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Kindness and Cellular Aging:
A Pre-Registered Experiment Testing the Effects of Prosocial Behavior on Telomere Length and Well-Being

A Dissertation submitted in partial satisfaction
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by

Megan Fritz

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Dissertation Committee:
Dr. Sonja Lyubomirsky, Chairperson
Dr. David Funder
Dr. Kate Sweeny

The Dissertation of Megan Fritz is approved:

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Committee Chairperson

University of California, Riverside

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ABSTRACT OF THE DISSERTATION

Kindness and Cellular Aging:
A Pre-Registered Experiment Testing the Effects of Prosocial Behavior
on Telomere Length and Well-Being

by

Megan Fritz

Doctor of Philosophy, Graduate Program in Psychology
University of California, Riverside, June 2019
Dr. Sonja Lyubomirsky, Chairperson

Empirical research suggests that prosocial behavior (i.e., doing acts of kindness for others) leads to improvements in psychological well-being and physical health, including mortality. However, little work has focused on identifying the underlying biological mechanisms that may mediate the relationship between prosociality and physical health. In this pre-registered experiment, I tested whether a 4-week kindness intervention that has been demonstrated in previous work to elicit changes in pro-inflammatory gene expression (Nelson-Coffey, Fritz, Lyubomirsky, & Cole, 2017) could shift a related psychobiomarker of health – namely, leukocyte telomere length – as well as improve psychological health. Across a diverse community sample ($N = 230$), participants who performed three kind acts for other people each week for 4 weeks did not demonstrate hypothesized shifts in telomere length (i.e., reduced rates of shortening), nor in well-being and related constructs, relative to those who tracked daily activities (i.e., controls). Exploratory analyses revealed that, relative to controls, participants who performed three self-focused acts (i.e., kindness to the self) also did not demonstrate shifts in telomere

length, but did report greater declines in loneliness across the intervention period, relative to controls, although this effect attenuated at the 2-week follow-up. However, participants who performed kind acts for others showed reductions in loneliness through the 2-week follow up, relative to controls. In conclusion, the salubrious effects of prosocial behavior may not likely to be due to the inhibition of cellular aging. However, kindness holds promise as a plausible intervention to alleviate the growing public health crisis of loneliness.

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> Goodness…is far more difficult to explain than evil, and just as complicated.
> – Margaret Atwood, *The Blind Assassin*

Kindness is vital to the survival of humankind. Prosocial behavior (i.e., helping or sharing to benefit another at some cost to the self) has been studied through the lens of evolutionary psychology (e.g., kin selection, reciprocal altruism), moral philosophy (e.g., generosity, religiosity), affective science and neuroscience (e.g., compassion, empathy), human development (e.g., prosocial development), and social psychology and behavioral economics (e.g., cooperative or helping behavior). Other-oriented prosocial behavior confers benefits not just for the target of kindness, but also for the actor. Correlational studies suggest that prosocial behavior is associated with such positive outcomes as well-being, romantic relationship formation, self-rated physical health, and mortality (Anderson et al., 2014; Gruenwald, Liao, & Seeman, 2012; Meier & Stutzer, 2008; Musick, Herzog, & House, 1999; Stavrova & Ehlebracht, 2015).

Kindness and Well-Being

Experimental studies suggest that prosocial behavior causally produces a variety of downstream benefits. Prosocial spending (e.g., donating money) robustly improves well-being, with some researchers proposing that this effect is culturally universal (Aknin et al., 2013). Meta-analyses suggest that prosociality-based interventions confer well-being benefits for the actor, with small-to-medium effects (Cohen's $d = 0.28$; Curry, Rowland, Van Lissa, Zlotowitz, McAlaney, & Whitehouse, 2018). Specifically, interventions employing acts-of-kindness paradigms have been shown to boost

psychological flourishing and well-being (Nelson et al., 2015; Nelson, Layous, Cole, & Lyubomirsky, 2016) and increase peer acceptance (Layous, Nelson, Oberle, Schonert-Reichl, & Lyubomirsky, 2012).

Most research testing kindness interventions has focused on their capacity to boost well-being, with less attention to whether engaging in prosocial behavior may ameliorate psychological distress. Yet, doing kindness is also expected to improve such outcomes as loneliness and stress. Volunteering has been associated with reductions in social isolation (McGarvey, Jochum, Davies, Dobbs, & Hornung, 2019), and 2 hours of volunteering per week may offset the increases in loneliness among recently widowed older adults (Carr, Kail, Matz-Costa, & Shavit, 2018). Prior research suggests that experimentally inducing prosocial behavior may lead to greater feelings of social connection (e.g. Fritz et al., 2019) and psychological flourishing, which includes social well-being (e.g., Nelson et al., 2016). Doing acts of kindness for others may thereby promote warmth and closeness, such that one feels embedded in a web of social connections, and thus less isolated and lonely. Additionally, individuals who provide support to friends and coworkers are more likely to receive support, as others around them reciprocate those efforts (Chancellor, Margolis, Jacobs Bao, & Lyubomirsky, 2018; Uehara, 1995). Thus, providing help *to* others may serve to garner help *from* others in return, potentially reducing stress, as tasks may be completed more easily, and alleviating loneliness, as individuals realize they are cared about and valued.

Kindness and Health

In addition to its potential to boost psychological health, prosocial behavior has been associated with better physical health and lower morbidity and mortality. Much of this work has focused on support-giving and volunteering behavior. Regular volunteerism has been related to a number of positive physical health outcomes, including reduced cardiovascular disease risk and mortality (Brown et al., 2009; Burr, Han, & Tavares, 2015; Crick, 1996; Konrath, Fuhrel-Forbis, Lou, & Brown, 2012; Layous et al., 2012; Penner, Dovidio, Piliavin, & Schroeder, 2005). Social support-giving has similarly been linked to improvements in physical health outcomes. Providing emotional and instrumental support to others predicts reduced mortality in older adults across a 5-year period (Brown, Nesse, Vinkour, & Smith, 2003). Additionally, support-giving was associated with lower blood pressure across a 24-hour period (Piferi & Lawler, 2006). However, this work is primarily correlational in nature, limiting generalizability and causal inference.

One potential pathway through which prosocial behavior could impact health is through the mammalian caregiving system, a neurobiological system composed of brain regions involved in maternal behavior and reward (e.g., ventral striatum, septal area, amygdala) and neurohormones associated with caregiving (e.g., oxytocin). The caregiving system has been associated with reduced physiological threat responding and has been shown to be activated during support-giving tasks (Inagaki & Eisenberger, 2012).

Experimental work directly testing the effects of induced prosocial behavior interventions on biological health has been limited. One exception, a study from our laboratory, found that participants randomly assigned to perform prosocial acts for others over a 4-week period showed significant reductions in proinflammatory genetic profiles (Nelson-Coffey, Fritz, Lyubomirsky, & Cole, 2017). Other correlational research supports the idea that prosocial behavior may impact biological indicators of health. For example, across a 6-week intervention, although practicing gratitude did not predict decreases in proinflammatory cytokines, increases in support-giving (i.e., emotional and instrumental support) did (Moieni et al., 2018). Taken together, these findings suggest that helping behavior may positively impact human health through biological and genomic pathways.

Telomeres: A Literature Review

Telomere biology. One promising area for further research is investigating the relationship between prosocial behavior and the length of human telomeres. Telomeres are DNA-protein structures that serve as protective end caps for eukaryotic chromosomes; they function to maintain chromosomal stability across cell replications (Blackburn, 2000; Blackburn, Epel, & Lin, 2015). Telomeres progressively shorten with each somatic cell replication; thus, over an individual's lifespan, telomere length normatively shortens, until cell senescence (i.e., cell death) begins (Blackburn et al., 2015). Importantly, work suggests that telomere lengthening is not only possible, but that the overall shortening process may be non-linear; in other words, telomeres likely shorten over time in an oscillatory manner (Epel, 2012).

Telomeres are highly responsive to threats and/or damage to the genome, and may serve as "first responders" to protect DNA (Blackburn et al., 2015). Importantly, telomere length is associated with diseases of aging (Codd et al., 2014), and current literature suggests that telomere shortening may serve as both a catalyst and a consequence of disease (Blackburn et al., 2015). Leukocyte telomere length (LTL) in particular is a measure of immune cell senescence, a process that may influence the progression of inflammation-related disease such as cardiovascular disease (Blackburn et al., 2015).

