

# Social Network Trajectories in Myocardial Infarction Versus Ischemic Stroke

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**Background**—Changes in social networks are rarely examined before and after various diseases because of insufficient data. CHS (The Cardiovascular Health Study) offers an opportunity to compare social network trajectories surrounding well-adjudicated myocardial infarction (MI) or stroke events. We tested the hypothesis that social networks will be stable after MI and decrease after stroke.

**Methods and Results**—We examined trajectories of the Lubben Social Network Scale score (LSNS, range 0–50) before and after vascular events over 11 years. The LSNS assesses engagement in family networks, friends' networks, and social supports. We used a linear mixed model with repeated measures and fixed effects to compare the change in social network score before and after events in 395 people with MI and 382 with ischemic stroke. Over a mean of 12.4 years of follow-up for MI and 11.1 years for stroke, we examined an average of 4 social network scores for each participant. We controlled for sociodemographics, baseline cognitive function, and comorbidities. The participants' mean age was 73.5, 51% were women, and 88% were non-Hispanic white. After MI, the social network trajectory remained stable compared with the baseline trajectory (–0.06 points per year, adjusted  $P=0.2356$ ). After stroke, the social network trajectory declined compared with the baseline trajectory (–0.14 points per year, adjusted  $P=0.0364$ ).

**Conclusions**—Social networks remained stable after MI and declined after stroke. This small and persistent decline after adjustment for potential confounders is notable because it deviates from stable network trajectories found in CHS participants and is specific to stroke. (*J Am Heart Assoc.* 2018;7:e008029. DOI: 10.1161/JAHA.117.008029.)

**Key Words:** epidemiology • myocardial infarction • social environment • social support • stroke

The social impact of cardiovascular diseases is substantial and understudied.<sup>1</sup> Contraction of patients' social network occurs after stroke.<sup>2</sup> However, there are insufficient data to track individual social networks before and after well-adjudicated cardiovascular events to understand the change from baseline trajectories. Comparison across vascular diseases is also lacking. CHS (The Cardiovascular Health

Study), a population-based longitudinal study of coronary heart disease and stroke in adults aged  $\geq 65$  years, offers the opportunity to study vascular events and social network change.

Among all participants in CHS, social networks are stable over time. One study showed there was no individual change over 5 years in family network contact, close friend network contact, or appraisal support.<sup>3</sup> Another study showed that social network scores did not differ significantly between white and black participants.<sup>4</sup> Nonetheless, increased levels of social isolation in CHS participants were associated with higher odds of loneliness.<sup>5</sup> Small social networks in participants were also associated with brain atrophy, including ventricular enlargement and white matter hyperintensities, on magnetic resonance imaging.<sup>6</sup>

In this study, we tested whether social networks would change compared with the stable baseline after vascular events. Cognitive and physical health have been shown to be particularly important to achieving and maintaining better network characteristics.<sup>7</sup> Our a priori hypothesis, therefore, was that stroke more than myocardial infarction (MI) would affect social network trajectories.

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## Clinical Perspective

### What Is New?

- Social networks of participants in the CHS (Cardiovascular Health Study) remained stable after myocardial infarction and declined after ischemic stroke.
- This pattern reflects the unique effects of stroke on socialization, likely driven by language, cognitive, and sociocultural factors.

### What Are the Clinical Implications?

- Social networks should be monitored after stroke, and patients should be counselled about this pattern.
- Social network interventions are needed to help patients maintain their social ties.

## Methods

The data, analytic methods, and study materials will be made available to other researchers for purposes of reproducing the results or replicating the procedure. Because of the sensitive nature of the data collected for this study, requests to access the data set from qualified researchers trained in human subject confidentiality protocols may be sent to CHS at <http://chs-nhlbi.org>.

## Participants

Considered for these analyses were participants who had an MI or ischemic stroke while enrolled in CHS and had at least one follow-up visit after the vascular event (Figure 1). Potential participants were sampled from Medicare eligibility lists in 4 states.<sup>8</sup> Briefly, the cohort is comprised of 5888 participants; 5201 participants were enrolled in 1989–1990, and an additional 687 black participants were enrolled in 1992–1993. Baseline data measured at the entrance of the study were obtained from interviews, clinical examinations, medical record abstraction, and publicly-released Medicare claims data, as previously described.<sup>8</sup> Each study center's institutional review board approved the study, and all participants provided informed, written consent.

