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Connecting social environment variables to the onset of major specific health outcomes

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Objective: The present research examined the effects of the social environment on the onset of specific health ailments. Design: Using data from the Health and Retirement Study, we examined participants' responses to social environment questions in 2006 as predictors of onset of different health conditions over the next four years.
Main Outcome Measures: Healthy participants ($n = 7514$) reported on their number of social partners, interaction frequency, positive social support and negative social support with respect to both their family and friends. These variables were used to predict onset of seven conditions in 2010: high blood pressure, heart condition, lung disease, cancer, stroke, diabetes and arthritis.
Results: Logistic regressions indicated that the social environment provided some predictive value for onset of most health outcomes, with more positive and less negative social support appearing to buffer against onset. Social environmental variables related to friendships appeared to play a greater role than the family indicators. However, no variable proved universally adaptive, and social indicators had little value in predicting onset of chronic conditions.
Conclusion: The current findings point to the potential for the social environment to influence later health, while demonstrating the nuanced role that our social lives play with respect to health.

Keywords: social environment; ailment onset; social support; social network size

One of the most frequently investigated topics in health psychology is the role of social networks and social support on health (see, for a review, Cohen, Gottlieb, & Underwood, 2000; Miller, Chen, & Cole, 2009; Seeman, 1996; Smith & Christakis, 2008). Part of the reason for this interest is that, as yet, a clear and consistent 'answer' has not been found regarding when and how social variables influence health outcomes (Seeman, 1996). Instead, accruing studies on the topic point to ever-increasing nuance in the findings. For instance, the potential benefits of social relationships may depend both on the aspect of the social environment under study (support, network size, interaction quality), as well as the outcome of interest (e.g. Banks, Berkman, Smith, Avendano, & Glymour, 2010; Walen & Lachman, 2000). Given these points, it is surprising that few studies have examined the role of multiple social aspects on different health outcomes, instead tending to focus on single variables on both ends.

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Such work thus limits the ability to address questions such as whether family or friend relationships have a wider influence on health outcomes, or if the propensity for our social environments to buffer or exacerbate our risks for disease differs based on the ailment under study.

Moreover, research tends to focus on concurrent associations with social environments and health, and not on addressing whether social environments predict who is more prone to contracting the ailment over time. Such questions are important for disentangling the effect of the social environment on past health concerns from its influence on one's vulnerability for future problems. The current study addressed these concerns using longitudinal data from the long-running Health and Retirement Study (HRS) of adults in the United States. Specifically, we examined the roles of family and friend relationships on whether adults developed multiple ailments, including cardiovascular and respiratory issues, cancer, stroke and arthritis. In both cases, we examined the role of four different aspects of family and friend relationships: number of close contacts, interaction frequency, positive social support and negative social support.

Social environment influences on physical health outcomes

The social environment consistently proves one of the most important predictors of health and well-being. For our purposes, we focus on the role of family and friend relationships when describing participants' social environments. The need for close friends and social contacts has been supported by research demonstrating how these variables predict greater longevity (see, Holt-Lunstad, Smith, & Layton, 2010, for a review), and work nominating social isolation as a prominent risk factor for mortality across different species (see, Cacioppo, Hawkley, Norman, & Berntson, 2011).

And yet, the potential remains for social environment variables, like having a large social network, to *negatively* impact health (Smith & Christakis, 2008). One way social experiences may lead to poorer health is that they may influence the propensity for health or unhealthy actions. For instance, there is the potential for obesity to 'spread' throughout one's friends and family (e.g. Christakis & Fowler, 2007), nominating social networks as a potential resilience and risk factor depending on with whom one associates. In addition, it is well known that our friends can significantly influence whether or not we use substances (Hawkins, Catalano, & Miller, 1992; Newcomb & Bentler, 1989), particularly during the adolescent years. Moreover, our social environments can play a role in whether and how we use health services (e.g. Bosworth & Schaie, 1997). Accordingly, the research on health behaviours has provided significant advances towards understanding the nuance in the relationships between one's social environment and his or her health.

