covariates that remained significant were BMI ($\beta = -0.06$, $SE = 0.02$, $p = .01$) and health status ($\beta = -0.47$, $SE = 0.12$, $p < .001$). Finally, depressive symptoms did not predict vigorous PA either ($\beta = -0.16$, $SE = 0.10$, $p = .14$). Only age ($\beta = -0.12$, $SE = 0.04$, $p = .002$) and health status ($\beta = -0.59$, $SE = 0.15$, $p < .001$) remained significant covariates in this case. Thus, health status was the only significant predictor for mild, moderate and vigorous PA in cross-sectional analyses. When conducting longitudinal analyses, depressive symptoms in Wave 9 did not significantly predict mild PA at later wave 13 ($\beta = -0.06$, $SE = 0.09$, $p = .52$). Significant covariates predicting mild PA were baseline mild PA ($\beta = 0.05$, $SE = 0.02$, $p = .002$), gender ($\beta = -0.75$, $SE = 0.26$, $p = .004$) and age ($\beta = -0.17$, $SE = 0.04$, $p < .001$) only.
Similarly, moderate PA was not predicted by depressive symptoms across time ($\beta = 0.01$, SE = 0.09, $p = .94$). Significant covariates in this case included baseline moderate PA ($\beta = 0.05$, SE = 0.02, $p = .004$), age ($\beta = -0.11$, SE = 0.04, $p = .008$), ethnicity ($\beta = 1.24$, SE = 0.59, $p = .04$) and BMI ($\beta = -0.06$, SE = 0.03, $p = .04$). Vigorous PA at wave 13 was not predicted by earlier depressive symptoms ($\beta = 0.06$, SE = 0.12, $p = .62$). Only baseline vigorous PA ($\beta = 0.06$, SE = 0.02, $p = .006$), age ($\beta = -0.14$, SE = 0.05, $p = .01$) and health status ($\beta = -0.60$, SE = 0.19, $p = .002$) remained significant covariates in this analysis. Baseline PA and age were the only significant predictors for mild, moderate and vigorous PA across time. In sum, when using the categorical PA variable, the economic theory of depression was not supported in cross-sectional or longitudinal analyses.

The second part of hypothesis 5 posited that higher depressive symptomatology would predict higher CRP levels whereas PA would moderate this relationship. This was tested using Process similarly to moderation analyses described above. Moderation analyses using the continuous PA variable, CRP values from wave 9 and depressive symptoms within the same wave were performed with 227 participants. Results showed that PA as measured by the continuous PA variable did not moderate the relationship between depressive symptoms and CRP within the same wave ($\beta = -0.003$, SE = 0.011, $p = .82$). All covariates were non-significant in this cross-sectional moderation analyses. Similarly, when using the continuous PA variable in moderation analyses predicting CRP on wave 11 (There was no sufficient data for analyses spanning from wave 9 to 12) with a sample size of 173 participants, physical activity did not moderate the relationship between depressive symptomatology and CRP ($\beta = -0.01$, SE
Covariates that were significant in this longitudinal moderation analysis were baseline CRP ($\beta = 0.22$, SE = 0.06, $p = .0002$) and ethnicity ($\beta = 4.19$, SE = 1.70, $p = .02$). Moderation analyses using the categorical PA variable, depressive symptoms values from wave 9 and CRP within the same wave were performed with 248 participants. Results for mild PA showed that PA was not a moderator between depressive symptomatology and CRP ($\beta = 0.01$, SE = 0.03, $p = .68$). The only covariate that was significant in this cross-sectional moderation analysis was gender ($\beta = 1.77$, SE = 0.83, $p = .03$). When evaluating moderate PA, results showed that PA did not moderate the relationship between depressive symptomatology and CRP ($\beta = 0.02$, SE = 0.03, $p = .49$). Similarly to moderation analyses with mild PA, the only covariate that was significant was gender ($\beta = 1.68$, SE = 0.8, $p = .04$). Finally, results when using the vigorous PA variable showed that PA did not moderate the relationship between depressive symptomatology and CRP ($\beta = 0.04$, SE = 0.07, $p = .56$). Only gender was significant in this cross-sectional moderation analyses with the vigorous PA ($\beta = 1.61$, SE = 0.81, $p = .048$).

