Social Network Characteristics and Cognition in Middle-Aged and Older Adults

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We examined the relationship between social network characteristics and global cognitive status in a community-based sample of 354 adults aged 50+ and with Mini-Mental State Examination (MMSE) scores of 28+ at baseline. Multivariate analyses indicated that interaction in larger social networks related to better maintenance of MMSE scores and reduced odds of decline to population-based lower quartile MMSE scores at follow-up 12 years later. At follow-up, higher levels of interpersonal activity (more frequent contacts in larger social networks) and exposure to emotional support independently related positively to MMSE. The findings suggest that interaction in larger social networks is a marker that portends less cognitive decline, and that distinct associational paths link interpersonal activity and emotional support to cognitive function.

Structural features of social networks such as size and frequency of contact with others can be considered indices of social integration or engagement, whereas functional features refer to specific qualitative support functions (e.g., informational, instrumental, and self-esteem or emotional support; Cohen & Wills, 1985). Those functions, and others that are not, perhaps, expressly supportive, could promote cognitive health in later life. For example, engagement might introduce life experiences that are cognitively complex and stimulating. Cognitive challenge is associated with higher cognitive functioning (Hultsch, Hammer, & Small, 1993), and it might create brain reserve (in the manner of education, e.g., Katzman, 1993) and delay the clinical expression of dementia (Verghese et al., 2003; Wilson et al., 2002). Emotional support might buffer against physiological stress (Seeman, Berkman, Blazer, & Rowe, 1994; Uchino, Holt-Lunstad, Uno, Betancourt, & Garvey, 1999), a possible risk factor for maintenance of cognitive function (e.g., Lupien et al., 1994, 1998; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997).

Other functions of engagement that benefit cognitive health are also possible (see reviews relevant to functions in Barrera, 1986, Cohen, 1988, and Cohen & Wills, 1985), and some of these may relate to cognitive stimulation and emotional support. Examples include the provision of economic and information resources; a sense of purpose, community, and stability; and opportunities to develop self-efficacy or learn coping strategies against stress.

In the present study, we examined longitudinally whether interactions in larger social networks related to maintenance of global cognitive status. Larger networks might provide more opportunity for emotional support or diverse experiences with the potential to produce cognitive stimulation or other functions related to cognitive health. Given the available archival data, we then used cross-sectional analyses to explicitly demonstrate whether social networks might provide both emotional support and the potential for cognitive stimulation as independent benefits to cognition.

We measured network size as the number of persons with whom there was a personal relationship and with whom the respondent kept in touch by phone or visit most of the time. In our cross-sectional analyses, we determined the frequency of those contacts and the amount of emotional support received. Germane to the review that follows, we consider that these measures reflect amounts of exposure to the potential benefits of social networks to cognitive health.

Two recent community-based longitudinal studies relate social network features and functions to incident cognitive declines—that by Bassuk, Glass, and Berkman (1999), and that by Seeman, Lusignolo, Albert, and Berkman (2001). A comparison of these studies illustrates the use of measures that include exposure levels, and their possible value in revealing associations with cognition. In the study by Bassuk and colleagues, an index incorporating the number and/or frequency of network interactions or activities embodies levels of exposure and predicted decline in global mental status over 12 years. In the study by Seeman, Lusignolo, and colleagues, frequency of emotional support represents levels of exposure and predicted decline on a composite of cognitive abilities over 7.5 years. However, a measure of network size with no index of the extent of actual social interaction did not predict decline in Seeman’s study, and a single binary (yes–no) item for emotional support did not predict decline in Bassuk’s study, possibly in part because these measures do not sufficiently differentiate levels of exposure to beneficial network functions. Seeman and colleagues note the issue of interactions and issues related to exposures in the network as well as other differences in the studies such as sample characteristics, and the measures of structural and functional features and cognitive function.

Other community-based longitudinal studies suggest that the level of exposure is potentially important. Wang, Karp, Winblad, and Fratiglioni (2002) found that daily–weekly, but not less frequent, participation in social activities was associated with reduced risk of dementia 6.4 years later. Fratiglioni, Wang, Ericsson, Maytan, and Winblad (2000)
found that frequent satisfying contacts with children and relatives or friends contributed to a formula that related to decreased risk of dementia over about 3 years.

