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Objectives: Telomere length (TL) is a robust indicator of cellular aging. TL erosion has been associated with exposure to social and traumatic stressors. Loneliness and perceived social support are strongly linked to increased morbidity and mortality, but have yet to be investigated in relation to TL after extreme stress. The present study examined whether loneliness and lack of perceived social support following wartime captivity may be associated with TL as repatriated prisoners of war (ex-POWs) enter old age and contribute to its prediction. ***Method:*** A cohort of Israeli ex-POWs from the 1973 Yom Kippur War (n = 83) were assessed. Questionnaires were utilized to assess loneliness and perceived social support 18 years after the repatriation (T1), and Southern blotting was used to measure TL 24 years later (T2). A zero-order Pearson correlation test and a hierarchical regression analysis were utilized in order to examine the research questions. ***Results:*** Loneliness and lack of perceived social support each significantly predicted shorter TL in later life, and together added 25.8% to the overall explained variance. ***Conclusions:*** This is the first study to empirically demonstrate that loneliness and lack of perceived social support in early adulthood may be associated with shorter TL during transition to old age in a population that has endured extreme stress. Although the study design precludes causal inferences, several psychobiological mechanisms may explain the findings. The potential clinical significance of social deficits for longevity and heath in related populations is therefore addressed, and an agenda for future investigations is suggested.

Keywords: telomeres, wartime captivity, loneliness, social support, stress

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Telomeres are DNA nucleoprotein complexes located at the ends of chromosomes that protect the stability and integrity of the chromosomes (e.g., prevent the end of the linear chromosomal DNA from being recognized as a broken end; Blackburn, Epel, & Lin, 2015). Telomeres shorten with every consecutive DNA replication, and therefore telomere length (TL) is considered a robust marker of cellular aging (e.g., Müezzinler, Zaineddin, & Brenner, 2013). Shorter telomeres are associated with a weaker immune system and altogether higher vulnerability to age related diseases and mortality (e.g., Kaszubowska, 2008). Moreover, evidence indicates that this relation is reciprocal: telomere shortening can both facilitate disease etiology and progression and be a result of age-related diseases (Blackburn et al., 2015). Notwithstanding, the mechanisms of telomere attrition and its association with morbidity and mortality remain only partially understood. In this respect, the role that stress-related factors play in this process is rapidly gaining researchers' attention.

The link between stressful and traumatic experiences and TL erosion has been initially identified *in vitro* in relation to caregiver stress (e.g., Epel et al., 2004). Recent studies have also demonstrated that TL shortening is associated with combat stress (e.g., Bersani et al., 2016; Zhang et al., 2014), social stressors (e.g., Oliveira et al., 2016), and childhood stressors (e.g., Price, Kao, Burgers, Carpenter, & Tyrka, 2013), to name but a few. Studies

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seeking to uncover mechanisms linking stress to TL and identify factors that may exacerbate or mitigate this process are therefore an imperative. Nevertheless, such endeavors are in their infancy. A domain that seems to hold promise in this respect is the psychosocial (e.g., Hawkley et al., 2005; Uchino et al., 2012; Uchino et al., 2015).

The salutogenic effects of favorable social relationships are well documented (e.g., Holt-Lunstad, 2018; Holt-Lunstad, Robles, & Sbarra, 2017) and have been shown to predict mortality better than several well-established risk factors such as smoking, obesity, alcohol consumption, and physical exercise (Holt-Lunstad, Smith, & Layton, 2010). Thus, the constituting components of social relationships and their relation to health outcomes are constantly being further nuanced and differentiated (e.g., Gottlieb & Bergen, 2010; Holt-Lunstad et al., 2010). Such refinements are realized as an imperative for guiding intervention (Holt-Lunstad, 2018). In relation to TL it has therefore been argued that such fine-tuning is a "crucial next step" (Uchino et al., 2012, p. 531) for health psychology.