Telomere psychology. The determinants of telomere length include both heritable and non-heritable factors, and telomere length has been shown to correlate with individual differences. For example, telomere length correlates negatively with neuroticism (Schoormans, Verhoeven, Denollet, van de Poll-Franse, & Pennix, 2018; van Ockenburg, De Jonge, Van der Harst, Ormel, & Rosmalen, 2014), and prospective work suggests that conscientiousness measured in childhood predicts longer telomeres 40 years later among women (Edmonds, Côté, & Hamson, 2015). However, research shows a mixed pattern of association between other individual differences and telomere length. For example, telomere length is positively associated with optimism in some studies (Schutte, Palanisamy, & McFarlane, 2016), but not others (O'Donovan et al., 2010). Agreeableness was positively correlated with telomere length in a large community sample from the Netherlands (Schoormans et al., 2018) but, in a cohort study of older adults from Finland, associations differed by gender, such that agreeableness correlated positively with telomere length for women and negatively with telomere length for men (Savolainen, Eriksson, Kajantie, Pesonen, & Räikkönen, 2015). One study found that

longer telomeres are associated with both low depression and defensiveness, as well as with high hostility and anxiety, within the same sample (Starnino, Busque, Tardif, & D'Antono, 2016).

Beyond typically stable individual difference variables, telomere length is also associated with health behaviors, chronic stress, and other social and psychological experiences (Epel, 2009). Cross-sectional studies have revealed relationships between shortened telomeres and body mass index, smoking, and physical activity, but longitudinal data have shown mixed associations between changes in telomere length and these behaviors across time (e.g., Weichser, Bojesen, & Nordestgaard, 2014). Meta-analyses suggest a negative association between depression and leukocyte telomere length (Ridout, Ridout, Price, Sen, & Tyrka, 2016; Schutte & Malouff, 2014). Shortened telomere length has been linked with lower social support and feeling ambivalent about social ties (Carroll, Diez Roux, Fitzpatrick, & Seeman, 2013; Uchino et al., 2012), as well as with both perceived stress and objective chronic stress (Damjanovic et al., 2007; Epel et al., 2004; Puterman, Lin, Blackburn, O'Donovan, Adler, & Epel, 2010). Childhood adversity predicts shortened telomere length in adulthood (Kananen et al., 2010; Puterman et al., 2016). Accelerated telomere shortening has been identified as a possible mechanism explaning heightened mortality and morbidity in adults with mood disorders (Simon et al., 2006) and in nondepressed African American men experiencing racial discrimination (Chae et al., 2016). Telomere length, as a proxy for risk of cognitive decline, has also been shown to moderate the effectiveness of a cognitive training intervention on cognition, such that those in the lowest tertile of telomere length reported

the biggest benefits (Sindi et al., 2017). Telomere length has even been associated with economic delay discounting and risk proneness (Yim et al., 2016), linking impulsive behavior with cellular aging and mortality.

Research has begun to illuminate whether protective factors may be associated with longer telomere length, but the results appear mixed. Well-being predicts longer LTL among patients with chronic heart failure (Huzen et al., 2010), but not among elderly men in the Netherlands and Greece (Rius-Ottenheim et al., 2012). Multisystem resiliency (i.e., a composite of engagement in healthy behaviors, strong social connections, and low emotional suppression) moderates the cross-sectional relationship between depression and short telomere length in adults with cardiovascular disease (Puterman et al., 2013). Likewise, vigorous physical exercise has been identified as a possible buffering factor in the relationship between perceived stress and telomere length (Puterman et al., 2010). Longer telomere length has been associated with environmental factors, such as green spaces (Woo, Tang, Suen, Leung, & Wong, 2009) and having fewer neighborhood stressors (Ellaway, Dundas, Robertson, & Shiels, 2019), as well as social and developmental factors, such as being raised with responsive parenting styles (Asok, Bernard, Roth, Rosen, & Dozier, 2013) and being married (Mainous et al., 2011). Some work suggests that earning a higher income may be associated with longer telomeres (Yen & Lung, 2013), but other studies have found no association between income and telomere length (Needham et al., 2013).

In sum, much of the work examining the associations between telomere length and psychological and social factors offers conflicting findings. Furthermore, it is

primarily cross-sectional or longitudinal, rather than experimental. Moreover, the existing longitudinal work typically examines associations with change in telomere length measured years or decades apart (e.g., Aviv et al., 2009; Chen et al., 2011; Ehrelenbach et al., 2009; Harris et al., 2016; Martin-Ruiz, Gussekloo, van Heemst, Zglinicki, & Westendorp, 2005; for an exception in which telomere attrition in women is assessed over 1 year, see Puterman, Lin, Krauss, Blackburn, & Epel, 2015). As a result, many vital questions remain inadequately answered. Specifically, little is known about the normative rates of telomere shortening in healthy (non-clinical) adult samples across relatively brief time periods, as well as whether interventions to enhance protective factors may ameliorate shortening (Sanders & Newman, 2013). To address such questions, as well as to disentangle which factors are associated with telomere shortening and which factors are causal, it is critical to develop interventions to test whether changes in the social, psychological, and behavioral domains might lead to longer telomeres—and, if so, whether these changes are sustainable or impactful for downstream health outcomes (Epel, 2012).

Telomere interventions. To this end, some intervention trials have been conducted to test whether psychological and/or behavioral interventions may positively influence telomere length. Dietary interventions, including supplementation with polyunsaturated fatty acids (Kiecolt-Glaser et al., 2013), have shown promise in lengthening telomeres. Physical exercise interventions, however, have generally not shown predicted effects (for a review, see Arsenis, You, Ogawa, Tinsley & Zuo, 2017).

Psychological approaches have also demonstrated some promise in improving telomere outcomes. For example, meditative practices (e.g., loving-kindness meditation, mindfulness meditation), lifestyle interventions (including stress management and social support components), and psychosocial interventions have produced increases in telomerase, an enzyme that counteracts telomere shortening, or reductions in the rate of telomere shortening (Biegler, Anderson, Wenzel, Osann, & Nelson, 2012; Epel et al., 2009; Hoge et al., 2013; Jacobs et al., 2011; Ornish et al., 2013; Schutte & Malouff, 2014).

These results are promising, but several key methodological factors are worth noting. First, intervention-based telomere studies generally use small sample sizes (e.g., *n*s of 30-40 per experimental condition, as in Jacobs et al., 2011, Kiecolt-Glaser et al., 2013), which are likely underpowered to detect effects (for a methodological review, see Aviv, Valdes, & Spector, 2006) and are less likely to replicate. Additionally, covariates differ widely between studies, and the use of varying combinations of control variables may suppress or overestimate associations with telomere length, may limit comparability among studies, and could promote the use of questionable research practices (John, Loewenstein, & Prelec, 2012).

Additional considerations pertain to study and intervention design. Many intervention-based studies use quasi-experimental methodologies, as participants self-select into meditation retreats (e.g., Conklin et al., 2018). Further, these studies generally employ highly intensive and multicomponent interventions, which limit the ability to identify key causal components. Finally, a number of intervention studies only assess

telomere outcomes at posttest, which prevents the ability to rule out baseline differences between groups. Results from these designs are important and informative, but randomized controlled trials are necessary in order to identify which components of these interventions are causally responsible for the observed changes in telomere length.

For example, a 3-month meditation retreat, during which participants meditated for approximately 6 hours per day, led to higher posttest telomerase relative to matched wait-list controls (Jacobs et al., 2011). However, this study lacked a pretest measure of telomerase, used a sample of frequent meditators who were able to relocate to an in-residence retreat center for 3 months, and involved a high financial and time cost for participants. Additionally, many components of this intervention may account for the telomerase findings, including increased social connection and lack of occupational stress.

Ornish and colleagues (2013) reported results from a pilot study in which a 3-month comprehensive lifestyle intervention involving aerobic exercise, stress management, and dietary changes was associated with increases in telomerase activity among 24 cancer patients. Participants in a 3-week intensive meditation retreat (n = 28) showed strong effects on telomere lengthening relative to wait-list controls (n = 34) (Conklin et al., 2018), Indeed, the average increase in telomere length among retreat participants was equivalent to the magnitude of decline normally observed across a 4-year period (Cohen's d = 0.51 for posttest telomere length scores).

Taken together, the above findings suggest that behavioral interventions may influence cellular aging. However, the relatively small sample sizes indicate that the

results should be interpreted with caution. The complex and expensive interventions may render key mechanisms difficult to detect, findings relatively nongeneralizable, and replication efforts challenging to attempt. Additionally, given that most interventions require trained interventionists (e.g., meditation or lifestyle coaches) to deliver, successful interventions may not be accessible to the general public. Thus, community-based randomized controlled trials are needed to test these effects in a broader population, with well-powered, pre-registered studies needed to provide strong support for the initial findings. Finally, to arm individuals with tools to improve their own biological health, researchers should aim to develop low-cost, accessible, self-administered, relatively brief, and nonstigmatizing interventions to impact telomere length.

Present Study

Theoretical work has called for more "shortitudinal" investigations—that is, panel studies that employ relatively shorter time lags—in order to estimate the optimal timing for longitudinal interventions (Dormann & Griffin, 2015). This is especially important, as most panel studies employ time lags that are longer than ideal, which may obfuscate effects (Dormann & Griffin, 2015). Telomere research is generally characterized by long-lag designs (e.g., measurements over the course of multi-year waves). However, telomeres can show change over very brief time intervals, even within 48 hours (e.g., Garrett-Bakelman et al., 2019). More shortitudinal studies would provide clarity on the ideal time lag for telomere measurement.

