General Fatalism and Diabetes Fatalism as Predictors of Diabetes Treatment Adherence

Esmeralda Ibette Nuñez

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General Fatalism and Diabetes Fatalism as Predictors of Diabetes Treatment Adherence

by

Esmeralda Ibette Nuñez

A Thesis submitted in partial satisfaction of the requirements for the degree Doctor of Philosophy in Clinical Psychology

June 2016
Each person whose signature appears below certifies that this thesis in his/her opinion is adequate, in scope and quality, as a thesis for the degree Doctor of Philosophy.

Hector M. Betancourt, Professor of Psychology

Patricia M. Flynn, Assistant Clinical Research Professor

Holly E. R. Morrell, Professor of Psychology
ACKNOWLEDGEMENTS

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ABSTRACT OF THE THESIS

General Fatalism and Diabetes Fatalism as Predictors of Diabetes Treatment Adherence

by

Esmeralda Ibette Nuñez

Doctor of Philosophy, Graduate Program in Psychology
Loma Linda University, June 2016
Dr. Hector M. Betancourt, Chairperson

Greater conceptual distinction between fatalism as a cultural value orientation (general fatalism) and disease-specific fatalism is necessary to evaluate fatalism as a predictor of health behaviors such as diabetes treatment adherence. We tested an integrated, theory-driven structural model of relationships among income, education, age, gender, general fatalism, diabetes fatalism, and type 2 diabetes treatment adherence, as measured by hemoglobin a1c levels (hba1c), in a sample of mainstream-Chileans (n=229) and indigenous people of Chile known as the Mapuche (n=134). Hypotheses were that higher income and education would predict more general fatalism and diabetes fatalism across ethnic groups and higher general fatalism would indirectly predict higher hba1c through diabetes fatalism. Multi-group structural equation modeling comparing ethnic groups revealed excellent fit of models (Mapuche: CFI = .995, $\chi^2$ (24, n = 229) = 27.26, $p = .29$, $\chi^2$/df = 1.14, RMSEA = .024, 90% CI (.000, .061), $R^2 = 0.148$; mainstream-Chilean: CFI = 1.000, $\chi^2$ (24, n = 134) = 22.15, $p = .57$, $\chi^2$/df = .92, RMSEA = .000, 90% CI (.000, .064), $R^2 = 0.043$) and partial supported of study hypotheses. Fewer years of education significantly predicted general fatalism and lower income significantly predicted diabetes fatalism across ethnic groups. Higher general fatalism was a significant indirect predictor of hba1c through diabetes fatalism only for mainstream-Chileans. This study highlights
the importance of measuring fatalism as a cultural value and disease-specific fatalism as antecedents of health behavior. Findings also underscore the necessity of studying how social structural factors influence health behavior through culture and disease-specific beliefs.
CHAPTER ONE
INTRODUCTION

Fatalism has been conceptualized as a cultural value orientation (general fatalism) that reflects a social group’s general understanding of how humans should relate to nature and whether people should try to control events in their environment or be subjugate to them (Kluckhohn & Strodtbeck, 1961). Contrastingly, disease-specific fatalistic beliefs are beliefs that death is inevitable when a disease is present, that some health outcomes are outside human control and are predetermined, and that health outcomes are controlled by external forces, powerful others, or chance (Egede & Bonadonna, 2003; Powe et al., 2003; Straughan & Seow, 1998). Researchers have described cancer-specific fatalism as stemming from the broader concept of fatalism, but more clarification is needed in terms of how general fatalism and disease-specific fatalism may differentially relate to health behaviors (Heiney, Gullatte, Hayne, Powe, & Habing, 2015).

Fatalism and Ethnicity

Latinos have often been represented in the research literature as holding more fatalistic values than individuals of other ethnic groups. In other words, Latinos have been understood as more likely to value a relationship between humans and nature in that humans do not try to control their environment. However, some investigators have noted the lack of evidence for the notion that Latinos are inherently more fatalistic than other ethnic groups and highlighted the assumption as a limitation of fatalism research (Abraído-Lanza et al., 2007). Powe and Finnie (2003), have explained that ethnic minority groups are high in cancer fatalism because of social circumstances of less
control with increased barriers to health that increase the odds of dying of cancer. Understanding social structural influences of disease-specific fatalism can help researchers better understand that certain ethnic minority groups are not inherently fatalistic, but may have more fatalistic health beliefs as a consequence of experiencing social circumstances of less control.

The connection between disease-specific fatalism and health behaviors among Latinos and other ethnic minorities is more consistently supported than the relationship between general fatalism and health behaviors. In a review of literature investigating the relevance of fatalism in the study of Latina’s cancer screening behavior Espinosa researchers found 11 quantitative studies that directly measured the relationship between fatalism and cancer screening behaviors in Latinas (Espinosa de los Monteros & Gallo, 2011). Two of the 11 studies reviewed used a measure of general fatalism created by Cuéllar and authors as a predictor of cancer screening behavior (Cuéllar, Arnold, and González, 1995; Randolph, Freedman, & Freedman, 2002; Teran, Baezconde-Garbanati, Marquez, Castellanos, & Belkic, 2007). One study revealed that more general fatalism significantly predicted less cancer screening behavior, while the other study found no relationship between general fatalism and cancer screening. Contrastingly, six out of the seven studies that measured disease-specific fatalism as a predictor of cancer screening found a statistically significant relationship such that higher levels of cancer fatalism predicted less cancer screening. In their discussion, the reviewers noted that “in regards to what would be most predictive of health behavior, it is not clear whether fatalism should be measured as a global trait or merely as a disease specific belief regarding the disease of interest” (p.316).
Fatalism and Diabetes Treatment Adherence