Another route taken by researchers has been to examine the potential for our social environments to influence our development and ability to cope with an ailment. Again, this literature points to the potential double-edged nature of our social environment. On one hand, perceived social support positively predicts physical health (e.g. Berkman, Glass, Brissette, & Seeman, 2000; Cohen, 2004), psychological well-being (e.g. Turner, 1981; Winefield, Winefield, & Tiggemann, 1992), cognitive functioning (e.g. Seeman, Lusignolo, Albert, & Berkman, 2001) and even adaptive personality development (Hill, Payne, Jackson, Roberts, & Stine-Morrow, in press). However, studies also have

demonstrated instances where support was unrelated or even positively related to markers of ill-being (Barrera, 1986; Bolger, Zuckerman, & Kessler, 2000; Coyne & Bolger, 1990), which may depend in part on whether the support was perceptible by the recipient (Bolger et al., 2000; Maisel & Gable, 2009). Accordingly, this line of work further underscores the lack of a consistent linkage between the social environment and health. Moreover, this literature points to the clear need to examine multiple aspects of the social environment simultaneously, as even seemingly similar indicators, such as visible and invisible received support, lead to drastically different health outcomes.

The current study took an alternative approach by examining whether social indicators predict the onset of specific health ailments. Such a strategy provides two important advances to the field. First, it allows further understanding of the mechanisms involved, given that there are known lifestyle differences that precede specific ailments. For instance, if a social indicator predicts participants' risk for lung disease but not diabetes, it would suggest a greater potential for that variable influences health through pathways such as smoking activity rather than eating behaviour. Alternatively, if the variable influences one's risk for both outcomes to a similar extent, then that social aspect may be predicting health through a pathway shared by both ailments, such as, in this example, exercise behaviour.

Second, by predicting the later onset of a disease, the current study provides a clearer understanding of the long-term or longitudinal effect of the social environment. Focusing instead on the co-occurrence of social environment and health (e.g. Banks et al., 2010) may obscure the potential risk or resilience provided by the social environment. Having an ailment certainly can influence an individual's number of social contacts or perceptions of social support, for instance via inclusion in a support group targeting similarly ailed individuals. As such, it becomes valuable to go beyond cross-sectional relations, to understand how the social environment predicts one's proneness to or protection from different health ailments. That said, the previous cross-sectional research has provided valuable direction regarding which health ailments may be most influenced by the social environment, a point we turn to next.

Social environment and specific health ailments

While several studies have focused on mortality, given its clearly objective nature and importance, a significant literature has examined the role of the social environment on other specific health outcomes. This direction has been motivated by literature reviews pointing to the potential for variables like social support to influence a number of physiological processes (e.g. Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Indeed, this work has linked social support to biomarkers of cardiovascular, endocrine and immune functioning, setting the stage for linking the social environment to the occurrence and onset of the specific health ailments associated with these systems.

For instance, using cross-sectional data from a similar sample as the current study, work has demonstrated linkages between markers of social support and social networks on specific health outcomes (Banks et al., 2010). Findings suggested that American men with broader social networks were more likely to report having heart disease, while social networks appeared to help buffer American women from ailments such as diabetes, heart disease and hypertension, but it led to greater risk for obesity. Moreover,

having fewer negative interactions with a social network was associated with lower rates of obesity, diabetes and lung functioning amongst both sexes, while the role of positive interactions appeared to play a lesser role on health. However, as the authors note, these cross-sectional associations fail to prove the full story of whether and how the social environment influences the onset specific physical health outcomes. Instead, these associations may reflect changing social environments in response to developing these health maladies.

Research is needed that moves beyond single-time assessments towards understanding whether social variables predict onset of different ailments. As of yet, most research towards that end has centred on heart disease and hypertension (e.g. Kawachi et al., 1996; Orth-Gomér, Rosengren, & Wilhelmsen, 1993; Reed, McGee, Yano, & Feinleib, 1983; see, for a review, Lett, Blumenthal, Babyak, Strautman, Robins, & Sherwood, 2005). Most of these studies though focused on a single aspect of the social environment, which may lead to very different conclusions regarding health risks, as noted by the distinction between social networks and negative social interactions above (Banks et al., 2010). Moreover, while the importance of heart disease is clear, given that it remains one of the most common causes of mortality (Hoyert & Xu, 2012), studying only heart-related ailments negates the ability to see whether the effect of social variables differs across health ailments. For instance, does the social environment influence risk for heart-related issues more than it does for other mortality risks or prominent chronic conditions?

