

# A 3-D Model of Health Decline: Disease, Disability, and Depression among Black and White Older Adults\*

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*Much of the research on health decline in older adulthood has specified a single causal direction between two health domains, often measured at the same time point, making it difficult to disentangle the mechanisms of health decline over time. Using three waves of data over seven years from older residents of North Carolina, a 3-D model is used to examine the temporal ordering of general health decline and the pathways of influence across three health domains (disease, disability, and depression). In addition, we test whether the 3-D model of health decline is similar for Black and White older adults. Results indicate that both prevalent and incident disease lead to increases in depression and that prevalent disease leads to greater disability three years later. Depression, in turn, leads to disease, identifying a cycle of health decline that is, for the most part, similar for Black and White older adults. The effect of widowhood is more deleterious to the health of Black older adults than for their White counterparts.*

Health decline in older ages is often a multidimensional process where multiple domains such as physical function and mental health deteriorate simultaneously or within a short time of each other. Verbrugge and Jette (1994) argue

that health decline, which includes disablement, rarely affects only a single body system. Rather, its scope includes cognitive, emotional, physical, sensory, and social pathologies that limit the routine and desired behaviors of an individual. While we know that disability, disease, and depression can affect each other over time, it is difficult to disentangle the causality (and sometimes reciprocal causality) between such domains. Some important longitudinal work has helped to identify these processes, finding that physical illness and poor mental health are related over time (Aneshensel, Frerichs, and Huba 1984; Ormel et al. 2002). However, much of this work has specified only unidirectional relationships and has not separated the disease, disability, and depression domains.

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In addition, there has been relatively little research on Black-White differences in the process of health decline over time. A stream of research has focused on the interplay of mental and physical health over time, particu-

larly their contribution to systemic health decline at advanced ages (e.g., Beekman et al. 1997; Reynolds, Crimmins, and Saito 1998), but much of this work has been conducted on all-White samples of older adults. The relationships among disease, disability, and depression over time may vary by race. For example, does depression lead to greater disability over time? If so, is it the case for both Black and White older adults? The present research seeks to identify the processes of health decline with repeated measures of these three health domains using a prospective study of more than 4,000 Black and White community-dwelling older adults. We draw from research on multidimensional health decline and racial health disparities in later life to help frame our analysis.

## THEORY AND EVIDENCE

### *Multidimensional Nature of Health Decline*

The physical and mental domains of health are very difficult to separate and consider independently. Research has shown that depression and physical disability are not merely correlated, but may actually exacerbate one another over time (Ormel et al. 2002). Bouts of depression are often seen among adults with chronic health conditions (Wilkinson and Lynn 2001). Additionally, the mere presence of depression may affect the trajectory and potential recovery of the person, even when the depression develops after the health condition (Carney and Freedland 2001; Cronin-Stubbs et al. 2000). This interrelatedness between physical and mental health problems is especially acute among older adults, who are more likely to experience both (Aneshensel et al. 1984; Ormel et al. 2002).

Two issues are pivotal to advancing our understanding of how mental and physical health may contribute to broader health decline. First, the strong association between depression and poor physical health may be indicative of reciprocal relations. As adults age, there is a higher likelihood of having more than one health condition at any given time. These comorbid conditions can, in turn, accelerate disability (Ferraro, Farmer, Wybraniec 1997; Guralnik et al. 1991). When considering the role of mental health, it is possible that depression could be an intervening factor between disease and disability. In addition, depressed affect may trigger psychoneuroimmunological responses that are dele-

terious to physical health (Kiecolt-Glaser et al. 2002). Ultimately, disease, disability, and depression may interact to create feedback loops, and “loops like this can become vicious spirals” (Verbrugge and Jette 1994:7).

When testing one direction of this relationship only, the researcher assumes—perhaps unknowingly—that the alternative process is not operant. As such, estimates of the presumed relationship between the two may be misleading. Much of the existing research has examined one causal direction only. Some studies have found that higher levels of depression are associated with higher levels of disability (Lyness et al. 1999; Penninx et al. 1999; Steffens et al. 1999; Oslin et al. 2000; Simons et al. 2000; Geerlings et al. 2001). A few researchers have considered the opposite pathway finding that poor physical health leads to greater depression (Beekman et al. 1997; Stump et al. 1997).

The relationship between these domains of health decline among older adults may take time to develop and the effects may not be seen immediately. For this reason, it can be difficult to discern specific mechanisms with a cross-sectional sample (Campbell 1988). A number of longitudinal studies have examined the lagged effect of one domain on the other, finding significant unidirectional causal relationships (Geerlings et al. 2001; Oslin et al. 2000; Simons et al. 2000). However, only a few studies have examined the simultaneous relationship of both the physical and mental health domains on each other over time (exemplars include Aneshensel et al. 1984 and Ormel et al. 2002). From this previous work, we know that depression and physical illness are intricately linked over time in reciprocal loops (Aneshensel et al. 1984). In addition, age contributes little to the health decline process; the presence of functional limitations is a stronger predictor of depression than age (Berkman et al. 1986). However, this refers only to relationships between mental and physical health measured at one time point. The timing and links between these health domains have not been clearly established.

The second issue to consider is that there may be an unidentified common cause of both depression and poor physical health, creating a spurious relationship between the two domains. Risk factors such as social isolation, poverty, obesity, or the side effects of medication have been shown to cause independently heightened depression and worsened physical health (Carney and Freedland 2001; Carpenter, Hasin,

and Allison 2000; Stansfield 1999). Failing to identify key social and biological precursors to declines in mental and physical health such as these may lead to an overestimate of the degree of influence on each other. Much of the previous research evaluating the relationship between depression and poor physical health has not tested for common causes of both health outcomes simultaneously.