Moderation analyses with the categorical PA variable, CRP values from wave 12 and depressive symptoms on wave 9 were done with 189 participants. Mild PA did not moderate the relationship between depressive symptomatology and CRP ($\beta = 0.002$, SE = 0.03, $p = .996$). Covariates that were significant in this longitudinal moderation analyses were baseline CRP ($\beta = 0.21$, SE = 0.06, $p = .0002$) and ethnicity ($\beta = 3.72$, SE = 1.48, $p = .01$). When evaluating moderate PA, this variable was not a moderator in the relationship between depressive symptomatology and CRP ($\beta = 0.007$, SE = 0.03, $p = .83$). Similarly to previous analyses, covariates that were significant in longitudinal moderation analyses with moderate PA were baseline CRP ($\beta = 0.20$, SE = 0.06, $p = .0003$) and ethnicity ($\beta = 3.68$, SE = 1.47, $p = .0134$).
Results from analysis with the vigorous PA variable were not significant indicating that PA did not moderate the relationship between depressive symptoms and CRP ($\beta = -0.03$, SE = 0.06, $p = .534$). Similarly with mild and moderate levels, covariates that were significant were baseline CRP ($\beta = 0.21$, SE = 0.06, $p = .0003$) and ethnicity ($\beta = 3.68$, SE = 1.48, $p = .0136$). In sum, baseline CRP and ethnicity were significant covariates for longitudinal moderation analyses using both PA variables but not for cross-sectional analyses. Moderation as stated in Hypothesis 5 was not supported in any of the analyses.

**Hypothesis 6.** *Moderation analysis with depressive symptoms as predictor, loneliness as outcome variable and frequency of social contact as the moderator.*

Hypothesis 6 stated that frequency of social contact would moderate the relationship between depressive symptomatology and loneliness, and this would be different depending on the sample. In the case of HRS sample, it was hypothesized that higher frequency of contact with friends instead of family would mitigate feelings of loneliness in individuals with depressive symptoms. When conducting cross-sectional analyses in Wave 9 with a sample of 2662 participants, and examining frequency of contact with people in general, results showed that frequency of social contact did not moderate the relationship between depressive symptoms and loneliness ($\beta = -0.0002$, SE = 0.0002, $p = .442$). Gender ($\beta = -0.38$, SE = 0.16, $p = .0194$) and health status ($\beta = 0.49$, SE = 0.08, $p < .0001$) were significant covariates. A more targeted moderation analysis with 2463 participants looking at frequency of contact with friends only showed that frequency of contact was not a significant moderator either ($\beta = 0.0000$, SE = 0.0004, $p = .9411$). Similarly to general analysis, gender ($\beta = -0.45$, SE = 0.16, $p = .0058$) and
health status ($\beta = 0.44$, $SE = 0.08$, $p < .0001$) were significant covariates. When conducting longitudinal analysis examining frequency of social contact with people in general and depressive symptoms on Wave 9, and loneliness in Wave 13 in a sample of 891 participants, results showed that frequency of social contact did not moderate the relationship between depressive symptoms and loneliness ($\beta = 0.001$, $SE = 0.0004$, $p = .126$). Only covariate that remained significant was health status ($\beta = 0.68$, $SE = 0.16$, $p < .0001$). Similarly, when looking at frequency of contact with friends with a sample size of 835, results showed that frequency of contact did not moderate the relationship between depressive symptoms and loneliness ($\beta = 0.0004$, $SE = 0.001$, $p = .6739$) and the only significant covariate was health status ($\beta = 0.68$, $SE = 0.16$, $p < .0001$). Thus, hypothesis 6 was not supported in the case of the HRS sample.

**Hypothesis 7. Mediation analysis with loneliness as predictor, depressive symptoms as outcome variable and perceived social support as the mediator**