Current study

The HRS is unique in that it has recently provided the opportunity to not only assess multiple social domains, but also several aspects of these domains, that map onto the more frequently examined indicators in the field, such as network size and social support. The current study employed longitudinal data from the HRS to advance our understanding of the social environment's effect on specific health ailments in three important ways. First, we addressed the topic longitudinally, by predicting the likelihood that initially, 'healthy' individuals were more likely to contract a specific ailment over time. Second, we did so by using assessments of the separate roles for family and friends, as well as indicators of network size (number of close associates), interaction frequency, positive social support and negative social support. Based on the previous studies (e.g. Banks et al., 2010), we predicted that interaction frequency and negative social support would play a stronger role on health outcomes (positively and negatively, respectively) across the board than network size and positive social support. Third, we tested the value of the social environment across seven different specific health outcomes, including those indicative of heart health (high blood pressure or heart condition), major non-heart-related causes of death (lung disease, cancer and stroke), as well as chronic conditions with wide-spanning occurrence (diabetes and arthritis). In this respect, we not only advance the extant literature by looking at different heart-related ailments, but also move beyond this domain towards understanding the potential role on other prominent ailments. Given the known linkage between social variables and mortality (Holt-Lunstad et al., 2010), we predicted that aspects of the social environment should influence risk for the non-heart-related major mortality risks, but it was unclear whether these variables would predict risk for chronic ailments like diabetes and arthritis.

Method

Participants

Participants ($N = 7514$; 59% female; $M_{age} = 68.0$, SD = 10.7) come from the HRS, a longitudinal representative sample of ageing American adults (Juster & Suzman, 1995). The HRS was initiated in 1991 with new cohorts added continually (Total N for HRS = 25,760). Participants in the current study were given a social environmental questionnaire in 2006. Social environmental scales were included in a leave behind packet, which participants returned by mail. The response rate for the self-administered questionnaires was 74%. Ethnicity was 83% Caucasian, 13% African American, 2% Hispanic and 2% Others. Participants had completed on average 12.7 years of education (SD = 3.1), and 62.9% reported currently working for pay. The mean income for all participants with 2006 and 2010 data was $41,032 (SD = $55,383). Participants were contacted again by phone in 2010, four years after the initial social environmental assessment.

Procedure and measures

Initial social environment questions determined whether or not the participants had family members or friends available to them. For family, participants were asked if 'they had any immediate family, for example, brother or sister, parents, cousins or grandparents?' For friends, participants were asked, 'do you have any friends?' Out of the initial sample, 556 participants reported not having family members and 346 participants did not have any friends.

For participants who reported having family and friends, four components of social environments were assessed in 2006: network size, interaction frequency, negative support and positive support. Each component was assessed separately for family and friends. For network size, participants were asked to list the 'number of family members/friends with close relationships'. If participants failed to respond to this item or the question above about having any friends or family, they were excluded from the analyses. Amount of weekly interaction was assessed by creating a composite of three questions asking, 'On average, how often do you do the following with any of your family members/friends, not counting any who live with you: meet up, speak on the phone, write or email'. For each response, participants could indicate 5 different choices ranging from 'three or more times a week' to 'less than once a year to never'. The weekly interaction composite demonstrated less-than-ideal reliability for a three-item measure for both family ($\alpha = .51$) and friends ($\alpha = .52$), but these reliabilities may be expected given the small number of items. Furthermore, it is unclear whether one should expect individuals to employ each of these mediums similarly when contacting their social networks. Indeed, HRS study administrators have cautioned researchers against expecting a high degree of internal consistency for these reasons (Smith et al., 2013).

Three items were used to assess positive social support (e.g. 'How much can you rely on them if you have a serious problem?'). Negative social support was assessed with four items (e.g. 'How often do they make too many demands on you?'). These items are taken from previous work linking support (or lack thereof) to depressed mood (Schuster, Kessler, & Aseltine, 1990) and have also been employed in the English Longitudinal Study of Ageing (Cheshire et al., 2012). Participants rated each question on a

Table 1. Correlations between social environment variables, organised by family and friend domains.