In the present study, we seek to explore whether a shortitudinal prosocial behavior intervention could positively impact human telomere length. We employed a paradigm (i.e., performing acts of kindness for others) that has been shown elsewhere to improve psychological flourishing (Nelson et al., 2016) and to elicit adaptive changes in gene expression (Nelson-Coffey et al., 2017) relative to a neutral control activity (i.e., tracking daily activities), with one significant modification (i.e., eliminating a condition involving doing kind acts "for the world").

We hypothesized that individuals who performed kind acts for others would demonstrate significant reductions in the rates of telomere shortening relative to controls (pre-registration available on the Open Science Framework at https://osf.io/93ck7/). Furthermore, we predicted that the effect of performing acts of kindness for others on telomere length change would be mediated by increases in social connectedness and decreases in loneliness and perceived stress.

Finally, we predicted that, relative to participants who performed a neutral task, those randomly assigned to perform acts of kindness for other individuals would improve in psychological well-being. Specifically, we hypothesized that our kindness for others group would report improvements in well-being (e.g., greater life satisfaction, more psychological flourishing, and less loneliness) across the intervention period, relative to controls. We also predicted that participants who performed kind acts for others would report increases in weekly measures of social connection and positive affect, and decreases in weekly measures of negative affect and perceived stress.

Method

Participants

Community adults ($N = 230$) were recruited through email advertisement, flyers, and community fairs to participate in a study of positive activities and health. Participants were eligible if they were 21 years of age or older and not currently taking antidepressant medication, and received $100 compensation for completing all timepoints. The majority of participants (73%) identified as female (M_{age} = 34.8 years, SD = 11.23, range = 21-83), white (42.6%), and highly educated (52.2%). See Table 1 for baseline sample characteristics.

Design and Procedure

Participants were randomly assigned to participate in one of three possible conditions that varied only with respect to their activity instructions. Participants in the *kindness-to-others* group were instructed to perform three acts of kindness for other people, all three in one day, each week for 4 weeks. Participants in the *kindness-to-self* group were instructed to perform three acts of kindness for themselves, all three in one day, each week for 4 weeks. Finally, participants in the control group were instructed to keep track of their usual daily activities on one day each week for 4 weeks. Each week during the intervention period (see Figure 1), participants logged on to the study website (either in-lab [i.e., at T_1 and T_5] or from home [i.e., T_2, T_3, and T_4]) to complete outcome measures, report on the previous week's activities, and to receive instructions for the following week's activities (T_1 through T_4 only). Finally, at the 2-week follow up (T_6),

participants logged on to the study website to complete one final assessment of outcome measures.

At the first and fifth time points (i.e., baseline and post-intervention), participants came in to the lab to provide a small dried blood spot (DBS) sample via finger prick for analysis of telomere length and to complete self-report measures of dependent (e.g., mental health flourishing, life satisfaction) and mediating (e.g., social connection, affect) psychological variables. Immediately following the completion of these questionnaires, participants were provided intervention instructions for the respective activity to which they had been randomly assigned (see Appendix A for full intervention instructions).

At all other time points (i.e., T_2, T_3, T_4, and T_6), participants were emailed web links in order to complete weekly surveys on their home computers or mobile devices (and to be debriefed; T_6 only).

Measures

Psychological measures.

Life satisfaction. At T_1, T_5, and T_6, participants reported their current satisfaction with their life in general using the Satisfaction With Life Scale (SWLS; Diener, Emmons, Larsen & Griffin, 1985). The SWLS consists of five questions (e.g., "In most ways my life is close to my ideal," "I am satisfied with my life"), which are rated on 7-point Likert-type scales (1 = *strongly disagree,* 7 = *strongly agree).* Across measurements in this study, Cronbach's αs ranged from .87 to .90.

Flourishing. The Mental Health Flourishing-Short Form (MHC-SF; Keyes, 2002) measures the extent to which participants are experiencing flourishing mental health. At

T_1, T_5, and T_6, participants responded to 14 items (e.g., "How often did you feel that you liked most parts of your personality?") assessing components of emotional, social, and personal well-being on a scale from 0 (*never*) to 5 (*every day*). Cronbach's αs ranged from .89 to .94 across timepoints.

Loneliness. The Revised UCLA Loneliness Scale (Russell, Peplau, & Cutrona, 1980) is a 20-item measure assessing perceived loneliness. At T_1, T_5, and T_6, participants indicated how frequently each item described them on a 4-point Likert-type scale (0 = *never*, 3 = *often*). Items include "I feel in tune with people around me," "My social relationships are superficial," and "I feel isolated from others." Across time points, Cronbach's αs ranged from .92 to .94.

Positive and negative affect. Weekly emotions were assessed using the Affect-Adjective Scale (Diener & Emmons, 1984). This 9-item measure taps a range of positive emotions (happy, pleased, joyful, enjoyment/fun) and negative emotions (worried/anxious, angry/hostile, frustrated, depressed/blue, unhappy). At all timepoints, participants rated the extent to which they have experienced the emotions in the past week on a 7-point Likert scale (0 = *not at all,* 1 = *slightly,* 2 = *somewhat,* 3 = *moderately,* 4 = *much,* 5 = *very much,* 6 = *extremely*). Across time points, Cronbach's αs ranged from .90 to .95 for positive emotions, and .82 to .89 for negative emotions.

Social connectedness. We assessed participants' feelings of social connectedness at each timepoint with the relatedness subscale from the Balanced Measure of Need Satisfaction (BMPN; Sheldon & Hilpert, 2011). This questionnaire includes a 6-item measure (with 3 positively worded and 3 negatively worded items each) to assess

connectedness over the past week, such as "I felt a sense of contact with people who care for me." Participants rated their level of agreement with each item on 5-point Likert-type scale (1 = *no agreement,* 3 = *some agreement,* 5 = *much agreement).* Cronbach's αs ranged from .71 to .85 across all measurements.

Perceived stress. At each timepoint, participants reported their level of stress using an adapted version of the 10-item Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983). Using a 5-point Likert-type scale (1 = *never*, 5 = *very often*), participants responded to items asking about the frequency of stress, such as "How often in the past week have you felt you could not cope with all the things you have to do?" and "How often in the past week have you felt unable to control the important things in your life?" Cronbach's αs ranged from .87 to .90 across all measurements.

Biological Measures.

Leukocyte telomere length (LTL). Genomic DNA was extracted from six 3-mm diameter cutouts from dried blood spot samples using QIAamp DNA Investigator Kit (cat# 56504) according to manufacturer's instructions. DNA concentration was quantified by PicoGreen Assay using ThermoFisher NanoDropTM 3300. The average DNA concentration was 4.5 ng/µl.

The telomere length measurement assay is adapted from the published original method by Cawthon (2002; Lin et al., 2010). The telomere thermal cycling profile consists of the following: Cycling for T(telomic) PCR: Denature at 96°C for 1 minute; denature at 96°C for 1 second, anneal/extend at 54°C for 60 seconds, with fluorescence data collection, 30 cycles. Cycling for S (single copy gene) PCR: Denature at 96°C for 1

minute; denature at 95°C for 15 seconds, anneal at 58°C for 1 second, extend at 72°C for 20 seconds, 8 cycles; followed by denature at 96°C for 1 second, anneal at 58°C for 1 second, extend at 72°C for 20 seconds, hold at 83°C for 5 seconds with data collection, 35 cycles. The primers for the telomere PCR are tel1b [5'-CGGTTT(GTTTGG)$_5$GTT-3'], used at a final concentration of 100 nM, and tel2b [5'-GGCTTG(CCTTAC)$_5$CCT-3'], used at a final concentration of 900 nM. The primers for the single-copy gene (human beta-globin) PCR are hbg1 [5'-GCTTCTGACACAACTGTGTTCACTAGC-3'], used at a final concentration of 300 nM, and hbg2 [5'-CACCAACTTCATCCACGTTCACC-3'], used at a final concentration of 700 nM. The final reaction mix contains 20 mM Tris-HCl, pH 8.4; 50 mM KCl; 200 mM each dNTP; 1% DMSO; 0.4x Syber Green I; 22 ng E. coli DNA per reaction; 0.4 Units of Platinum Taq DNA polymerase (Invitrogen Inc.) per 11 microliter reaction; 7 ng of genomic DNA. Tubes containing 26, 8.75, 2.9, 0.97, 0.324 and 0.108ng of a reference DNA (from Hela cancer cells) are included in each PCR run so that the quantity of targeted templates in each research sample can be determined relative to the reference DNA sample by the standard curve method. The same reference DNA was used for all PCR runs.