How general fatalism and disease-specific fatalism relate to treatment adherence in individuals with type 2 diabetes, the people of focus in the current research study, is unclear. In a review of culturally relevant issues among Hispanic individuals with diabetes, Caban and Walker (2006) emphasize the lack of consistency in how fatalism relates to diabetes self-management across studies. The authors defined fatalism in more global terms, but included both studies that investigated general fatalism and diabetes fatalism in their review. Studies published subsequently to this review that assess fatalism and diabetes treatment adherence are few in number and generally use measures of diabetes fatalism rather than measures of general fatalism (Egede et al., 2003; Egede & Ellis, 2010; Osborn, Bains, & Egede, 2010; Walker et al., 2012). Researchers of one study found that diabetes fatalism directly predicted self-care behavior and indirectly predicted glycemic control as measured by hba1c blood levels (Osborn et al., 2010). In another study on the independent effects of diabetes fatalism on self-reported treatment adherence and self-care behaviors in type 2 diabetes, investigators reported that more diabetes fatalism significantly predicted less medication adherence, diet, exercise, and blood sugar testing (Walker et al., 2012).

Sociostructural Determinants of Fatalism

As previously mentioned, investigators have emphasized sociostructural factors as significant determinants of disease-specific fatalism, such that lower socioeconomic status tends to predict higher levels of disease-specific fatalism. Other demographic factors, including age and gender, have been found to have an association with disease-
specific fatalism though less consistently than income and education (Flynn, Betancourt, & Ormseth, 2011; Straughan et al., 1998). Studies discussing sociostructural predictors of general fatalism are also limited in number. Researchers of one study found that the relationship between Mexican identity and general fatalism was mediated by education, income, and occupational prestige (Ross, Mirowsky, & Cockerham, 1983). In the absence of guidance from multiple studies, researchers have suggested that multiple demographic factors must be carefully considered as potential sources of influence on culture values, health beliefs, and health behaviors (Betancourt & Flynn, 2009).
CHAPTER TWO

THE PRESENT STUDY

The purpose of this study was to emphasize the conceptual distinction between general fatalism and disease-specific belief and to clarify how these two fatalism constructs are related to social structural factors and health behavior. More specifically, we examined how general fatalism and diabetes fatalism predicted diabetes treatment adherence in a sample of patients with type 2 diabetes. We used hba1c blood levels as a proxy variable for diabetes treatment adherence. In medical settings, the hba1c blood test is used as a biologic marker of whether a treatment plan is effective or to demonstrate the effect of medication, diet, and exercise choices (American Diabetes Association, 2014). In focusing on hba1c, Gonzalez and Schneider (2011) have note that information about specific patient behavior within the domains of medication, diet, and exercise adherence is lost. However, using a biologic marker of treatment adherence has the advantages of circumventing both the concern of bias in self-report measures and the uncertainty surrounding the validity of existent, commonly-used self-report adherence measures (Bennett Johnson, 1992; McNabb, 1997; Gonzalez et al., 2013).

The study sample is composed of mainstream-Chileans and the largest indigenous group in Chile known as the Mapuche. This sample is of particular interest for the study of fatalism and diabetes because the rate of diabetes mortality in Chile has been rising rapidly since the 1970s (Albala, Vio, Kaiin, & Uauy, 2001). The Mapuche, who at one point had the world’s lowest diabetes prevalence rate of under one percent, with increasing urbanization are now demonstrating rapid increases in the prevalence of diabetes (Carrasco et al., 2004; Pérez-Bravo et al., 2001).
We argue that studying general fatalism as a disease–specific fatalism within an integrated framework is critical to understanding how social structural factors influence these cultural values and health beliefs, and in turn, how all these factors predict diabetes treatment adherence across ethnic groups. The proposed relationships among study variables were based on Betancourt’s model for the study of culture, psychological processes, and behavior adapted for the study of health behavior (see Figure 1; Betancourt et al., 2009). This is theory-driven, integrated model is useful in understanding how sociostructural, cultural, and psychological factors may influence health behaviors. Researchers have suggested that an integrative model is essential for the study of social and psychological determinants of health behavior (Gallo, Smith, & Cox, 2006). A critical underlying principle of Betancourt’s model is that predictors of health behavior are structured from most distal to most proximal. Variables more proximal to behavior are theorized to have a greater impact on behavior. Another important aspect of the model is that culture may affect health behavior directly or indirectly through more proximal psychological processes, such as idiosyncratic beliefs and emotions that are experienced at the individual level. The most distal determinants of health behavior within this model are social categories such as socioeconomic status, ethnicity, age, and gender. These factors may not directly relate to health behavior but represent direct sources of cultural variation. A strength of Betancourt’s model is that it highlights the argument that ethnicity is not culture in and of itself, but instead is one of multiple sources of variation in culture. Considering multiple social structural factors as influences of culture may help researchers avoid inaccurate conceptions of ethnic minorities.
Figure 1. Betancourt’s Model of Culture and Behavior Adapted for Health Behavior
Hypothesized relationships among general fatalism, diabetes-specific fatalism, and diabetes treatment behavior were also guided by attitude-behavior relations theory (Zen & Fishbein, 1977; Ajzen & Fishbein, 2005). Researchers in this area have demonstrated that general cognitions (i.e., attitudes, values, beliefs) may inconsistently relate to specific behaviors but cognitions more specific to a behavior of interest tend to be more predictive of that behavior. This principle is exemplified by the inconsistency of the relationship between general fatalism and disease-specific health behaviors and the more consistent relationship between disease-specific fatalism and disease-specific health behaviors. Investigators have suggested that general cognitions can relate to specific health behaviors in certain circumstances, however this relationship can be missed if potential moderating/mediating variables are not considered. In the case of the relationship between general fatalism and diabetes treatment adherence, it is critical to consider the potential mediating effect of diabetes-specific fatalism. A measure of diabetes-fatalism is more likely to directly predict diabetes treatment adherence because it assesses beliefs that are specific to diabetes. We suggest that having general fatalism will relate to type 2 diabetes treatment adherence through diabetes fatalism. Although researchers have acknowledged disease-specific fatalism as stemming from the concept of fatalism more generally, to our knowledge no research group has examined the relationships among sociostructural factors, general fatalism, disease-specific fatalism in a single model.

