Abstract

Subjective life expectancy is a powerful predictor of a variety of health and economic behaviors. This research expands upon the life expectancy literature by examining the influence of familial health histories. Using a genetic/environmental model, we hypothesize that individuals' assessments of their life expectancies will be linked to the health of first-degree and second-degree relatives, with same-sex relatives' health exercising a stronger effect than that of opposite-sex relatives. Multivariate analyses based on data from a 2009 survey merged with familial health records (N=1,019) confirm that the health experiences of same-sex, first-degree relatives are linked to respondents' subjective life expectancy. The relationship between the health experiences of second-degree relatives and subjective life expectancy is much less pronounced. These findings have the potential to not only to inform our understanding of health behaviors, but also to encourage communication between patients and health professionals aimed at promoting preventative behaviors.

Key words:

 subjective life expectancy, family health histories

Family, Frailty, and Fatal Futures?
Own-Health and Family-Health Predictors of Subjective Life Expectancy

Only a few generations ago, individuals likely viewed their longevity as a function of events that took place during their lifetimes (e.g., infectious diseases, occupational risks, military service). As the leading causes of death have shifted from infectious to chronic diseases and as scientific knowledge about genetic and environmental bases of longevity has expanded, individuals have adjusted the way they estimate how long they are likely to live ("subjective life expectancy" hereafter). As we learn more about the human genome and what it implies for health and longevity, the weight of family health histories and gene-environment interactions in the assessment of subjective life expectancy is likely to grow. While some research on subjective life expectancy has examined the influence of a person's lifetime health experiences as well as parental health and longevity, much remains to be discovered regarding the impact of own-health and family-health histories. In this study, we focus on gender differences, expand on the age range of individuals typically surveyed, and add depth to the measurement of both a respondent's own-health history and that of first- and second-degree relatives.

What We Know About Subjective Life Expectancy

Research has demonstrated that subjective life expectancy is a powerful predictor of a variety of health and economic behaviors including smoking (Balia, 2011; Carbone, 2005; Sloan, Taylor, & Smith, 2001), consumption and savings decisions (Li, Zhang, & Zhang, 2007; Post & Hanewald, 2010; Salm, 2010), and retirement timing (Hurd, Smith, & Zissimopoulos, 2004; van Solinge & Henkens, 2010). Thus, understanding subjective life expectancy's determinants elucidates the direct and indirect influences on these behaviors as well. Yet, our knowledge of the correlates of subjective life expectancy remains limited.

The literature on the presumed determinants of subjective life expectancy gives priority to four factors: socio-demographic characteristics, healthy and unhealthy lifestyles, various aspects of a person's physical health status and mental outlook, and the longevity of parents. There is a well-documented connection between several socio-demographic characteristics and *objective* longevity (Peracchi & Perotti, 2011), and evidence suggests that individuals consider these relationships when estimating their own life expectancy (Benitez-Silva & Ni, 2008; Delavande & Rohwedder, 2011; Halvorsen, Selmer, & Kristiansen, 2010; Liu, Tsou, & Hammitt, 2007; Mirowsky & Ross, 2000; Peracchi & Perotti, 2011). The relationships appear to be relatively complex in the case of minority samples (Bulanda & Zhenmei, 2009; Irby-Shasanmi, 2012).

Prior research on the socio-demographic links to subjective life expectancy has focused almost exclusively on samples of middle-aged and older individuals (Benitez-Silva & Ni, 2008; Biro, 2012; Halvorsen et al., 2010; Hurd & McGarry, 2002; Liu et al., 2007). While life expectancy may be a more salient concept for older individuals, many choices about health and lifestyle with long-term consequences are made when individuals are young (e.g., smoking, diet, exercise) and may be driven in part by subjective life expectancy. Thus, it is important to identify factors associated with subjective life expectancy at a variety of ages.

The Role of Own Health and Family Health Histories

Various aspects of physical and mental health status are also related to subjective life expectancy. Own-health status has been operationalized by asking individuals about their overall health (Benitez-Silva & Ni, 2008; Hurd & McGarry, 2002; Liu et al., 2007) and/or their disease history (Benitez-Silva & Ni, 2008; O'Brien, Fenn, & Diacon, 2005; Peracchi & Perotti, 2011), sometimes linking data from clinical health records as well (Halvorsen et al., 2010; Liu et

al., 2007). The research reveals, not surprisingly, that higher levels of overall self-reported physical health are associated with reports of longer subjective life expectancy (Benitez-Silva & Ni, 2008; Ferraro & Kelley-Moore, 2001; Hurd & McGarry, 1995; Liu et al., 2007; Mirowsky & Ross, 2000; Peracchi & Perotti, 2011), while negative health events are associated with lower subjective life expectancy (Benitez-Silva & Ni, 2008; Halvorsen et al., 2010; Liu et al., 2007; Peracchi & Perotti, 2011).