To control for batch variance, the T/S ratio for each sample was measured twice. T/S values were then adjusted based on initial systematic differences between the two runs. When the duplicate T/S values varied by more than 7% after adjustments, the sample was ran for a third or fourth time and the two closest values were reported. Samples were run in batches of 96 wells. The batch differences were adjusted by

repeating a subset of samples from each plate and subsequently adjusting runs 1 and 2 accordingly. Using this method, the average CV for this study was 2.15%.

Analytic Approach

We pre-registered our analytic plan, including our planned covariates for the biological analyses, on the Open Science Framework (https://osf.io/93ck7/); currently embargoed. See Supplementary Materials for additional measures not reported here. Based on the Shapiro-Wilk test, T/S values were normally distributed at baseline (W = 0.9917, p = .22 for null hypothesis), but not at posttest (W = 0.9812, p = .005 for null hypothesis). Thus, T/S ratio scores were log-transformed prior to analysis.

Results

Pre-Registered Analyses

Telomere length. Using the NLME package in R Studio, we ran a linear mixed-effects model analysis, adjusted for our pre-registered covariates (i.e., age, sex, BMI, illness symptoms at pretest and posttest, and hormone use), to examine the effect of condition on our primary biological outcome of change in LTL (see Figure 2). Contrary to our prediction, participants in the *other-kindness* condition did not report significant changes in LTL over time relative to controls, b = -0.01, p = 0.15.

Psychological outcomes. Using planned contrasts, we tested whether participants in the *other-kindness* condition reported greater change scores relative to the control group. Contrary to predictions, no significant differences between *other-kindness* and control were detected for flourishing, $t(224) = 0.66$, p = .51, life satisfaction, $t(224)$ = 0.30, p = .76, or loneliness, $t(223) = -0.91$, p = .37, for change scores between baseline

and posttest (see Table 2). A similar pattern emerged when examining change scores between baseline and follow-up, with no significant differences between *other-kindness* and control detected for flourishing, $t(128) = 0.74$, $p = .46$ life satisfaction, $t(133) = 1.56$, $p = .12$, or loneliness, $t(127) = -1.37$, $p = .17$.

Additionally, relative to controls, participants in the *other-kindness* condition did not report greater positive affect, $t(227) = 0.54$, $p = .59$, greater connectedness, $t(167) = -0.14$, $p = .89$, less negative affect, $t(227) = -0.45$, $p = .66$, or lower stress, $t(167) = 0.96$, $p = .34$, on average across the intervention period (e.g., averaged from T_2-T_5; see Table 3). Given the lack of association between experimental condition, our hypothesized potential mediator variables, and our outcomes, we did not proceed to test mediation models.

Exploratory Analyses

Telomere length. Across the full sample, telomere length increased from baseline, and a one-sample t-test on the change scores suggested this nonsignificant finding was marginal, $t(222) = 1.77$, $p = .08$. LTL did not differ by sex at baseline, $t(228) = 0.10$, $p = .92$, or posttest, $t(221) = 0.02$, $p = .96$.

A linear mixed-effects model analysis, adjusted for our pre-registered covariates (i.e., age, sex, BMI, illness symptoms at pretest and posttest, and hormone use) revealed that, relative to controls, participants in the *self-kindness* condition did not report significant changes in LTL over time, $b = -0.0007$, $p = 0.91$.

In light of the trend evident in Figure 2 (i.e., in the opposite direction from our pre-registered hypothesis), we further explored the effect of condition on LTL using regressed change analyses. Using this approach, and controlling for our pre-registered

covariates and baseline LTL, we found that participants in the *other-kindness* condition reported significantly lower LTL at posttest than did participants in the control condition, $b = -1.245 \times 10^{-2}$, $p = 0.05$. Participants in the *self-kindness* condition, however, did not report significantly different LTL than did controls, $b = -2.704 \times 10^{-3}$, $p = 0.67$. Additionally, collapsing across all three conditions, number of kind acts reported across the intervention period also marginally predicted lower posttest LTL, controlling for baseline LTL and our covariates, $b = -8.352 \times 10^{-4}$, $p = .08$.

We also found a significant condition by sex interaction ($b = 0.04$, $p = .008$), such that, relative to women, men reported longer posttest LTL, controlling for baseline LTL and our pre-registered covariates (see Figure 3). This interaction appears to be driven by women in the *other-kindness* condition declining in LTL, as when the analyses were run separately by sex, *other-kindness* predicted lower posttest LTL, controlling for baseline LTL and all covariates except sex, for women ($b = -0.024$, $p = .002$) but not for men ($b = 0.019$, $p = .12$).

Psychological outcomes.

Kindness to self. We used post-hoc contrasts to test whether the *self-kindness* differed from either the control group or the *other-kindness* group on our outcomes of interest. Relative to controls, participants in the *self-kindness* group reported significantly lower change scores (i.e., less loneliness) at the posttest, $t(223) = -2.06$, $p = .04$, but not at the follow up, $t(127) = -1.40$, $p = .17$. No other significant differences were detected between the *self-kindness* and control groups for change scores of flourishing or life satisfaction between baseline and posttest, ps > .58, or baseline and follow up, ps > .68.

Further, relative to controls, participants in the *self-kindness* condition did not report improved positive affect, connectedness, negative affect, or stress, on average, across the intervention period (*p*s > .09). Finally, no significant differences emerged between *self-kindness* and *other-kindness* on these outcomes, *p*s > .27 (see Tables 2 and 3).

Growth modeling. To further explore the trajectory of our psychological variables across multiple time points, we used multilevel growth curve modeling to account for repeated measures nested within participant. Relative to controls, participants in the *other-kindness* condition reported significant decreases in loneliness from baseline through the 2-week follow up, $\gamma_{11} = -0.06$, $p = .05$, and participants in the *self-kindness* condition reported a trend in the same direction, $\gamma_{12} = -0.05$, $p = .08$ (see Table 4 and Figure 4). However, relative to controls, participants in the other-kindness and self-kindness conditions did not report greater increases in in life satisfaction, *p*s > .13, or flourishing, *p*s > .44, across time.

Sex differences in psychological variables. Given the interaction of sex and condition on telomere length, we conducted exploratory analyses to identify whether parallel psychological processes might provide convergent support for sex differences from our intervention.

At baseline, relative to males, females were significantly less lonely, $t(228) = -2.51$, $p = .013$, and more satisfied with their lives, $t(228) = 2.44$, $p = .015$. No sex differences emerged at baseline for flourishing, $t(228) = 1.25$, $p = .21$, connectedness, $t(228) = 0.45$, $p = .66$, positive affect, $t(228) = 0.09$, $p = .93$, negative affect, $t(228) = 0.81$, $p = .94$, or stress, $t(228) = 0.18$, $p = .86$.

We found a main effect of sex on loneliness, such that, among control participants, males reported higher loneliness than did females at posttest, controlling for baseline, $b = 0.25$, $p = .0002$. However, there was a sex-by-condition interaction, $b = -0.31$, $p = .001$, which is the product of the aforementioned differences between sexes in the control condition and a relative lack of differences between sexes in the *other-kindness* condition (see Figure 5). We found a similar trend in the *self-kindness* group, $b = -0.19$ $p = .053$, again driven by differences in males in the control condition (see Figure 5).

We also found a main effect of sex on satisfaction with life, such that, within the control group, males reported smaller increases in life satisfaction than did females, $b = -0.70$, $p = .005$. Relative to females, males also reported lower average connectedness throughout the intervention period, $b = -0.31$, $p = .04$. No sex-by-condition interactions were detected for either life satisfaction or connectedness. No other differences emerged by sex for average positive affect, average negative affect, average stress, or change in flourishing, ps > .15.

Relationship between psychological variables and telomere length.

We explored raw and partial correlations (i.e., adjusted for our covariates) between baseline telomere length and participant demographic and psychological variables. We also examined whether change in any psychological variables across conditions correlated with change in telomere length from baseline to posttest. Tables 5-7 present these correlation matrices.

As we expected, LTL correlated negatively with age (r = -.45, p < .000). However, partial correlations between baseline LTL and baseline psychological characteristics, including well-being, loneliness, flourishing, positive affect, negative affect, stress, and social connection, were all nonsignificant (rs ranging from -.08 to .06, ps >.22). One exception was marginal: Baseline LTL correlated with baseline life satisfaction (r = .115, p = .09). Finally, partial correlations (controlling for age, gender, BMI, hormone use, and illness symptoms) between LTL change and change in well-being, loneliness, flourishing, positive affect, negative affect, stress, and social connection were all nonsignificant (with rs ranging from -.11 to .11, ps >.16).