## Follow-up

Potential events were identified through regular contact with participants or proxies. Data on incident and recurrent vascular events were collected at local sites and adjudicated by centralized end-point committees for MI and stroke, as detailed previously.<sup>9</sup> Stroke was classified as ischemic, hemorrhagic, or unknown.<sup>10</sup>

## Outcomes and covariates

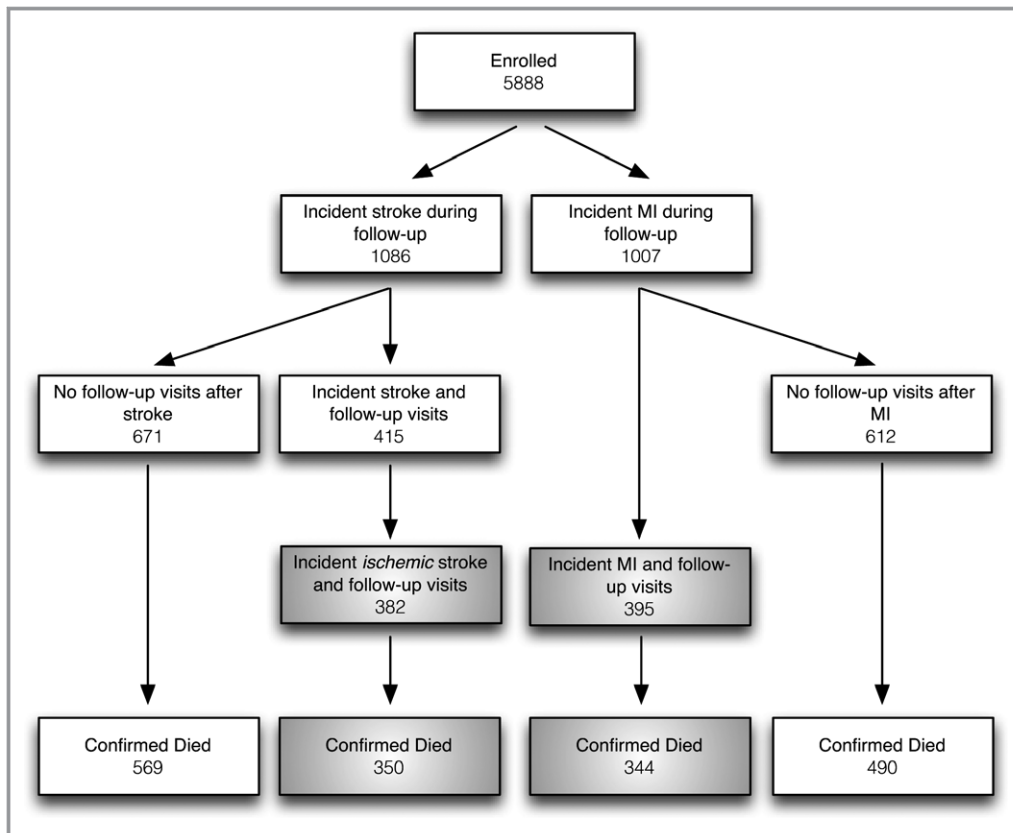
The outcome was the course of the Lubben Social Network Scale (LSNS) score over a maximum of 11 years of repeated measures in participants with MI and those with stroke. The LSNS is a validated composite measure of social networks among elderly populations, although we could not find specific validation in stroke or myocardial infarction patients. Nonetheless, it is used in other population-based studies such as the Honolulu Heart Study<sup>11</sup> and derived from the Berkman-Syme scale, which has been used in stroke patients.<sup>12</sup> The LSNS consists of 3 questions on family networks, 3 questions on friends' networks, and 4 questions on interdependent social supports. The last question includes living with a spouse, which is why marital status was not included as a covariate in our analysis. Scores range from 0 to 50, with higher scores indicating more frequent contact with larger networks.<sup>13</sup> In CHS, the LSNS was collected by a trained interviewer at enrollment and then years 3, 4, 5, 6, and 11.