Hypothesis 7 stated that loneliness will predict higher depressive symptomatology and that this relationship would be mediated by perceived social support whereas those who felt lonely but endorsed feeling more supported would experience less depressive symptomatology. Results from a slope-intercept model analysis showed good model fit of the data in a sample of 5693 participants. The various waves included in this analysis ranged from Wave 9 to Wave 13. Because of sample size, the Chi-Square indicator becomes uninterpretable. The CFI is .98; the TLI is .97; the RMSEA is .04, and the SRMR is .03. These results appear in Figure 5 and corresponding syntax for slope-intercept model appears in Appendix C.
Figure 5. Slope intercept model. CFI = .98; TLI = .97; RMSEA = .04; SRMR = .03. Loneliness and Perceived Negative Social Support from baseline wave, wave 9. Sample size of 5693 participants.
Parallel mediation analyses using Process were conducted on 2659 participants in cross-sectional analyses and on 1256 participants in longitudinal analyses. The two mediators, positive and negative perceived social support were assumed to be correlated but not influence each other causally. Bootstrapping was performed using Model 4 with 5000 iterations using the bias corrected method and 95% confidence intervals. Covariates included in mediation analyses included gender, age, ethnicity, race and health status. Baseline depression scores were included in longitudinal analyses. When performing mediation analysis within the same wave, the correlation between residuals for positive and negative perceived social support was .5206 while the correlation between either of these and depressive symptoms was zero. When examining indirect effects of loneliness on depressive symptoms, negative, but not positive, perceived social support was a significant mediator in the relationship between loneliness and depressive symptoms (β = 0.02, SE = 0.03, 95% CI [0.0115, 0.0247]). Negative perceived social support accounted for 13.94% of the total effect of loneliness on depressive symptoms. Significant covariates in cross-sectional mediation analyses were gender (β = 0.48, SE = 0.06, p < .0001) and health status (β = 0.51, SE = 0.03, p < .0001). Across waves, the correlation between residuals for positive and negative perceived social support was .5025 while the correlation between either of these and depressive symptoms was zero. Similarly, when conducting longitudinal analyses, only negative perceived social support was a significant mediator between loneliness and depressive symptoms (β = 0.01, SE = 0.004, 95% CI [0.0002, 0.0158]). Negative perceived social support accounted for 17.67% of total effect of the IV on the DV. Significant covariates in longitudinal mediation analyses were baseline depressive symptoms (β = 0.39, SE = 0.03, p < .0001), gender (β = 0.21, SE = 0.09, p = .024), ethnicity (β = -0.03, SE = 0.02, p =
.039), and health status (β = 0.29, SE = 0.05, p < .0001). Thus, negative perceived social support was a significant mediator in the relationship between loneliness and depression in both, cross-sectional and across time analyses. Cross-sectional and longitudinal mediation models appear in Figure 6 and Figure 7 respectively.

Figure 6. Cross-Sectional Mediation Model. Dotted line represents total effect.

Figure 7. Longitudinal Mediation Model. Dotted line represents total effect.
**Mexican Health and Aging Study Analyses**

**Hypothesis 3. Multiple Regression Analyses with depressive symptoms as outcome variable.**

Hypothesis 3, which relates to the inflammation theory of depression, argued that higher CRP levels at baseline would predict higher depressive symptomatology in later waves. Possibly influencing variables like baseline depression scores, BMI, gender, age, and health status were included in the regression analyses as covariates. A stepwise method was used for multiple regression analyses. Analyses were performed using wave 3 as baseline considering that previous waves did not have CRP data. There was a significant correlation between CRP and depressive symptoms within the same wave ($r = .40, p < .0001$) while there was not a significant correlation between CRP on wave 3 and depression on wave 4 ($r = .08, p = .17$). Results show that CRP did not predict depressive symptoms at a later wave regardless of controlling for other variables; the CRP variable for removed from final model ($\beta = 0.04, SE = 0.04, p = .35$). Only covariates that significantly predicted depressive symptoms at a later wave in final model were baseline depressive symptoms ($\beta = 0.38, SE = 0.08, p < .001$), and gender ($\beta = 0.83, SE = 0.40, p = .04$).
Hypothesis 4. Moderation analysis with CRP as predictor, depressive symptomatology as outcome variable and physical activity as the moderator.

Hypothesis 4 stated that the relationship between CRP and depressive symptomatology would be moderated by physical activity whereas those with high CRP levels and higher levels of physical activity would endorse fewer depressive symptoms compared to those with high CRP levels and less physical activity. Like with the HRS sample, Process was used to conduct a moderation analysis using Model 1. Bootstrapping was performed with 5000 iterations using the bias corrected method and 95% confidence intervals. Covariates included in moderation analyses using the PA variable were baseline depression for longitudinal analyses only, age, gender, BMI, and health status. Although there was some employment information for the MHAS sample, there was not a specific question asking whether the job involved physical activity; thus, the type of job variable was not included in these analyses like it was included for the HRS sample.

Moderation analyses using the PA variable, CRP values from wave 3 and depressive symptoms within the same wave were conducted with 179 participants. Results showed that physical activity did not moderate the relationship between CRP and depressive symptomatology within the same wave ($\beta = -0.06, SE = 0.09, p = .46$). Covariates that were significant in this cross-sectional moderation analyses were gender ($\beta = 0.81, SE = 0.36, p = .03$) and health status ($\beta = 1.01, SE = 0.24, p < .0001$). Similarly, when using the PA variable in moderation analyses predicting depressive symptoms on last wave for a sample size of 133 participants, physical activity did not moderate the relationship between CRP and depressive symptomatology ($\beta = 0.04, SE = 0.09, p = .64$). Covariates that were significant in this longitudinal moderation
analysis were baseline depression ($\beta = 0.36, SE = 0.09, p = .0001$) and gender ($\beta = 0.88, SE = 0.42, p < .04$). Thus, only gender was a significant predictor of depressive symptoms within and across waves when using PA as a moderator.