	1	2	3	4	5	6	7	8
No. of family members (1)	–							
Family interaction frequency (2)	.22*	–						
Positive family support (3)	.24*	.47*	–					
Negative family support (4)	−.03*	.03*	−.18*	–				
No. of friends (5)	.36*	.08*	.09*	−03*	–			
Friend interaction frequency (6)	.04*	.32*	.09*	.02	.17*	–	–	
Positive friend support (7)	.06*	.12*	.28*	−.02*	.18*	.37*	–	
Negative friend support (8)	.00	.00	−.08*	.46*	.00	.04*	−.07*	–
Mean	3.80	3.39	2.88	1.55	4.50	3.76	3.04	1.40
SD	5.34	1.11	0.86	0.63	6.05	1.07	0.75	0.52

*$p < .05$.

four-point scale ranging from ‘a lot’ to ‘not at all’. Alpha reliability was acceptable for positive social support (.86, .84) and negative social support (.78, .76) for both family and friends, respectively. As shown in Table 1, the correlations between these constructs were low to modest, and thus, we examined positive and negative social support separately. While these measures reflect the items available for friend and family support, similar items were asked about partners or spouses and for children. However, we focused on the family and friend items given the greater potential for participants to be without spouses or children.

Health measures were collected through telephone interviews in 2006 and 2010. Three different types of diseases were assessed: heart health, prominent mortality risks and chronic illnesses. Participants were asked, ‘Has a doctor ever told you that you have [specific illness]?’ Heart health illnesses were (a) high blood pressure or hypertension and (b) having a heart condition, including a heart attack, coronary heart disease, angina, congestive heart failure or other heart problems. Prominent mortality risks included (a) cancer or a malignant tumour, excluding minor skin cancer, (b) a chronic lung disease, such as chronic bronchitis or emphysema and (c) a stroke. Chronic illnesses were (a) diabetes or high blood sugar and (b) arthritis or rheumatism. Responses were coded as either *yes* (1) or *no* (0). For each common disease, we considered only the set of participants who reported not having that disease during the first set of interviews in 2006. As a result, the sample size differs for each disease. Illnesses more likely to occur (e.g. high blood pressure, arthritis) comprise a smaller sample, while illnesses less likely to occur (e.g. cancer, lung disease) have larger samples. Initial sample sizes ranged from 6642 individuals who never had a stroke to 2790 participants who never were diagnosed with arthritis. The likelihood of developing the disease across the study period also differed across disease category. The number of new cases ranged from 642 for high blood pressure to 233 incidences of stroke. See, Tables 2–4 for specific sample sizes and prevalence rates.

Data analytic plan

Our analyses below are grouped into three main categories. First, we examined the unique nature of the social environment variables by performing zero-order correlations on the indices at 2006. If indicators appear to assess separate constructs, we will pro-

Table 2. Predicting onset of heart health problems using single predictors, and then all predictors within a given domain (family or friends), controlling for age, sex, minority status and years of education.

	Single-predictor models			Domain models		
Predictor	B (s.e.)	Wald	Odds (95% CI)	B (s.e.)	Wald	Odds (95% CI)
High blood pressure (Initial n = 3040, New cases = 642, 21.1%)						
No. of close family	−.017 (.012)	2.06	.98 (.96–1.01)	−.017 (.012)	1.95	.98 (.96–1.01)
Family interaction	−.091 (.045)	4.01*	.91 (.84–1.00)	−.049 (.053)	.88	0.95 (.86–1.06)
Positive family support	−.119 (.058)	4.25*	.89 (.80–.99)	−.059 (.070)	.71	.94 (.82–1.08)
Negative family support	.059 (.079)	.56	1.06 (.91–1.24)	.055 (.084)	.43	1.06 (.90–1.25)
No. of close friends	−.010 (.010)	1.08	.99 (.97–1.01)	−.008 (.010)	.71	.99 (.97–1.01)
Friend Interaction	−.029 (.048)	.35	.97 (.89–1.07)	.011 (.054)	.05	1.01 (.91–1.12)
Positive friend support	−.116 (.069)	2.87	.89 (0.78–1.02)	−.087 (.077)	1.26	.92 (.79–1.07)
Negative friend support	.217 (.097)	5.04*	1.24 (1.03–1.50)	.219 (.101)	4.74*	1.25 (1.02–1.52)
Heart condition (Initial n = 5472, New cases = 534, 9.8%)						
No. of close family	−.014 (.012)	1.39	.99 (.96–1.01)	.007 (.012)	0.34	.99 (.97–1.02)
Family interaction	−.090 (.046)	3.78	.91 (.84–1.00)	−.076 (.055)	1.96	.93 (.83–1.03)
Positive family support	−.088 (.058)	2.28	.92 (.82–1.03)	−.025 (.070)	.12	.98 (.85–1.12)
Negative family support	.117 (.080)	2.12	1.12 (0.96–1.32)	.088 (.084)	1.08	1.09 (0.93–1.29)
No. of close friends	−.009 (.010)	.76	.99 (0.97–1.00)	−.007 (.010)	.50	.99 (.97–1.01)
Friend interaction	−.044 (.049)	.81	.96 (.87–1.05)	−.024 (.054)	.20	.98 (.88–1.09)
Positive friend support	−.071 (.069)	1.06	.93 (.81–1.07)	−.017 (.077)	.05	.98 (.85–1.14)
Negative friend support	.120 (.098)	1.49	1.13 (0.93–1.37)	.107 (.102)	1.09	1.11 (.91–1.36)