Perceived Social Support, Loneliness, Health, and Telomeres

Two social factors that may be differentiated in this respect are the subjective sense of being socially disconnected from others, which is captured by the concept of *perceived social isolation* or loneliness (J. T. Cacioppo & Cacioppo, 2014), and the subjective perception of the availability of necessary support provisions in the face of adversity, encapsulated in the phenomenon of *perceived social support* (Gottlieb & Bergen, 2010). Though the two concepts are not mutually exclusive and may be linked conceptually (Stein & Tuval-Mashiach, 2015b), they are nevertheless distinct. Thus, although some studies have viewed loneliness and perceived social support as complementary (e.g., Segrin & Passalacqua, 2010; Wang, Mann, Lloyd-Evans, Ma, & Johnson, 2018), others offered a competing view that compared the two factors' unique contributions to health outcomes. For instance, compared with loneliness, perceived social support was found to be a better predictor of disease outcomes among the elderly (Tomaka, Thompson, & Palacios, 2006) and a stronger predictor of mortality across studies (Holt-Lunstad et al., 2017). Therefore, it would seem that perceived social connection (or lack thereof) and perceived social support may function differently.

Several neural pathways may account for the buffering effect of perceived social support in the advent of stress (Ditzen & Heinrichs, 2014) and may be relevant to TL maintenance. These include the overall accommodation of the sympathetic nervous system in coping with the challenges of stress. Supported by empirical findings in the domain of fear extinction (e.g., Genud-Gabai Klavir, & Paz, 2013), it has been suggested that perceived social support may generate safety signals within the neural system, and these contribute to the extinction of fear learning and in the process affect cortical and amygdala activation (Ditzen & Heinrichs, 2014). The attenuation of the physiological stress response may lead to a lesser taxation of the cellular infrastructure and to some extent mitigate processes of TL erosion. Indeed, several studies (e.g., Brooks et al., 2014; Wiley, Bei, Bower, & Stanton, 2017) have indicated that psychosocial resources such as social support may attenuate recipients' allostatic loads (i.e., the cumulative wear and tear of physiological systems in the face of prolonged stress) and thus buffer the deleterious effects of stress on TL (Zalli et al., 2014).

Conversely, perceived social support was found to mitigate posttraumatic stress disorder (PTSD), anxiety, and depression in traumatized veterans (e.g., Neria, Besser, Kiper, & Westphal, 2010; Sripada, Lamp, Defever, Venners, & Rauch, 2016). All three psychiatric sequelae have been recognized as potential predictors of relatively shorter TL (Needham et al., 2015; Schutte & Malouff, 2015; Zhang et al., 2014). It therefore stands to reason that the contribution of perceived social support for their alleviation may result in TL maintenance.

It is noteworthy that the advantageous effects of social support for TL may depend on different factors. Empirical studies indicate that these may include the source of support (i.e., spousal support being beneficial but not so support from friends; Barger & Cribbet, 2016) and the age of support recipients (i.e., beneficial for old rather than young recipients; Carroll, Diez Roux, Fitzpatrick, & Seeman, 2013). It is therefore plausible that perceived support may relate to TL differently as a factor of the degree and nature of the stress involved.

Much like perceived social support, perceived social isolation has been repeatedly associated with detriments to health and longevity (e.g., J. T. Cacioppo & Cacioppo, 2014; Luo, Hawkley, Waite, & Cacioppo, 2012). From an evolutionary perspective, it is argued that humans, much like other social animals, depend on the togetherness of the group for survival (J. T. Cacioppo & Cacioppo, 2018). Concomitantly, it has been demonstrated across numerous studies in social neuroscience (e.g., J. T. Cacioppo, Cacioppo, Capitanio, & Cole, 2015; S. Cacioppo, Capitanio, & Cacioppo, 2014) that loneliness is wired into our physiological mainframe by evolutionary processes, serving as a signal that social ties must be strengthened. Though it has been suggested that loneliness may underlie TL erosion (Carroll et al., 2013; Hawkley et al., 2005), empirical investigations among older adults found no significant associations between loneliness and TL (Rius-Ottenheim et al., 2012; Schaakxs et al., 2016). However, these studies did not include individuals who have endured extreme stress.