The hypothesized relationships in this study were tested through the structural model presented in Figure 2. The demographic categories of education, income, gender, and age were tested as predictors of both general fatalism and diabetes fatalism based on
theory and previous research (Betancourt et al., 1993; Betancourt et al., 2009; Flynn et al., 2011; Powe et al., 2003; Straughan et al., 1998). Additionally, we tested diabetes fatalism as a direct predictor of hba1c blood levels. General fatalism was tested as both a direct predictor and indirect predictor of type 2 diabetes treatment adherence through diabetes fatalism. However, we hypothesized that higher cultural fatalism would indirectly relate to health behavior through disease-specific fatalism. Specific study hypotheses were as follows:

1. Income and education will directly predict general fatalism and diabetes fatalism in both mainstream-Chilean and Mapuche participants such that lower income and fewer years of education will predict higher levels of general fatalism and diabetes fatalism.

2. Diabetes fatalism will directly predict type 2 diabetes treatment adherence in both mainstream-Chilean and Mapuche participants such that higher levels of diabetes fatalism will predict higher/worse hba1c blood levels. General fatalism will indirectly predict hba1c blood levels through diabetes fatalism in both mainstream Chilean and Mapuche participants such that higher levels of general fatalism will predict higher levels of diabetes fatalism, which will, in turn, predict higher hba1c blood levels.
Figure 2. Proposed Structural Model of Relationships. Separate models will be run for mainstream-Chilean and Mapuche participants.
Method

Participants

As part of a larger research program investigating cultural and psychological factors relevant to diabetes management, multi-stage stratified sampling was used to obtain participants from demographic backgrounds conceived as sources of cultural variation from both public and private health clinics in the Araucanía region of Chile. A total of 394 type 2 diabetics were recruited (Mapuche; n = 146, mainstream-Chilean; n = 254). Potential participants were contacted by phone with the support of clinic directors to explain the purpose of the study. Inclusion criteria were being 18 years of age, having a diagnosis of diabetes for over one year, and being non-insulin dependent.

Materials

Ethnicity

Ethnicity was self-reported by participants as either Mapuche or mainstream-Chilean and used as a moderating variable in multi-group analyses.

Sociostructural Sources of Cultural Variation

Income, education, age, and gender were included in structural equation model as social structural sources of cultural variation. Participants indicated their age in years, number of years of education, and annual household income based on six categories (see Table 1). The variable for gender was dummy coded with females coded as 0 and males coded as 1.
General Fatalism

General fatalism was measured with Betancourt’s Fatalism Scale based on the work of cultural anthropologists Kluckhohn and Strodtbeck (1961). It consists of 10-items rated on a 7-point scale with ratings ranging from strongly disagree to strongly agree. The scale represents items designed to assess fatalistic value orientation. Higher scores on the scale represent stronger fatalistic values. See Appendix A for Betancourt’s Fatalism Scale. Confirmatory factor analysis revealed that all the items loaded on one factor of general fatalism. The scale was found to be highly reliable across ethnicity (mainstream-Chilean \( \alpha = 0.85 \), Mapuche \( \alpha = 0.84 \)).