Beyond a person's own health history and status, there is evidence that some aspects of family health history are related to subjective life expectancy, particularly paternal and maternal longevities (Biro, 2012; Hurd & McGarry, 2002; Liu et al., 2007). Other studies have provided mixed or null results with respect to parental health history (Benitez-Silva & Dwyer, 2005; Peracchi & Perotti, 2011).

Few studies have examined the relevance of other genetically-related relatives. Two empirical studies address the influence of family histories on subjective life expectancy by using summary-level measures of family health (Halvorsen et al., 2010; Robbins, 1988). Two other studies look at the relationship between the death of a sibling and subjective life expectancy, with one finding no effect (Hurd & McGarry, 2002) and the other finding that a sibling's death is linked to a decrease in a respondent's subjective life expectancy assessment (Biro, 2012). Finally, in a study conducted three decades ago, Hamermesh (1985) finds support for a positive relationship between subjective life expectancy and both grandparents' and parents' longevity.

To date, virtually all of the work regarding familial health histories has focused on the self-reported timing of a parent's, grandparent's and/or sibling's death. No one has examined the role of chronic disease histories for specific first- or second-degree relatives. Yet, familial

histories of heart disease, cancer, or some other serious chronic condition may also provide information that individuals use in assessing their subjective life expectancy.

The Potential for Gender-Specific Effects

In addition to a lack of attention to the full breadth of family health history in the literature, very little consideration has been given to the possibility that individuals weigh the health experiences of same-sex relatives more heavily than those of opposite-sex relatives. In some studies, the mother's and father's longevity are not distinguished from one another (Halvorsen et al., 2010; Hamermesh, 1985; Robbins, 1988). In other studies, mother's and father's longevity are treated separately, but there is no test for possible interaction effects with the sex of the respondent (Balia, 2011; Benitez-Silva & Ni, 2008; Biro, 2012). Hurd and McGarry (2002) selectively interact the respondent's sex with variables that denote a mother's death and a father's death, and they find that only in one instance is the interaction statistically significant (albeit reflecting a counterintuitive association). Peracchi and Perotti (2011) estimate separate models for women and men and find that men's (but not women's) assessments of their longevity are related to whether their mothers and their fathers are still alive. In contrast, Liu et al. (2007) find that Taiwanese males whose fathers have died and Taiwanese females whose mothers have died both report significantly lower assessments of the chances they will live to age 75, but these relationships disappear when the outcome is the respondent's assessment of her/his chances of living to age 85.

The Organizing Framework and Hypotheses

Although the analysis reported here is exploratory, it is guided by the framework that recognizes the influences of both genes and environment in chronic disease transmission and health outcomes (Jorde, Carey, & Bamshad, 2010; Rowe, 1994; Smith, Mineau, Garibotti, &

Kerber, 2009). In this framework, the experiences of older or similarly aged first-degree relatives (i.e., parents and siblings) provide strong signals regarding one's longevity because they share 50% of the genes with an individual and because they share the same social environment for an extended period of time. Thus, the potential for inheriting specific genes for serious conditions such as cancer, heart disease, or Alzheimer's is high among first degree relatives (Jorde et al., 2010). In addition, some environmental factors that affect longevity -- such as smoking (and/or second-hand smoke), exercise, and diet -- are likely formed during childhood. To the extent that both genes and environmental factors affect longevity, we hypothesize that an individual will take his/her primary cues regarding longevity by reflecting not only on his/her own health but also on the disease diagnoses and longevity of parents and/or siblings.

An individual shares fewer genes and typically fewer environmental factors with second-degree relatives (i.e., grandparents, aunts/uncles). Thus, while their health experiences may also influence an individual's subjective life expectancy, we hypothesize that the effect size, if present, will be smaller. In both cases, however, the prediction regarding the impact of familial predispositions is unambiguous as we hypothesize that a familial predisposition for disease is hypothesized to reduce an individual's subjective life expectancy assessment.