Targeting aging veterans who have endured the extreme stress of wartime captivity in early adulthood, the current study examined the unique (i.e., competing) contributions of loneliness and perceived social support in early adulthood for TL during the transition to old age. To the best of our knowledge, no previous study has investigated such associations in populations exposed to episodes of such extreme stress, wherein social connectedness and support are not only vastly needed but often critically thwarted (Stein & Tuval-Mashiach, 2015a). The current study, therefore, fills an important gap in the literature.

The Case of Repatriated Prisoners of War and the Current Study

Combat veterans, and particularly repatriated prisoners of war (ex-POWs), face interpersonal challenges during and after their extremely stressful and traumatic episodes. The pernicious physical and psychological torments of wartime captivity often entail humiliation, torture, brutal interrogations, deprivation, and protracted solitary confinements (Lieblich, 1994). These accrue to a potentially devastating and extremely traumatic experience (Hunter,

1993). Because of the highly interpersonal nature of the trauma, wartime captivity may result in severe detriments in social relationships thereafter (e.g., loneliness, insecure attachment, impaired marital relations; Stein, Snir, & Solomon, 2015). Furthermore, the long-lasting aftereffects of wartime captivity often entail psychopathological reactions such as PTSD (e.g., Rintamaki, Weaver, Elbaum, Klama, & Miskevics, 2009), anxiety and depression (e.g., Ginzburg, Ein-Dor, & Solomon, 2010), and insalubrious outcomes that may include more somatic complaints, increased morbidity and higher mortality rates (Solomon et al., 2014). For these reasons these populations may be most suitable for investigating the contribution of both loneliness and perceived social support to TL following extreme stress.

Undeniably, loneliness and lack of perceived social support among veterans are not mutually exclusive and may reciprocally implicate one another (Stein, 2017). An investigation seeking to unravel the toll that social deficits may exert on TL following such adversity must therefore consider loneliness and perceived social support in tandem, and evaluate their unique contributions. The current study investigated TL in a cohort of Israeli ex-POWs from the 1973 Yom Kippur War 42 years after repatriation in relation to their reports of loneliness and perceived social support nearly 24 years earlier. It was hypothesized that higher rates of loneliness and lower rates of perceived social support would be associated with shorter TL in later life (H1), and that both would add a unique contribution to the prediction of later life TL (H2).

Method

Participants

The present study capitalized on data from a longitudinal study focusing on the psychological and biological implications of war and captivity (Solomon et al., 2017). A cohort of 240 male Israeli veterans of the Israeli Defense Force (IDF) who fought in the 1973 Yom Kippur War and fell captive on the Egyptian and Syrian fronts was approached in 1991 (T1) and revisited four times, the last of which was in 2015 (T2 in the current study; for full details, see Solomon et al., 2017). Participant mean age at T2 was 63.6 years (SD = 3.7, range = 61–77). Twenty participants (22.7%) reported smoking, 10 (11.4%) reported substance abuse, 15 (17%) reported medications for sleep and anxiety, and 68 (79.1%) reported usage of medications for health problems.

Of the 158 who participated at T2, 101 were randomly summoned for medical examinations that included the TL tests. Of these, 99 (98%) agreed to complete the examinations and had valid data attained from the test. Considerations for summoning only a subsample of the participants related primarily to budget restrictions. Data was anchored to include only participants with TL data at T2, as explicated below. No education differences were found between those who agreed to participate in T2 compared with those who did not, $t(121) = 1.15$, $p = .35$. No differences were found with regard to their T1 PTSD, $t(276) = .93$, $p = .35$, or T1 depression, $t(161) = .74$, $p = .46$, in T1. T2 PTSD differences were found between participants who agreed to participate in filling in questionnaire for the T2 measurement but were not summoned to participate in the TL examination and those who participated in both filling in questionnaires and TL examination, $t(287) = 9.52$, $p < .001$. Participants who agreed to also do the TL (M = 9.93, SD = 6.06) test were higher in PTSD symptoms compared with those who did not (M = 3.09, SD = 5.44). The groups were no different in depression, $t(161) = .74$, $p = 46$.