It is important to identify the specific mechanisms among these three domains of health over several years. One study examined the relationship between depression and physical illness with repeated measures of both over time, but the latter was a single factor composed of disability days and number of illnesses (Aneshensel et al. 1984). The individual influence of disability and disease on subsequent depression is still unknown, so a logical next step would be to examine those concepts separately.

Thus, we propose a "3-D" model of health decline that examines the influence of disease, disability, and depression. Etiologically, we envision disease (or morbidity) as the main engine of health decline, but coupled with reciprocal relations between the three *d*'s. Each *d* is treated as both an independent and dependent variable although models of disability typically place disease as antecedent (Verbrugge and Jette 1994). Some diseases may not heighten disability, but even the medications or treatment regimens can disrupt functioning, including sleep and appetite. As such, disease and disability often precipitate a negative affective response. This may not necessarily lead to depression, but it raises the risk of bouts of depressive symptoms, perhaps leading to a cycle of health decline. Disease heightens disability and the risk of depression; each, in turn, may raise the risk of additional disease (comorbidity). We are unaware, however, of any study that examines such a 3-D model of disease, disability, and depression over time.

### *Race Differences in Health Decline*

Lawrence and Jette (1996) noted the dearth of studies with enough racial and ethnic variation to test for subgroup differences in the process and timing of disablement in particular, and, more generally, health decline. Scholars generally concur that Black older adults tend to have poorer physical functioning (Andresen and Brownson 2000; Kelley-Moore and Ferraro

2004; Mendes de Leon et al. 1997) and more prevalent health conditions (Manton and Stallard 1997) than White older adults. Unfortunately, little is known about whether the process of health decline is the same or similar across races because the majority of the existing research on the relationships between depression, physical function, and morbidity have been on samples of White older adults (e.g., Alexopoulos et al. 1996; Beekman et al. 1997; Prince et al. 1997; Simon et al. 1998; Leibson et al. 1999; Lyness et al. 1999; Penninx et al. 1999; Simons et al. 2000; Aneshensel et al. 1984; Reynolds et al. 1998; Allair et al. 1999).

The lack of clear evidence on race differences in the relationships among these three domains has deterred our ability to determine the mechanisms of health decline over time. Does depression contribute significantly to disability over time for White older adults but not for Black older adults, as has been shown in previous studies (Cummings, Neff, and Husaini 2003; Harralson et al. 2002; Leveille et al. 1998)? Or, consistent with another body of literature, might the relationships among these three health domains be similar across race groups (Blazer et al. 1998; Berkman et al. 1986)?

In addition, a number of studies have shown that certain risk factors for poor physical function over time are stronger predictors of health decline for either Black or White older adults. For example, body composition and fat distribution contribute to a greater risk of disability in White older adults relative to Black older adults (Perry et al. 2000). Moreover, socioeconomic factors seem to contribute to higher activities of daily living disability among Black older adults, but not White older adults (Mendes de Leon et al. 1995). A systematic examination of common risk factors may yield important information about the differences and similarities in the process of health decline for Black and White older adults; however, this has not been studied previously.

This article uses panel data from a seven-year community study to address two aims. First, we explicate the processes of health decline by disentangling the relationships between mental and physical health over time. Specifically, we focus on the multidimensional and complex relationships among three health domains over time: disease, disability, and depression. Using repeated measures of all three domains, we seek to identify how health declines. Are there, in

fact, “pernicious loops of dysfunction” among disease, disability, and depression among older adults over time (Verbrugge and Jette 1994)? Second, we test for racial differences in the interrelationships between disease, disability, and depression to determine if the process of health decline is similar for Black and White older adults. We seek to identify whether certain risk factors have greater influence on health decline over time for Black and White people. Given the substantial social and health inequality in American society, might the relationships between disease, disability, and depression—and their associated risk factors—be exacerbated for Black older adults?

## METHODS

### Sample

The data for this research are from the North Carolina Established Populations for the Epidemiologic Study of the Elderly (EPSE).

In 1986, a multistage, random sample of five counties was conducted, and 4,162 residents who were age 65 or older were interviewed. Black adults compose more than half of the baseline sample (54 percent). Subjects were reinterviewed yearly for seven years. The first, fourth, and seventh interviews were more extensive and were conducted in the home. The remaining interviews were conducted by telephone. See Cornoni-Huntley et al. (1993) for sampling procedures.

Table 1 shows all of the variables, coding, and descriptive statistics for the total sample and separately for Black and White respondents. There are three endogenous variables in these analyses: disease, disability, and depression. Depression was only measured during the three in-home interviews, so the other two outcome variables are used only at those waves. For these analyses, the three surveys conducted in the home are referred to as wave 1, wave 2, and wave 3, with three years between waves. The sample N for these analyses is 3,642. This does not equal the total wave 1 sample due to