Based on research regarding parents, we anticipate that not all relatives are created equal. Specifically, subjective life expectancy may be sensitive to the subject's gender and the gender of their relatives. In the case of cancer, for example, the recurrence risk for a gender-specific disease is well established (Teerlink, Albright, Lins, & Cannon-Albright, 2012). Thus, daughters likely take cues from their female kin on matters unique to women such as female reproductive cancers (i.e., breast, ovarian, cervical). Sons will likely do the same with their fathers on reproductive cancers (i.e., prostate, testicular). These sex-specific associations do not prevent an

individual from considering the health and longevity of opposite sex relatives, but it is likely that the health experiences of same-sex kin will be weighted more heavily.

An individual also gains insights about their life expectancy based on his/her own health experiences. The diagnosis of a serious, life threatening disease for a given individual can affect her/his subjective life expectancy assessments in two ways. First, it may lead an individual to reduce her/his life expectancy forecast because of the physical changes s/he experiences and/or because of information s/he learns about survival probabilities from a medical professional or from the medical literature. Second, the diagnosis may lead an individual to change her/his behaviors (e.g., stopping smoking, losing weight) that could mute the impact of the disease on his/her life expectancy. Thus, the magnitude of the effect associated with an individual diagnosis is somewhat ambiguous, but the negative sign prediction is not.

Contributions of the Current Study

The research done to date typically shows that demographics, life-style choices, self-assessed personal health, and the longevity of parents all appear to shape expectations about subjective life expectancy among older individuals. We build on the existing literature by assessing whether the hypotheses generated by the gene-environment framework are supported by our data. Our analyses make use of objective, detailed data on own and familial health histories, including first-degree and second-degree family members' chronic disease diagnoses and ages at death. The empirical work allows for the possibility of gender effects by analyzing females and males separately and distinguishing between male relatives' and female relatives' health experiences. Finally, our hypotheses are tested using data from a sample of individuals who represent a broad age range rather than only adults who are middle aged and older.

Methods

Data and Measures

Unique data from two sources are linked to test the hypotheses posed in the current study which has the approval of the University's Institutional Review Board. Information on subjective life expectancy comes from [university] Retirement Planning Survey (URPS). The URPS was designed to assess [university] employees' retirement planning knowledge, priorities, perceptions, and behaviors in the aftermath of the economic recession of 2008-09. Included in the survey was the following question regarding subjective life expectancy: *What is your best guess about the percent chance that you will live to age 85 or older where 0% represents absolutely no chance and 100% represents absolute certainty that you will live to at least age 85?* The question was taken from the HRS and it was benchmarked at age 85 rather than age 75 given the education level of the typical URPS respondent.

All [university] benefits-eligible employees with valid email addresses (N=9,747) were invited to participate online in the URPS during October 2009. Publicity efforts and participation incentives resulted in 3,000 people submitting completed surveys for an overall cooperation rate of 32.1%. Sixty-five percent of the 3,000 URPS respondents were female and the median respondent age was 44 years. As a point of comparison, as of October 2009, 58% of all university employees were female and the median employee age was approximately 42. Thus, the survey respondents generally reflect the larger population of university benefits-eligible employees in terms of gender and age.

Detailed clinical data on familial health histories come from the Utah Population Database (UPDB). The UPDB is a shared resource located at the [university]. For 35 years, researchers have used this resource to identify and study health issues within a family context. The core component of UPDB is an extensive set of Utah family histories, in which family

members are linked to demographic (i.e., birth, death, marriage, and divorce records) and medical information. Central to the current investigation, the UPDB includes state-wide medical information on cancer diagnoses, hospital inpatient discharges, and causes of death. Most families living in Utah are represented in the UPDB, and individuals in the same family pedigrees are linked to one another with their familial relationship identified.

Utah death certificates and the U.S. Social Security Death Index are linked to the UPDB and provide the needed information on age and cause of death for first and second-degree relatives of the URPS respondents. In addition, diagnoses of specific health conditions for both living and deceased URPS family members come from three UPDB sources: (1) the Utah Statewide Inpatient Hospital Discharge Data, (2) the Utah Cancer Registry, and (3) The [university] Health Sciences Center.

In accordance with the University's Institutional Review Board, consent for linkage was requested of the 2,795 respondents who provided contact information when completing the URPS survey. Of those, 81 declined and of the 2,714 who agreed to be part of this study, 2,669 respondents linked to one or more data sources in the UPDB, for a linkage rate of 98.3%. Linkage of the URPS survey data to UPDB records was done by the Pedigree and Population Resource (PPR) staff at the Huntsman Cancer Institute and a de-identified file was returned to the researchers for analysis. For the purposes of the current analyses, the sample is further restricted to those URPS respondents who linked to both a biological mother and a biological father in UPDB. Thus, the final data set contained 1,119 respondents, 682 women and 337 men. For those equations where we include information about second-degree relatives, the samples are modestly smaller, at 630 and 318 respectively.