The participants were relatively healthy though a small proportion of them reported health problems: 20 (24.4%) reported having diabetes, eight (9.6%) reported kidney diseases, four (4.9%) reported cancer, three (3.8%) reported a tumor, three (3.6%) had undergone a stroke, 19 (22.6%) had heart failure, seven (8.8%) reported pulmonary problems, and 34 (40%) reported high blood pressure.

Potential participants were drawn from updated IDF files and approached by phone. At every assessment point, participants signed an informed consent form prior to participation. The battery of questionnaires was administered in medical centers in central Israel. The collection of telomere data was done via blood samples extracted as part of a thorough medical examination (approximately 3 to 4 hr long) at the Tel-Aviv Sourasky Medical Center, the results of which were given to the participants. Blood samples were analyzed at the Rabin Medical Center. The study was approved by the Tel Aviv University Institutional Review Board prior to each wave of measurement and by the Sourasky Medical Center's Helsinki Committee prior to the last assessment.

Measures

Background variables and demographics. Given that previous studies have indicated that TL may be implicated by depression and PTSD, as well as age, body mass index (BMI), smoking and substance abuse, these were assessed at T2, as were demographic variables. Although smoking and substance abuse were dichotomous (yes was coded as 1 and no was coded as 0), BMI was coded as a continuous variable.

Loneliness. Loneliness was measured only at T1 via the commonly used UCLA loneliness scale (Russell, Peplau, & Cutrona, 1980). Initially, the questionnaire consists of 20 items relating to social connection and disconnection. Participants were asked to indicate how often they had experienced these feelings on a four-point scale ranging from 1 (*not at all*) to 4 (*very often*). The total score of the scale was the mean of all items after having reversed the positively worded items. High scores reflect greater levels of loneliness. Cronbach's alpha in the current sample was .89, indicating high internal consistency.

Perceived social support. Perceived social support was assessed only at T1 by a seven-item questionnaire devised in our laboratory on the basis of Mueller's (1980) social network approach. Items reflected the availability of expressive and instrumental support (e.g., emotional help, financial support) from participants' network members (e.g., "There are people in my surrounding that I can speak to openly even about the most intimate things," "I have friends who will remain real friends even if I get into trouble"; for the full questionnaire, see the online supplemental material). Respondents were asked to indicate the personal relevance of each statement on a four-point scale 1 (*not at all*) to 4 (*very often*). Perceived social support was calculated by averaging answers to the seven questions. To achieve a consistency with loneliness the measure was reversed. Thus, higher scores indicate the lack of perceived social support rather than its presence. Cronbach's alpha was .87, indicating high internal consistency.

PTSD symptomatology. A questionnaire based on the PTSD Inventory (PTSD-I; Solomon et al., 1993) was used to assess PTSD symptoms at both time points. At T2, four items were added to the initial 17 items of the questionnaire to reflect *DSM–5* symptoms of PTSD (reexperiencing, avoidance, negative alterations in cognition, and hyper-arousal and reactivity; American Psychiatric Association, 2013). Participants rated each symptom/item as it has been experienced in the previous month on a four-point Likert scale ranging from 1 (*not at all*), to 4 (*almost always*). Endorsement of a symptom was considered when an item was rated 3 or 4. PTSD symptoms were computed as a continuous variable indicating the number of symptoms endorsed. The PTSD-I has shown strong reliability and convergent validity when compared with diagnoses based on structured clinical interviews (Solomon et al., 1993) and has shown high internal consistency in its updated form (Solomon et al., 2017). In the current study, the internal consistency was high ($\alpha = .9$).

Depression. Depression was assessed at both time points using the six-item depression subscale of the commonly used Symptom Checklist-90 (Derogatis, 1977). Participants were asked to indicate how frequently they experienced each symptom in the last 2 weeks on a five-point Likert scale ranging from 1 (*not at all*) to 5 (*extremely*). Depression was calculated by averaging responses on all items. High internal consistency was found in this study ($\alpha = .9$).