**TABLE 1. Variables, Coding, and Descriptive Statistics**

Variables	Coding	Total Sample, N = 3,642	Black Adults, N = 1,988	White Adults, N = 1,654
Prevalent disease W1	7 prevalent conditions	1.28 (1.06)	1.29 (1.06)	1.27 (1.05)
Incident disease W2	7 incident conditions since W1	.64 (.68)	.68 (.68)	.60 (.67)***
Incident disease W3	7 incident conditions since W2	.61 (.69)	.64 (.67)	.57 (.72)**
Disability W1	Ranges from 0 (none) to 14 (high)	.56 (1.57)	.61 (1.62)	.51 (1.51)
Disability W2	Ranges from 0 (none) to 14 (high)	.83 (2.05)	.87 (2.02)	.78 (2.07)
Disability W3	Ranges from 0 (none) to 14 (high)	1.09 (2.48)	1.23 (2.60)	.93 (2.31)**
Depression W1	Ranges from 0 (low) to 20 (high)	3.11 (3.41)	3.25 (3.35)	2.95 (3.48)**
Depression W2	Ranges from 0 (low) to 20 (high)	2.19 (3.29)	2.28 (3.21)	2.07 (3.38)*
Depression W3	Ranges from 0 (low) to 20 (high)	2.08 (3.25)	2.19 (3.31)	1.94 (3.16)*
Female	1 = female; 0 = male	.65	.65	.65
Black	1 = Black; 0 = White	.54	—	—
Age	Ranges from 64 to 105	73.55 (6.72)	73.62 (6.85)	73.48 (6.57)
Lives in rural area	1 = yes; 0 = otherwise	.44	.44	.45
Education	1 = 8 or fewer years; 6 = postcollege	2.03 (1.45)	1.74 (1.37)	2.37 (1.47)***
Income	1 = less than \$1,000; 11 = more than \$40,000	5.36 (2.32)	4.53 (1.88)	6.36 (2.42)***
Currently employed	1 = yes; 0 = otherwise	.12	.11	.12
Religious service attendance	1 = never; 6 = more than once per week	4.00 (1.72)	4.15 (1.57)	3.82 (1.86)***
Close relatives	0 = none; 3 = 5 or more	2.13 (1.11)	2.15 (.02)	2.12 (.03)
Lives alone	1 = yes; 0 = otherwise	.38	.35	.41***
Obese	1 = yes; 0 = otherwise	.25	.33	.17***
Incontinence	1 = never; 5 = all the time	2.02 (1.12)	2.01 (1.12)	2.03 (1.13)
Current smoker	1 = yes; 0 = otherwise	.17	.16	.18
Past smoker	1 = yes; 0 = otherwise	.26	.23	.30***

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

Source: North Carolina Established Populations for the Epidemiologic Study of the Elderly, 1986–1992.

Notes: Numbers are means with standard deviations in parentheses. Significant differences between Black and White adults were tested with  $t$ -tests for continuous and ordinal variables and chi-square for binary variables. W1 = wave 1; W2 = wave 2; W3 = wave 3.

item missing on the three dependent variables.<sup>1</sup> Community-dwelling samples such as this one tend to underestimate health problems in an older adult population.

*Disease* at baseline is a measure of prevalent health conditions, and disease at waves 2 and 3 are measures of incident health conditions. Prevalent disease is a sum of seven existing health conditions asked of the respondent at baseline. Interviewers asked, "Since the age of 50, have you ever been told by a doctor, nurse, therapist, or medical assistant that you had . . . ?" These conditions were heart attack, stroke, cancer, diabetes, high blood pressure, broken or fractured hip, and other broken bones. Those who had the condition were given a score of 1 and all others a score of 0. The conditions were then summed to create a prevalent disease measure ranging from 0 to 7. At subsequent waves, respondents were asked to report their new health conditions in the previous year.

Disease at waves 2 and 3 reflects counts of incident diseases in the previous three years and includes new reported health conditions at the interim telephone interviews. We selected this measurement scheme of disease for two reasons. Investigators using panel data must carefully consider the temporal ordering of phenomena. A prevalent or cumulative measure of morbidity would confound tests of causality because health conditions could be "predicted" by disability or depression that technically occurred afterward.

The second reason we have chosen to use incident disease at waves 2 and 3 is that some health conditions such as broken bones can heal with time and no longer contribute to other domains of health such as physical functioning. Continuing to include them in a cumulative measure of morbidity would in fact not accurately reflect the long-term health process. Indeed a strength of EPESE is that one can become nonsymptomatic (i.e., healed). Some surveys use the "ever had" question form to probe diseases. In those situations, disease, by definition, cannot decline. We believe that the EPESE approach and our application of it is a sensitive and valid method.

*Disability* was measured with seven domains of activities of daily living: walking, bathing, grooming, dressing, eating, transferring from a bed to a chair, and using the toilet. Respondents reported whether they were currently able to perform the function independently (0), with some help (1), or not at all (2). The seven functions were then summed, creating a

summary score of physical function ranging from 0, meaning able to perform all seven activities independently, to 14, meaning unable to perform any of them ( $\alpha = .89$ ). Disability was measured identically at all three waves.

For *depression*, respondents were asked a series of 20 "yes or no" questions about their general feelings and mood in the past week from the Centers for Epidemiologic Studies Depression subscale ( $\alpha = .83$ ; Radloff 1977). Questions included, "Have you felt restless in the past week?" and "Have you felt people were unfriendly?" Those who responded affirmatively were coded as 1 and all others were coded as 0. Four statements were reverse coded. The 20 binary variables were then summed, creating one score of depressive mood, with a possible range of 0 (low) to 20 (high). The questions remained consistent across the three surveys.

Persons with a body mass index greater than or equal to 30 are classified as obese and identified in a binary variable with a value of 1. All others are coded 0. Current smokers and past smokers are also dichotomous variables where 1 equals the name of the variable and 0 for all others. Another health indicator measured at baseline was incontinence, which was assessed by asking respondents how often they had difficulty holding their urine. Categories ranged from never (1) to all of the time (5).

Socioeconomic status is measured with education, income, and being currently employed. Education ranges from fewer than eight years (1) to postcollege (6). Total household income has 11 categories, ranging from less than \$1,000 (1) to greater than \$40,000 (11). Current employment is a binary variable with those working having a value of 1 (all others 0).

To control for the effect of social integration on subsequent health, four indicators are used in these models. Religious service attendance is an ordinal variable ranging from never (1) to more than once per week (6). Those who are currently widowed and those who live alone are identified with binary variables where 1 equals the name of the variable and all others are coded 0. Respondents were asked how many relatives they had to which they feel really close. Categories range from none (0) to five or more (3).