In constructing measures of the respondent's health and their familial health histories, we limit our measures to the six leading causes of deaths in the United States (National Center for Health Statistics, 2010) that are also thought to have a genetic and/or an environmental component (U.S. Department of Health and Human Services, 2012a). These are ischemic heart disease, cerebrovascular disease, cancer, chronic obstructive pulmonary disease (COPD), diabetes, and cerebral degenerations. These diagnoses are obtained from the ICD9 and ICD10 codes available in the UPDB. Diagnoses and death record information is limited to those events reported through September 2009, the month before the URPS survey was administered.

Analysis Plan

We estimate six alternative multivariate models separately for women and men. The first model contains information on how many of the six health conditions their mother and father have each had. The second model expands the measurement of family health history to include the percentage of male and female first- and second-degree relatives who have been diagnosed with one or more of these six health conditions. Next we replace diagnosis information with information regarding relatives' ages at death. In the third model, we include variables that capture whether the respondent's mother or father died before age 76 and in the fourth model we add in similar variables for the respondent's grandparents. In the fifth model we expand the measurement of age at death to include the percentage of all male and female first and second-degree relatives who died prior to age 76. Finally, in the last model, we focus exclusively on parental disease diagnoses and parental longevity. All six models control for the respondent's socio-demographic and economic characteristics and whether s/he has ever been diagnosed with one of the six health conditions noted above.

Results

Variable definitions and descriptive information on the URPS sample by gender appear in Table 1. On average, both female and male respondents link to five first-degree relatives in UPDB. As expected, both females and males link to more second-degree relatives than first-degree with the average female respondent linking to 14 second-degree relatives while the corresponding number for the average male respondent is 16.

[Insert Table 1 about here]

A little over seven percent of the males have ever been diagnosed with one of the six conditions that are the leading causes of death in the United States while only 5.2 percent of the women have had such a diagnosis. By virtue of being older, the respondents' mothers and fathers have a higher prevalence of such diagnoses. For female respondents, 34% of their fathers and 26% of their mothers have had one or more of the six diagnoses while the figures for males are 43% and 30%, respectively. For a given individual, the average percentage of first- and second-degree relatives with one or more of the six diagnoses is low but ranges as high as 50 percent of first-degree male and female relatives. Larger percentages of fathers than mothers have died before age 76, and this is also true when comparing grandfathers to grandmothers. These gender differences are not surprising given the gender differences in U.S. life tables (Hamermesh, 1985; Hurd & McGarry, 1995, 2002; Peracchi & Perotti, 2011; Perozek, 2008).

Next, we focus on cause of death among first- and second-degree relatives. Here we observe that in the case of first-degree relatives, only a small fraction of the survey participants have had one or more first-degree relatives – male or female – die from one of the six selected diseases. This is not surprising given the age of the respondents and the fact that first-degree relatives are limited to parents and siblings who may still be living. In the case of second-degree

relatives the fractions are higher. as would be expected as this measure includes older

grandparents.

Given the censored distribution of the dependent variable, multivariate estimation was

done using both tobit and ordinary least squares (OLS) estimation routines in SAS 9.3 (proc qlim

and proc reg). The estimated coefficients and the associated tests of statistical significance do

not substantially differ across the two estimation approaches and thus we present the OLS

estimates because of their ease of interpretation. Tobit estimates are available from the authors

upon request. To avoid the undue influence of outliers, we examined the studentized residuals

and Cook's D statistics used to detect influential observations (Belsey, Kuh, & Welsch, 1980).

We excluded those cases where the studentized residuals exceeded 2.0 in absolute value. Tables

2 and 3 contain the OLS results of the six different models for females and males, respectively.

[Insert Tables 2 and 3 about here]

Across all six models we include controls for age, education, marital status, number of

children, and household income. We are unable to control for race/ethnicity as the data set does

not contain this information. However, it is worth noting that the vast majority of employees at

the university are White non-Hispanic.

Models 1 and 2 examine the impact of familial histories of disease diagnoses on

subjective life expectancy. For model 1, parents' diagnoses have no significant relationship

with females' subjective life expectancy; however, in the case of males, there is a statistically

significant negative relationship between a father's disease diagnosis and the son's estimate.