_Hypothesis 5. Moderation analysis with depression as a predictor, CRP as outcome variable and physical activity as the moderator._

The first part of Hypothesis 5 stated that higher depressive symptomatology at baseline would predict lower level of physical activity in subsequent waves according with the economic theory of depression. A binary logistic regression analysis was conducted on 4330 participants for cross-sectional analyses and on 1251 participants for longitudinal analyses. Covariates included in these binary logistic regression analyses were baseline depression for longitudinal analyses only, age, gender, BMI, and health status. The correlation between depression and physical activity within the same wave was .10 ($p < .001$) while the correlation between baseline depression and later PA frequency on last wave was no longer significant ($r = .028, p = .32$). Results showed that depressive symptoms did not significantly predict frequency of PA at a later wave when including covariates ($\beta = -0.04, SE = 0.03, p = .17$). However, PA at a later wave was predicted by baseline PA ($\beta = -0.41, SE = 0.15, p = .007$), gender ($\beta = -0.54, SE = 0.15, p < .0001$), age ($\beta = 0.06, SE = 0.02, p = .001$), and health status ($\beta = 0.25, SE = 0.09, p = .008$). Similarly, depressive symptoms did not predicted PA in cross-sectional analysis either ($\beta = 0.03, SE = 0.02, p = .071$). Significant covariates were gender ($\beta = -0.71, SE = 0.07, p < .0001$), age ($\beta = 0.05, SE = 0.01, p < .0001$), and health status ($\beta = 0.20, SE = 0.05, p < .0001$).
The second part of hypothesis 5 posited that higher depressive symptomatology would predict higher CRP levels whereas physical activity would moderate this relationship. This was tested using Process similarly to moderation analyses described above. Moderation analyses with the PA variable, CRP values and depressive symptomatology within the same wave were conducted with 179 participants. Results showed that PA did not moderate the relationship between depressive symptoms and CRP within the same wave ($\beta = -0.06, SE = 0.41, p = .88$). Only health status was a significant covariate ($\beta = -1.46, SE = 0.63, p = .02$). Similarly, when using the PA variable in moderation analyses predicting CRP on wave 3 (wave 3 is the only wave in MHAS dataset containing CRP information; thus baseline CRP values could not be used for this analysis) for a sample size of 189 participants, physical activity was not a significant moderator in the relationship between depressive symptomatology and CRP ($\beta = 0.47, SE = 0.43, p = .27$). None of the covariates were significant in this longitudinal moderation analysis.

**Hypothesis 6.** Moderation analysis with depressive symptoms as predictor, loneliness as outcome variable and frequency of social contact as the moderator.

Hypothesis 6 stated that frequency of social contact would moderate the relationship between depressive symptomatology and loneliness, and this would be different depending on the sample. In the case of MHAS sample, it was hypothesized that higher frequency of contact with family instead of friends would mitigate feelings of loneliness in individuals with depressive symptoms. Two variables were created based on data gathered in MHAS sample that reflected frequency of contact with family members; the first variable referred exclusively frequency of contact with parents while the second variable reflected frequency of contact using spouse or romantic partner.
information, children and relatives in general. Extended family is particularly important to consider in Hispanic samples. The variable for frequency of contact with friends was a single continuous variable. When conducting longitudinal analyses using first and last wave with a sample of 117 participants, and examining frequency of contact with mother, father or both parents, frequency of contact with family member did not moderate the relationship between depression and loneliness ($\beta = 0.001, SE = 0.0004, p = .063$). Only age of participant was a significant covariate in this longitudinal analysis ($\beta = 0.18, SE = 0.05, p = .0006$). When using the second variable referring to frequency of contact with partner, children and relatives for a sample of 1256 participants, results of longitudinal analysis showed that frequency of contact did not significantly moderate the relationship between depression and loneliness. Similarly to analyses using first variable, only age of participant was a significant covariate ($\beta = 0.04, SE = 0.07, p = .19$). In the case of frequency of contact with friends and neighbors, longitudinal analyses were performed on 856 participants, and frequency of contact with friends and neighbors was not a significant moderator either like previous analyses ($\beta = 0.001, SE = 0.002, p = .77$). Age remained a significant covariate ($\beta = 0.0002, SE = 0.0002, p = .44$) in longitudinal analyses using frequency of contact with friends as the moderator.

**Hypothesis 7. Mediation analysis with loneliness as predictor, depressive symptoms as outcome variable and perceived social support as the mediator**

This hypothesis was not possible to test with the MHAS sample considering that the variable loneliness as measured with the three item questionnaire only appeared in this last wave, but not
on previous waves. So, it was not possible to use baselines loneliness in a slope intercept model as a predictor of depression in future waves.