The primary explanatory variable was time of follow-up assessment in years. Covariates included in the models were demographic variables, vascular risk factors, depression, and cognitive status. Sociodemographics assessed at baseline included self-reported age, sex, race-ethnicity, and level of education. Personal income was defined as total family income before taxes from all sources in the past 12 months and was categorized into <\$12 000, \$12 000 to \$34 999, and ≥\$35 000. Vascular risk factors included hypertension, diabetes mellitus, and cardiac disease, as previously defined.<sup>10</sup> Depression was measured by the Centers for Epidemiologic Studies Depression (CES-D) scale, using a cutoff score of >9 for a high depressive symptom burden. Cognition was measured by the 100-point Modified Mini-Mental State Examination score.

## Statistical Analysis

The distributions of baseline characteristics and follow-up times, frequencies of MI and stroke events during follow-up, and frequencies of LSNS score assessments before and after events were examined.

The goal of the analysis was to determine whether the slope of network scores differed before and after vascular events. Due to correlations among repeated measures of outcomes in the same participant, linear mixed models were used to estimate the time trend or slope of network scores over time. The covariance structure of the model was variance components. The primary covariate was time of follow-up, and the parameter term associated with this covariate signified the slope of network scores. The model included interactions between time-varying postevent status variable and time of



**Figure 1.** Flow diagram of study participants. Grayed boxes are participants included in trajectory analysis. Of note, 175 patients had both events (86 had MI after stroke and 89 had stroke after MI) and were included in both trajectory analyses. Confirmed dead were during the mean follow-up of 12.4 years for MI and 11.1 years for stroke. MI indicates myocardial infarction.

follow-up. This allowed for a different slope before and after vascular events (MI and stroke) as follows:

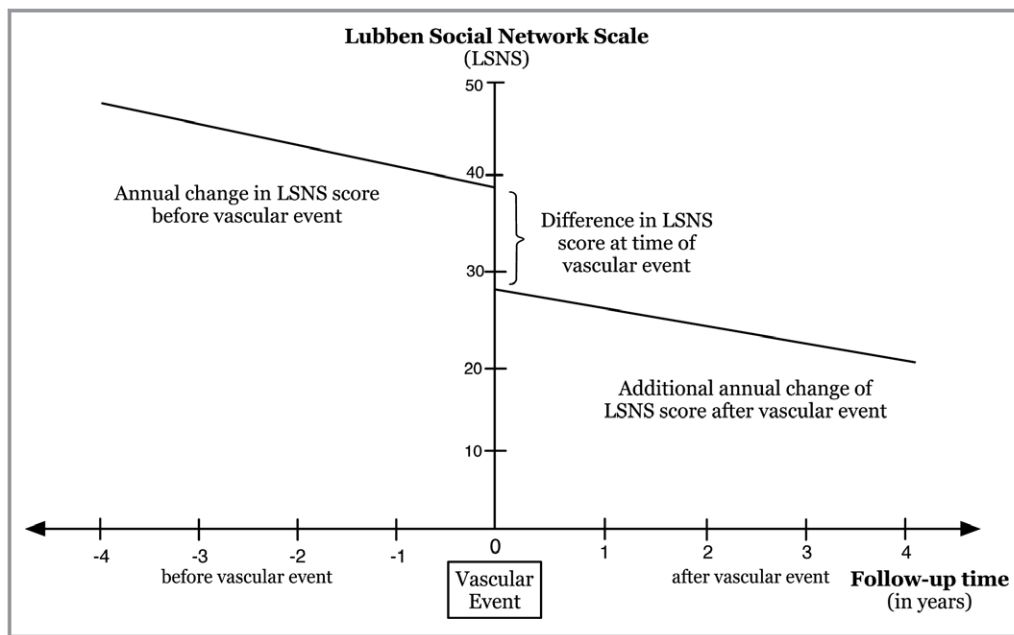
$$Y = \text{intercept} + \beta_1 \times \text{time} + \beta_2 \times \text{postMI} + \beta_3 \times \text{time} \times \text{postMI} + \beta_4 \times \text{postStroke} + \beta_5 \times \text{time} \times \text{postStroke},$$