Diabetes Fatalism

Diabetes fatalism was measured using the 6-item Diabetes Fatalism Scale. The scale was developed with Mapuche and mainstream-Chilean type 2 diabetes patients based on the bottom-up mixed methods cultural research approach to instrument development (see Betancourt, Flynn, Riggs, & Garberoglio, 2010). This method begins with observations with members of the population of interest (e.g. Mapuche and mainstream-Chilean patients), which are derived through interviews. Based on the interview responses, close-ended items are developed and compiled into a quantitative scale. The resulting scale represents items designed to assess fatalistic beliefs about diabetes. Higher scores on the scales represent higher levels of diabetes-specific fatalistic beliefs. Confirmatory factor analysis revealed that the items loaded onto one factor. The scale demonstrated adequate reliable across ethnic groups (mainstream-Chilean \( \alpha = 0.71 \), Mapuche \( \alpha = 0.72 \)). See Appendix A for Diabetes Fatalism Scale.
**Diabetes Treatment Adherence**

Hba1c blood percentage was used as a biological index of diabetes treatment adherence. The measure is obtained using a sample of blood and indicates an individuals’ average blood glucose (blood sugar) control for the past 2 to 3 months. Hba1c is used as an indicator of whether a treatment plan is effective or to demonstrate the effect of medication, diet, and exercise choices. Values range from about 5% to 14%. Individuals without diabetes normally have an hba1c range from 4.5 to 6%. Diabetes is usually diagnosed at 6.5% or higher. Diabetes treatments usually aim to maintain an hba1c level of 7%. Higher hba1c blood levels suggest lower diabetes treatment adherence and more risk for diabetes complications (American Diabetes Association, 2014).

**Covariates**

This study relied primarily on participant self-report. As such, social desirability, as measured by the 13-item Marlowe-Crowne Social Desirability Scale, was assessed as a covariate (Crowne & Marlowe, 1960).

**Procedure**

Individuals were recruited between September 2011 and February 2012 through private and public health centers in Temuco, Chile and rural areas of the region. Healthcare personnel assisted in recruitment as well as flyers posted and distributed in public and private healthcare facilities. Potential participants were contacted by phone with the support of clinic directors to explain the purpose of the study, including the inclusion/exclusion criteria. Those interested in participation contacted the research office.
and were further screened for eligibility. They were informed that participation would involve 30-45 minute questionnaire and a free hba1c blood test. They would be compensated 5,000 pesos ($10 U.S.) for their time. Those who met inclusion criteria were scheduled for participation at a research facility at the Universidad de la Frontera, School of Medicine. Those who lived in rural areas reported to an office space provided at a local health clinic.

Once at the research facility, in 4-6 participants per sessions, participants were again read the purpose of the study and guided through informed consent. Once written consent was obtained, participants were given the questionnaire. Participants were surveyed about a variety of diabetes-related beliefs and behaviors as well as some experiences within the Chilean healthcare system. All participants were encouraged to ask questions. If a participant was unable to read, a research assistant guided the individual through the questionnaire in a private setting. Upon completing the questionnaire, participants were then given an hba1c blood test and their heights and weights were measured. Finally, participants were given the results of their hba1c test, debriefed, and paid.

This study was funded by CONICYT (National Commission for Scientific and Technological Research, Government of Chile; FONDECYT Project #1090660 to Dr. H. Betancourt, P.I.), and approval for the study was obtained from the public university ethics committee for research and the regional office of the Chilean Ministry of Health (SEREMI de Salud, Region de La Araucanía).
Statistical Analyses

Before testing the study hypotheses, data were screened for missing data, multivariate outliers, and assumptions for multivariate normality (Kline, 2011). Confirmatory factor analyses (CFA) using Bentler’s (2006) structural equation modeling were performed for Betancourt’s Fatalism Scale and Diabetes Fatalism Scale and verified that these scales factored into single-independent, though correlated factors.

In order to test the strength of relationships among study variables across ethnic groups, the study hypotheses were tested using Bentler’s multi-group structural equation modeling with Bentler’s (2006) structural equations program (EQS), maximum likelihood method of estimation. Based on theory and previous research, we reasoned that ethnicity in and of itself would not be a significant predictor of general fatalism nor diabetes-fatalism. We tested separate, but identical structural equation models for mainstream-Chilean and Mapuche participants in order to establish structural invariance. Income, education, age, and gender were included in the tests of models as social structural sources of cultural variation. Fit were accessed using non-significant $\chi^2$ goodness-of-fit statistic, a ratio of less than 2.0 for the $\chi^2/df$ ratio (Tabachnick & Fiddell, 2007), a Comparative Fit Index (CFI) of .95 or greater, Root Mean Square Error of Approximation (RMSEA) of less than .08 (Browne & Cudeck, 1993), and RMSEA upper-bound confidence interval below .10 (Kline, 2011). Modifications of the hypothesized models were performed based on results from the Lagrange multiplier (LM) test and the Wald test in combination theory and research.
CHAPTER THREE

RESULTS

Preliminary Analyses

The final analyses included 229 mainstream-Chilean and 134 Mapuche type 2 diabetes patients. Thirty-one participants were missing data on one or more of the items from noted multi-item scales or key covariates such as the gender of the health professional and were eliminated from subsequent analysis. Less than 5% of cases were missing data for any single variable, which Kline (2011) notes is of little concern in a large sample especially if pattern of missingness is nonsystematic. A missing value analysis and a Little’s Missing Completely at Random test did not indicate statistical deviation from randomness for the sample ($p=0.241$). There were no statistically significant differences between the omitted and retained sample in terms of ethnicity, gender, age, income, education, and hba1c blood levels.