Turning to model 2, we see additional evidence of the gender-specific effects for males as an

increase in the percentage of first-degree, male relatives who have been diagnosed with at least

one of the six conditions is associated with a statistically significant decline in subjective life

expectancy. For instance, if a male has three first-degree male relatives (e.g., a father and two brothers) and one of them is diagnosed with one of the six conditions, this would lead to an estimated 28.38 percentage decline in the chance of living to age 85 (-.86*33 = 28.38), holding other factors constant.

Models 3-5 test the relationship between a familial history of early death and subjective life expectancy. Again these models provide supportive evidence of gender-specific effects and they reveal evidence in support of our hypothesis that first-degree relatives' experiences matter more than second-degree relatives' experiences. Focus first on model 3 where only the parents' death information is included. On average, a woman whose mother died before age 76 lowers her estimated chance of living to age 85 by 25.24 percent compared to a comparable woman whose mother died after age 75 or whose mother is still alive, *ceteris paribus*. If her father died before age 76, she only reduces her chance of living to age 85 by 4.4 percent (p<.05). In the case of males, the magnitude of the estimated relationships are reversed with the death of a father before age 76 being linked to a 19.49 percent reduction in the male's estimated chance of living to age 85 while the estimated coefficient associated with a mother's death before age 76 is only -4.79 and the estimated effect is statistically insignificant.

Model 4 includes variables that measure whether one or both grandmothers and one or both grandfathers died before age 76. While there is no statistically significant impact of these covariates on males' subjective life expectancy, the early death of one or both grandmothers is linked to a decline in the females' estimated longevity. We also find support for our hypothesis that first-degree relatives should have a larger impact than second-degree relatives when focusing on females. The coefficients suggest that the early death of a mother is linked to an

11.88 percent reduction in the female respondent's subjective assessment of her chance of living to age 85 while the death of a grandmother is linked to only a 3.85 percent reduction.

The gender-specific percentages of all first- and second-degree relatives who have died from one of the six conditions are included as independent variables in model 5. For the females, we observe statistically significant effects of second-degree female relatives and first-degree male relatives – although the estimated magnitude of the effect is much larger for the female second-degree relatives than for the first-degree male relatives. In the case of the males, we again observe the importance of male first-degree relatives' health experiences as they relate to males' subjective life expectancy assessments. Female relatives' experiences and second-degree male relatives' experiences, in contrast, appear to have no impact.

Model 6 includes variables that capture both the parents' disease diagnoses and death prior to age 76. As in other models, the gender-specific effects are strong with the mother's early death dominating in the case of female respondents and the father's health experiences and early death dominating in the case of male respondents.

Finally, it should be noted that across all six models, the variable that measures whether the respondent has been diagnosed with one or more of the six health conditions has a consistently strong, statistically significant relationship with subjective life expectancy. For females the estimates range from -12.88 to -29.34, while for males the range is -12.67 to -17.29. This reaffirms the findings of past research that has examined the impact of disease diagnoses on subjective life expectancy (Benitez-Silva & Ni, 2008).

Study Limitations

The conclusions drawn from our analyses should be tempered with the acknowledgement of our exploratory study's limitations. Specifically, our empirical work is based on a sample of

individuals who, on average, are highly educated and work for a single employer. As such, our findings may not extrapolate to groups with different socio-demographic characteristics. In addition, the survey data used in this study do not include questions regarding the respondent's race/ethnicity or smoking status. The omission of these variables likely causes minimal bias in our estimates, however, as the majority of university employees are White Non-Hispanic and smoking rates in the state are among the lowest in the nation. Our survey also did not query respondents about their chances of living to a range of ages (e.g., 75, 85), although multiple age benchmarks are often used in the literature (Balia, 2011; Hamermesh, 1985; Liu et al., 2007; Peracchi & Perotti, 2011). Finally, the relatively small sample sizes available for analysis may have reduced the statistical power of the study, thus making the findings conservative.

Discussion and Conclusions

With the above caveats in mind, we turn now to a discussion of our findings. To place our exploratory family history results in context, it is useful to begin with the effects of own-health on subjective life expectancy. Parallel to previous studies that have used self-assessments of overall health status (Bulanda & Zhenmei, 2009; Peracchi & Perotti, 2011) or specific health conditions (Benitez-Silva & Ni, 2008; Biro, 2012; Halvorsen et al., 2010; Hurd & McGarry, 2002), we find that objective data on own-health is strongly linked to subjective life expectancy. Specifically, respondents who have experienced one or more of six serious health events offer much lower probabilities of living to age 85. For the average woman in our sample, having one or more of these six illnesses drops her self-assessed chance of living to 85 by roughly 25 to 30 percentage points. For men, the drop is in the range of 15-20 points. Based on prior research, these dramatic results are to be expected; what is noteworthy is that some of the effects of family

history are estimated to be of similar magnitude when they are entered in equations that control for own-health.