where “time” is equal to follow-up time, “postMI” equal to 0 if the time of follow-up was before the MI, and 1 if after the MI, and similarly for “postStroke”. “ $\beta_1$ ” estimated annual change in social network score before events, “ $\beta_2$ ” estimated change in network score at the time of MI, “ $\beta_3$ ” estimated additional annual change in network score after MI, “ $\beta_4$ ” estimated change in network score at the time of stroke, and “ $\beta_5$ ” estimated additional annual change in network score after stroke. For “ $\beta_2$ ” and “ $\beta_4$ ”, the change in network score was not at the exact time of the event due to survey timing but measured within a mean of 9 months after MI and 7 months after stroke. The *P*-values are linked to the betas. Therefore, the *P*-value for “ $\beta_1$ ” assesses whether the slope before the event is different than 0; *P*-value for “ $\beta_2$ ” and “ $\beta_4$ ” compares the mean scores before and after the vascular event, and *P*-value for “ $\beta_3$ ” and “ $\beta_5$ ” assesses whether the annual change

of scores after vascular events are different than the baseline trajectory (see Figure 2 for conceptual depiction).

In model building, we sequentially added groups of potential confounders, including demographic variables, vascular risk factors, and cognitive and mood factors. Each of these blocks were tested one-by-one to avoid over-fitting. Time, age, and mini-mental status exam scores were added as continuous variables. Sex, high school education, income, history of coronary artery disease, and depression were added as categorical variables. These potential confounders were determined based on prior literature describing factors that may influence network change.<sup>7,14,15</sup>

Additional analyses were completed to examine mechanisms of the social network trajectory change, selection bias, and handling of patients who had both MI and stroke. To examine whether depression and cognition were mediators between stroke and declining social networks, we tested for interactions among (1) depression, stroke, and time and (2) cognition, stroke, and time. To examine selection bias or informative censoring, we compared network scores and demographics of participants included in the analysis versus those who did not have follow-up.



**Figure 2.** Conceptual depiction of the course of social network scores before and after vascular event. In the analysis, the “Annual change in LSNS score before vascular event”, or baseline trajectory, is compared with 0. The “Difference in LSNS score at time of vascular event” compares the mean scores before and after the vascular event. The “Additional annual change of LSNS score after vascular event” is compared with the baseline trajectory. LSNS indicates Lubben Social Network Scale.

Finally, we compared trajectories after single events of MI and stroke by categorizing patients based on the first event to occur, and not using any data after the second event occurs.

## Results

Among the 5888 participants, 395 had a MI during 12.4 years (range 1.4–21.5), and 382 had a stroke during a mean follow-up of 11.1 years (range 1.2–21.5). Of patients with MI, the mean age was 73, 44% were women, 89% were non-Hispanic white, 70% completed high school. Of patients with stroke, the mean age was 74, 58% were women, 87% were non-Hispanic white, and 68% completed high school (Table 1). During the observation period, 175 participants had both MI and stroke (86 had MI after stroke and 89 had stroke after MI), and their data contributed to both trajectory analyses. In comparing those included versus excluded in the analysis, we found no difference in baseline network score (32.29, SD=7.57 versus 32.86, SD=7.32;  $P=0.1526$ ). However, participants included in the analysis were older (73.7, SD=5.6 versus 72.6, SD=5.3;  $P=0.0001$ ), more likely to be men (50% versus 41%,  $P=0.0002$ ), and more likely to be white race (88% versus 83%,  $P=0.0117$ ) (Table 2). During the observation period, the mean number of LSNS assessments was 4 for both groups of participants. Per protocol, the measurements occurred at enrollment and then years 3,

4, 5, 6, and 11. More assessments occurred before the vascular event (means of 2.7 for MI and 2.5 for stroke) than after the event (means of 1.3 for MI and 1.2 for stroke). There was no evidence of non-linearity in the time trend. Although each participant had few measurements, the variation in the time of follow-up enabled reliable slope estimates.

We compared the trajectory of network scores before and after vascular events (Table 3, Figure 3). In the adjusted model, the annual change in the network scores before vascular events, or baseline trajectory, did not differ from 0 ( $-0.01$  points per year,  $P=0.3872$ ). At the time of MI and stroke, the mean network score before the vascular event was not significantly different than after the vascular event (MI:  $-0.10$ ,  $P=0.7762$ ; stroke:  $-0.64$ ,  $P=0.1192$ ). After MI, the social network trajectory remained stable compared with the baseline trajectory ( $-0.06$  points per year,  $P=0.2356$ ). After stroke, the social network trajectory declined compared with the baseline trajectory ( $-0.14$  points per year,  $P=0.0364$ ).