Table 1 presents demographic information of final sample. Overall, Mainstream-Chilean participants reported significantly more years of education ($t(359) = -10.08, p < .001$) and yearly household income ($t(359) = -8.22, p < .001$) than Mapuche participants. Mainstream-Chilean participants had a significantly lower Body Mass Index (BMI) than Mapuche participants ($t(359) = 2.44, p < .05$), but the groups did not differ significantly in terms of hba1c blood glucose levels. The percentage of male and female participants was significantly different between ethnicities ($c^2(1, N = 363) = 7.59, p < .01$).
Table 1. Final sample demographics based on ethnicity.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mapuche (n =134)</th>
<th>Mainstream-Chilean (n =229)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>58.84(13.06)</td>
<td>57.32(13.82)</td>
</tr>
<tr>
<td>Education*</td>
<td>5.41(4.37)</td>
<td>10.12 (4.23)</td>
</tr>
<tr>
<td>BMI*</td>
<td>32.44(6.44)</td>
<td>30.87(5.44)</td>
</tr>
<tr>
<td>Hba1c</td>
<td>7.46(2.24)</td>
<td>7.08 (1.83)</td>
</tr>
<tr>
<td>Income*</td>
<td>n(%)</td>
<td>n(%)</td>
</tr>
<tr>
<td>0-150</td>
<td>108(80.60)</td>
<td>94(41.00)</td>
</tr>
<tr>
<td>151-250</td>
<td>13(9.70)</td>
<td>59(25.80)</td>
</tr>
<tr>
<td>251-500</td>
<td>12(9.00)</td>
<td>53(23.10)</td>
</tr>
<tr>
<td>501-1000</td>
<td>1(0.70)</td>
<td>20(8.70)</td>
</tr>
<tr>
<td>1000-1500</td>
<td>0(0.00)</td>
<td>1(0.40)</td>
</tr>
<tr>
<td>above 1500</td>
<td>0(0.00)</td>
<td>2(0.90)</td>
</tr>
<tr>
<td>Gender*</td>
<td>95(70.90)</td>
<td>129(56.30)</td>
</tr>
</tbody>
</table>

Note. *p < .05

Table 2 includes the means and standard deviations for the study variables. The table also includes the correlations among the study variables. Fischer’s r-to-z test of difference demonstrated two significantly different bivariate correlations based on ethnicity, confirming the need to conduct a test of invariance. The correlation between age and hba1c were significantly different across ethnicities (z= -2.41, p < .05). Additionally, the correlation between gender and Parcel 2 of the diabetes fatalism measure were significantly different across ethnicities (z= -2.40, p < .05).

As previously mentioned, social desirability was examined as a potential covariate prior to using structural equation modeling. Social desirability did not significantly covary with any other study variable.
Table 2. Intercorrelations, means, and standard deviations as a function of ethnicity