We find overall support for the hypothesis that the health and longevity of relatives affects subjective life expectancy, with the experience of first-degree relatives having a much stronger impact than that of second-degree relatives. Our findings are consistent with a handful of other studies that have reported larger first-degree relatives' effects and smaller (or non-existent) second-degree relatives' effects (Biro, 2012; Hamermesh, 1985; Hurd & McGarry, 2002). Given that the genetic makeup as well as the social environment of parents more closely overlaps with that of respondents, this finding makes sense. Among men, for example, having a father who died before the age of 76 reduces the perceived probability of living to age 85 by almost twenty percentage points. From a genetic perspective, the health history of grandparents and other second-degree relatives can also be relevant to actual longevity. It is therefore noteworthy that respondents – especially men -- do not appear to rely on this information very heavily. Perhaps respondents view information on the health experiences of second-degree relatives as too unreliable in that information passed down by family members may be only partially correct and become even more distorted by time.

We also find consistent evidence of gender-specific familial health effects with females taking their cues from their mothers' and grandmothers' ages at death whereas males appear to be taking their cues from their fathers' and other first-degree male relatives' health and longevity experiences. Recall that most previous studies do not test for gender-specific parental effects (Balia, 2011; Benitez-Silva & Ni, 2008; Biro, 2012; Halvorsen et al., 2010; Hamermesh, 1985; Robbins, 1988). Of the three that do test for such effects, our findings are consistent with two studies (Liu et al., 2007; Peracchi & Perotti, 2011) and counter to the findings of one study (Hurd

& McGarry, 2002).   The sex-specific patterns that we observe make sense to the extent that some diseases (e.g., breast and prostate cancer) afflict only members of one sex. But, the risk of other conditions (e.g., heart disease and diabetes) with hereditary and lifestyle components increases if either parent had the condition. Whether individuals rely too much or too little on the gender-specific health experiences of their relatives in assessing their own subjective life expectancy is an open question that merits future research.

Two unfolding technological advances are likely to enhance the link between family health histories and subjective longevity in the future. First, advances in human genome research are increasing the number and accuracy of genetic tests for adult-onset diseases (Jorde et al., 2010). As these tests grow in number and their costs decline, individuals will have the potential to forecast their life expectancies with greater accuracy and at earlier ages. Second, the increasing use of electronic health records has the potential to help narrow the gap between what individuals think they know about their family health histories and what is actually true about these histories. Of course, there are issues of health privacy to be resolved, but the potential exists for medical professionals to have access to more accurate information about family health histories than they -- and their patients -- do currently. Advances in genetic testing and electronic health records thus have the potential to lead to more refined and complete family health portraits.

Given the growing importance of family health history information, it is not surprising that public health officials are actively encouraging individuals to gather as much of this information as possible and create a "family health portrait" that may be shared with their health care providers (U.S. Department of Health and Human Services, 2012b).   Such portraits may contain information from a wide range of sources including genetic tests, clinical diagnoses,

causes of death as noted on death certificates, and oral health histories obtained from living family members. While our research suggests that respondents do not rely on family health history much beyond first-degree relatives, the rich information from such family portraits may be better utilized when interpreted by health professionals who can accurately assess disease risk and encourage early detection and prevention strategies. Indeed, health professionals are probably in the best position to accurately recognize familial disease patterns and give advice regarding familial risk of a specific disease (Yoon, Scheuner, & Khoury, 2003).

The question of how clinicians can best help patients to process family health history information appropriately is important. We know that self-perceived health is an important predictor of objective mortality risk, even after controlling for specific health conditions. Likewise, self-perceived health is an important indicator of subjective life expectancy (Bulanda & Zhenmei, 2009; Peracchi & Perotti, 2011).   As individuals' family health portraits become more refined and complete, it will be important for health care professionals to communicate the implications of family health histories to patients in ways that motivate them to take appropriate preventive actions without precipitating negative social-psychological consequences.   Thus, it is vital that both researchers and clinicians gain a better understanding of the underlying mechanisms that link family health histories to both objective and subjective patient outcomes.