Additional analyses were completed to examine mechanisms of the social network trajectory change, selection bias, and handling of patients who had both MI and stroke. To examine whether depression and cognition were mediators between stroke and declining social networks, we completed an interaction analysis. Interactions between depression, stroke, and time ( $\beta -0.09$ ; 95% confidence interval,  $-0.36$ –

**Table 1.** Baseline Characteristics of Participants, According to Adjudicated Event

Characteristic	MI (n=395)	Stroke (n=382)	P Value*
Age, mean (SD), y <sup>†</sup>	73 (5)	74 (6)	0.0288
Female, n (%)	174 (44)	220 (58)	0.0002
Race, n (%)			0.4047
Non-Hispanic white	351 (89)	332 (87)	
Non-white	44 (11)	50 (13)	
At least high school education, n (%)	275 (70)	259 (68)	0.5845
Yearly income, n (%)			0.0930
<\$12 000	95 (25)	118 (33)	
\$12 000 to \$34 999	194 (52)	175 (48)	
≥\$35 000	85 (23)	70 (19)	
History of coronary heart disease, n (%)	76 (19)	106 (28)	0.0051
Depressed (CES-D score >9), n (%)	58 (15)	55 (15)	0.8992
Baseline modified Mini-Mental State Examination score, mean (SD)	87 (13)	82 (18)	0.0001

CES-D indicates Centers for Epidemiologic Studies Depression Scale.

\*Of note, 175 patients had both MI and stroke, and their data were included in both groups. Therefore, differences between groups may be underestimated.

<sup>†</sup>Age at start of the study.

0.17,  $P=0.4945$ ) were not significant. Interactions between cognition, stroke, and time ( $\beta$  0.03; 95% confidence interval,  $-0.17$ – $0.23$ ,  $P=0.7900$ ) were not significant, suggesting neither depression nor cognition were explanations of the social network decline after stroke.

To examine individuals who had both MI and stroke, we categorized all participants based on the first event and censored any data after the second event. For example, for a patient with first MI and then stroke, we analyzed all network scores before and after the MI up to the time of stroke. The slope after MI compared with baseline was  $-0.07$  every year (confidence interval,  $-0.18$ – $0.04$ ,  $P=0.1864$ ), and the slope after stroke compared with baseline was  $-0.15$  (confidence interval,  $-0.30$ – $0.00$ ,  $P=0.0529$ ). The results were similar to our main analysis, but did not reach significance.

## Discussion

The results support the hypothesis that social networks remain stable after MI, but decrease after stroke. Compared with a stable baseline network score before the vascular event, participants with stroke experienced a significant network score decline of  $-0.14$  points for every year after stroke. This small and persistent decline after adjustment for potential confounders is notable because it deviates from stable network trajectories found in CHS participants, and is specific to stroke. We did not discover a mechanism in this study; depression and cognition scores did not interact with the diagnosis of stroke and time to explain the social network decline. Speculated mechanisms supported by the literature are described below.

**Table 2.** Comparison of Baseline Characteristics of People Included and Excluded in Analysis

Characteristic	Included in Analysis (n=661)*	Excluded Due to No Follow-Up After Event (n=1040) <sup>†</sup>	P Value
LSNS at baseline, mean (SD)	32.29 (7.57)	32.86 (7.32)	0.1526
Age, mean (SD)	73.66 (5.58)	72.63 (5.28)	0.0001
Male, n (%)	333 (50.38)	428 (41.15)	0.0002
White race (vs non-white), n (%)	580 (87.75)	866 (83.27)	0.0117
High school education, n (%)	457 (69.14)	726 (69.81)	0.7698
Depressed (CES-D score >9), n (%)	92 (13.98)	132 (12.73)	0.4580

CES-D indicates Centers for Epidemiological Studies Depression Scale; LSNS, Lubben Social Network Score.

\*For Lubben Social Network Score, sample size was n=596.

<sup>†</sup>For Lubben Social Network Score, sample size was n=892.