<table>
<thead>
<tr>
<th>Study Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2. Gender</td>
<td>0.136</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>-</td>
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<tr>
<td></td>
<td>(0.226**)</td>
<td>-</td>
<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3. Education</td>
<td>-0.556**</td>
<td>0.109</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td></td>
<td>(-0.260**)</td>
<td>(0.122)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4. Income</td>
<td>-0.169</td>
<td>0.209**</td>
<td>0.569**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(-0.151*)</td>
<td>(0.193**)</td>
<td>(0.523**)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5. General Fatalism</td>
<td>0.039</td>
<td>-0.087</td>
<td>-0.234**</td>
<td>-0.246**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Parcel 1</td>
<td>(0.118)</td>
<td>(-0.084)</td>
<td>(-0.298**)</td>
<td>(-0.180**)</td>
<td>-</td>
<td>-</td>
<td>-</td>
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</tr>
<tr>
<td>6. General Fatalism</td>
<td>0.118</td>
<td>-0.093</td>
<td>-0.383**</td>
<td>-0.318**</td>
<td>0.654**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Parcel 2</td>
<td>(0.175**)</td>
<td>(-0.068)</td>
<td>(-0.428)</td>
<td>(-0.225**)</td>
<td>(0.740**)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(0.148)</td>
<td>(0.039)</td>
<td>(-0.320**)</td>
<td>(-0.216**)</td>
<td>(0.361**)</td>
<td>(0.422**)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7. Diabetes</td>
<td>0.171*</td>
<td>-0.028</td>
<td>-0.258**</td>
<td>-0.180*</td>
<td>0.223**</td>
<td>0.263**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Fatalism Parcel 1</td>
<td>(0.067)</td>
<td>(0.067)</td>
<td>(-0.289**)</td>
<td>(-0.245**)</td>
<td>(0.344**)</td>
<td>(0.403**)</td>
<td>0.528**</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8. Diabetes</td>
<td>0.087</td>
<td>-0.194*</td>
<td>-0.218**</td>
<td>-0.290**</td>
<td>0.187*</td>
<td>0.257**</td>
<td>(0.488**)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Fatalism Parcel 2</td>
<td>(0.067)</td>
<td>(0.067)</td>
<td>(-0.289**)</td>
<td>(-0.245**)</td>
<td>(0.344**)</td>
<td>(0.403**)</td>
<td>0.528**</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9. Diabetes</td>
<td>0.155</td>
<td>-0.071</td>
<td>-0.312**</td>
<td>-0.344**</td>
<td>0.148</td>
<td>0.383**</td>
<td>(0.536**)</td>
<td>0.509**</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Fatalism Parcel 3</td>
<td>(0.130*)</td>
<td>(-0.008)</td>
<td>(-0.363)</td>
<td>(-0.266**)</td>
<td>(0.228**)</td>
<td>(0.410**)</td>
<td>0.084</td>
<td>(0.567**)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10. Hba1c</td>
<td>-0.296**</td>
<td>0.036</td>
<td>0.067</td>
<td>-0.074</td>
<td>0.124</td>
<td>0.158</td>
<td>(0.126)</td>
<td>0.143</td>
<td>0.097</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(-0.041)</td>
<td>(0.092)</td>
<td>(-0.113)</td>
<td>(-0.074)</td>
<td>(-0.03)</td>
<td>(-0.025)</td>
<td>5.31</td>
<td>(0.093)</td>
<td>(0.087)</td>
<td>-</td>
</tr>
<tr>
<td>M</td>
<td>58.84</td>
<td>-</td>
<td>5.41</td>
<td>1.30</td>
<td>5.97</td>
<td>5.62</td>
<td>5.31</td>
<td>4.67</td>
<td>5.16</td>
<td>7.46</td>
</tr>
<tr>
<td>(57.32)</td>
<td>-</td>
<td>(10.12)</td>
<td>(2.05)</td>
<td>(5.33)</td>
<td>(4.77)</td>
<td>(4.28)</td>
<td>(4.12)</td>
<td>(4.27)</td>
<td>(7.08)</td>
<td>-</td>
</tr>
<tr>
<td>SD</td>
<td>13.06</td>
<td>-</td>
<td>4.37</td>
<td>0.66</td>
<td>1.21</td>
<td>1.51</td>
<td>1.83</td>
<td>2.05</td>
<td>1.80</td>
<td>2.24</td>
</tr>
<tr>
<td>(13.82)</td>
<td>-</td>
<td>(4.23)</td>
<td>(1.08)</td>
<td>(1.34)</td>
<td>(1.70)</td>
<td>(1.77)</td>
<td>(1.94)</td>
<td>(1.96)</td>
<td>(1.83)</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. Correlations, means, and standard deviations for mainstream-Chilean participants \((n = 229)\) are listed in parentheses below those for Mapuche participants \((n = 134)\). Boldface indicates that the groups differ significantly at \(p < .05\). *\(p < .05\) level (2-tailed); **\(p < .01\) level (2-tailed).
Structural Equation Modeling

Before conducting multi-group structural equation modeling, data screening revealed a violation of multivariate normality for both ethnic groups. Consequently, we used a corrected normal theory method of analysis with original data and reported ML robust test statistics, corrected model statistics that are robust against non-normality (Kline, 2011). The tested model demonstrated good fit for mainstream-Chilean participants, but poorer fit for Mapuche participants (mainstream-Chilean: $CFI = 0.997$, $\chi^2 (23, n = 229) = 24.97, p = 0.35 \chi^2/df = 1.08, \text{RMSEA} = .019, 90\% \text{CI (.000, .059)}, R^2= 0.041$; Mapuche: $CFI = 0.951$, $\chi^2 (23, n = 134) = 37.78, p = 0.03 \chi^2/df = 1.64, \text{RMSEA} = .069, 90\% \text{CI (.023, .107)}, R^2= 0.029$). Based on the Lagrange test, theory, research, logic, structural paths from age to diabetes fatalism and gender to general fatalism were dropped from the models for both groups. Based on the Wald test and theory suggesting that in some instances population factors may relate directly to health behavior, a direct path was added from age to hba1c. The resulting model is presented in Figure 3. The model for fit the data well for mainstream-Chilean participants (CFI = .995, $\chi^2 (24, n = 229) = 27.26, p = .29, \chi^2/df = 1.14, \text{RMSEA} = .024, 90\% \text{CI (.000, .061)}, R^2= 0.043$). The model also fit the data well for Mapuche participants (CFI = 1.000, $\chi^2 (24, n = 134) = 22.15, p = .57, \chi^2/df = .92, \text{RMSEA} = .000, 90\% \text{CI (.000, .064)}, R^2= 0.148$).
Figure 3. Final Structural Model of Relationships. Mapuche: CFI = 1.000, $\chi^2$ (24, n = 134) = 22.15, $p = .57$, $\chi^2$/df = .92, RMSEA = .000, 90% CI (.000, .064), $R^2$ = 0.148; mainstream-Chilean; CFI = .995, $\chi^2$ (24, n = 229) = 27.26, $p = .29$, $\chi^2$/df = 1.14, RMSEA = .024, 90% CI (.000, .061), $R^2$ = 0.04.
Test of Research Hypotheses

The first hypothesis concerning the direct effects of income and education on general fatalism and diabetes fatalism was partially supported. For both Mainstream-Chilean and Mapuche participants only years of education significantly predicted general fatalism (Mainstream-Chilean $\beta = -0.41$; Mapuche $\beta = -0.37$). This relationship was in the hypothesized direction, with less years of education predicting more general fatalism. Lower income and less years of education significantly predicted more diabetes fatalism only for Mainstream-Chilean participants (Income $\beta = -0.16$; Education $\beta = -0.19$).