Finally, several demographic variables are included in these models. Rural residents, females, and Black adults are identified with individual binary variables having the value of 1 for the name of the variable (others are

coded 0). Age is measured in years and ranges from 64 to 105. Several other variables were tested in preliminary models and eliminated because they did not significantly predict disease, disability, or depression. They included currently married, home ownership, primary labor career, primary professional career, and number of living children.

### *Analysis Plan*

The analysis proceeds in four stages. First, descriptive statistics are calculated for the total sample and then separately for Black and White older adults. Significant differences in means by race are calculated with *t*-tests for continuous and ordinal variables and with chi-square tests for binary variables. The second stage of the analysis is to test the measurement model with just the nine endogenous variables. Repeated measures of disease, disability, and depression are regressed on all three measures at the previous wave and correlated within the same wave. In the third stage of analysis, the measurement model is extended to the full model with exogenous covariates. This model tests whether the relationships between disease, disability, and depression hold after controlling for demographic, socioeconomic status, and other health indicators.

The final stage of analysis tests for race differences in the full model. Multigroup models are tested for Black and White older adults to determine if the process of health decline and its risk factors are similar by race. We do this by estimating the Black and White older adult subsamples as separate groups and constrain one path at a time to be equal between the two groups. To determine significant differences in risk factors, we individually test the paths between endogenous health domains and the paths from exogenous covariates to the health domains. Even when a covariate significantly predicts an outcome for one group and not the other, the confidence intervals may overlap. A significant increase in chi-square per one degree of freedom (greater than or equal to 3.84) indicates a significant difference in that relationship between Black and White older adults. We present the parameter estimates for all models with unstandardized coefficients and standard errors.

All three outcomes are discrete count variables with non-normal distributions. The result-

ing estimates may be sensitive to such departures from normality so we use nonparametric correlations to generate the input covariance matrix in the final models based on polychoric and polyserial correlations. In addition, we conduct a series of diagnostic model tests such as examining the residuals. In the final model, these values were generally small and were not drastically different from each other (Bollen 1989). In the results, we present the root mean square error of approximation (RMSEA) for each model, which is calculated from the residuals. We also examine and present fit measures such as the Incremental Fit Index because they are standardized and not dependent on total sample size or degrees of freedom (Kelloway 1998).

### *Attrition*

There is one important issue that spans all of the models in this analysis. Attrition, due primarily to death, is extensive in this sample. Just over 2,500 of the original 4,162 respondents participated in the last interview. Failing to account for nonrandom attrition and other mechanisms of selection typically produces estimates on a relatively privileged sample in terms of health, socioeconomic status, and cognitive ability, leading to potentially biased results (Lillard and Panis 1998).

To adjust the estimates of the measurement model for this selection effect, the analysis proceeds with a multigroup model containing complete and incomplete data (Allison 1987; McArdle 1994; McArdle and Hamagami 1991; Bollen 1989). This model contains three groups, each with a different pattern of missingness. Group 1 has complete data for all three waves. Group 2 has complete data on the first two waves and is missing on the third wave. Group 3 has complete data on the first wave only.<sup>2</sup>

The estimated matrix is an augmented moment matrix calculated from a covariance matrix and a vector of means. Group 1 has estimates for each element. Groups 2 and 3 have estimates only for the nonmissing elements. The missing covariances are replaced with 0 and the missing variances and means with the value of 1. These values are essentially placeholders so that the shape and size of the matrices and vectors are consistent across the three groups. The fully constrained maximum likelihood

model has invariant factor loadings ( $\lambda$ ), parameter estimates ( $\beta$ ), and measurement error ( $\theta$ ) across groups (Bollen 1989).<sup>3</sup> Relaxing these constraints for the measurement error and factor loadings tests the degree to which the three groups differ from each other (McArdle 1994). This multigroup method is used for all of the models in this article.

## RESULTS

Table 1 shows the descriptive statistics of all of the variables in the analysis for the total sample and then separately for Black and White adults. Disability systematically increased and depression systematically decreased over the three waves, but there were some differences by race. Black adults had significantly higher disease incidence at waves 2 and 3 and significantly higher depression at all three waves compared to their White counterparts. Disability was significantly different only at wave 3, with Black adults having more physical limitations.

Prior to estimating the measurement model with all nine of the endogenous variables, it is necessary to examine the influence of attrition on the estimates of the variables. Table 2 shows the mean, standard deviation, and sample size for each of the three groups defined by their pattern of attrition: present at all three waves (group 1), present only at waves 1 and 2 (group 2), and present only at wave 1 (group 3). The means for disease, disability, and depression are significantly lower for group 1 with complete data, supporting the earlier assertion that attrition is a nonrandom process. Indeed,

the means on all three wave 1 variables are significantly higher for group 3 than for the other two groups. As a note, only four of the estimates differed by race: incident disease at waves 2 and 3, disability at wave 3, and depression at wave 3. Black adults were significantly higher on each of the four outcomes, indicating that those who remained in the sample were in significantly worse health than the White adults who remained in the sample.

The measurement model, with the multigroup procedure for sample attrition, allows disease and depression to predict disability at subsequent waves while disability simultaneously predicts disease and depression. All within-wave measures are correlated. These correlations are not presented in Table 2, but are briefly described here. All of the within-wave correlations at baseline are significant and positive, meaning that the presence of any of the three health domains is associated with higher levels of the other domains ( $p < .01$ ). However, once the lagged measures of disease, disability, and depression were used to predict the later health measures, within-wave correlations at waves 2 and 3 became nonsignificant.<sup>4</sup>

The results of the causal measurement model are presented in Table 3, using all three groups. Almost every path is significant, and all are in a positive direction, indicating that the presence of one is a positive predictor of the presence of another three years later. Prevalent disease, incident disease, and depression predict higher disability, and at the same time, disability predicts higher incident disease and depression at a later time point.