Although measures with exposure levels might be more likely to reveal associations with cognition, significant relationships between binary indices of social engagement and cognition do appear in the literature (e.g., single and living alone as a risk factor for dementia in Fratiglioni et al., 2000). In addition, a cross-sectional analysis using older volunteers living independently in the community (Arbuckle, Gold, Andres, Schwartzman, & Chaikelson, 1992) and a recent case-control study using dementia cases (Seidler, Bernhardt, Nienhaus, & Frolich, 2003) appear to demonstrate independent paths without exposure levels on some measures. For example, Arbuckle and associates found that level of support satisfaction directly linked to general intelligence whereas support network size (absent an exposure index) directly linked to performance on certain memory tasks.

In summary, research suggests that there are at least two functions of social engagement that might be protective of cognitive health in later life—that is, cognitive stimulation and the emotional benefits of support. When significant associations between measures of social ties that imply these or other functions and cognition have not emerged, one reason might be that levels of exposure were not included (e.g., Bassuk et al., 1999, and Seeman, Lusignolo, et al., 2001; see also Fabrigoule et al., 1995, and some of the measures in Fratiglioni et al., 2000).

Given our available data, we performed longitudinal analyses to determine whether interactions in larger networks related to better maintenance of global cognitive status at follow-up about 12 years later. Again, exposures in larger networks might offer more opportunity for emotional support, cognitive stimulation, or other beneficial functions. At follow-up, we used measures that captured (1) more frequent contacts in larger networks—a plausible marker for high exposure to diverse experiences and resources—suggesting functions of engagement that are distinctive of emotional support in the strictest sense (e.g., informational or cognitive stimulation), and (2) levels of exposure to emotional support. We hypothesized that these measures would each positively and uniquely relate to cognition because they probe exposure levels and denote putatively distinct functions. Although we could not ascertain that those functions were causally related to cognition, the demonstration of associational paths would suggest that possibility.

### Methods

#### Sample

We used baseline data at Wave 1 (1981) and follow-up data an average of 12.4 years later during Wave 3 (1993–1996) of the Epidemiologic Catchment Area (ECA) survey at the Baltimore site. Wave 2 was conducted in 1982. The study was approved by the Committee on Human Research at the Bloomberg School of Public Health, Johns Hopkins University. Background and methodology of the ECA survey have been described in detail elsewhere (Eaton & Kessler, 1985). Of 4,238 Wave 1 participants drawn from eastern Baltimore by using probability sampling methods and 3,481 interviews successfully completed, 1,920 were available for reinterview at Wave 3 (73% of survivors; see Eaton et al., 1997). Of 881 participants who met the age and Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) criteria for inclusion in the data analyses (age ≥ 50 and MMSE ≥ 28 at Wave 1), we used 354 in the longitudinal data analyses (420 were lost to follow-up and 107 had missing longitudinal data, including 88 with missing Wave 3 MMSE scores). Of these 354 individuals, about 5.4% had missing cross-sectional data at Wave 3 and were deleted from the analyses for which data were missing (see n values in Table 1). Table 1 compares the criterion age–MMSE assessed and nonassessed participants on the variables used in the study. In summary, of some of the statistically significant comparisons, the assessed participants were younger, had more years of formal education, included a greater percentage of females, and had higher baseline MMSE scores than the nonassessed participants.