Telomere length. TL was measured at T2 by utilizing the Southern blot (as described in Uziel et al., 2007). Telomeres were measured in total white blood cells obtained from 10 ml of blood. Cell composition was not measured. Genomic DNA was extracted (ArchivePure; 5-prime) according to the manufacturer's instructions and quantified (NanoDrop; Thermo). DNA, 5 mg, was digested for 16 hr with *RSAI* and *HINFI*, (TTAGGG length assay; Roche). DNA integrity was examined by running it separately on an agarose gel (not shown) as a routine procedure prior to the Southern Blotting process. The digested DNA was separated by gel electrophoresis (0.6% agarose), de-purinated by HCl 0.25M, denatured with alkaline denaturing solution (NaOH 0.5M, NaCl 1.5M) and then neutralized (Tris 0.5M, NaCL 3M). Subsequently, the DNA was capillary-transferred onto a positively charged whatman nylon membrane (Roche) for 16 hr. The DNA was then UV-cross-linked (120mJ) to the membrane and incubated for 16 hr with DIG-labeled TL probe $(CCCTAA)_4$. The membrane underwent washes as follows: twice in Stringent wash buffer I (2X SSC, 0.1% SDS) for 5 min at reaction time (RT), twice in Stringent wash buffer II (0.2X SSC, 0.1% SDS) for 15 min at 50 °C, in 1X maleic acid buffer (supplied by the TTAGGG length assay kit; Roche) for 5 min, in blocking solution (kit) for 30 min at RT, in Anti-DIG-AP solution for 30 min at RT, twice in washing buffer (kit) for 15 min at RT and, finally, in detection solution (kit) for 5 min at RT. The membrane was then applied with ~40 drops of CSPD substrate and exposed to a sensitive film for 1.5 hr. After development, the film was scanned and quantified by the Quantity One software (Versadoc; BioRad). To calculate TL, each signal was segmented and its intensity was measured. TL was calculated according to the following equation:

$$\frac{\sum (OD_i)}{\sum (OD_i/L_i)},$$

where OD_i is the chemiluminescent signal and L_i is the length of the telomere at position i.

Analytic Strategy

Participant fluctuation is an inherent part of longitudinal study designs and must be addressed to avoid potential sampling biases (Collins, Schafer, & Kam, 2001). The data were anchored to include only participants with TL data at T2 ($n = 83$). However, there was a small amount of missing data in age and BMI (3.4%) and with a few of the loneliness and perceived social support items (10.2% to 17.5%). Little's missing completely at random test revealed that the data were missing at random, $\chi^2(4) = 3.09$, $p = .38$. SPSS 24 was used for a maximum likelihood estimation procedure (Schafer & Graham, 2002).

Zero-order Pearson correlations were calculated between TL, age, BMI, PTSD symptoms, depression, lack of perceived social support and loneliness. In order to assess the predictive significance of loneliness and perceived social support for later life TL a hierarchical linear regression for the prediction of TL at T2 was conducted. Because of considerations of statistical power related to the sample size, the amount of variables that could be entered into the regression was limited. Thus, covariates included only variables measured at the time of TL measurement (i.e., T2) and the individual contribution of each health problem could not be entered into the regression. The covariates: age, BMI, depression, and PTSD, and dummy variables of smoking and substance abuse (1 = yes, 0 = no) were entered in Step 1, loneliness and perceived social support at T1 were entered in Step 2. The social factors were entered into the regression together because they are conceptually related to one another and because there was no theoretical justification for a specific order of inserting one before the other.

Results

TL ranged between 9.90 kilo base pairs (kb) and 2.50 kb ($M = 5.32$; $SD = 1.5$). Preliminary analyses were performed to ensure that there was no violation of the assumption of normality. The data were then examined using recommended skew values of less than 2 and kurtosis values of less than 7. Skew values between 2 and 3 and kurtosis values less than 10 were considered moderately nonnormal (Curran, West, & Finch, 1996). Although all variables were in the recommended acceptable range for skewness (0.74) and kurtosis (−0.4), six outliers were identified in the TL measures, signified by large gaps in the histogram and a *Z* score greater or lower than 3.33 (long telomeres ranged from 8 to 9.90 kb and shorter telomeres ranged from 2.5 to 3.2 kb, whereas most of the sample ranged between 3.7 to 6.5 kb, as reflected in the mean value). The values of the kurtosis and skewness remained unaffected by the outliers. Moreover, the outliers were within the documented range of human TL variability (Samassekou, Gadji, Drouin, & Yan, 2010). Therefore, the outliers were not excluded from the analyses. To determine whether outliers had significant effects concerning the research questions, analyses were performed also after Winsorizing the outliers (i.e., exclude them from the sample). The results indicated that though coefficients changed slightly, the explained variance and significance remained the same.