Finally, we tested whether, collapsed across conditions, participants who reported the greatest gains in well-being also reported differential levels of LTL change. Using regressed change analyses to predict post-intervention LTL, controlling for baseline LTL and our pre-registered covariates, we found that, collapsed across conditions, neither changes in loneliness, $b = -9.71 \times 10^{-3}$, p = .31, nor flourishing, $b = -6.83 \times 10^{-3}$, p = .21, nor life satisfaction, $b = -6.46 \times 10^{-3}$, p = .81, predicted change in LTL across the intervention period. Similarly, across all conditions, change in LTL was not predicted by average negative affect, b = -0.0028, p = .43, average positive affect, b = 0.0033, p = .31, average stress, b = -0.001, p = .89, or average connectedness, b = .0004, p = .95.

Finally, we also compared changes in LTL across time for participants in the highest versus lowest quartiles of improvements in well-being, loneliness, and flourishing—that is, those who reported the greatest gains versus the greatest declines from baseline to posttest in these outcomes, collapsed across all conditions. Relative to

the lowest quartile, those in the highest quartile of change in loneliness, b = -0.004, p = .15, flourishing, b = -0.002, p = .42, and life satisfaction, b = 0.002, p = .52, did not report differential levels of LTL change across time.

Discussion

To our knowledge, this study is the first pre-registered experimental investigation to test the effect of a positive psychological intervention on telomere length across time. We are aware of two other pre-registered studies; both are observational and employ a single assessment of telomere length. The first, partially supporting hypotheses, found an indirect association between maternal depressive symptoms and shorter infant telomere length (Nelson, Allen, & Laurent, 2018). The second, contrary to predictions, showed no association between years of caregiving and telomere length in a sample of 1233 Filipino adults (Rej, Tennyson, Lee, & Eisenberg, 2019).

Our hypothesis with regard to telomere length change was not supported using our pre-registered analytic plan. In fact, exploratory analyses suggested a pattern in the opposite direction—that engaging in kindness to others may have led to *shorter* telomere length. It is possible that engaging in kindness to others backfired by inadvertently eliciting stress or other negative psychological states that may have contributed to telomere attrition. However, given the small and inconsistent size of this effect and the lack of parallel psychological findings (e.g., involving stress or negative affect) in this and other studies using this paradigm, the results are more likely to be a result of chance and/or telomeric measurement error than true shortening. Indeed, given the relatively large number of exploratory analyses we ran (46 tests in total; see Supplementary

Materials for those not reported here), we likewise suspect that the interaction between sex and condition on telomere length to be a product of chance.

Across conditions, we found a trend toward telomere elongation across time in our study. Although this finding is likely noise, it is important to note that telomere elongation has been reported elsewhere (e.g., Aviv et al., 2009), and one longitudinal study reported telomere elongation in 46% of the sample across a 5-year span (Berglund et al., 2016). Although biological explanations for this phenomenon have been proposed (e.g., involving increases in telomerase), such findings may reflect measurement error, even when the coefficient of variation is low, such as in our study (Chen et al., 2011).

Importantly, not all leukocytes are telomerically equal. Subsets of lymphocytes from the same individual may show different lengths of telomeres (Lin et al., 2010). Importantly, leukocyte composition is sensitive to acute stressors, and even a relatively brief (i.e., 5-minute) activation of the sympathetic nervous system can elicit lymphocyte subtype mobilization and redistribution (e.g., by increasing NF-κB relative to other types of leukocytes; Richlin, Arevalo, Zack, & Cole, 2004). This phenomenon, termed "pseudo-lengthening" (Epel, 2012), could provide one plausible explanation for the overall trend toward telomere lengthening from baseline to posttest in our sample. Specifically, the initial blood draw may have served as an acute stressor, eliciting leukocyte redistribution, to which the participant was habituated by the 4-week follow-up.

Some theoretical work based on existing evidence (e.g., Bateson & Nettle, 2018) has challenged the notion that behaviors influence telomeres by suggesting that a reverse

causal model, in which telomere length may influence selective adoption of behaviors in humans and birds, is more likely. Although our experimental results do not support this causal path, one account of the null impact of our intervention on telomere length could be that the widely assumed causal direction (e.g. that behaviors influence telomere length) is incorrect.

Psychological Outcomes

Our pre-registered hypotheses regarding kindness to others and changes in well-being were also not supported. However, exploratory analyses revealed a pattern of results suggesting that engagement in either kindness activity led to improvements in loneliness across time. This finding supports our prediction that engaging in kindness to others may lead individuals to feel closer and more connected to others in their lives. Regarding this effect in the self-kindness condition, it is possible that these participants may have engaged in self-directed activities (e.g., getting a manicure, going to a movie) alongside friends or spouses, which caused them to feel less isolated and more integrated into their social groups.

Additionally, we found that participants in the other-kindness group reported changes across time in loneliness, but only when the follow-up time period was included (i.e., via growth curve modeling). One plausible explanation for this effect is that changes in loneliness may take longer than our 4-week intervention period to germinate. In our study, kindness recipients may have responded positively to our participants (e.g., by expressing gratitude or reciprocating the support), which may have led to a recursive prosocial spiral between our participants and the recipients of their kind acts, ultimately

improving relationships. This dynamic process may not have been detected by the participant or captured with our loneliness measure until after the formal intervention period had ended. However, the loneliness-alleviating capacity of prosocial behavior is particularly exciting, given that social isolation is a public health concern and has been identified as a target for intervention efforts (e.g., Holt-Lunstad, Robles, & Sbarra, 2017).

We also found an interaction between gender and condition, such that men who performed kindness for others showed the biggest improvements in loneliness across time. Given that males were lonelier at baseline than were females, and that men may be less likely than women to engage in certain kinds of support-giving (Eagly, 2009), our intervention may have been more powerful and novel among this potentially vulnerable group. Additionally, this effect appears to be driven by males in the control group *increasing* in loneliness across time, and it is possible that our intervention served to buffer against this trajectory. However, given the relatively small number of male participants in each of these cells (n = 21 in control; n = 21 in *other-kindness*) and the exploratory nature of this analysis, this finding warrants replication in a larger sample.

Limitations and Future Directions

Our shortitudinal trial occurred over the course of just 4 weeks, and employed a relatively low touch intervention. As most telomere intervention studies are highly intensive and multicomponent, it is possible that our intervention was not potent enough or lengthy enough to elicit the predicted changes in telomere length. Although the same paradigm has successfully shifted biological outcomes in past work (Nelson-Coffey et al., 2017), perhaps a stronger dosage of our prosocial behavior intervention may be required

to impact telomere biology. It is also possible that our 1-month time lag was too short to detect changes in telomere length. Future work should strive to establish the optimal dosage for kindness interventions and the optimal time lag for measuring telomere length.

Our hypotheses regarding improvements in psychological variables were largely unsupported. One potential reason that we did not replicate the psychological flourishing finding reported by Nelson and colleagues (2016) is that, given our budgetary constraints for blood assays, our sample size was approximately half as large. Thus, we may have been underpowered to detect psychological effects. Notably, however, prior work using this prosocial behavior paradigm has found biological effects even in the absence of self-reported psychological effects (e.g., Nelson-Coffey et al., 2017).

Concluding Words

We conducted the first pre-registered positive psychological (i.e., prosocial behavior) intervention aimed at impacting telomere health. Our pre-registered hypotheses regarding telomere length and psychological well-being were not supported. Notably, even participants who reported the biggest gains in well-being and related constructs from our intervention failed to show parallel shifts in telomere length. Further, collapsing across experimental conditions, neither single timepoint measures nor pre-post changes in psychological variables were significantly correlated with telomere health. Exploratory analyses, however, suggested that performing kindness may alleviate loneliness, thereby highlighting prosocial behavior as a potentially useful—brief, self-delivered, low-cost, and scalable—intervention for addressing a key public health concern.