### Table 1. Comparison of Assessed and Nonassessed Participants Aged 50+ with MMSE Scores of 28+

<table>
<thead>
<tr>
<th>Variable</th>
<th>Assessed Sample (n = 354)</th>
<th>Nonassessed Sample (n = 527)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMSE W1</td>
<td>29.1 (0.8, 28–30)</td>
<td>28.9 (0.8, 28–30)***</td>
</tr>
<tr>
<td>MMSE W3</td>
<td>26.5 (2.8, 10–30)</td>
<td>—a</td>
</tr>
<tr>
<td>Network sizeb W1</td>
<td>6.8 (2.0, 0–10)</td>
<td>6.2 (2.4, 0–10)***</td>
</tr>
<tr>
<td>Network sizeb W3</td>
<td>6.4 (2.2, 0–10)</td>
<td>—a</td>
</tr>
<tr>
<td>Mean age W1</td>
<td>61.3 (69, 50–81)</td>
<td>66.0 (92, 50–91)***</td>
</tr>
<tr>
<td>Mean years</td>
<td>10.4 (2.8, 0–17)</td>
<td>9.6 (2.8, 0–17)***</td>
</tr>
<tr>
<td>education W1</td>
<td>(n = 526)</td>
<td></td>
</tr>
<tr>
<td>Gender (% female)</td>
<td>68.6</td>
<td>61.7*</td>
</tr>
<tr>
<td>Race (% white)</td>
<td>79.1</td>
<td>83.1</td>
</tr>
<tr>
<td>Physical disabilitya W1</td>
<td>9.3 (0.9, 9–17)</td>
<td>9.6 (1.5, 9–24)***</td>
</tr>
<tr>
<td>Physical disabilitya W3</td>
<td>9.9 (2.0, 9–22)</td>
<td>—f</td>
</tr>
<tr>
<td>Dysphoriaa W1</td>
<td>1.1 (1.2, 0–7)</td>
<td>1.2 (1.4, 0–9)</td>
</tr>
<tr>
<td>Emotional supporta W3</td>
<td>19.2 (5.2, 6–27)</td>
<td>(n = 341)</td>
</tr>
<tr>
<td>Frequency of contactb W3</td>
<td>7.3 (1.9, 1–10)</td>
<td>—k</td>
</tr>
<tr>
<td>Interpersonal activityb W3</td>
<td>13.8 (3.4, 2–20)</td>
<td>(n = 345)</td>
</tr>
<tr>
<td>Cerebrovascular disease-riska W3 (% lifetime)</td>
<td>63.8</td>
<td>—m</td>
</tr>
<tr>
<td>Alcohol use disorderb W3 (% lifetime)</td>
<td>7.3</td>
<td>—</td>
</tr>
</tbody>
</table>

**Notes:** W1 and W3 refer to measures taken at Waves 1 and 3; MMSE = Mini-Mental State Examination.

There were missing data for some assessed participants on frequency of contact and interpersonal activity (present n = 345) and emotional support (present n = 341); see the Methods section. For nonassessed participants, Wave 3 data are not reported and comparisons not attempted because of drop-outs and missing data (see Methods section). In these cases, ns are given in specific notes for the number of scores available.

a n = 19; b higher scores reflect more engagement; c n = 28; d Mann–Whitney tests conducted because of heterogeneity of variance yielded p values of at least this magnitude; e higher scores reflect more disability; f higher scores reflect more dysphoria; g n = 65; h higher scores reflect more support; i n = 28; k n = 30; l n = 28; m n = 28; n from a derived variable at Wave 3.

*χ² p < .05; *** p < .001; **** p < .0005.
Measures of Cognition and Social Networks

We computed cognitive change by using total MMSE scores (0–30). The MMSE item asking which floor of the building was not included at Wave 3, and a scoring accommodation was made. At both waves, of the items to spell world backward and to count serial sevens, the one producing the highest total MMSE score for the participant was used. In addition, at both waves, participants were asked to name two main streets nearby and not location by county. The assessed social network factors differed at the two waves in that frequency of contact and level of emotional support measures were available at Wave 3 but not at Wave 1. At both waves, the introductory instructions stipulated that the social network items referred to persons with whom the respondent had a personal relationship and kept in touch most of the time, and that the researchers were particularly interested in the previous 6 months. Respondents were cautioned to not include persons they did not know well.

Network size Wave 1.—Two survey items asked (1) the number of relatives and family members outside the household and (2) the number of friends and neighbors with whom the respondent kept in touch by phone or visits. The categories of response for each of these items were 0, 1, 2–3, 4–5, 6–10, and 11+; these were given scores of 0–5, and summed (range of possible scores = 0–10).

Network size Wave 3.—This was assessed as at Wave 1.

Frequency of contact Wave 3.—Two additional items at Wave 3 asked the frequency of contact by phone or “getting together,” scored 0–5 (never, less than once/month, about once/month, few times/month, few times/week, and most every day) for each of the two categories of relationships (relatives or family and friends or neighbors; range of possible scores = 0–10).