The second hypothesis that diabetes fatalism would directly predict diabetes treatment adherence was partially supported. Higher diabetes fatalism significantly predicted higher hba1c for Mainstream-Chilean participants ($\beta = 0.25$). Diabetes fatalism did not significantly predict hba1c for Mapuche participants, however, the relationship between diabetes fatalism and hba1c was in the expected direction and was trending toward significance ($\beta = 0.16$, $p<.10$).

The third hypothesis that higher levels of general fatalism would indirectly predict lower diabetes treatment adherence through higher levels of diabetes fatalism was also partially supported. For both Mainstream-Chilean and Mapuche participants higher levels of general fatalism significantly predicted higher levels of diabetes fatalism (Mainstream-Chilean $\beta = 0.45$ Mapuche $\beta = 0.32$). General fatalism had a significant indirect effect on hba1c through diabetes fatalism for Mainstream-Chilean participants (indirect $\beta = 0.11$, $p < .05$).
Test of Structural Invariance

Because preliminary analyses revealed that ethnicity moderated the correlations between age and hba1c, a test of structural invariance was conducted. Review of the LM test of equality constraints statistics revealed significant between-group differences in the path from age to hba1c. The magnitude of this effect was stronger for Mapuche participants as compared to mainstream-Chilean participants. Once the path constraint from age to hba1c was released the fit demonstrated by the resulting model improved. The fit of this model was once again comparable to the configural model, suggesting that no other paths should be released (see Table 3).

Table 3. Model summary for tests of configural, measurement, and structural invariance across ethnicity

<table>
<thead>
<tr>
<th>Model</th>
<th>S-BX²</th>
<th>df</th>
<th>CFI</th>
<th>RMSEA(90% CI)</th>
<th>ΔS-BX²a</th>
<th>p</th>
<th>Δdf</th>
<th>ΔCFI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Configural Invariance</td>
<td>47.291</td>
<td>48</td>
<td>1</td>
<td>0(0.000,0.047)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Measurement Invariance</td>
<td>47.933</td>
<td>51</td>
<td>1</td>
<td>0(0.000,0.043)</td>
<td>0.493756949</td>
<td>0.92</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Structural Invariance</td>
<td>70.789</td>
<td>61</td>
<td>0.989</td>
<td>0.03(0.000,0.056)</td>
<td>24.32047234</td>
<td>0.023</td>
<td>13</td>
<td>-0.011</td>
</tr>
<tr>
<td>Partial Structural Invariance</td>
<td>62.706</td>
<td>60</td>
<td>0.997</td>
<td>0.016(0.000,0.049)</td>
<td>15.64723554</td>
<td>0.208</td>
<td>12</td>
<td>-0.003</td>
</tr>
</tbody>
</table>

Note. Configural invariance model has no constraints; measurement invariance model factor loadings constrained across ethnicities; structural invariance model constrained factor loadings and 10 structural paths; partial structural invariance model constrained factor loadings and 9 structural paths, released path from ‘Age’ to ‘Hba1c’.
CHAPTER FOUR

DISCUSSION

The results of this study provide clarification regarding two conceptual issues within fatalism research and further the study of fatalism in relation to type 2 diabetes treatment adherence. The first of these issues is the lack of conceptual differentiation in the research literature between measures of fatalism as a global, cultural value orientation and fatalism as a disease-specific belief. In the current study, higher levels of diabetes fatalism significantly predicted higher hba1c blood levels for Mainstream-Chilean participants. Though this relationship was not statistically significant for Mapuche participants, it was in the anticipated direction and trended toward significance. General fatalism did not have a significant direct effect on diabetes treatment adherence, but instead related to hba1c through more specific fatalistic beliefs about diabetes. This finding is in accordance with the relationship between suggested among these variables by Betancourt’s model and attitude-behavior principles. General cognitions may inconsistently predict specific behaviors because the relationships among general cognitions and specific behaviors are often mediated/moderated by cognitions that are more specific to that behavior. To our knowledge, no publication to date has assessed these constructs simultaneously in relation to diabetes treatment adherence. Furthermore, no previous study to our knowledge has proposed a framework for how general fatalism and disease-specific fatalism both relate to health behavior.

This study highlights the importance of making the conceptual distinction between general fatalism and diabetes fatalism and measuring both constructs in relation to health behaviors. General fatalism was not a significant direct predictor of hba1c in
this study. Had our model only included a measure of general fatalism, it may have seemed that general fatalism does not relate to diabetes treatment adherence. However, including general fatalism and diabetes fatalism in our study revealed how diabetes fatalism directly and significantly predicted specific fatalistic beliefs about diabetes across ethnicities, while general fatalism indirectly predicted hba1c through diabetes fatalism for mainstream-Chilean participants.

Another critical reason for differentiating general fatalism and diabetes fatalism is for the purpose of better understanding the social structural determinants of general fatalism and diabetes fatalism. In the current study, fewer years of education significantly predicted more general fatalism across ethnic groups. Similarly, lower income significantly predicted more diabetes fatalism across ethnicities. These findings lend support to researchers who have argued that fatalism is related to social circumstances of less control rather than ethnicity in and of itself.