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Table 1.
Baseline Sample Characteristics

	Control	Self-Kindness	Other-Kindness	*p**
Age (mean ± SD years)	37.02 (13.04)	33.18 (9.94)	33.86 (9.84)	.07
Sex (% Female)	75.0%	73.0%	70.8%	.84
Race/ethnicity (% self-identified)				.93
Asian/Asian American	9.5%	12.2%	13.9%	
Black/African American	3.6%	5.4%	5.6%	
Hawaiian/Pacific Islander	1.2%	1.4%	2.8%	
White	46.4%	41.9%	38.9%	
Hispanic/Latinx	32.1%	32.4%	25.0%	
Middle Eastern	1.2%	2.7%	5.6%	
Other/More than One	4.8%	4.1%	6.9%	
American Indian/Alaskan Native	1.2%	0.0%	1.4%	
Education level				.52
(1) Did not finish high school	0.0%	2.7%	2.8%	
(2) High school diploma	1.2%	4.1%	1.4%	
(3) Some College	15.5%	14.9%	13.9%	
(4) 4-Year Degree	26.2%	31.5%	26.4%	
(5) Graduate or Professional Degree	57.1%	43.2%	55.6%	
Employment** (% Yes)				
(1) Full-time	59.5%	63.5%	58.3%	.80
(2) Part-time	9.5%	10.8%	11.1%	.94
(3) Full-time college/university student	25%	23%	25%	.95
(4) Self-employed	2.4%	4.1%	2.8%	.82
(5) Unemployed	4.8%	4.1%	5.6%	.91
(6) Retired	2.4%	0.0%	1.4%	.42
(7) Other	4.8%	5.4%	5.6%	.97
Hormone use (% Yes)	22.6%	25.7%	31.9%	.41
Baseline sickness (% Yes)	8.3%	8.1%	2.8%	.30
Posttest sickness (% Yes)	6.0%	6.8%	4.2%	.79
Body mass index	26.24 (6.05)	28.85 (8.13)	26.09 (6.88)	.03*
Life Satisfaction (mean ± SD)	4.82 (1.32)	4.92 (1.17)	4.73 (1.12)	.61
Flourishing (mean ± SD)	3.27 (0.66)	3.30 (0.72)	3.28 (0.61)	.95
Happiness (mean ± SD)	4.81 (1.33)	4.99 (1.21)	4.88 (1.16)	.67
Loneliness (mean ± SD)	1.97 (0.52)	2.01 (0.52)	2.00 (0.61)	.91
Baseline log LTL (mean ± SD)	0.149 (.075)	0.151 (.086)	0.153 (.088)	.96

*Omnibus test statistic from ANOVA (continuous variables) or χ^2 (categorical variables).
** Employment categories were not mutually exclusive.

Table 2.
Planned Contrasts and Post-Hoc Tests by Condition and Time Point for Pre-Post Measures

	Kindness to Others *Contrast 1: +1 Contrast 2: +1 Contrast 3: 0*		**Kindness to Self** *Contrast 1: 0 Contrast 2: -1 Contrast 3: +1*		**List Daily Activities (Control)** *Contrast 1: -1 Contrast 2: 0 Contrast 3: -1*		*Contrast 1*			*Contrast 2*			*Contrast 3*		
	M(SD)	*n*	*M(SD)*	*n*	*M(SD)*	*n*	*t*	*p*	*r*	*t*	*p*	*r*	*t*	*p*	*r*
Post-Intervention															
Life Satisfaction	4.62 (1.37)	71	4.71 (1.44)	73	4.68 (1.48)	83	-0.23	.82	.02	-0.35	.73	.02	0.14	.89	.01
Flourishing	3.33 (0.70)	71	3.26 (0.75)	73	3.26 (0.71)	83	0.61	.54	.04	0.56	.57	.04	0.03	.97	.00
Loneliness	1.95 (0.57)	70	1.94 (0.56)	73	1.99 (0.56)	83	-0.39	.70	.03	0.10	.92	.01	-0.50	.62	.03
Follow Up															
Life Satisfaction	5.09 (1.24)	39	5.16 (1.27)	42	5.03 (1.34)	55	0.20	.84	.02	-0.24	.81	.02	0.47	.64	.04
Flourishing	3.35 (0.81)	38	3.38 (0.81)	40	3.30 (0.85)	53	0.31	.76	.03	-0.13	.90	.01	0.45	.66	.04
Loneliness	1.82 (0.62)	38	1.92 (0.58)	39	1.94 (0.56)	53	-0.94	.35	.08	-0.79	.43	.07	-0.09	.93	.01

$^{\dagger}p \leq .10$. $^{*}p \leq .05$. $^{**}p \leq .01$. $^{***}p \leq .001$.

Table 3.
Planned Contrasts and Post-Hoc Tests by Condition and Time Point for Weekly Measures

	Kindness to Others *Contrast 1: +1 Contrast 2: +1 Contrast 3: 0*		**Kindness to Self** *Contrast 1: 0 Contrast 2: -1 Contrast 3: +1*		**List Daily Activities (Control)** *Contrast 1: -1 Contrast 2: 0 Contrast 3: -1*		*Contrast 1*			*Contrast 2*			*Contrast 3*		
	M(SD)	*n*	*M(SD)*	*n*	*M(SD)*	*n*	*t*	*p*	*r*	*t*	*p*	*r*	*t*	*p*	*r*
Week 1 / Baseline															
Positive Affect	4.63 (1.16)	72	4.83 (1.15)	74	4.67 (1.11)	84	-0.26	.79	.02	-1.10	.27	.07	0.88	.38	.06
Negative Affect	2.87 (1.09)	72	2.94 (1.14)	74	2.80 (1.09)	84	0.36	.72	.02	-0.42	.68	.03	0.80	.43	.05
Connectedness	3.81 (0.73)	72	3.70 (0.80)	74	3.86 (0.73)	84	-0.41	.69	.03	0.90	.37	.06	-1.34	.18	.09
Perceived Stress	2.64 (0.66)	72	2.66 (0.66)	74	2.62 (0.72)	84	0.17	.87	.01	-0.17	.86	.01	0.35	.73	.02
Week 2															
Positive Affect	4.63 (1.19)	62	4.65 (1.32)	62	4.67 (1.06)	75	-0.23	.83	.02	-0.11	.91	.01	-0.10	.92	.01
Negative Affect	2.68 (1.00)	62	2.81 (1.04)	62	2.52 (0.93)	75	0.97	.34	.07	-0.73	.47	.05	1.73	.09†	.12
Connectedness	3.85 (0.74)	62	3.66 (0.86)	62	3.86 (0.81)	75	-0.07	.94	.00	1.30	.20	.09	-1.43	.15	.09
Perceived Stress	2.68 (.64)	62	2.70 (0.73)	62	2.60 (0.72)	75	0.67	.50	.05	-0.14	.89	.01	0.83	.41	.06
Week 3															
Positive Affect	4.79 (1.22)	56	4.90 (1.33)	61	4.85 (1.24)	79	-0.29	.77	.02	-0.48	.63	.03	0.22	.83	.02
Negative Affect	2.57 (1.05)	56	2.66 (1.13)	61	2.52 (0.92)	79	0.25	.81	.02	-0.50	.62	.04	0.79	.43	.06
Connectedness	3.92 (0.68)	56	3.81 (0.81)	61	4.01 (0.77)	79	-0.69	.50	.05	0.75	.45	.05	-1.52	.13	.11
Perceived Stress	2.60 (0.67)	56	2.53 (0.68)	61	2.48 (0.69)	79	1.03	.30	.07	0.54	.59	.04	0.47	.64	.03
Week 4															
Positive Affect	4.65 (1.39)	54	4.93 (1.35)	60	4.75 (1.31)	72	-0.40	.69	.03	-1.08	.28	.08	0.75	.46	.06
Negative Affect	2.49 (1.18)	54	2.59 (1.10)	60	2.55 (1.14)	72	-0.26	.80	.02	-0.45	.65	.03	0.21	.83	.02
Connectedness	3.95 (0.81)	54	3.89 (0.84)	60	3.95 (0.76)	72	-0.01	.99	.00	0.36	.72	.03	-0.39	.69	.03
Perceived Stress	2.63 (0.71)	54	2.58 (0.70)	60	2.48 (0.76)	72	1.14	.26	.08	0.36	.72	.03	0.79	.43	.06
Week 5 / Posttest															
Positive Affect	4.89 (1.21)	71	5.00 (1.26)	73	4.72 (1.33)	83	0.83	.41	.06	-0.53	.59	.04	1.39	.17	.09
Negative Affect	2.31 (0.99)	71	2.48 (0.97)	73	2.47 (1.16)	83	-0.96	.34	.06	-0.98	.33	.07	0.06	.96	.00
Connectedness	4.03 (0.76)	71	4.03 (0.70)	73	4.02 (0.78)	83	0.09	.93	.01	.01	.99	.00	0.08	.94	.01
Perceived Stress	2.50 (0.76)	71	2.51 (0.70)	73	2.56 (0.82)	83	-0.44	.66	.03	-0.07	.95	.00	-0.37	.71	.02

Week 6 / Follow Up															
Positive Affect	5.18 (1.44)	39	4.98 (1.42)	42	4.93 (1.35)	55	0.86	.39	.07	0.64	.53	.06	0.19	.85	.02
Negative Affect	2.64 (1.37)	39	2.65 (1.06)	42	2.60 (1.09)	55	0.18	.86	.02	-0.04	.97	.00	0.23	.82	.02
Connectedness	4.09 (0.86)	39	3.89 (0.86)	41	3.90 (0.87)	55	1.07	.29	.09	1.01	.31	.00	-0.02	.99	.00
Perceived Stress	2.50 (0.83)	38	2.52 (0.69)	40	2.49 (0.75)	53	0.09	.93	.01	-0.11	.91	.01	0.21	.83	.02

$^{\dagger}p \leq .10$. $^{*}p \leq .05$. $^{**}p \leq .01$. $^{***}p \leq .001$.