Emotional support Wave 3.—These items asked how much respondents believed spouse or partners, relatives, and friends cared about them, could rely on them for help for a serious problem, and could relax—be themselves with them. The items were scored 0–3 for increasing support each from spouse or partner, other relatives, and friends, and the items were summed across the three items and the relationship categories. There were 190 participants with missing data for the items relating to emotional support from a spouse or partner, and these items were scored as zero. There were 195 persons in the sample who were not currently married. No currently married persons had missing data for these items unless they were not living with their spouse (one case). The summed scores had a possible range of 0 to 27.

Control Measures

Cognition at baseline.—To the extent that low MMSE scores reflect diminished capacities that result in smaller networks, the central issue of the contribution of larger networks to the maintenance of normal cognitive function could be obscured. Thus, we included only persons who scored 28 or above on the MMSE at baseline in the analyses. According to population-based norms established with data from the five-site ECA survey (Crum, Anthony, Bassett, & Folstein, 1993), 28 is the mean and median score for our sample mean baseline age (as well as our sample mean age plus mean education level).

By comparison with our selection criteria, Seeman, Lusignolo, and colleagues (2001) used the upper tertile of scores on the Short Portable Mental Status Questionnaire (SPMSQ; Pfeiffer, 1975) and delayed recall of a short story as cognitive criteria (they had physical criteria as well) in constituting a subsample of relatively high-functioning persons from a community-based sample aged 70–79 years (SPMSQ criterion = 6 of 9 correct; see Berkman et al., 1993, the MacArthur Studies of Successful Aging). Bassuk and associates (1999) studied noninstitutionalized persons aged 65+ years old and excluded persons with SPMSQ scores < 7 at the beginning of an assessment interval from analyses of decline in that interval.

Control covariates.—The covariate measures in the analyses were cerebrovascular disease or risk (CVD), age, education, depressive symptomatology at testing, race, gender, physical disability, and alcohol use disorder (abuse or dependence). Previous research relates these covariates to the network predictors or cognition; for example, links to cognition obtain with CVD (Logrosino, Kang, & Grodstein, 2004; Prencipe et al., 2003), sociodemographic variables (Graham et al., 1997; O’Connor, Pollitt, Treasure, Brook, & Reiss, 1989), and depressive symptomatology (Dufouil, Fuhrer, Dartigues, & Alperovitch, 1996).

We categorized CVD as being present if the respondent indicated having ever had either high sugar levels or diabetes, stroke, high blood pressure, or heart trouble at Wave 3. We assessed physical disability from nine items extracted from the ECA survey at Waves 1 and 3 relating to basic activities such as bathing, dressing, use of arms to reach, and use of fingers to grasp or handle—each scaled from 1 to 3 such that 1 = performance of the task without difficulty, 2 = performance with difficulty, and 3 = inability to perform. Five participants had one disability item response of “don’t know,” and we assigned these responses a value of 2. We measured dysphoria at both Waves with three items on the General Health Questionnaire (Goldberg & Hillier, 1979) that asked respondents to self-assess feelings of unhappiness and depression, hopelessness, and worthlessness over the past few weeks (scored 0 to 3 for each of the three items indicating absent, as much as usual, more than usual, much more than usual). The internal consistency of these items was $\alpha = .74$ at Wave 1 and $\alpha = .62$ at Wave 3. We derived the presence of lifetime alcohol use disorder from the Diagnostic Interview Schedule—a structured interview administered by trained laypersons (see Robins, Helzer, Ratcliff, & Seyfried, 1981), which was based on Diagnostic and Statistical Manual of Mental Disorders, third edition, revised (DSM-III-R; American Psychiatric Association, 1987) criteria at Wave 3.

There were 226 participants who reported having (had) one or more indicators of CVD. There were 26 participants who indicated a history of stroke, with 20 of these reporting that the most recent occurrence was more than 1 year prior to the Wave 3 interview, 5 reporting 6–12 months prior, and 1 reporting an indeterminate recency. The mean (standard deviation) Wave 3
Table 2. Bivariate and Adjusted Associations With MMSE Change