*$p < .05$.

ceed by examining their influence on the onset of specific illness. Second, we performed a series of logistic regressions, predicting whether one has the illness at 2010 from each social environment indicator, controlling for other known influences on health, including age, sex, minority status and years of education. These analyses provide an exploration of the extent to which the social environment may prove a risk factor for these different illnesses. Research has described the social environment as potentially having both a direct effect and buffering role (see, e.g. Cohen & Wills, 1985) on health outcomes. Given the relative novelty of the work, we focused on

examining their general predictive value to provide initial insights into the potential role of social support and network variables on illness onset. Significant effects therefore could provide evidence for either a direct effect or a buffer interpretation, and we do not make any claims towards either front. Third, to gain greater insight into which aspects of a given social network are most valuable, we performed these analyses again only with all indicators for a given domain in the model. In other words, we predicted onset of each health outcome again, only with all friendship indicators in the same model, and then with all family indicators in the same model.

Results

Correlations between social environment variables

Prior to testing their effects on health outcomes, we examined the correlations between different social environment variables, in order to demonstrate their distinct nature. Table 1 presents these zero-order correlations across the family and friend domains. Clearly, none of the different social indicators were synonymous in nature, as all correlations were below .5 in magnitude, and most well below this threshold. Within the family domain, the average magnitude was only .20, and an even lower value was found for the friend domain indicators (.14). Therefore, these different variables do appear to be picking up on distinct elements of the social environment, and thus, we proceeded to examine their separate effects on the health outcomes of interest.

Predicting onset from single social indicators

Next, we tested the relationships between indicators of the social environment and the onset of specific health outcomes. Given the novel nature of several of these analyses, we first performed single logistic regression analyses for each social indicator on each outcome of interest, controlling for age, gender, minority status (dichotomised as white or minority) and education. In Tables 2–4, the results for the single indicator models are presented in columns 2–4, while columns 5–7 present the results when including all indicators from one social domain in the same model. Accordingly, while each table organises the social variables together by domain, social indicators initially are analysed separately, and then, we discuss below the results of combining these variables into a single model.

With respect to the heart health outcomes, shown in Table 2, a number of social environmental factors predicted future onset of an illness. For all significant results below, we provide the reduction or increase in the odds for onset of the ailment per a one-unit increase in the predictor of interest. Again, these results are largely exploratory, given the novelty of the work, and thus, the findings should be interpreted as such. Only one friend indicator proved significant, as having more negative friend support predicted an increased risk for high blood pressure (24% increase in odds). Two effects were evidenced in the family domain. Having more family interactions led to a 9% reduction in the risk of onset for high blood pressure. In addition, having positive family support predicted a reduced risk for high blood pressure (11% reduction).

With respect to the other prominent mortality risks (Table 3), only one family effect was evidenced. Having positive family support reduced the odds for lung disease (16%

Table 3. Predicting onset of prominent mortality risks using single predictors, and then all predictors within a given domain (family or friends), controlling for age, sex, minority status and years of education.