Model statistics indicate a very good fit. Chi-square is 250.08 with 36 adjusted degrees of

**TABLE 2. Descriptive Statistics for Endogenous Variables in the Groups Defined by Attrition**

Variables	Group 1, Responded at W1, W2, and W3	Group 2, Responded at W1 and W2	Group 3, Responded at W1 Only
Prevalent disease W1	1.20 (1.01)	1.39 (1.09)	1.63 (1.21)
Disability W1	.35 (1.21)	.90 (1.97)	1.34 (2.51)
Depression W1	2.96 (3.14)	3.37 (3.53)	3.63 (3.78)
Incident disease W2	1.32 (1.08)	1.46 (1.16)**	—
Disability W2	.55 (1.62)	2.05 (3.59)	—
Depression W2	2.39 (3.34)	1.44 (2.94)	—
Incident disease W3	1.38 (1.11)**	—	—
Disability W3	1.09 (2.48)**	—	—
Depression W3	2.08 (3.25)*	—	—
N	2,501	706	435

\*  $p < .05$ ; \*\*  $p < .01$

Source: North Carolina Established Populations for the Epidemiologic Study of the Elderly, 1986–1992.

Notes: Numbers are means with standard deviations in parentheses. Significance indicates statistically significant differences between Black and White older adults. W1 = wave 1; W2 = wave 2; W3 = wave 3.

**TABLE 3. Maximum Likelihood Estimates of the Measurement Model for Disease, Disability, and Depression**

Independent Variables	Incident Disease W2	Disability W2	Depression W2	Incident Disease W3	Disability W3	Depression W3
Prevalent disease W1	.39*** (.02)	.09*** (.03)	.33*** (.05)	—	—	—
Incident disease W2	—	—	—	.30*** (.02)	.13*** (.03)	.25*** (.04)
Disability W1	.07*** (.02)	.32*** (.02)	.16** (.04)	—	—	—
Disability W2	—	—	—	.05*** (.01)	.43*** (.02)	.03 (.03)
Depression W1	.05*** (.01)	.03*** (.01)	.22*** (.02)	—	—	—
Depression W2	—	—	—	.04*** (.01)	.04** (.01)	.23*** (.02)
R <sup>2</sup>	.30	.46	.26	.32	.56	.25

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

Source: North Carolina Established Populations for the Epidemiologic Study of the Elderly, 1986–1992.

Notes: Numbers are slopes with standard errors in parentheses. Total N = 3,642. Model statistics: chi-square = 250.08; adjusted degrees of freedom = 36; root mean square error of approximation = .038; goodness-of-fit index = .98; W1 = wave 1; W2 = wave 2; W3 = wave 3.

freedom.<sup>5</sup> Other fit statistics are excellent: the goodness-of-fit index is .98. The root mean square error of approximation (RMSEA) is .038, also signifying a good overall model fit. Obtaining this good model fit, however, required the relaxation of several matrix invariance constraints across groups. While the factor loading and parameter matrices remained invariant for all three groups, measurement error did not. Groups 2 and 3 had significantly stronger within-wave measurement error than group 1. Allowing these terms to correlate produced a better model fit and significantly better chi-square value ( $p < .001$ ).

The next stage of analysis tests the measurement model while controlling for covariates. Again, all three groups are entered into the model simultaneously. Table 4 presents these results. The chi-square value is 340.81 with 41 adjusted degrees of freedom. The RMSEA is .04, but still within the range of a well-fitting model. Figure 1 presents all three endogenous health domains over time and shows the significant paths between each possible pair of variables, including within-wave correlations. For ease of presentation, the relationships between exogenous covariates and the health outcomes were eliminated from the figure. Examining the covariates, disability no longer significantly predicts incident disease or depression at either wave. Prevalent disease at baseline predicts significantly higher depression at wave 2, and inci-

dent disease at wave 2 predicts higher depression at wave 3. Depression also predicts greater incident disease at the next wave. Prevalent disease at wave 1 predicts higher disability at wave 2. Incident disease does not lead to higher disability over time. After controlling for independent covariates, depression and disability do not significantly predict each other over time.

Higher prevalent disease at wave 1 is found among women, Black respondents, older adults, those who live in a rural area, and those who have been widowed. Lower socioeconomic status is associated with the presence of more health conditions: Those with lower incomes or less education have higher prevalent disease. More frequent attendance at religious services and having more close relatives is associated with having more health conditions at wave 1. Being obese, being incontinent, currently smoking, and being a past smoker are all strong predictors of higher prevalent disease. Incident disease at waves 2 and 3 share all of the same predictors except for education, which does not significantly predict disease at wave 2.

Disability is predicted by itself at earlier waves and by higher prevalent disease, as noted earlier. Females, those not currently employed, and those who are incontinent have higher disability at baseline. The latter two variables consistently predict change in disability by waves 2 and 3, as does widowhood. In addition, older age and not living alone lead to increases in disability by wave 3.<sup>6</sup>