As is evident in Table 1, zero-order Pearson correlations revealed that lack of perceived social support and loneliness were both negatively correlated with TL (see Figure 1). Age was negatively correlated with PTSD and depression symptoms. Depression was positively correlated with PTSD, lack of perceived social

Table 1
Correlation Matrix

Variable	1	2	3	4	5	6	7
1. TL	—						
2. Age	−.07	—					
3. BMI	.11	−.05	—				
4. Depression	−.09	−.22*	−.04	—			
5. PTSD symptoms	.06	−.24*	−.01	.74***	—		
6. Lack of PSS	−.37***	.02	−.05	.24*	.17	—	
7. Loneliness	−.43***	−.13	−.06	.28**	.16	.35**	—

Note. TL = telomere length; BMI = body mass index; PTSD = posttraumatic stress disorder; PSS = Perceived social support.
* $p < .05$. ** $p < .01$. *** $p < .001$.

support, and loneliness. Lack of perceived social support and loneliness were positively correlated.

The hierarchical linear regression used to determine the unique and combined contribution of loneliness and perceived social support to the prediction of later life TL yielded a significant model, $F(8, 78) = 4.69$, $p < .000$, $R^2 = 32.5\%$, specifically, as evident in Table 2, save for smoking that was associated with shorter TL, the covariates failed to significantly contribute to the prediction of TL in the final analysis. Though depression contributed significantly to shorter TL in Step 1, it was no longer significant in Step 2. Importantly, both loneliness and lack of perceived social support at T1 significantly and negatively predicted TL at T2, and added 23.6% of explained TL variance in the ex-POWs' later life.

Discussion

The current study examined whether perceived social isolation (i.e., loneliness) and perceived lack of social support in early adulthood are associated with TL in later life in a sample that has been exposed to extreme stress such as wartime captivity. As hypothesized (H1), loneliness and lack of perceived social support 18 years after repatriation were both significantly and negatively associated TL 24 years later. Moreover, as hypothesized (H2), both factors uniquely contributed to later life TL. Interestingly, PTSD and depressive symptoms were not related to TL, and they did not contribute to its prediction. These findings are inconsistent with previous studies among older adults, wherein no significant associations between loneliness and TL were found (Rius-Ottenheim et al., 2012; Schaakxs et al., 2016) as well as findings that associate shorter TL with depression (e.g., Schutte & Malouff, 2015) and PTSD (e.g., Zhang et al., 2014). In an attempt to may account for the differences in findings, it may be speculated that the extreme interpersonal nature of the traumatic stressors of wartime captivity gives rise to a greater vulnerability to social threats in its aftermath (Stein et al., 2015), which in turn increases also cellular vulnerability that hamper TL maintenance.

Nevertheless, the preceding findings are consistent with a formidable body of literature indicating that the insalubrious effects of low perceived social support have a psychobiological infrastructure (Ditzen & Heinrichs, 2014) and that the deleterious effects of loneliness may be associated with multiple behavioral, neurological, endocrine, and cellular adjustments (e.g., J. T. Cacioppo & Cacioppo, 2018), several of which may implicate TL. For instance, loneliness has been associated with poor sleep (e.g., Matthews et al., 2017), which in turn has been associated with shorter TL (Jackowska et al., 2012). Finally, health behaviors associated with loneliness (e.g., smoking; Shankar, McMunn, Banks, & Steptoe, 2011) may also be associated with shorter TL (e.g., Puterman, Lin, Krauss, Blackburn, & Epel, 2015).