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Unadjusted Model*</th>
<th>Adjusted Modelb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Robust</td>
<td>Adjusted Modelb</td>
</tr>
<tr>
<td></td>
<td>( B )</td>
<td>( SE )</td>
</tr>
<tr>
<td>Baseline network size</td>
<td>0.13</td>
<td>0.06</td>
</tr>
<tr>
<td>( \Delta )Network size:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>W1–W3</td>
<td>–0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Education</td>
<td>0.31</td>
<td>0.06</td>
</tr>
<tr>
<td>Age</td>
<td>–0.11</td>
<td>0.02</td>
</tr>
<tr>
<td>Race—White</td>
<td>1.55</td>
<td>0.45</td>
</tr>
<tr>
<td>( \Delta )Dysphoria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increase at W3</td>
<td>–0.68</td>
<td>0.55</td>
</tr>
<tr>
<td>Decrease at W3</td>
<td>0.32</td>
<td>0.34</td>
</tr>
<tr>
<td>( \Delta )Physical disability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increase at W3</td>
<td>–1.08</td>
<td>0.49</td>
</tr>
<tr>
<td>Decrease at W3</td>
<td>–0.08</td>
<td>0.75</td>
</tr>
<tr>
<td>Baseline MMSE</td>
<td>0.66</td>
<td>0.18</td>
</tr>
<tr>
<td>Cerebrovascular disease–risk present</td>
<td>0.13</td>
<td>0.31</td>
</tr>
<tr>
<td>Gender—female</td>
<td>–0.28</td>
<td>0.30</td>
</tr>
<tr>
<td>Alcohol use disorder—present</td>
<td>–0.19</td>
<td>0.55</td>
</tr>
</tbody>
</table>

Notes: For categorical variables, the indicator term(s) are given and compared with the reference class, which for change terms is absence of change. A positive coefficient indicates better maintenance of Mini-Mental State Examination (MMSE) scores. W1 and W3 refer to measures taken at Waves 1 and 3; \( \Delta \) = change.

* Predictors are entered with baseline MMSE. \( ^{b} \) Equation for the adjusted model is \( F(13, 340) = 5.92, p < .0005, R^2 = .27. \)
* Less increase–more decrease in interwave network size relates to decreased Wave 3 MMSE.
* \( p < .05; ** p < .01; *** p < .001; **** p < .0005. \)

MMSE score for the 20 cases was 26.7 (2.2) compared with 26.5 (2.8) for those participants without stroke. The mean Wave 3 MMSE for the 5 cases was 24.6 (3.6). There were 26 persons who had alcohol use disorder. At Wave 1, 307 participants were without disability as were 241 at Wave 3. At Wave 1, 159 participants were nondysphoric, as were 189 at Wave 3.

Analyses

We conducted the multivariate analyses by using simultaneous linear or logistic regression. We used Huber–White–sandwich robust standard errors in the linear models because of heteroscedasticity in the residuals. We conducted all analyses by using the STATA 7.0 statistical package (Stata Corporation, College Station, TX).

RESULTS

Baseline Network Size and Cognitive Change

(Longitudinal Analyses)

The mean MMSE at Wave 1 was 29.1 (SD = 8; range = 28–30). At Wave 3 the mean was 26.5 (SD = 2.8; range = 10–30). MMSE change (\( \Delta \)MMSE) was \( M = -2.6 \) (SD = 2.7, with a range of –19 to +2).

We conducted the longitudinal analyses to determine whether interactions in larger baseline networks predicted interwave \( \Delta \)MMSE. We computed change in MMSE as residualized change with baseline MMSE scores entered as a predictor of Wave 3 MMSE scores.

The covariates were \( \Delta \)physical disability and \( \Delta \)dysphoria, MMSE at baseline, lifetime presence of alcohol disorder and CVD status as of Wave 3, age, education level, gender, and race (White or non-White). We entered disability change as increased (\( n = 59 \)) or decreased (\( n = 13 \)), with unchanged as the reference (\( n = 282 \)); dysphoria change was entered as increased (\( n = 43 \)) or decreased (\( n = 61 \)), with unchanged as the reference (\( n = 250 \)). Change indicated that the interwave scores differed by 2+ points (a difference of approximately 1+ SDs). We also included a term for \( \Delta \)network size to assess its association with \( \Delta \)MMSE in the interwave interval.

The interwave decrease in MMSE scores was statistically greater than zero, paired \( t(353) = 18.03, p < .0005. \) The bivariate and fully adjusted associations of the predictors with \( \Delta \)MMSE are given in Table 2. In the adjusted model, a linear effect obtains for baseline network size (\( p = .006; \) effect size = .06). The beta weight for network size indicates that its contribution to the explained variance is relatively small. For example, age accounts for about 3.2 times the variance that network size does. The model also shows that less increase–more decrease in interwave network size relates to decreased Wave 3 MMSE (\( p = .03; \) effect size = –.06), indicating an independent linear “concurrent” association between these two variables.