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Table 1. Descriptive Statistics

Variable	Defintion	Females (N=677)		Males (N=337)	
		Mean / Proportion	St. Dev.	Mean / Proportion	St. Dev.
chance of living to 85	Estimated percentage chance of living to age 85	67.75	27.05	59.10	29.29
age	Age in years	41.16	12.57	42.03	12.46
education	Education in years	15.49	1.79	16.35	2.14
married	1=married, 0=otherwise	0.59		0.82	
kids	Number of children ever born	1.44	1.64	2.39	2.05
income	Household income in 2009 (in $10,000s/yr)	7.29	4.02	8.37	4.49
father's age	Father's age when respondent was born	31.22	6.47	31.20	6.27
mother's age	Mother's age when respondent was born	28.66	5.83	28.83	5.90
respondent ill	1=respondent has been diagnosed with ≥ 1 of the six diseases, 0=otherwise	0.05		0.07	
mother ill	Total number of diagnoses respondent's mother has had	0.36	0.68	0.40	0.70
father ill	Total number of diagnoses respondent's father has had	0.48	0.78	0.62	0.83
mother died before 76	1=mother died before age 76, 0=otherwise	0.12		0.11	
father died before 76	1=father died before age 76, 0=otherwise	0.21		0.15	
grandmother died before 76	1=one or both grandmothers died before age 76, 0=otherwise	0.29		0.28	
grandfather died before 76	1=one or both grandfathers died before age 76, 0=otherwise	0.43		0.44	
% male FDR[a] with chronic conditions	% of male first-degree relatives with ≥ 1 of the six chronic diseases	3.51	7.53	3.95	7.58
% female FDR[a] with chronic conditions	% of female first-degree relatives with ≥ 1 of the six chronic diseases	2.49	6.61	2.98	7.45
% male SDR[b] with chronic conditions	% of male second-degree relatives with ≥ 1 of the six diseases	2.43	3.55	2.36	3.33
% female SDR[a] with chronic conditions	% of female second-degree relatives with ≥ 1 of the six chronic diseases	1.92	3.17	2.13	3.17
% male FDR[a] cause of death	% of male first-degree relatives who died from 1 of the six diseases	6.71	18.95	8.52	22.52
% female FDR[a] cause of death	% of female first-degree relatives who died from 1 of the six diseases	6.18	19.09	4.85	17.36
% male SDR[b] cause of death	% of male second-degree relatives who died from 1 of the six diseases	12.13	17.18	11.96	13.59
% female SDR[b] cause of death	% of female second-degree relatives who died from 1 of the six diseases	9.76	14.43	9.74	13.23

Total FDR[a] and SDR[b]	Total number of first- and second-degree relatives	18.79	10.45	20.78	11.42

[a] FDR = first-degree relatives
[b] SDR = second-degree relatives

Table 2. Parameter Estimates for the Multivariate Regressions: Females

Variable	Model 1 (N=639) Coef.	S.E.	Model 2 (N=594) Coef.	S.E.	Model 3 (N=638) Coef.	S.E.	Model 4[a] (N=537) Coef.	S.E.	Model 5 (N=593) Coef.	S.E.	Model 6 (N=637) Coef.	S.E.
Intercept	42.46	8.01	47.33	8.80	51.08	7.98	51.20	9.09	44.12	8.55	50.02	8.05
age[b]	-0.01	0.09	0.01	0.09	0.07	0.08	0.10	0.09	0.15	0.10	0.11	0.09
age squared[b]	0.02	0.01***	0.02	0.01	0.02	0.01**	0.02	0.01**	0.02	0.01***	0.02	0.01**
education	1.37	0.52**	1.00	0.55*	1.12	0.52**	1.18	0.57**	1.58	0.54***	1.25	0.52**
married	3.51	2.04*	3.94	2.15*	3.45	2.02*	2.69	2.21	3.06	2.13	2.81	2.01
kids	0.32	0.61	0.09	0.63	0.40	0.60	-0.01	0.65	-0.17	0.63	0.68	0.60
income	0.44	0.27*	0.53	0.28**	0.20	0.26	0.35	0.28	0.38	0.27	0.14	0.26
respondent ill	-29.34	4.05***	-27.93	4.15***	-25.24	3.92***	-23.92	4.21***	-25.43	4.09***	-27.49	3.85***
mother ill	0.18	1.37									0.83	1.35
father ill	-1.20	1.16									-1.66	1.14
% male FDR[c] with chronic conditions			-0.19	0.12								
% female FDR[c] with chronic conditions			-0.13	0.14								
% male SDR[d] with chronic conditions			-0.16	0.26								
% female SDR[d] with chronic conditions			-0.20	0.29								
Total FDR[c] and SDR[d]			0.07	0.09					0.00	0.09		
mother died before 76					-12.88	2.74***	-11.88	2.90***			-12.72	2.76***
father died before 76					-4.40	2.26**	-2.77	2.44			-4.58	2.26**
grandmother died before 76							-3.85	2.11*				
grandfather died before 76							-1.94	1.92				
% male FDR[c] cause of death									-0.09	0.05*		
% female FDR[c] cause of death									-0.04	0.05		
% male SDR[d] cause of death									-0.05	0.05		
% female SDR[d] cause of death									-0.25	0.07***		
Adjusted-R^2	0.11		0.11		0.12		0.12		0.12		0.13	
F-Statistic	9.54**		6.87**		10.84**		6.64**		7.59**		9.71**	