**Discussion**

The interpretation of findings that follows pertains only to results obtained for analyses using the HRS sample considering prior results for measurement invariance in Study 1 that make comparison across groups or interpretability of results for MHAS sample unfeasible. The inflammation theory of depression as tested following hypothesis 3 and using multiple regression was supported. These results are consistent with prior literature that demonstrate the role of inflammation on onset and maintenance of depressive symptoms. Moderation analyses using two different PA variables in cross-sectional and longitudinal analyses showed that PA was not a significant moderator in the relationship between inflammation and depressive symptoms. Thus, this might be the result of poor modeling in which case the role of PA in the relationship between CRP and depressive symptoms needs to be reconceptualized using a different model, or it could be the case that the effects of PA are not substantially large to influence the various levels of this relationship between CRP and depression in older adults. The first part of hypothesis 5 referring to the economic theory of depression was partially supported in cross-sectional analyses and only when using first PA variable, which was a continuous variable. Partially significant results might indicate that further conceptualization of the economic theory of depression might be necessary especially as related to conservation of resources, or in the case of PA, energy. Also, since results were not supported longitudinally with either PA
variable might indicate that conservation of energy might be short lived, and it might vary in the long term depending on the episodic nature of depressive symptoms and individuals’ own cognitive and behavioral coping strategies to manage depressive symptoms. Moderation analyses in hypothesis 5 were not significant indicating like in prior hypothesis that effects of PA might not be large enough to influence relationship between depression symptoms and CRP, or that the role of PA in reducing depressive symptoms might not be an indirect effect. In the case of hypothesis 6, frequency of contact with friends was not a significant moderator in the relationship between depressive symptoms and loneliness.

For hypothesis 7, a slope intercept model showed good fit and subsequent mediation analysis evidenced that negative, but not positive, perceived social support was a significant mediator between loneliness and depressive symptoms both cross-sectionally and longitudinally. One interpretation of this finding is that the relationship between loneliness and current and future depressive symptoms might be explained through the effect of negative perceived social support, which seems to be a potential mechanism of action in this relationship.

One of the limitations of the study is that variables were constricted depending on available data, and some of the variables were not exactly the same across samples. In addition, some of the variables were dichotomous in nature or were composed of a few items, which might limit the amount of information that is available for analysis and interpretation. In other cases, variables that were available might not be the best representation of a specific construct. For example, interlukin-6 (IL-6) might be a better indicator of inflammation than CRP according to past literature; however, this biomarker
was not available in analyzed data. Furthermore, the variable loneliness in HRS and MHAS datasets involved items from the UCLA Loneliness Scale; however, none of the items measures distress, which according to literature on loneliness is a crucial component to differentiate loneliness from social isolation. Although analyzing community data has its advantages to discover the way in which variables interact in the population at large, the main variable of interest, depressive symptoms was present at low levels in both samples; thus, the relationships hypothesized in the Multisystemic Geriatric Depression Cycle might show in clinical samples or at higher levels of depression. Also to the CESD-8 measure itself might not be the best measure to capture depression in older adults, especially the very old. As stated above the CESD was originally created for people under 65, and the CESD-8 lacks any items that capture somatic complaints, which are important component of depression in old age. Physical activity and exercise are different variables at a conceptual level; PA variables in these study might have been too broad to capture the specific benefits or the type of exercise in influencing depressive symptoms and related constructs.

Future directions for the HRS and its sister studies like the MHAS should involve a greater coordination in the measures that they administer to make sure they are consistent across samples to support harmonization efforts and reduce the number of possible confounders. The main goal should be to leave culture as the main factor that differs among samples. Although it was noticed during the course of these studies that recent waves in the HRS and MHAS studies have an increasing number of similar variables, it is also clear that these efforts are still quite novel and need further refinement. Furthermore, one covariate that emerged as significant in most analysis was health status; this variable was already part
of the proposed Multisystemic Geriatric Depression Cycle; however, none of the hypotheses on the current projects looked at the direct effects of this variable on other variables in the model. Several prior studies have found a relationship between health status and depressive symptoms (Chang-Quan et al., 2010; J. Liu, Wei, Peng, & Guo, 2021; Palladino, Tayu Lee, Ashworth, Triassi, & Millett, 2016); thus, results from the present study confirm evidence in prior literature. Finally, this project confirmed past findings regarding the inflammation theory of depression and the mediating role of perceived social support in the relationship between loneliness and depression. Considering the increasing body of evidence supporting these relationships, mitigation of global inflammation and reducing perceived negative social support on older adults may mitigate both physiological and psychological morbidity. Finally, this project contributes to the growing body of literature on harmonization of HRS and its sister studies. Comparative cross-cultural research is paramount in the age of globalization, which will warrant a more transparent and accurate understanding of true cultural differences in various constructs like depression in older adults. Toward these goals, the current findings provide a limited but meaningful source of empirical evidence.
CHAPTER FOUR: CONCLUSION