**Table 3.** Unadjusted and Adjusted Models of Social Network Trajectories Before and After Vascular Disease

Variable	Unadjusted Model			Adjusted Model		
	Change in LSNS Score	95% CI	P Value	Change in LSNS Score	95% CI	P Value*
Annual change of LSNS score before stroke or MI	−0.001	−0.033, 0.031	0.9429	−0.01	−0.05, 0.03	0.3872
Difference in LSNS score at time of MI	0.09	−0.50, 0.68	0.7561	−0.10	−0.79, 0.59	0.7762
Difference in LSNS score at time of stroke	−0.94	−1.70, −0.18	0.0169	−0.64	−1.44, 0.16	0.1192
Additional annual change of LSNS score after MI	−0.07	−0.17, 0.03	0.1349	−0.06	−0.16, 0.04	0.2356
Additional annual change of LSNS score after stroke	−0.14	−0.26, −0.02	0.0332	−0.14	−0.27, −0.01	0.0364
Covariates						
Age, y				−0.22	−0.25, −0.19	<0.0001
Male				−0.44	−0.80, −0.08	0.0167
Non-white race				−0.57	−1.08, −0.06	0.0287
High school education				−0.92	−1.34, −0.50	<0.0001
Income in past 12 mo \$12 000 to \$34 999 <sup>†</sup>				2.08	1.63, 2.53	<0.0001
Income in past 12 mo >\$35 000 <sup>†</sup>				3.21	2.66, 3.76	<0.0001
History of coronary artery disease				0.86	0.36, 1.36	0.0008
Depression				−2.61	−3.12, −2.10	<0.0001
Baseline modified Mini-Mental State Examination score				0.04	0.02, 0.06	0.0001

CI indicates confidence interval; LSNS, Lubben Social Network Scale; MI, myocardial infarction.

\*Please see the Methods section for details on P-value derivation in the linear mixed models.

<sup>†</sup>Reference category is <\$12 000.

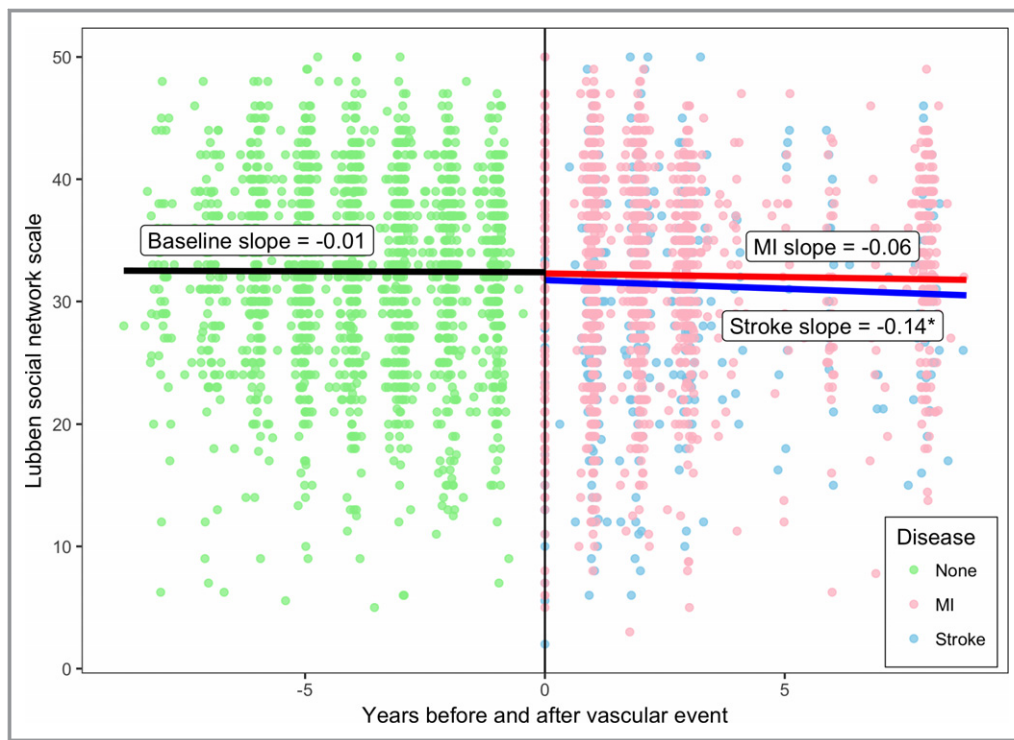
One possibility is that neurological injury, unlike myocardial damage, leads to reduced network group size due to cognitive constraints. This idea is supported by the social brain hypothesis and neuroimaging findings of CHS participants. The social brain hypothesis is an evolutionary theory which posits that a primate's social network group size is proportional to neocortical volume.<sup>16</sup> Accordingly, the human neocortex allows a person to have coherent personal relationships with ≈150 people. This number is reduced after neurological injury where patients experience difficulty to maintain social relationships.<sup>7,17</sup> Neuroimaging findings in a subset of CHS participants support the notion in a cross-sectional analysis by showing people with small social networks were more likely to have severe ventricular enlargement and white matter hyperintensities.<sup>6</sup> This preliminary finding requires further longitudinal analysis in CHS participants to better understand the relationship of brain structure and network structure.