We used logistic regression with an end-point MMSE cutoff score \( \leq 26 \) (\( n = 128 \)) to further illustrate the relationship between baseline network size and maintenance of MMSE. This is the population-based lower quartile cutoff score for our sample mean education and end-point age (Crum et al., 1993). In the fully adjusted model, for a given network size, the odds of a score \( \leq 26 \) were 84% those for that size \( -1 \) (odds ratio or \( OR = .84, p = .01. \) The odds of a score \( \leq 26 \) for a given interwave change in network size were 115% those for a change with one unit more increase–less decrease (\( OR = 1.15, p = .02; \) complete models for these and subsequent summarized results are available on request from George W. Rebok).

Eighteen Wave 3 MMSE scores could be considered outliers, that is, \( \leq \{Q1 – [1.5 \ (Q3 – Q1)]\}; \) MMSE \( \leq 20. \) A reanalysis of the data in a linear regression excluding these scores showed the reported associations for maintenance of MMSE with baseline network size (\( p = .02; \) network size change (\( p = .02. \) A reanalysis excluding scores \( < 24 \) (the often-used indicator of possible dementia; \( n = 45 \)) revealed a significant positive linear association between baseline network size and maintenance of MMSE (\( p = .04. \)).

Network Measures and Cognitive Function at Wave 3

(Cross-Sectional Analyses)

The longitudinal analyses established that a relationship exists between interactions in larger social networks and maintenance of MMSE. However, we were not able to specify which function(s) of those interactions might benefit cognitive health. We had measures of frequency of contact and levels of emotional support in addition to network size at Wave 3, and we used these data to assess the possibility of distinct independent functions of social networks that contribute to cognitive health. As explained earlier, we hypothesized that more frequent contact in larger networks would reflect...
a function relative to cognition that is independent of emotional support. In order to test this hypothesis, we created the variable of interpersonal activity by summing network size and frequency of contact. Note that, whereas the network size variable was constructed to include only those persons with whom there was contact most of the time, it did not stipulate the frequency of interaction. Considering minimum and mean values of frequency and size across tertiles of interpersonal activity scores, we found that higher tertiles of this variable had more frequent contacts and larger networks.

Table 3 contains the correlations between all the network measures at Wave 3, and those measures with cognition. We conducted a series of linear regression analyses by using the network measures to predict cognition, adjusting for age, gender, education, race, CVD, alcohol use disorder, physical disability, and dysphoria. Tertiles of physical disability and dysphoria each represented the smallest difference in $n$s between the reference classes of no disability and no dysphoria, and two successively higher levels of each.

In an initial series of these analyses, each regression model contained a single network measure entered as a continuous and a categorical variable (the latter, tertiles with the smallest differences in $n$s) to detect linear and nonlinear associations between the network measure and MMSE. For example, the network size models were (1) $\text{MMSE} = \text{network size (continuous)} + \text{covariates}$ and (2) $\text{MMSE} = \text{network size (categorical)} + \text{covariates}$. We report the results for the categorical variables for the top tertile only. In these and subsequent analyses, all of the models were significant ($p < .0005$).

In the initial series, frequency of contact was the only network measure that did not significantly predict MMSE: continuous model $F(11, 333) = 7.57$ and 8.37, frequency of contact $\beta = .10, p > .05$; categorical model $F(12, 332) = 6.71$ and 7.36, frequency of contact $\beta = .07, p > .21$. (Two $p$ values were obtained for each continuous and categorical network measure because there were two alternative criterion tertile arrangements for the physical disability covariate, resulting in somewhat different $p$ values for the network features, depending on which arrangement was entered.) Thus, frequency of social contact by itself might not provide diverse experiences with the potential for cognitive stimulation (e.g., frequent contact could be with one person), or ensure emotional support, or substantively tap other functions of social interaction that relate positively to cognitive health (see Fratiglioni et al., 2000, for a comparable finding for mere frequency of contact).