Overall findings and implications

Measurement invariance findings were that responses to the CESD-8 scale in the HRS sample were not comparable to responses to the CESD-8 scale in the MHAS sample. Although this could be due to multiple reasons, a factor contributing to this failure to reach MI could be different responding tendencies to the same measure based on cultural and linguistic differences between American and Mexican populations. Depression in older adults typically present with fewer cognitive-affective symptoms and greater somatic complaints such as sleep impairment and psychomotor symptoms than among younger and middle-aged adults (Alexopoulos, Bruce, Silbersweig, Kalayam, & Stern, 1999; Blazer, 2003). Furthermore, depression as a construct looks different across cultures with somatization symptoms being a primary presentation in Latin-American countries by contrast to cognitive symptoms being a primary presentation in North America. A study examining the original CESD administered to Mexican-Americans and non-Hispanic in Los Angeles metropolitan area whites found variability in responding patterns between both groups especially related to sleep disturbances. The authors concluded that conceptual equivalence had not been reached meaning that the CES-D measures similar but not identical dimensions of depression in these two populations (Golding & Aneshensel, 1989). The CESD-8 does not contain any item asking about physical symptoms or addressing somatization concerns, which suggests important questions on construct and content validity of the CESD-8 in Spanish speaking samples in general and the MHAS sample in particular. Although another brief screener, the CESD-10, has proven to be a good screener for depression in older adults (Andresen, Malmgren, Carter, & Patrick, 1994;
Irwin, Artin, & Oxman, 1999), it is unclear the extent to which the same can be said about the CESD-8, especially as it relates to older adults in other non-English speaking or collectivistic cultures. Also, it is worth noting that the samples used for the CESD-10 studies included either clinical or highly educated samples, and they might not be representative of community-dwelling older adults.

Because CESD-8 measurement invariance did not hold between HRS and MHAS samples, only results related to the HRS sample could be interpreted meaningfully. A longitudinal multivariate regression analysis showed that inflammation predicted depressive symptoms in the HRS sample, and this significant relationship was consistent with the inflammation theory of depression. Another significant finding in study 2 was that negative perceived social support mediated the relationship between loneliness and depression. These results corroborate those obtained by Liu and colleagues (L. Liu et al., 2016) in a Chinese sample using a different measure of perceived social support, the Perceived Social Support Scale (PPSS); thus, the effect of perceived social support in the relationship between loneliness and depression seems to hold independently of cultural and linguistic differences.

It is worth noting that PA did not seem to moderate the bilateral relationship between inflammation and depressive symptoms in the Multisystemic Geriatric Depression Cycle model. The economic theory of depression was partially supported in cross-sectional analyses suggesting that a categorical assessment of PA might be inadequate. The relationship between depression and physical activity from the perspective of the economic theory of depression is not well supported over time by results in this study. This could be the result of inefficiencies with the PA variables that were used or a need for
reconceptualizing long term effects of depressive symptoms in physical activity levels in the Multisystemic Geriatric Depression Cycle. For example, the economic theory of depression would predict a reduction in exercise levels; however, PA variables in our analyses referred not only to exercise activity but PA in general. It might be possible that exercise as opposed to physical activity might be a better predicting variable. Given observed low rates of depression in our samples in general, the impact of non-clinical depressive symptoms might not be substantial enough to impact overall activity (e.g. fixing things around the house, gardening, walking), but could have been for vigorous exercise, which in the HRS and MHAS dataset appears as categorical. Furthermore, the HRS and the MHAS are community studies. The average rate of depressive symptoms endorsement in these non-clinical samples as measured by the CESD were quite low, and future research with clinical samples may provide contrasting evidence. It might be possible that the impact of PA in the relationship between CRP and depression or its relationship with depression alone become relevant only when inflammation or depressive symptoms are at clinical levels.

The movement towards harmonization among HRS sister studies is an important enterprise that is costly with regards to time and personnel, and it will require further refinement informed by research. As it was noted during completion of both studies described above, the measures and questions in the HRS and MHAS dataset do not completely map into one another especially in earlier waves. For example, while some measures including the CESD are part of all waves for both samples, the questions to create other variables like PA, perceived social support, loneliness and frequency of contact with
family or friends were not the same across questionnaires even in later waves. As results showed, even for those variables were questions seemed to be the same as it is the case with the CESD-8, the instrument used might not be appropriate to use cross-culturally from a psychometric standpoint. Thus, the MHAS longitudinal project should consider changing the current depression scale in future waves to account for the particular way in which depression manifests in Mexican older-adults. For example, the Hamilton Rating Scale for Depression includes items related to somatic symptoms, so this or similar scales could be considered (Dunlop et al., 2020).