* p< .1   **p<.05   ***p< .001

[a]This equation also contains two dummy variables that take on a value of one if data on one-to-three grandparents is missing. Estimates based on regressions that restricted to the sample to respondents with complete information on all four grandparents paralleled the estimates presented here and are available from the authors upon request.

[b] Age has been centered on the mean and then squared so as to avoid potential collinearity problems between age and age-squared (Glantz & Slinker, 1990).

[c]FDR = first-degree relatives

[d] SDR = second-degree relatives

Table 3. Parameter Estimates for the Multivariate Regressions: Males

Variable	Model 1 (N=326) Coef.	S.E.	Model 2 (N=306) Coef.	S.E.	Model 3 (N=322) Coef.	S.E.	Model 4[a] (N=278) Coef.	S.E.	Model 5 (N=304) Coef.	S.E.	Model 6 (N=324) Coef.	S.E.
Intercept	25.19	12.32	37.76	12.50	24.73	11.53	16.81	13.51	24.07	12.13	32.82	12.14
age[b]	-0.39	0.16***	-0.30	0.17*	-0.22	0.15	-0.30	0.17*	-0.25	0.17	-0.24	0.16
age squared[b]	0.01	0.01	0.02	0.01	0.02	0.01	0.03	0.01**	0.02	0.01*	0.01	0.01
education	1.93	0.78***	1.30	0.79*	2.05	0.75*	2.09	0.82**	2.17	0.78	1.66	0.76**
married	4.85	4.28	7.83	4.38*	4.95	4.18	8.37	4.77*	7.21	4.32*	3.40	4.22
kids	0.88	0.85	0.59	0.88	1.01	0.82	0.81	0.90	0.51	0.88	1.25	0.83
income	-0.08	0.40	-0.04	0.40	-0.14	0.39	-0.21	0.46	-0.16	0.41	-0.01	0.39
respondent ill	-16.50	5.80***	-16.29	5.81**	-17.29	5.58***	-12.67	5.94**	-14.94	5.74**	-16.11	5.60***
mother ill	3.30	2.31									3.79	2.24*
father ill	-5.08	1.89**									-5.40	1.86***
% male FDR[c] with chronic conditions			-0.87	0.20***								
% female FDR[c] with chronic conditions			-0.16	0.20								
% male SDR[d] with chronic conditions			-0.33	0.48								
% female SDR[d] with chronic conditions			-0.74	0.47								
Total FDR[c] and SDR[d]			-0.02	0.14					-0.06	0.14		
mother died before 76					-4.79	4.57	-3.31	4.99			-7.03	4.67
father died before 76					-19.49	4.11***	-19.64	4.48***			-19.16	4.12***
grandmother died before 76							1.80	3.48				
grandfather died before 76							1.52	3.41				
% male FDR[c] cause of death									-0.23	0.07***		
% female FDR[c] cause of death									-0.05	0.08		
% male SDR[d] cause of death									-0.07	0.11		
% female SDR[d] cause of death									-0.12	0.12		
Adjusted R-squared	0.10		0.14		0.15		0.13		0.14		0.16	
F	5.12**		5.05**		7.19**		4.29**		4.98**		6.62**	

* p< .1   **p<.05   ***p< .001

[a]This equation also contains two dummy variables that take on a value of one if data on one-to-three grandparents is missing. Estimates based on regressions that restricted to the sample to respondents with complete information on all four grandparents paralleled the estimates presented here and are available from the authors upon request.

[b] Age has been centered on the mean and then squared so as to avoid potential collinearity problems between age and age-squared (Glantz & Slinker, 1990).

[c]FDR = first-degree relatives

[d] SDR = second-degree relatives