In contrast, network size was a significant predictor of MMSE scores: continuous model $F(11, 342) = 7.05$ and 6.53, network size $\beta = .15, p = .004$; categorical model $F(12, 341) = 6.29$ and 5.85, network size $\beta = .13$ and .12, $p \leq .03$. So was interpersonal activity: continuous model $F(11, 333) = 8.41$ and 9.09, activity $\beta = .14, p < .006$; categorical model $F(12, 332) = 7.78$ and 8.59, activity $\beta = .15, p < .009$. So too was emotional support: continuous model $F(11, 329) = 7.07$ and 7.83, support $\beta = .15, p < .005$; categorical model $F(12, 328) = 6.35$ and 7.11, support $\beta = .18$ and .19, $p < .004$.

Then, in the next series of analyses, we paired interpersonal activity and network size each with emotional support to assess independent paths with cognition in adjusted models. That is, the paired interpersonal activity and support models were (1)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Network Size + Frequency–Contact</th>
<th>Emotional Support</th>
<th>Wave 3 MMSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency–Contact</td>
<td>.42****</td>
<td>.34****</td>
<td>.20****</td>
</tr>
<tr>
<td>(Interpersonal Activity)</td>
<td>.66****</td>
<td>.16***</td>
<td>.13*</td>
</tr>
<tr>
<td>Network size + frequency–contact</td>
<td>.30***</td>
<td>.18***</td>
<td>.25***</td>
</tr>
</tbody>
</table>

Table 3. Whole Sample Correlations Between Network Measures and Wave 3 MMSE

**Notes:** MMSE = Mini-Mental State Examination.

Any differences in $p$ values when Spearman rho correlations are used are given in specific lettered notes: * $p < .01$; ** $p < .05$; *** $p < .02$; **** $p < .005$.

MMSE = interpersonal activity (continuous) + emotional support (continuous) + covariates, and (2) MMSE = interpersonal activity (categorical) + emotional support (categorical) + covariates. We likewise paired network size and emotional support.

The results of these analyses showed that, when we paired network size and emotional support as continuous measures, they were not both significantly related to MMSE scores—continuous model $F(12, 328) = 7.31$ and 8.07; network size $\beta = .10, p < .08$, and support $\beta = .11, p = .06$. They also were not both significant when paired as categorical variables—categorical model $F(14, 326) = 5.84$ and 6.53; network size $\beta = .09, p < .14$, and support $\beta = .16, p \leq .02$. However, the top tertiles of interpersonal activity and emotional support were both significant when these features were paired as categorical variables: $F(14, 326) = 6.33$ and 7.09; activity $\beta = .13$ and .12, $p < .04$, and support $\beta = .16$ and .17, $p < .01$; effect size $= .26$ for activity and .35 for support, with, in these models, MMSE $SD = 2.6$. When we paired them as continuous variables, activity and support were both significant given one of the disability tertile arrangements—$F(12, 322) = 7.09$; activity $\beta = .11, p = .047$ and support $\beta = .12, p = .04$—but not the other arrangement—$F(12, 322) = 7.75$; activity $\beta = .10, p = .06$ and support $\beta = .12, p = .04$. The results for the categorical variables indicate that more frequent contact in larger networks (most marked in the top tertile of interpersonal activity) and higher levels of emotional support have independent positive associations with cognition, as we hypothesized. We repeated the analyses in a subsample excluding MMSE scores $< 24$, and there was no evidence to support the hypothesis.

**Discussion**

The longitudinal models showed that interactions in larger social networks at Wave 1 related to better maintenance of MMSE at Wave 3 and reduced odds of decline from the population-based median cutoff score (at minimum) to a lower quartile score. This association could reflect benefits of greater exposure to variety or novelty in resources or experiences, increased opportunities for emotional support, or other functions. We did not have the Wave 1 measures necessary to partition out effects associated with emotional support longitudinally. We cannot estimate the extent to which the
The association between baseline size and MMSE change manifested independent of a significant concurrent interwave change in both size and MMSE. This concurrent change might represent counterdirectional or bidirectional effects involving network size and cognition. In this regard, although baseline MMSE scores indicated that the sample was initially high functioning, it is possible that some participants were in decline at Wave 1 if we consider underlying cognitive capacities that did not register as lower MMSE scores at Wave 1 (related issues are addressed in, e.g., Coyle, 2003; Geerlings, Jonker, Bouter, Ader, & Schmand, 1999; and Katzman, 1995), which might affect baseline size. The conservative position is that interaction in a larger network is a marker that portends less decline in global cognitive function as measured by the MMSE.