One primary mechanism that may be at the epicenter of these processes involves the over activation of the hypothalamic–pituitary–adrenocortical (HPA) axis (e.g., cortisol awakening responses). The HPA plays a pivotal role in reactions to anticipated stress and may be implicated by social factors such as perceived support (e.g., Hostinar, Sullivan, & Gunnar, 2014) and loneliness (e.g., J. T. Cacioppo et al., 2015; Okamura, Tsuda, & Matsuishi, 2011; Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004; Zilioli et al., 2017). This heightened activation of the HPA in the face of perceived social isolation has been explained by the evolutionary perspective as a result of a shift in "emphasis from mutual aid and protection to self-preservation in a potentially hostile world" (J. T. Cacioppo & Cacioppo, 2018, p. 25). Lonely individuals evince heightened vigilance to social threats and do not benefit from the soothing sense of safety provided by the group. Thus, their sympathetic nervous system remains alert and ready for encountering potential threat. Evidence in previous studies indicates that such a heightened activation of the HPA axis may result in an overall more taxing allostatic load and shorter TL (Tomiyama et al., 2012). Conversely, evidence suggests that this process may be partially mitigated by increasing social resources and support (Brooks et al., 2014; Zalli et al., 2014), possibly by generating safety signals that may attenuate the activity of the HPA system (Ditzen & Heinrichs, 2014).

Other potential pathway related to the allostatic burden may include the toll that protracted pain exerts on the physiological system. Empirical evidence indicates that chronic pain may be conducive to a cumulative allostatic load (Sibille, McBeth, Smith, & Wilkie, 2017) and shorter TL (Sibille, Witek-Janusek, Mathews, & Fillingim, 2012). Loneliness has been conceptualized as a psychological pain (Stein & Tuval-Mashiach, 2015b) and has been shown to share neural circuitries with physical pain (e.g., Eisenberger, 2012). Therefore, it is plausible that the pain that loneliness entails is also related to its association to TL.

Finally, studies among animals and humans have also found that loneliness is associated with proinflammatory conditions (e.g., Karelina & DeVries, 2011; Jaremka et al., 2013) and a diminished immune system (e.g., Pressman et al., 2005). This inflammatory vulnerability has been associated with the activation of the conserved transcriptional response to adversity (CTRA; Slavich & Cole, 2013): a pattern of upregulated proinflammatory immune response gene activity and down-regulated antiviral immune response gene activity intended to facilitate wound healing and limit infection. Anticipating physical or social threat, the combination of neurobiological activation of leukocyte inflammatory genes and the inhibition of innate antiviral genes activates the CRTA. What may have been in the distant past an adaptive response to potentially injuring physical threat and bacterial infection, has become less adaptive in contemporary social environments wherein (a) actual threats of injury and bacterial infection are less prevalent, (b) threats of socially mediated viral infections are on the rise, and (c) social threats such as social disconnection may activate the CTRA in the absence of an actual physical threat (J. T. Cacioppo