A second possibility is the unique effects of certain stroke syndromes on communication and social life. Studies show that contact with friends and involvement in social activities are reduced after stroke due to communication and physical disabilities, fatigue, relocation, depression, and stigma.<sup>2</sup> Therefore, social networks become more family-oriented, although family unit relations may also become strained.<sup>2</sup> The network transformation does not appear to be as dramatic for MI survivors. Detrimental effects of living alone or reduced

perceived social support on outcomes after MI have been reported.<sup>18</sup> However, dynamic shifts in social network structure and composition are not reported in the cardiovascular literature, and psychosocial interventions after MI have failed to improve outcomes.<sup>19</sup> Therefore, stroke may have a particular effect on sociality that makes patients more vulnerable to social network shrinkage, isolation, and loneliness.

These findings have implications for optimization of recovery after vascular events. In both MI and stroke, social isolation is a well-established independent risk factor for poor recovery.<sup>18,20</sup> The reasons are both biological effects, such as increased inflammation and decreased neurogenesis,<sup>21,22</sup> and pragmatic effects in terms of the necessity of instrumental and emotional support to engage with health care and to persevere. Our results suggest that existing social isolation before vascular events may be worsened by induced network constriction after vascular events, particularly for stroke. In other words, a vicious cycle of disease-causing network constriction may result, which if not rectified, leads to socially mediated poor outcomes. Identifying and intervening on the process of network constriction during recovery offers one avenue of optimizing functional and patient-oriented outcomes.<sup>1</sup>

Our study has limitations. Patients with consecutive MI and stroke were included in both groups in the model. We did this because postdisease trajectories occasionally include a second vascular event and censoring such data would introduce a



**Figure 3.** Social network trajectories in MI vs stroke. After MI, social network trajectories remained stable compared with baseline trajectories ( $-0.06$  points per year, adjusted  $P=0.2356$ ). After stroke, social network trajectories declined compared with baseline trajectories ( $-0.14$  points per year, adjusted  $P=0.0364$ ).  $*P<0.05$ . MI indicates myocardial infarction.

selection bias. Although the CHS is a large prospective database, it includes participants from only 4 US counties enrolled in 1989 to 1993. The findings may not be generalizable to other populations. Nonetheless, social network size and patterns have been shown to be stable in the last 30 years even with the use of social media.<sup>23</sup> Differences in demographics (age, sex, and race) of those included in the analysis versus those excluded suggest a possible role of non-random censoring. However, key mechanistic variables (eg, baseline LSNS, education level, and depression) were not different across groups. The LSNS is not a granular measure of social network structure or composition, and it was not recorded at regular time intervals for every participant. Therefore, the network scale offers a coarse signal with variability among participants. Changes in covariates could not be included because covariates were assessed at entrance into the study only. Finally, the social network decline may be underestimated due to death and lack of follow-up measurements of sicker patients in both groups. We would argue that this underestimation biases towards the null hypothesis because severe stroke more than severe MI likely has a greater effect on social networks and follow-up rates.

Strengths of the study include the design of repeated measures of network scores before and after well-adjudicated vascular events. CHS is also one of the largest samples of

community-dwelling older adults in a population with random selection of participants to approximate the US population distribution by age and sex. Lastly, the comparison between MI and stroke, illnesses that affect comparable populations, is useful to understand the specific effects of vascular diseases on network change.

In a large, prospectively followed cohort of community-dwelling older adults, we found that social networks remained stable after MI and declined after stroke. Future work on mechanisms of this pattern are needed, including studies of differences between people of varying ages and race, the relationship of neuroimaging changes and social networks, and factors that encourage resilience. These findings show the natural history of social network trajectories in vascular diseases, which may be used as a baseline for future studies aiming to improve social networks.

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## Disclosures

None.

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