There were significant independent effects of education, and race, in addition to age, on the maintenance of cognitive function in the whole-sample linear and logistic regression models (see Table 2 for the linear model). Higher levels of education might create brain reserve and then better maintenance of cognitive function (Katzman, 1993). In addition, lower levels of education and non-White race index lower socioeconomic strata, and, as reviewed in Seeman and Crimmins (2001), socioeconomic status is inversely related to a broad spectrum of health outcomes (including cognitive health; see Lynch, Kaplan, & Shema, 1997), with paths of effect that have not been completely identified.

The results of the cross-sectional analyses confirmed our hypothesis that more frequent contacts in larger networks and level of emotional support independently relate positively to cognitive health. The results of Arbuckle and colleagues (1992) and Seidler and colleagues (2003), in their respective ways, also imply the existence of independent paths linking emotional support and other functions of social engagement with cognition.

It is important that our subsample cross-sectional analyses that excluded MMSE scores < 24 did not reveal independent paths. Thus, our partitioned social network functions might reflect causes or effects that distinguish normal from morbid cognitive ability, and not functioning within a normal range. However, the MMSE is itself meant to detect possible clinical cognitive dysfunction and not gradations of normal abilities, so that it would be worthwhile to determine whether our network characteristics relate to these normal gradations. Interestingly, using a battery of cognitive tests in a cross-sectional analysis on their baseline, relatively high-functioning sample, Seeman, Lusignolo, and colleagues (2001) found an independent (of emotional support) positive association with the frequency of social demands or conflict, which they interpret as possibly indexing complex social interactions. Similarly, as reported earlier, the cross-sectional analyses of Arbuckle and associates (1992) support independent paths using measures of general intelligence and memory in persons living independently in the community. More finely tuned cognitive measures in the present study might also yield a greater effect for network size than that obtained in the longitudinal analysis (see Table 2).

Whether more frequent phone or visit contacts in larger networks produce or reflect better cognitive functioning is open to question. If causal of better cognition, the benefit might derive from experiencing more variety, novelty, or challenge from events (e.g., trips to museums or other cities, new recreational or church activities; see, e.g., Murrell, Norris, & Chipley, 1992), or from handling informational or interactional complexities that arise more often or prominently with frequent encounters in larger networks (e.g., scheduling dates and events, reaching for the best way to communicate thoughts, anticipating the responses of others). Situations such as these conceivably stimulate cognition. Significantly decreased risk for the clinical expression of dementia has been associated with higher levels of participation in cognitive activities such as reading and playing board games (Vergheese et al., 2003; Wang et al., 2002; Wilson et al., 2002). We speculate as to the mechanism(s), and direction, of effect for more frequent contact in larger networks, but if it involves higher levels of cognitive stimulation, then our results would be consistent with those longitudinal findings, considering, too, the apparent contribution of MMSE scores < 24 to our results.

As we noted previously, there is a possible link between emotional support, decreased physiological stress (cardiovascular or neuroendocrine response), and cognitive health. In addition, positive effects of support on lifestyle or behaviors (e.g., eating, smoking, exercise, adherence to medication regimens) might contribute to cognitive health (see review pertinent to social support and health behaviors, and biopsychosocial model in Seeman & Crimmins, 2001).

Given the long prodromal period before the expression of clinical symptomatology in Alzheimer’s disease, longitudinal studies with relatively short follow-up intervals, let alone cross-sectional studies, are particularly vulnerable to a directionality confound (see, e.g., Fabrigoule et al., 1995 and Friedland et al., 2001). However, counterdirectional effects do not refute the existence of distinct pathways in our study.

It is noteworthy that the assessed persons were significantly younger, more highly educated, more physically able, and had significantly higher MMSE scores and larger networks at baseline than the nonassessed persons who met our age and MMSE criteria. The generalizability of our results may thus be restricted.

In summary, we found evidence that interaction in larger networks at baseline was positively related to maintenance of global cognitive function about 12 years later. We also showed that more frequent contact in larger networks has a distinctive positive path of association with cognition, apart from an emotional support function. Research that more closely specifies these associations will help to inform strategies for practical preventive interventions with aging populations.

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