Social Relationships and Physiological Determinants of Longevity across Human Life Span

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Abstract

This study addresses the question of how the biophysiological mechanisms underlying the health impacts of social relationships unfold over the human life span. Drawing on data from four nationally representative longitudinal samples of the U.S. population, this research examined the associations of social integration, social support and strain with biomarkers of physiological stress response across immune, cardiovascular, and metabolic systems from adolescence and young adulthood (Add Health) to middle (MIDUS) and late adulthood (NSHAP and HRS). Social integration predicted lower risks of physiological dysregulation in a dose-response manner across the life course samples. Low social support as well as high social strain moderately increased the risks in some stages. In addition to the important roles of social connections in the emergence of aging related disease throughout the life course, the study also reveals physiological vulnerabilities to social stress that may be specific to relationship stressors and life course stages.

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Extended Abstract

Introduction

A defining characteristic of human society is that lives are intertwined through social relationships. Full social participation is considered a fundamental human need which can be conceptualized by examining engagement in social networks and receipt of social support. Lack of social connections is shown to be important contributors to inequality in health and survival chances. The questions remain as to why social relationships have health impacts, when these impacts may emerge, and how long they may last. The social relationship gradient in longevity can be understood as arising from a biological process in which social experiences get under the skin by altering physiological stress response. Deficits in social relationships such as social isolation or low social support can lead to chronic activations of immune, neuroendocrine, and metabolic systems that lie in the pathways leading to cardiovascular, malignant, and other common aging related diseases. Furthermore, social experiences may be biologically embedded early on in life. Chronic stress associated with relationship deficits is created by continuous exposures to chains of risk in the process of accumulation over the life course. At the same time, the emergence of chronic diseases usually takes many decades due to the long latency after the initial risk exposures. Therefore, extensively longitudinal data and analyses are imperative to the understanding of how the connection of social relationships and healthy longevity unfolds over the human life span.

This study synthesizes previous research on social relationship and health and carries out the first longitudinal model of how social relationships matter for physiological health across the life course. We make three unique contributions. First, drawing on data from an array of nationally representative longitudinal samples of the U.S. population, this research implements an innovative life course design that begins at the earliest developmental stage (adolescence) in which the physiological consequences of key social relationship patterns begin to manifest and traces all subsequent life stages (early, middle, and late adulthood) to depict the life-long process of stress response cascades such relationship pattern

initiate. It thus offers an unprecedented fuller view of the evolvement of the biophysiological mechanisms underlying the social gradient in health across the human life span than any previous studies of a particular sample or single life stage alone. Second, this study used refined measurements of social relationships that encompass both structural-quantitative and functional-qualitative dimensions of social interaction across all datasets where information on social networks and social support were collected. It examined both the social network size and the perceived social support or strain to capture the degree of social integration and social resources that provide resilience to moderate stressors impact on the body. Third, the study examined multiple objectively measured biomarkers that indicate functioning of diverse bodily systems including inflammation, cardiovascular function, and energy metabolism that are key physiological determinants underlying common diseases of aging and longevity.

Data and Methods

We used longitudinal data from the following four nationally representative NIH studies that collectively cover all stages of the life course. Data for the *adolescence* and *young adulthood* come from 10,901 participants in the National Longitudinal Study of Adolescent Health (Add Health) aged 12-17 at Wave I (1994-95) and followed up at aged 24-32 in Wave IV (2008-09). Data for *mid adulthood* come from 939 respondents aged 25-74 in the National Survey of the Midlife Development in the United States (MIDUS) surveyed at Wave I (1995-96) and followed up at Wave II (2004-09). Data for *late adulthood* come from two studies, including 1,700 participants aged 57-85 in the National Social Life, Health, and Aging Project (NSHAP) at Wave I (2005-06) and followed up at Wave II (2010) and 4,323 participants aged 50 and older in the Health and Retirement Study (HRS) at the baseline year of 1992 and followed up every two years to 2008.

We included four biomarker measurements that were collected in all the above studies at the follow-up surveys. The immune function and inflammation was assessed by the *C-reactive protein* (hsCRP). The cardiovascular function was assessed by the *diastolic and systolic blood pressure* (BP).

The metabolic function was assessed by *waist circumference* indicating body composition and also by the *Body Mass Index* (BMI) based on measured weight and height. We employed both the continuous measures (endophenotypes) that directly indicate physiological functioning in response to stress and categorical measures based on clinical cut points that indicate the corresponding disease outcomes such as *chronic inflammation*, *hypertension*, *abdominal obesity*, and *obesity*.

We used social relationship measures available at the baseline in all studies and also at the follow-ups when available (for MIDUS and HRS). We measured *social integration* by social network size that summarized the number of social ties across relationship domains including marital status, frequency of interaction with friends and relatives, religious attendance, membership in social organizations, and volunteering. There are slight variations in the specific items included in each study sample, with the social integration index values ranging from 0 – 6. We measured *social support* as perceived by respondents from family, friends, school (for Add Health), and spouse, as well as the summary measures of total support. We also measured *social strain* from the corresponding relationships and constructed a similar total strain scale. Support variables were not available for the HRS; and strain variables were not available for the Add Health or HRS. We assessed both continuous and categorical variables in regression analyses to account for linear and threshold effects.

We examined the associations between social relationship characteristics assessed at prior points in time and continuous outcomes of biomarkers at the follow-up surveys using the OLS regression models and categorical outcomes of biomarkers using generalized linear models, including ordinal logit models for inflammation and logistic models for the others. For each outcome variable, we estimated both the basic models adjusting for age, sex, and race and the full models adjusting for all the other covariates including socioeconomic status, health behaviors, chronic conditions, and medications. All analyses were conducted in Stata 12 and adjusted for survey design effects and nonresponse using sampling weights.

Results

Social integration was found to predict better physiological functioning and lower risks of physiological dysregulation in a linear or dose-response fashion across all life stage study samples. Individuals with a higher number of social ties showed significantly lower values of log(CRP), systolic BP, waist circumference, and BMI. More socially integrated individuals also had decreased odds of elevated inflammation, hypertension, central obesity, and overall obesity. Adjusting for social, behavioral, and other health related covariates reduced some but not all of these associations. We found age variation in these associations that also vary by biomarkers. The protective effects of social integration were particularly large at young adulthood against inflammation and at adolescence against central and overall obesity, whereas the benefits of social integration were particularly prominent at old age against high systolic BP and hypertension. We did not find any significant results regarding the associations for middle age.

While the associations of biomarkers with objective and quantitative dimension of social relationships are consistently strong, those with perceived social support and strain are more modest, nonlinear, and varied by relationship domains. Low social support (from friends, spouse, and in total) was associated with higher BMI values and increased odds of abdominal and overall obesity in middle age. And high social strain (from friends and in total) was predictive of higher log(CRP) values and increased odds of inflammation in middle age. High social strain (from family, friends, spouse, and in total) also predicted larger waist circumferences and increased odds of central and overall obesity in old age. Adjusting for the covariates diminished most of these associations.

These results shed new light on the biological mechanisms and life course processes by which social relationships impact health. They suggest physiological vulnerabilities to social stress that may be specific to relationship stressors and life course stages. Disrupting the social relation and physiological dysregulation connection can directly arrest early progression toward chronic diseases and delay the disease onset or lessen the disease burden in late life. The findings, therefore, provide a strong scientific basis for effective prevention and intervention that will lead to further improvement in life expectancy.