Several unexpected and significant relationships arose in this investigation and warrant further investigation. That age had a significant negative association with lower hba1c for Mapuche participants was one such unexpected result. Possible explanations of this finding are that older Mapuche participants may adhere more to their diabetes treatment or that younger Mapuche participants tend to have a lifestyle more similar to that of mainstream-Chileans because of trends toward urbanization. These findings highlight the complexity of the relationships among social structural factors, culture, health beliefs, and health behavior. Additionally, these findings point to the need to consider a broad range of social structural factors and demographic categories, beyond ethnicity, that can uniquely affect the culture and health beliefs of different social groups.
An additional query that this study brings forward is the question of why diabetes fatalism significantly predicted hba1c blood levels for Mainstream-Chilean participants but not for Mapuche participants. In the present study, we did not make any a priori hypotheses about psychological variables because of the primacy of making the conceptual distinction between general fatalism and disease-specific fatalism and testing how general fatalism and disease-specific fatalism relate to health behavior. However, Betancourt’s model highlights the importance of psychological factors as direct predictors of health behavior and as potential mediators between cultural factors and health behavior. Flynn and authors (2011) found that cancer screening fatalism was a significant indirect predict of clinical breast exam adherence through the psychological factor of emotions for Latino women but not Anglo women. Researchers have also shown demonstrated that self-efficacy, defined as an individual’s belief in his/her capacity to execute behavioral change that reflects confidence in his/her ability control personal motivation, behavior, and the social environment, is a critical psychological predictor of a variety of health behavior including diabetes self-management (Abubakari, Cousins, Thomas, Sharma, & Naderali, 2015; Bandura, 2004). Future researchers should investigate emotions related to diabetes fatalism, self-efficacy, and other psychological factors as potential mediators of the relationship between diabetes fatalism and diabetes adherence.

Limitations of this research include reliance on hba1c as a proxy for diabetes adherence. Using a biological indicator of adherence has the advantage of not being subject to the biases of self-report measures, however, hba1c is influenced by other genetic and physiological factors besides behavioral compliance. In using hba1c, we are
unable to report how general fatalism and diabetes fatalism relate to specific aspects of adherence such as engaging in medication, eating, and exercise behaviors recommended for individuals with type 2 diabetes. In a post-hoc analysis, general fatalism and diabetes fatalism generally had no significant correlation with self-reported diabetes adherence in the domains of medication, exercise, and diet as measured by the Summary of Diabetes Self-Care Activities (SDSCA) across mainstream-Chilean and Mapuche participants. Similarly, the correlations between self-reported type 2 diabetes treatment adherence and hba1c were generally nonsignificant. These correlations could not attributed to the effect of social desirability. However, one possible explanation for this inconsistency goes back to attitude-behavior theory. The SDSCA anchors individuals to answer questions based on the past 7 days of adherence behavior, thus violating measurement compatibility when relating the SDSCA with diabetes fatalism, which has no time anchor, and hba1c, which is a general biological marker of 3-month adherence. The SDSCA, though often used, has related inconsistently to other diabetes adherence measures across studies (Gonzalez et al., 2011).

In the future, researchers should consider looking at how sociostructural factors, general fatalism, and diabetes fatalism relate to behavioral measures of diabetes adherence and hba1c across time. It is important to note that our data were collected in a cross-sectional design. Longitudinal data would provide stronger evidence for the causal relationships proposed among sociostructural factors, general fatalism, diabetes fatalism, and type 2 diabetes treatment adherence. In addition to studying these relationships in a longitudinal study, researchers interested in how fatalism relates to behavior across
different social groups should further investigate fatalism in relation to such social categories as gender and age.
REFERENCES


APPENDICES

APPENDIX A

General Fatalism Scale (1 to 7 Likert scale)

Parcel 1 (Unpredictability)
1. The future appears to be totally unpredictable
2. When things begin to happen in life that you want, something happens and things change
3. Life is so uncertain that it’s best to take things day by day
4. There are so many ups and downs in life that you can’t know what will happen in the end
5. There are so many things that can happen that it’s difficult to plan for the future

Parcel 2 (Predestination)
1. In real life there is little that you can do to change things
2. Success depends on destiny, like being in the right place at the right time
3. Something that is going to happen will happen, no matter what you do
4. Destiny determines how successful one will be in life, therefore there is not much else you can do about it
5. Each person has a set time to live, and when that time is over, it’s over

Diabetes Fatalism Scale (1 to 7 Likert scale)

When the diabetic DOES NOT follow treatment it is because:

Parcel 1
1. Diabetes leads to death and there is not much that can be done about it
2. You think that everybody dies from something, and if it is not from diabetes, it will be from something else.

Parcel 2
1. When you follow your diet you do not feel different
2. If you are destined to have diabetes, then you will suffer no matter what

Parcel 3
1. You believe that no matter what you do, you are not going to get better
2. In the end, what happens with diabetes is in the hands of God

Self-Reported Adherence

Diet: On average, during the last month, how many days per week did you follow your diet?

Exercise: How many of those seven days did you do at least 30 minutes of continuous exercise? (including walking)

Medication: On how many of those days did you take your prescribed drugs for diabetes?