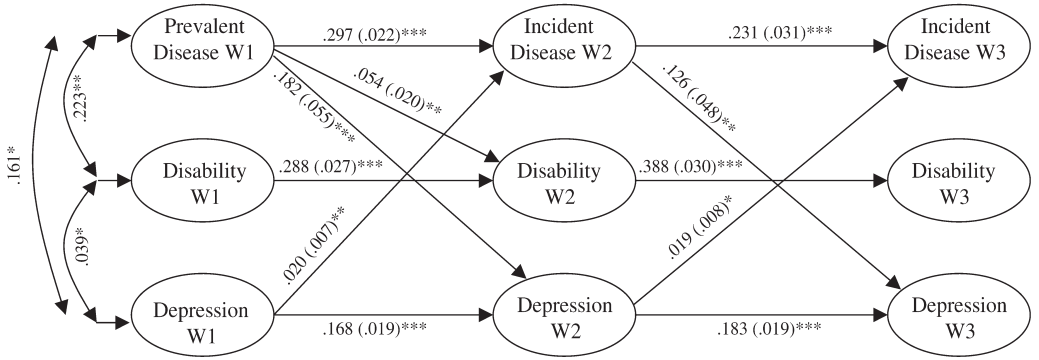
TABLE 4. Maximum Likelihood Estimates of Disease, Disability, and Depression over Seven Years

Independent Variables	Prevalent Disease W1	Incident Disease W2	Incident Disease W3	Disability W1	Disability W2	Disability W3	Depression W1	Depression W2	Depression W3
Prevalent disease W1	—	.297*** (.022)	—	—	.054*** (.020)	—	—	.182*** (.055)	—
Incident disease W2	—	—	.231*** (.031)	—	—	.063 (.045)	—	—	.126** (.048)
Disability W1	—	.010 (.023)	—	—	.288*** (.027)	—	—	.093 (.060)	—
Disability W2	—	—	-.002 (.018)	—	—	.388*** (.030)	—	—	-.011 (.042)
Depression W1	—	.020** (.007)	—	—	.015 (.008)	—	—	.168*** (.019)	—
Depression W2	—	—	.019* (.008)	—	—	.018 (.013)	—	—	.183*** (.019)
Female	.191*** (.040)	.202*** (.043)	.166*** (.046)	.077* (.037)	.049 (.049)	.093 (.076)	.602*** (.118)	.307** (.111)	.263* (.106)
Black	.123** (.042)	.175*** (.045)	.172*** (.048)	.07 (.038)	.092 (.051)	.149 (.079)	.372** (.123)	.266* (.116)	.223* (.110)
Age	.015*** (.001)	.003** (.001)	.003** (.001)	.001 (.001)	.001 (.001)	.002* (.001)	.005*** (.001)	.003** (.001)	.002 (.001)
Rural area	.123** (.045)	.123** (.048)	.119* (.050)	-.019 (.041)	.029 (.054)	.061 (.083)	.316* (.131)	.220 (.123)	.153 (.117)
Income	-.016** (.006)	-.014* (.006)	-.017* (.007)	-.005 (.005)	-.004 (.007)	-.008 (.011)	-.034* (.017)	-.016 (.016)	-.020 (.015)
Education	-.034** (.013)	-.022 (.014)	-.032* (.015)	-.011 (.012)	-.011 (.016)	-.012 (.025)	-.018 (.039)	-.020 (.037)	-.001 (.035)
Currently employed	-.144 (.076)	-.054 (.082)	.003 (.086)	-.234*** (.070)	-.243** (.093)	-.439** (.142)	-.557* (.224)	-.332 (.210)	-.197 (.200)
Widowed	.126** (.045)	.111* (.048)	.134** (.051)	.031 (.041)	.120* (.055)	.203* (.083)	.529*** (.131)	.175 (.123)	.159 (.117)
Religious service attendance	.021* (.008)	.022* (.008)	.022* (.009)	-.005 (.007)	-.007 (.009)	.002 (.014)	.044* (.022)	.027 (.021)	.025 (.020)
Close relatives	.040** (.014)	.032* (.015)	.039* (.016)	.012 (.013)	.014 (.017)	.033 (.041)	.047 (.041)	.027 (.038)	.038 (.036)
Lives alone	.115 (.106)	.092 (.113)	.089 (.119)	-.011 (.097)	-.143 (.129)	-.584** (.196)	.330* (.153)	.161 (.221)	.162 (.226)
Obese	.178** (.059)	.168** (.063)	.168* (.067)	.096 (.054)	.124 (.072)	.144 (.110)	.409* (.173)	.288 (.162)	.238 (.154)
Incontinence	.070*** (.015)	.048** (.016)	.057*** (.017)	.054*** (.013)	.044* (.018)	.090*** (.027)	.245*** (.043)	.138*** (.041)	.097* (.039)
Current smoker	.289*** (.076)	.201* (.082)	.248** (.086)	.010 (.069)	-.022 (.093)	-.046 (.142)	.876*** (.223)	.613** (.209)	.667*** (.199)
Past smoker	.212*** (.066)	.170** (.066)	.195** (.070)	.031 (.057)	.037 (.076)	.018 (.115)	.621*** (.182)	.238 (.170)	.282 (.162)
R <sup>2</sup>	.264	.824	.847	.115	.285	.371	.220	.281	.263

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ 

Source: North Carolina Established Populations for the Epidemiologic Study of the Elderly, 1986–1992.

Notes: Numbers are unstandardized slope coefficients with standard errors in parentheses. Total N = 3,642; chi-square = 340.81; adjusted degrees of freedom = 41; root mean square error of approximation = .043; goodness-of-fit index = 1.00; W1 = wave 1; W2 = wave 2; W3 = wave 3.

**FIGURE 1. Model of Health Decline over Seven Years for Black and White Older Adults Adjusted for Covariates**

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

Source: North Carolina Established Populations for the Epidemiologic Study of the Elderly, 1986–1992.

Notes: Two-headed arrows display correlations and single-headed arrows display unstandardized slope coefficients. Standard errors are in parentheses. W1 = wave 1; W2 = wave 2; W3 = wave 3. All of the cross-lagged paths and within-wave correlations were estimated. After controlling for covariates, some paths became nonsignificant. For clarity of presentation, these paths were eliminated from the figure.