	Single-predictor models			Domain models		
Predictor	B (s.e.)	Wald	Odds (95% CI)	B (s.e.)	Wald	Odds (95% CI)
Cancer (Initial N = 6076; New cases = 343, 5.7%)						
No. of close family	.006 (.013)	.19	1.01 (.98–1.03)	.009 (.013)	.44	1.01 (.98–1.03)
Family interaction	−.062 (.058)	1.17	.94 (.84–1.05)	−.079 (.068)	1.34	.92 (.81–1.06)
Positive family support	.008 (.073)	.01	.99 (0.86–1.14)	.029 (.087)	.11	1.03 (.87–1.22)
Negative family support	.022 (.103)	0.05	1.02 (0.84–1.25)	.037 (.108)	0.12	1.04 (.84–1.28)
No. of close friends	.007 (.009)	.64	1.01 (.99–1.03)	.010 (.009)	1.20	1.01 (.99–1.03)
Friend interaction	−.128 (.061)	4.41*	0.88 (.78–.99)	−.181 (.068)	7.02*	.84 (.73–.95)
Positive friend support	.083 (.088)	0.87	1.09 (.91–1.29)	.128 (.096)	1.77	1.14 (.94–1.37)
Negative Friend Support	−.130 (.132)	.97	.88 (0.68–1.14)	−.079 (.135)	0.35	.92 (.71–1.20)
Stroke (Initial N = 6642; New cases = 233, 3.5%)						
No. of close family	.018 (.012)	2.47	1.02 (1.00–1.04)	.020 (.012)	2.74	1.02 (1.00–1.04)
Family interaction	.054 (.066)	0.65	1.06 (.93–1.20)	.031 (.077)	.17	1.03 (.89–1.20)
Positive family support	.010 (.086)	.01	1.01 (.85–1.20)	−.014 (.103)	.02	.99 (.81–1.21)
Negative family support	.222 (.113)	3.85	1.25 (1.00–1.56)	.219 (.120)	3.31	1.24 (.98–1.58)
No. of close friends	−.008 (.015)	.31	.99 (.96–1.02)	−.003 (.014)	.04	1.00 (.97–1.03)
Friend interaction	−.030 (.071)	.18	.97 (0.84–1.12)	.012 (.079)	.02	1.01 (0.87–1.18)
Positive friend support	−.205 (.098)	4.38*	.81 (0.67–0.99)	−.200 (.110)	3.27	.82 (.66–1.02)
Negative friend support	.202 (.140)	2.09	1.22 (0.93–1.61)	.190 (.146)	1.68	1.21 (.91–1.61)
Lung disease (Initial N = 6359; New cases = 245, 3.9%)						
No. of close family	−.012 (.017)	.54	.99 (.96–1.02)	−.001 (.017)	.00	1.00 (.97–1.03)
Family interaction	−.125 (.067)	3.44	.88 (.77–1.01)	−.112 (.079)	2.00	.89 (.77–1.11)
Positive family support	−.174 (.083)	4.43*	.84 (.72–.99)	−.092 (.100)	.84	.91 (.75–1.11)
Negative family support	−.179 (.114)	2.48	1.20 (.96–1.50)	.125 (.119)	1.10	1.13 (.90–1.43)
No. of close friends	−.014 (.015)	.83	.99 (.96–1.02)	−.005 (.014)	.14	1.00 (.97–1.02)
Friend interaction	−.207 (.070)	8.89*	.81 (.71–.93)	−.231 (.078)	8.68*	.79 (.68–.93)
Positive friend support	−.104 (.099)	1.10	.90 (.74–1.09)	.036 (.110)	.10	1.04 (.84–.129)
Negative friend support	.364 (.131)	7.72*	1.44 (1.11–1.86)	.382 (.136)	7.94*	1.47 (1.12–1.91)

*$p < .05$.

Table 4. Predicting onset of chronic conditions using single predictors, and then all predictors within a given domain (family or friends), controlling for age, sex, minority status and years of education.