Table 4

Model Parameters (Standard Errors) and Goodness of Fit for Linear Change for Loneliness from Baseline to Follow-Up (T_6).

	Effect	Parameter	Loneliness	
			Model 1: Unconditional Growth	Model 2: Kindness Condition vs Control
Fixed Effects	Intercept	γ_{00}	2.02 (0.40)***	1.97 (0.07)***
	Time	γ_{10}	-0.03 (0.01)*	0.006
	Other-Kindness	γ_{01}	-	0.09 (0.10)
	Self-Kindness	γ_{02}	-	0.08 (0.10)
	Time * Other-Kindness	γ_{11}	-	-0.06 (0.03)*
	Time * Self-Kindness	γ_{12}	-	-0.05 (0.03)†
Random Effects	Level 1	σ^2_{ε}	0.03	0.03
	Level 2	σ^2	0.27	0.28
		σ^2	0.01	0.01
Goodness of Fit	Deviance		432.57	427.60
	Akaike Information Criterion		444.57	447.60
	Bayesian Information Criterion		470.81	491.34
	$\Delta\chi^2$			4.97
	Δdf			4

Note. In Model 1, the intercept parameter estimate (γ_{00}) represents average loneliness at baseline across the sample. In Model 2, the intercept parameter estimate (γ_{00}) represents average loneliness for those in the control condition.

$^{\dagger}p \leq .10$. $^{*}p \leq .05$. $^{**}p \leq .01$. $^{***}p \leq .001$.

Table 5

Correlations Among All Study Measures

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Baseline																								
1. Telomere Length	-																							
2. Age	-.45***	-																						
3. Loneliness	-0.04	-.03	-																					
4. Life Sat	0.15*	-.11†	-.45***	-																				
5. Flourish	-0.05	.14*	-.63***	.54***	-																			
6. PA	0.07	-.04	-.53***	.53***	.69***	-																		
7. NA	0.08	-.18**	.40***	-.46***	-.50***	-.45***	-																	
8. Connected	0.01	.08	-.60***	.51***	.61***	.60***	-.52***	-																
9. Stress	0.06	-.21***	.43***	-.48***	-.63***	-.58***	.66***	-.57***	-															
Posttest																								
10. Telomere Length	.89***	-.44***	-.02	.20**	-.03	.10	.09	.02	.06	-														
11. Loneliness	.03	.03	.88***	-.46***	-.61***	-.50***	.43***	-.52***	.40***	.03	-													
12. Life Sat	.11†	-.09	-.47***	.74***	.53***	.47***	-.43***	.42***	-.44***	.13†	-.61***	-												
13. Flourish	-.03	.12†	-.57***	.49***	.77***	.59***	-.45***	.47***	-.53***	-.05	-.66***	.67***	-											
14. PA	-.00	.02	-.41***	.40***	.49***	.59***	-.27***	.36***	-.35***	.02	-.53***	.58***	.68***	-										
15. NA	.11†	-.20**	.32***	-.24***	-.32***	-.22***	.49***	-.30***	.45***	.11†	.43***	-.42***	-.54***	-.46***	-									
16. Connected	.03	.04	-.51***	.36***	.41***	.41***	-.35***	.48***	-.40***	.03	-.63***	.50***	.61***	.63***	-.60***	-								
17. Stress	.10	-.18**	.36***	-.34***	-.46***	-.39***	.51***	-.40***	.67***	.11†	.48***	-.55***	-.66***	-.60***	.74***	-.63***	-							

Follow Up																								
18. Loneliness	.01	-.06	.82***	-.47***	-.54***	-.46***	.44***	-.54***	.35***	.00	.90***	-.63***	-.67***	-.55***	.43***	-.62***	.49***	-						
19.Life Sat	.14	-.07	-.47***	.77***	.54***	.50***	-.39***	.43***	-.44***	.19*	-.52***	.83***	.62***	.57***	-.33***	.52***	-.53***	-.60***	-					
20.Flourish	.02	.01	-.50***	.48***	.70***	.62***	-.38***	.45***	-.40***	.05	-.57***	.67***	.84***	.71***	-.37***	.54***	-.54***	-.57***	.71***	-					
21. PA	.05	-.03	-.43***	.47***	.49***	.65***	-.23**	.38***	-.37***	.11	-.50***	.64***	.68***	.73***	-.34***	.53***	-.54***	-.50***	.69***	.79***	-				
22. NA	.02	-.13	.33***	-.43***	-.38***	-.32***	.51***	-.30***	.43***	-.01	.41***	-.48***	-.56***	-.43***	.59***	-.47***	.56***	.41***	-.54***	-.58***	-.54***	-		
23.Connected	.11	-.07	-.47***	.53***	.50***	.48***	-.34***	.47***	-.34***	.13	-.53***	.58***	.60***	.52***	-.42***	.64***	-.53***	-.53***	.70***	.67***	.71***	-.61***	-	
24.Stress	-.04	-.13	.38***	-.43***	-.50***	-.47***	.49***	-.38***	.61***	.02	.49***	-.57***	-.68***	-.53***	.56***	-.60***	.72***	.49***	-.63***	-.69***	-.66***	.75***	-.65***	-

$^{\dagger}p \leq .10$. $^{*}p \leq .05$. $^{**}p \leq .01$. $^{***}p \leq .001$.

Table 6

Raw Correlations Between Change in Telomere Length and Change in Psychological Measures

	1	2	3	4	5	6	7	8
1. Telomere Length Change	-							
2. Loneliness Change Score	-.08	-						
3. Life Satisfaction Change Score	-.02	-.40***	-					
4. Flourishing Change Score	-.09	-.36***	.42***	-				
5. Average Positive Affect	.07	-.18**	.32***	.24***	-			
6. Average Negative Affect	-.06	.21**	-.29***	-.28***	-.52***	-		
7. Average Connectedness	.01	-.25***	.29***	.22**	.71***	-.72***	-	
8. Average Stress	-.05	.15†	-.28***	-.20**	-.66***	-.81***	-.72***	-

†$p \leq .10$. *$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

Table 7

Partial Correlations Between Change in Telomere Length and Change in Psychological Measures

	1	2	3	4	5	6	7	8
1. Telomere Length Change Score	-							
2. Loneliness Change Score	-.04	-						
3. Life Satisfaction Change Score	-.03	-.43***	-					
4. Flourishing Change Score	-.11	-.36***	.47***	-				
5. Average Positive Affect	.05	-.10	.31***	.27**	-			
6. Average Negative Affect	-.01	.19*	-.30***	-.30***	-.55***	-		
7. Average Connectedness	.00	-.21*	.25**	.24**	.73***	-.75***	-	
8. Average Stress	-.06	.15†	-.27***	-.24**	-.68***	.80***	-.74***	-

Note. Partial correlations controlled for age, sex, BMI, hormone medication use, and illness symptoms at baseline and posttest.
†$p \leq .10$. *$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

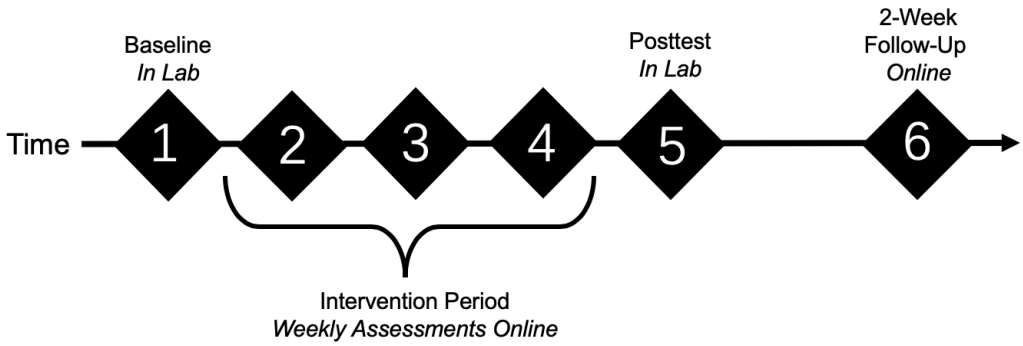

Figure 1. Study timeline.

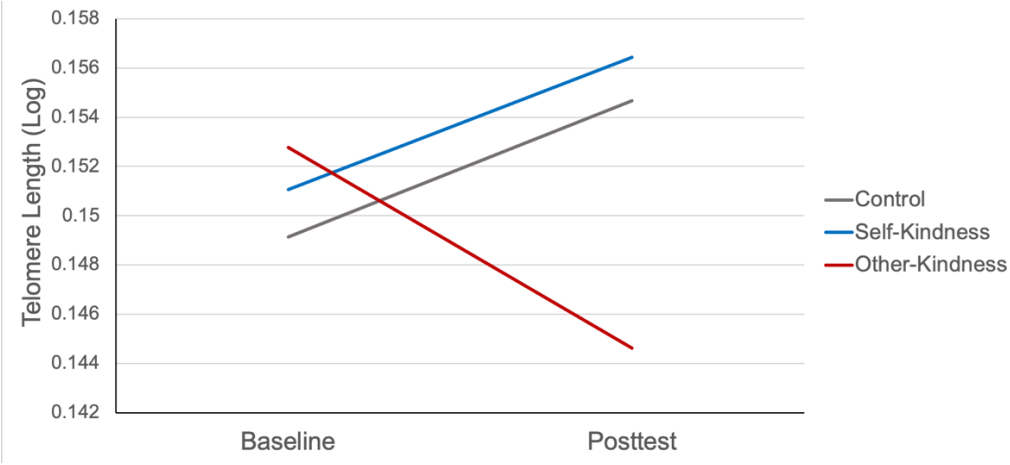

Figure 2. Change in LTL by condition.