Higher depression at baseline is found among women, Black respondents, older adults, those who live in a rural area, those who have been widowed, and those who attend religious services more frequently. Higher income and current employment are associated with lower levels of depression. Health indicators of obesity, incontinence, current smoking, and being a past smoker all predict higher depression scores. Increases in depression by wave 2 are predicted only by being female, Black, older, incontinent, and a current smoker. Greater increases in depression by wave 3 are found among women, Black adults, those who are incontinent, and current smokers.

The final stage of analysis estimates the relationships among disease, disability, and depression separately for Black and White older adults, using the multigroup procedure described earlier to account for nonrandom attrition.<sup>7</sup> This stage of analysis has two purposes: (1) to determine if the relationships among the three health domains are consistent by race and (2) to test if there are differences in the risk factors for health decline between Black and White older adults. The model is estimated for two subsamples: Black and White older adults.

The estimates indicate that, after controlling for covariates, the causal relationships among the three endogenous health domains over time for the Black subsample are consistent with the White subsample. Even the majority of the

predictors are similar across groups. There are, however, a few notable exceptions. Living in a rural area and being obese each increase the risk of incident disease for White older adults more than for Black older adults. Widowhood is associated with higher disease for Black older adults at baseline and over time, but being widowed does not heighten risk of prevalent or incident disease for White older adults.

## DISCUSSION AND CONCLUSION

Health decline is a complex and dynamic process in older adulthood, with mental and physical health often simultaneously exacerbating the other. This study had two primary aims in seeking to better understand health decline. First, we set out to disentangle the multidimensional and reciprocal relationships between mental and physical health over time. Second, we tested whether the process of health decline is similar for Black and White older adults by testing for racial differences in the interrelationships among disease, disability, and depression. Our 3-D model of health decline used repeated measures of three domains (disease, disability, and depression) over a period of seven years to identify the temporal ordering of general health decline and determine the chains of risk between these domains. These results indicate that, after adjusting estimates

for covariates, there is a reciprocal relationship between disease and depression over time. Disability is predicted by prevalent diseases at baseline, but disability does not lead to incident diseases or to depression during the span of the study.

Our proposed 3-D model provided a unique opportunity to examine the temporal ordering of health decline because we did not specify an a priori causal chain among disease, disability, and depression. Thus, it is possible to elucidate the complex and reciprocal relationships between mental and physical health in general health decline. For instance, we found that prevalent disease at baseline leads to both higher depression and higher disability over time, and incident disease caused greater depression at later waves.

Although social scientists generally agree that health decline is a complex multidimensional process, many previous longitudinal investigations of health decline only specify unidirectional causality (e.g., Geerlings et al. 2001; Oslin et al. 2000; Simons et al. 2000). The net effect is a possible overestimation of the relationship in one direction and a possible unidentified relationship in the other direction.

To counter this, the present article tested the cross-lagged paths between disease, disability, and depression over time and simultaneously accounted for the within-wave associations among the three domains. We found that the baseline within-wave correlations were significant and positive, but at later waves when previous health measures were controlled, these correlations were not significant. This underscores our argument that, for many older adults, health decline is a process that occurs over several years rather than instantaneously. Failing to account for such a time lag may yield significant correlations between the domains at a single time point, but does not reflect the actual processes at work.

These results also showed a positive association between a health domain and its previous measurement. For example, heightened levels of depression led to greater depression three years later. These positive and significant lagged effects are not uncommon, particularly among variables measured identically over time. Two relationships, however, are of particular interest. First, despite an apparent decrease in mean depression scores over time, we found a positive association between age and depression. This relationship appeared only after we

adjusted our estimates for nonrandom attrition, indicating that the relationship between age and depression may be stronger for those at highest risk of death or institutionalization. The results of our multigroup analysis demonstrated that attrition was more likely among those who were older, exhibited more depressive symptoms, had greater disability, and had multiple health problems. Inclusion of older adults who are both older and more likely to be depressed in panel studies may help elucidate the dynamics of health decline in later life. Our finding of a positive relationship between depression and age is consistent with other research using these North Carolina EPESE data (George and Lynch 2003). In a related finding, we observed that the strength of the relationships among disease, disability, and depression diminished somewhat over time but that age was positively associated with the three domains. It is unlikely that advancing age itself increases risk of health decline, but rather age-related stressors such as widowhood or cognitive impairment. Future research should consider other triggers of health decline over time to tease out its relationship with age.

The second relationship of interest in these analyses is the positive relationship between prevalent disease and incident disease over seven years. Those who had more prevalent diseases at baseline were at risk of a greater number of incident diseases three years later. Thus, those who are experiencing health decline are likely to have even more health problems over time. One possible pathway between prevalent disease and greater incident disease is that some health conditions can lead to other conditions; for instance, hypertension is associated with heightened risk of heart disease. In effect, this article and others (e.g., Guralnik et al. 1991) confirm that the sickest continue to get sicker.

Before controlling for the independent covariates, the longitudinal relationships among these three mental and physical health domains demonstrated that disease, disability, and depression all led to higher levels of each other over time. After controlling for covariates, however, some of the relationships disappeared, namely, disability causing higher depression and greater incident disease. Conversely, depression and incident disease no longer predicted disability over time. At first glance, it may seem logical to conclude that depression and diseases are not associated with disability and vice versa. In actuality, the previously significant relationships

become nonsignificant only after including covariates such as obesity, current employment, incontinence, and widowhood that are important predictors of health status. These independent variables may be common causes for disease, disability, and depression, rendering the spurious relationship between certain health domains nonsignificant. As we noted in the beginning, this article is one of the first to estimate the impact of key independent variables on the relationships among the three health domains simultaneously, making it possible to separate and identify the causal relationships between mental and physical health in the process of health decline over time.