	Single-predictor models			Domain models		
Predictor	B (s.e.)	Wald	Odds (95% CI)	B (s.e.)	Wald	Odds (95% CI)
Diabetes (Initial N = 5681; New cases = 430, 7.6%)						
No. of close family	.011 (.010)	1.03	1.01 (.99–1.03)	.010 (.011)	.81	1.01 (.99–1.03)
Family interaction	.093 (.051)	3.28	1.10 (.99–1.21)	.076 (.059)	1.64	1.08 (.97–1.20)
Positive family support	.022 (.066)	0.12	1.02 (.90–1.16)	−.022 (.079)	.08	.98 (.84–1.14)
Negative family support	.127 (.087)	2.11	1.14 (.96–1.35)	.106 (.093)	1.29	1.11 (.93–1.34)
No. of close friends	−.008 (.011)	.51	.99 (0.97–1.01)	−.008 (.011)	.57	.99 (.97–1.01)
Friend interaction	.018 (.054)	.11	1.02 (0.92–1.13)	.017 (.060)	0.08	1.02 (.90–1.14)
Positive friend support	−.023 (.076)	.10	.98 (0.84–1.13)	−.027 (.086)	.10	.97 (.82–1.15)
Negative friend support	.257 (.102)	6.41*	1.29 (1.06–1.58)	.273 (.106)	.6.67*	1.31 (1.07–1.62)
Arthritis (Initial N = 2790; New cases = 601, 21.5%)						
No. of close family	−.017 (.013)	1.90	.98 (.96–1.01)	−.018 (.013)	1.84	.98 (.96–1.01)
Family interaction	.014 (.048)	.09	1.02 (0.92–1.11)	.073 (.055)	1.74	1.08 (0.97–1.20)
Positive family support	−.098 (.059)	2.73	0.91 (.81–1.02)	−.077 (.071)	1.17	.93 (.81–1.07)
Negative family support	.178 (.081)	4.84*	1.20 (1.02–1.40)	.164 (.085)	3.72	1.18 (1.00–1.39)
No. of close friends	−.014 (.011)	1.48	.99 (.97–1.01)	−.017 (.012)	1.96	.98 (.96–1.01)
Friend interaction	.002 (.050)	.00	1.00 (.91–1.11)	−.003 (.056)	.00	1.00 (.89–1.11)
Positive friend support	.048 (.072)	.44	1.05 (.91–1.21)	.063 (.079)	.64	1.07 (.91–1.25)
Negative friend support	.077 (.101)	.58	1.08 (.89–1.32)	.056 (.106)	.28	1.06 (.86–1.30)

*$p < .05$.

reduction). Four significant findings were evidenced in the friend domain. Having more friend interactions reduced risk for cancer (12% reduction) and lung disease onset (19% reduction). Positive friend support reduced risk for stroke onset (19% reduction), while negative friend support increased the risk for lung disease (44% increase).

Finally, in line with expectations, the social environment played little role with respect to the onset of either diabetes or arthritis, shown in Table 4. Negative family support increased the risk for arthritis (20% increase), while negative friend support did so for diabetes (29% increase). No other effects reached significance in the single-predictor models.

Predicting onset by social environment domain, including control variables

Next, we sought to examine which social variables may play the most important roles within a given domain, by including all indicators for family or friend relations in a single model. Columns 5–7 of Tables 2–4 present the results of these domain regressions. Only one remained a significant predictor of heart health in these fuller models. Negative friend support increased risk for the onset of high blood pressure (25% increase). The social environment played a somewhat more prominent role with respect to the other mortality risk outcomes, but primarily with respect to the friend domain. Friend interaction frequency remained a negative predictor of cancer (16% reduction) and lung disease onset (21% reduction). In addition, negative friend support remained a risk factor for onset of lung disease (47% increase). The only significant effect in the family domain was that negative family support remained a risk factor for stroke (28% increase). Finally, the domain models also found little to no influence of the social environment on risk for chronic conditions. The only significant effect evidenced was that negative friend support remained a risk factor for diabetes onset (31% increase).

Discussion

The current study sought to extend our knowledge of how the social environment influences physical health outcomes in three important ways. First, we examined how the social environment predicts onset of disease, rather than solely occurrence, demonstrating a number of significant influences. Second, we tested these models across a wide array of important ailments, showing that the social environment may play a greater role with respect to the onset of certain ailments (prominent mortality risks) than others (non-fatal, chronic conditions). Third, we examined four different aspects of the social environment for both the family and friend domains, suggesting that the role of friends may prove more influential than family social indicators. We discuss below our findings with respect to each of these points.

First, a significant advance of the current work is that it demonstrates the capacity for aspects of the social environment to predict whether one is prone to contracting an ailment over the next four years. Indeed, the single-predictor analyses suggested that social environment variables provided significant predictive value for all outcomes studied, except for having a heart condition. Almost all of these effects suggested at least a 10% fluctuation in the odds of onset for an ailment, by virtue of a one-unit increase in the predictor of interest and thus could have very meaningful significance

for the medical community. As such, these findings point to the potential for future research to examine whether these indicators proved significant predictors because they serve a more direct influence on physical health, or if they moderate other risk or resilience factors for the onset of these ailments. Moreover, if one follows the 'direct effect' perspective more closely, it becomes a question of when and how social relationships play an influence, pointing to the need to test some of the proposed pathways mentioned earlier (e.g. that friends could model healthier or unhealthier habits). However, speaking again to the nuanced role of one's social environment (e.g. Banks et al., 2010; Walen & Lachman, 2000), none of the social indicators studied proved universally beneficial. Therefore, again, it proves difficult to neatly summarise the role that one's social life may play on physical health.