Ethical considerations for this project involved compliance with sensitive data access use agreements in both HRS and MHAS websites. Some of these provisions included making no attempts to identify persons in used datasets, disclosing and publishing aggregates statistical summaries of the data, storing and using datasets in a secure computing environment among other conditions. This project was submitted for review to the Institutional Review Board of the University Central Florida, and IRB procedures were followed to completion during the duration of project proposal, data management and statistical analyses, and presentation of results. Other ethical considerations included avoiding arbitrary exclusion of participants in the study, choosing research methods that were consistent with the project goals and hypotheses’ driven, and ensuring results of the project were informative and contributed to the scientific community and existent literature.

The present project adds to the current literature in that to our knowledge, this is the first project that integrates the inflammation theory of depression and economic theory of depression
in a single model for older adults (>65), the Multisystemic Geriatric Depression Cycle. The impact of an active lifestyle on inflammation and depressive symptoms and vice versa may be further studied through the development and reconceptualization of variables composing this novel model for older adults’ health trajectories. Considering globalization and greater interconnectedness through the Internet and social media, future research would need to consider the impact of acculturation and homogeneity of experiences regardless of cultural background in the experience of depressive symptoms in older adults from future generations. Thus, current scales and instruments used in epidemiological studies might need to be replaced based on an evolving world population and unique cultural characteristics from participants in these studies overseas. Measurement invariance has become an invaluable tool informing appropriateness of using some scales with various populations. As such, this should become standard practice in future research endeavors comparing different constructs cross-culturally.
APPENDIX A: IRB LETTER
NOT HUMAN RESEARCH DETERMINATION

March 17, 2021

Dear Daniel Paulson:

On 2/12/2020, the IRB reviewed the following protocol:

<table>
<thead>
<tr>
<th>Type of Review:</th>
<th>Initial Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Title of Study:</td>
<td>Measurement Invariance in the Assessment of Mood Between American and Mexican Community Studies</td>
</tr>
<tr>
<td>Investigators:</td>
<td>Daniel Paulson, Manuel Herera Legon</td>
</tr>
<tr>
<td>IRB ID:</td>
<td>STUDY.0000.1144</td>
</tr>
<tr>
<td>Funding:</td>
<td>None</td>
</tr>
<tr>
<td>Grant ID:</td>
<td>None</td>
</tr>
</tbody>
</table>

The IRB determined that the proposed activity is not research involving human subjects as defined by DHHS and FDA regulations.

IRB review and approval by this organization is not required. This determination applies only to the activities described in the IRB submission and does not apply should any changes be made. If changes are made and there are questions about whether these activities are research involving human in which the organization is engaged, please submit a new request to the IRB for a determination. You can create a modification by clicking Create Modification / CR within the study.

If you have any questions, please contact the UCF IRB at 407-823-2901 or irb@ucf.edu. Please include your project title and IRB number in all correspondence with this office.

Sincerely,

Renea Carver
Designated Reviewer
APPENDIX B: MEASUREMENT INVARIANCE SYNTAXES
TITLE: Configural invariance for CES-D 8.

DATA:
FILE IS "MI.dat"; ! Data with two indicator variables.

VARIABLE:
NAMES ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8 LWAVE DATSET;
USEARIABLES ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8 DATSET;
CATEGORICAL ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8;
GROUPING IS DATSET (1=HRS 2=MHAS); ! Two groups are considered.
MISSING ARE ALL (-99);

ANALYSIS:
ESTIMATOR = wlsmv; ! Estimation for ordinal variables/default in Mplus
H1ITERATIONS = 3000;

MODEL:
y1 BY CESD1@1; ! Loading of phantom variable must be fixed to 1 for identification
y2 BY CESD2@1;
y3 BY CESD3@1;
y4r BY CESD4@1;
y5 BY CESD5@1;
y6r BY CESD6@1;
y7 BY CESD7@1;
y8 BY CESD8@1;
F1 BY y1-y8*; ! Factor loadings are estimated freely across groups!
[F1@0]; ! Factor variance is fixed to 0 for all groups
{CESD1-CESD8@1}; ! Factor scale is fixed to 1 in all groups
[y1-y8@0]; ! Intercept means are fixed to 0 in all groups
y1-y8@0; ! Residual variances are fixed to 0 in all groups