This 3-D model of health decline has the advantage of allowing researchers to examine how multiple health domains can exacerbate or counterbalance each other over time. Failure to account for the complexity of later-life health may lead to overestimating the influence of unidirectional relationships between two domains and virtually ignoring the dynamic interplay of mental and physical health over time. This model of health decline can be extended to include a number of other health domains, including cognition, social well-being, and subjective health. With the bevy of research demonstrating a mind-body connection, particularly among older adults (e.g., Clark, Long, and Schiffman 1999), researchers should cautiously consider whether cross-sectional or unidirectional specifications between these mental and physical domains appropriately capture the realities of health in older adulthood.

Our second aim was to test for Black-White differences in the interrelationships among disease, disability, and depression to determine if the process of health decline was similar for the two groups. Multigroup tests of relationships among the health domains over time indicate that Black and White older adults share similar processes of general health decline. There were no significant differences in the relationships between disease, disability, and depression for the Black and White subsamples. For example, depression leads to higher incident disease for both Black and White adults. From this we conclude that the process of health decline is consistent across race groups. There were, however, differences in some risk factors for health decline. White older adults who live in rural areas or are obese were more vulnerable to incident disease, which then led to greater subsequent depression. Black older adults who

have been widowed are more likely to experience health decline relative to White older adults.

Previous research has tested for racial differences in the relationship between two domains at a time (e.g., disability and depression). Among these dyads, some have found that mental health and physical health are not linked as closely—if at all—for Black older adults as for White older adults (Cummings et al. 2003; Harralson et al. 2002; Leveille et al. 1998). Our findings, however, are consistent with other research showing that there are no race differences in the relationships between mental and physical health (Steffens et al. 1999; Stump et al. 1997). Since much of this previous work tested only two health domains at a time, our article extended this research program by considering three health domains simultaneously (disease, disability, and depression).

It is important to note that the EPESE study design draws from community-dwelling persons 65 years of age or older (excluding institutionalized persons). Thus, it is readily anticipated that the distributions of at least the disability measures are skewed. Our results, however, are consistent with other research using the North Carolina EPESE (e.g., Mendes de Leon et al. 1997). The relative wellness of this sample may slightly underestimate the relationships among the health domains; however, we expect that the bias is small. In addition, this sample is from the central region of North Carolina. While this project benefits from such a large subsample of Black older adults (54 percent), the limited geographic area could restrict variability found in other regions of the country. Future studies should examine the mechanisms of health decline for older adults in other areas of the nation.

The findings in this article help to disentangle the mechanisms of health decline for Black and White older adults. Much of the previous research has focused on unidirectional causality between two health domains, sometimes measured at the same time point. We examined the interrelationships over time among three health domains—disease, disability, and depression—finding that both prevalent and incident disease cause worse depression and that prevalent disease leads to greater disability three years later. Depression causes both prevalent and incident disease over time, as well. These relationships among the three health domains over seven years are consistent across race groups, meaning that the processes of general

health decline are comparable for Black and White older adults. This similarity in health decline across race groups does not extend to all risk factors, however. Living in a rural area or being obese is more consequential to health decline for White older adults than for Black older adults. Likewise, widowhood accelerates health decline for Black older adults but not for their White counterparts. Understanding the multidimensional and complex relationships among the major domains of health over time helps in the development of interventions to slow the process of health decline among Black and White older adults.

## NOTES

1. The effective N does not equal the sample N because 173 respondents did not answer any of the questions on depression at one or more waves; 83 respondents did not answer three or more questions on depression at a given wave; 164 respondents were missing on two or more disease questions; and 86 were missing on disability at one or more waves.
2. It is possible to add a fourth group for those who responded in the first and third waves, but were missing on the second wave. However, the total number for this group is small ( $n = 14$ ), and Allison (1987) argues that when the group is small relative to the other groups and the pattern is not common, estimates are generally not affected by its non-inclusion.
3. The multigroup model can be estimated with either maximum likelihood or generalized least squares (GLS) estimation procedures (Bollen 1989). We tested the final models with both procedures. Although GLS does not require assumptions about the distribution of the dependent variables, its algorithm tends to produce less efficient estimators. The best-fitting models for our analysis were estimated with maximum likelihood. However, the substantive results (e.g., significant slope coefficients) were not different across estimation procedures, providing further confidence in our results.
4. The within-wave correlations between health domains in the measurement model are listed here. Only wave 1 correlations are significant. Wave 1: disability and prevalent disease = .21; prevalent disease and depression = .18; depression and disability = .10. Wave 2: disability and incident disease = .09; incident disease and depression = .07; depression and disability = .03. Wave 3: disability and incident disease = .07; incident disease and depression = .08; depression and disability = .04.
5. In multigroup models, the total number of degrees of freedom must be adjusted to account for the pseudovalues of the means, variances, and covariances in the incomplete data (Bollen 1989; Allison 1987). There were 9 means, 9 variances, and 42 covariances that were entered into the matrices for estimation (as place-holding zeros or ones), totaling 60 degrees of freedom. This was subtracted from the 96 model degrees of freedom. Adjusted-goodness-of-fit is not reported in these analyses because it is calculated based on the model degrees of freedom, which is artificially high in this model (Kelloway 1998).
6. The coefficient between age and disability in our article is small relative to other studies. This is probably because many previous studies use a categorical or even binary age variable, which, by definition, increases the size of the coefficient (one unit of the age variable may represent 10 or more years). The units of our age variable are individual years, so one unit change reflects the average amount of change in the outcome variables expected over one year.
7. For diagnostic purposes only, the Black and White subsamples, each with the three groups of complete and incomplete data, were tested in one large multigroup model to determine which covariates significantly differed by race. The results of these analyses are discussed in the text. Tables with these results are available from the first author upon request.

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