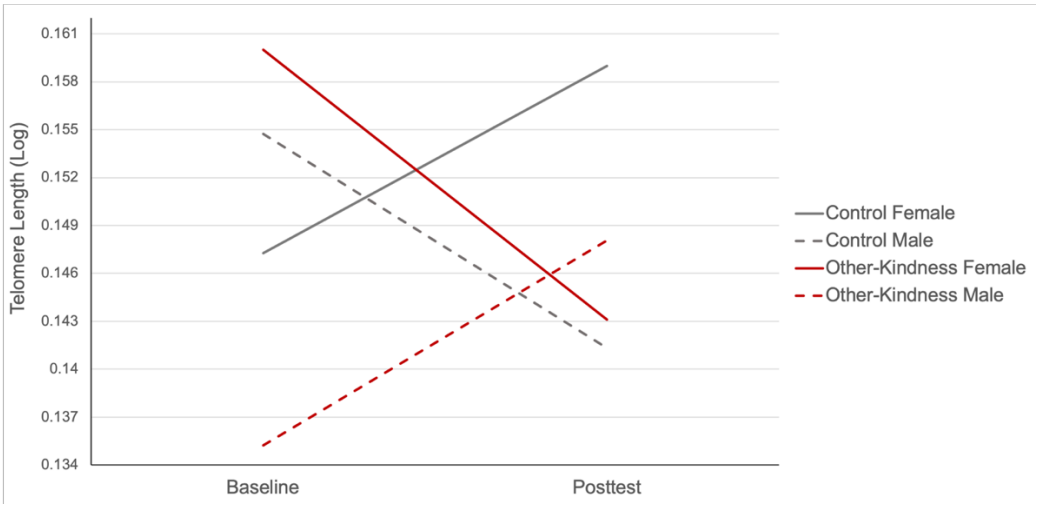

Figure 3. Change in LTL by condition and sex.

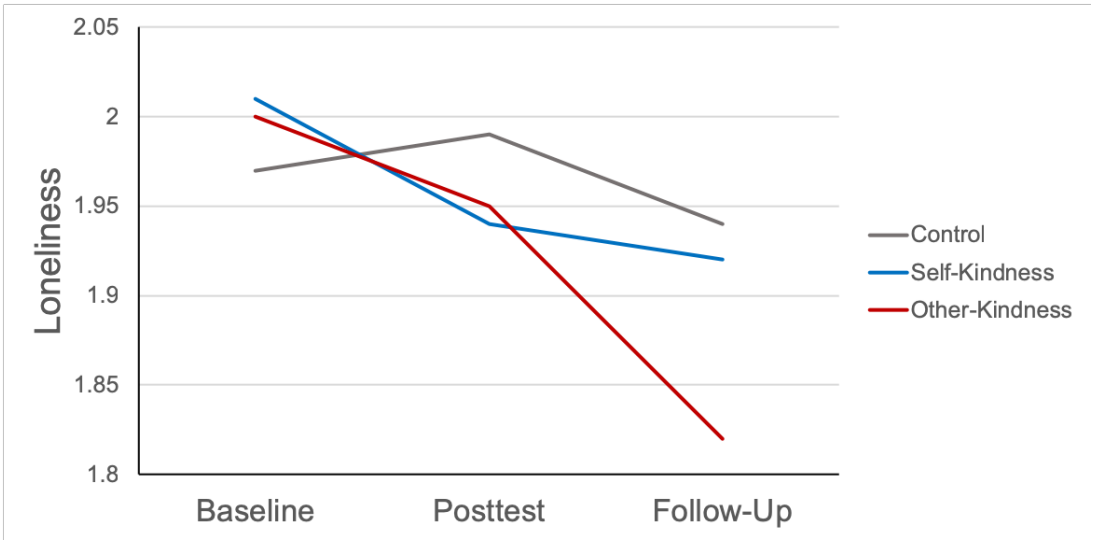

Figure 4. Loneliness by condition.

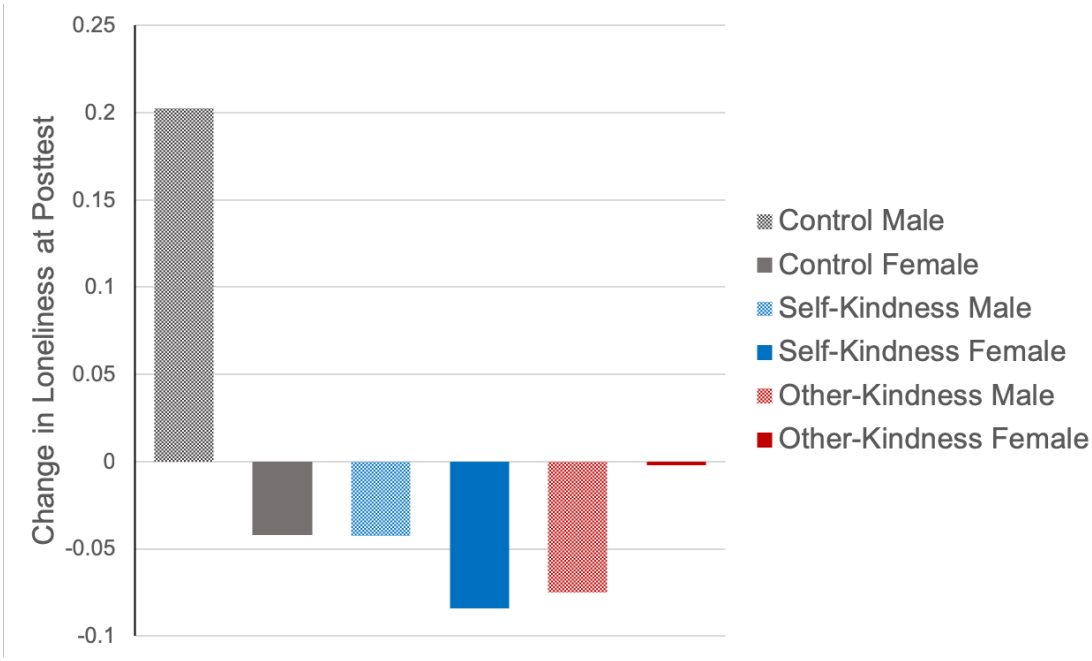

Figure 5. Loneliness change scores by condition and sex.

Appendix A.

OTHER-KINDNESS INTSTRUCTIONS

In our daily lives, we all perform acts of kindness, generosity, and thoughtfulness—both large and small—for others. Examples include cooking dinner for friends or family, doing a chore for a family member, paying for someone's coffee in line behind you, visiting an elderly relative, or writing a thank you letter. Tomorrow, you are to perform three nice things for others, all three in one day. These acts of kindness do not need to be for the same person, the person may or may not be aware of the act, and the act may or may not be similar to the acts listed above. Next week, you will report what nice things you chose to perform. Please do not perform any kind acts that may place yourself or others in danger.

SELF-KINDNESS INSTRUCTIONS

In our daily lives, we all perform acts of kindness for others, but we often neglect to do nice things for ourselves. Tomorrow, you are to perform three acts of kindness for yourself, all three in one day. These nice things that you do for yourself could be large (e.g., enjoying a day trip to your favorite hiking spot or a day at the spa) or they could be small (e.g., taking a 5-minute break when feeling stressed), but they should be something out of the ordinary that you do for yourself with a little extra effort. Examples include having your favorite meal, treating yourself to a massage, or spending time on your favorite hobby. These nice things for yourself do not need to be the same as the examples listed above, and although they may involve other people, they should be things that you do explicitly for yourself, not others.

CONTROL INSTRUCTIONS

Tomorrow, as you go about your day, please keep track of your activities. You do not need to remember who you are with or how you are feeling during that time. Instead, just try to remember factual information about what you are doing. Do not alter your routine in any way; simply keep track of what you do. When you log back in to the study, you will be asked to write an outline of what you did. For example: Morning: Ate breakfast, went to work, ate lunch with coworkers. Afternoon: Started a new project, held a meeting, went to the gym. Evening: Ate dinner, watched TV, went to bed. Only the facts are important.