Second, while each outcome was predicted by at least one of the social environment indicators examined, it was clear that these variables played different roles across the ailment categories examined. Multiple social indicators appeared to buffer against the onset of poor heart health or prominent mortality risks, a finding that coincides with the past literature nominating the social environment as protective against heart conditions or diseases (e.g. Kawachi et al., 1996; Orth-Gomér et al., 1993; Reed et al., 1983) and mortality risk (Holt-Lunstad et al., 2010). However, fewer effects were evidenced with respect to the onset of chronic conditions, in line with predictions. When exploring these outcomes further, one can see how the social environment could have both positive and negative influences. For instance, diabetes onset will be closely tied to diet and obesity, variables that, as noted above, could be influenced by our friends and family in ways that promote or harm our health (Christakis & Fowler, 2007). Therefore, it may prove difficult to find a consistent role for the social environment on onset of these conditions, although our domain models do suggest the potential for friendships to influence diabetes risk. Instead, for some chronic conditions, it may be more valuable to examine the social environment's role on the perceived pain or severity of the symptoms, as researchers have previously done with respect to arthritis (e.g. Affleck, Pfieffer, Tennen, & Fifield, 1988; Holtzman, Newth, & Delongis, 2004). That said, the current study is an advance in providing an initial investigation into the role of the social environment on onset of these outcomes.

Third, some trends were evidenced when comparing the friend and family variables, as well as comparing different social aspects. For instance, it appears that our friendships may play a more prominent role than our family relationships, which coincides with research suggesting that middle and older adults tend to strive towards having close friendships and may value them more than younger adults (Carstensen, Isaacowitz, & Charles, 1999; Lang, 2001). In addition, we found some support for the prediction that negative social support may play a particularly important role, as it did in previous work (Banks et al., 2010). Specifically, negative social support either from family or friends exacerbated the risk for four of the seven outcomes in the single regression models and three of these in the domain regression models. Alternatively, our markers of network size appeared to play little role, as the number of close family or friends failed to predict risk for onset of any disease. In sum, our results demonstrate that the relationships between one's social environment and health can and will differ based on the aspect of the environment that one assesses.

A few limitations of the current study can serve as directions for future research. First, our study relies upon participants' reports of physical ailments, and as such, it

would be valuable to supplement these findings with additional work using clinical diagnoses. Moreover, further advances can be made if researchers measure specific diagnoses, rather than broader categories such as 'heart condition', although with greater specificity will come the need for larger sample sizes. Second, it would be valuable to compare self-reports of social environments to the reports of family and friends, to attenuate potential self-report biases, as well as examine the possibility that these close others may pick up on elements that the reporter does not. Indeed, the literature on visible vs. invisible support clearly demonstrates the importance of including multiple reporters (Bolger et al., 2000). Third, the reliabilities for our interaction frequency measures were less than ideal, and thus, those results need to be replicated using longer, more reliable assessments. In addition, more work is needed to validate each of the current measures with additional samples and using more objective outcomes. In addition, the current research focused on perceptions of one's available social environment, given the social environmental measures of interest, and thus, future work should better examine the influence of not reporting any available support. Fourth, given that some of these diseases are comorbid in presentation, future work with multiple data waves is needed to examine whether the effect of the social environment on one health ailment may be mediated by its influence on a coinciding ailment. Even in this case, though, the interpretation of the results would here hold insofar that the social environment influences a precursor to the ailment in question. Fifth, again, the exploratory nature of this work warrants mention, leading to the need for future research to replicate these findings with a more focused and directed analytic strategy.

Moreover, by connecting these social variables to risks for onset of major ailments, it sets the foundation for future research that considers the social alongside other prominent predictors of health, in line with previous health psychology models (e.g. Adler & Matthews, 1994). For instance, personality traits are clear predictors of perceived social support (see, Swickert, 2009 for a review), as well as health risks (Hampson, 2012; Smith, 2006), and thus, it would be valuable to examine whether personality affects physical health through influencing one's social life. In addition, rather than employing them solely as control variables, researchers also should examine whether age and sex effects on onset risk can be explained by differences in one's social environment. In both cases, it would be valuable to collect additional waves of data to allow for more precise and sophisticated mediational tests, an opportunity that long-running longitudinal studies like the HRS will provide in the years ahead. As such, we can move closer towards understanding whether living a social life entails living a healthy one.

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