MODEL MHAS:
y1 BY CESD1@1; ! Loading of phantom variable must be fixed to 1 for identification
y2 BY CESD2@1;
y3 BY CESD3@1;
y4r BY CESD4@1;
y5 BY CESD5@1;
y6r BY CESD6@1;
y7 BY CESD7@1;
y8 BY CESD8@1;
F1 BY y1-y8*; ! Factor loadings are estimated freely across groups!
[F1@0]; ! Factor variance is fixed to 0 for all groups
{CESD1-CESD8@1}; ! Factor scale is fixed to 1 in all groups
[y1-y8@0]; ! Intercept means are fixed to 0 in all groups
y1-y8@0; ! Residual variances are fixed to 0 in all groups

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TITLE: Invariance of loadings, thresholds and intercepts for CES-D 8

DATA:
FILE IS "MI.dat"; ! Data with two indicator variables.

VARIABLE:
NAMES ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8 DATSET;
USEVARIABLES ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8 DATSET;
CATEGORICAL ARE CESD1 CESD2 CESD3 CESD4 CESD5 CESD6 CESD7 CESD8;
GROUPING IS DATSET (1=HRS 2=MHAS); ! Two groups are considered.
MISSING ARE ALL (-99);

ANALYSIS:
ESTIMATOR = wlsmv; ! Estimation for ordinal variables/default in Mplus
DIFFTEST = prop10.dat ! File to be considered from previous step
H1ITERATIONS = 3000;

MODEL:
y1 BY CESD1@1; ! Loading of phantom variable must be fixed to 1
y2 BY CESD2@1; ! for identification
y3 BY CESD3@1;
y4 BY CESD4@1;
y5 BY CESD5@1;
y6 BY CESD6@1;
y7 BY CESD7@1;
y8 BY CESD8@1;
F1 BY y1-y8*(L1-L8); ! Constrain loadings across groups!
[F1@0]; ! Factor means are fixed to 0 for all groups
{CESD1@1 CESD2@1 CESD3@1 CESD4@1 CESD5@1 CESD6@1 CESD7@1 CESD8@1}; ! Factor scale is fixed to 1 in first group
[y1-y8@0]; ! Intercept means are fixed to 0 in all groups
y1-y8@0; ! Residual variances constrain across groups
{CESD1$1*(T1); ! Thresholds are constrained to equality across CESD2$1*(T2); ! all groups via T1, T2...etc.
CESD3$1*(T3);
CESD4$1*(T4);
CESD5$1*(T5);
CESD6$1*(T6);
CESD7$1*(T7);
CESD8$1*(T8);

MODEL MHAS:
F1 BY y1-y8*(L1-L8); ! Constrain loadings across groups!
F1*; ! Factor variance is estimated in groups other than 1
{CESD1$1*(T1); ! Thresholds are constrained to equality across CESD2$1*(T2); ! all groups via T1, T2...etc.
CESD3$1*(T3);
CESD4$1*(T4);
CESD5$1*(T5);
CESD6$1*(T6);
CESD7$1*(T7);
CESD8$1*(T8);

SAVE DATA:
SAVEDATA:
DIFFTEST = prop11.dat;
APPENDIX C: SLOPE INTERCEPT MODEL SYNTAX
TITLE: LONELINESS & PERCEIVED SUPPORT PREDICT DEPRESSION
DATA: FILE = MediationSocialSupport.dat;
VARIABLE: NAMES = WAVE R9AGEY_E R13AGEYE RAGENDER RAHISPAN RARACEM R9CESD R10CESD R11CESD R12CESD R13CESD PSUPPVT9 PSUPNV9 LONELY9; MISSING = ALL (-99);
USEVARIABLE = R9AGEY_E RAGENDER R9CESD R10CESD R11CESD R12CESD R13CESD PSUPNV9 LONELY9;
ANALYSIS: TYPE=GENERAL; ESTIMATOR=ML; MATRIX=COVARIANCE; ITERATIONS=1000;
MODEL:
I BY R9CESD@1 R10CESD@1 R11CESD@1 R12CESD@1 R13CESD@1; S BY R9CESD@0 R10CESD@1 R11CESD@2 R12CESD@3 R13CESD@4;
[I@0 R9CESD@0 R10CESD@1 R11CESD@2 R12CESD@3 R13CESD@4];
[I S];
I S ON R9AGEY_E RAGENDER PSUPNV9 LONELY9; PSUPNV9 ON LONELY9;
MODEL INDIRECT:
I IND PSUPNV9 LONELY9; S IND PSUPNV9 LONELY9;
LIST OF REFERENCES


Willis, R. J. (1999). Theory confronts data: How the HRS is shaped by the economics of aging and how the economics of aging will be shaped by the HRS. *Labour Economics, 6*(2), 119-145.