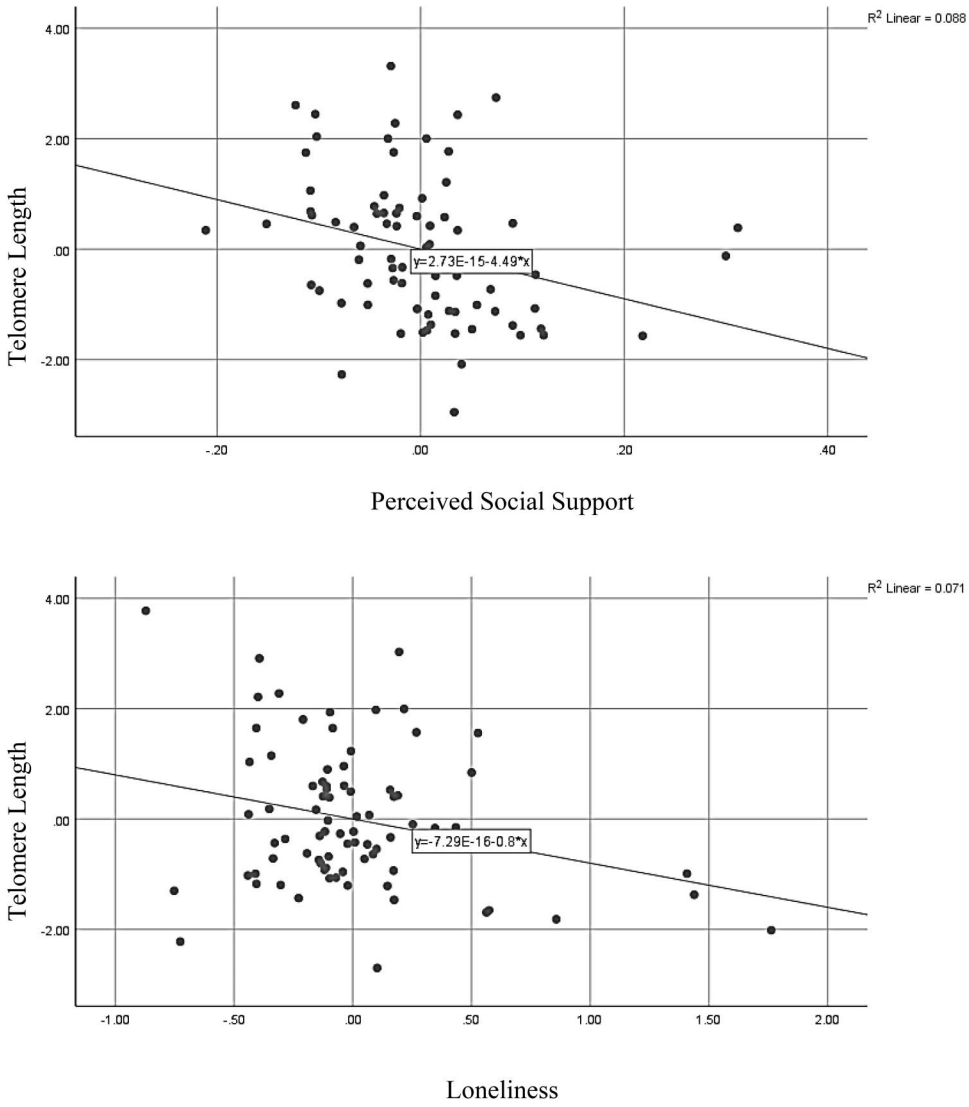

Figure 1. Adjusted scatterplots for telomere length, perceived social support and loneliness.

& Cacioppo, 2018; Slavich & Cole, 2013). Given that inflammation and a diminished immune system have been associated with TL erosion (e.g., Cohen et al., 2013; Effros, 2011; Jergović et al., 2014; Kaszubowska, 2008), the maladaptive activation of the CTRA in the face of perceived social threat (e.g., loneliness) may facilitate TL shortening.

Though tentative and in no way exhaustive, taken together the pathways delineated above demonstrate the multifarious manners in which social deficits such as loneliness and low perceived social support may gradually and cumulatively tax a person's cellular infrastructure and perhaps contribute to premature TL erosion. Future research should investigate the interplay between psychobiological and behavioral mechanisms underlying the link between loneliness, supportiveness and TL, and seek to expand the current findings by empirically establishing casual mechanisms that underlie such associations.

Trauma is related to health impediments via multiple pathways (e.g., sleep dysregulation, allostatic load, depression; e.g., McFarlane, 2010; Schnurr & Green, 2004). Furthermore, recent findings reveal relatively high mortality rates and worse health outcomes among ex-POWS in particular (Solomon et al., 2014). TL erosion associated with loneliness and lack of perceived social support may play a mediating role in these processes (i.e., social deficits and TL as sequential mediators of the relation between trauma and health and mortality) as well as an end product of trauma related health impediments (i.e., social deficits and health as sequential mediators of the relation between traumatic stress and TL). Indeed, the contemporary understanding is that TL and age related diseases are reciprocally implicated (Blackburn et al., 2015). Investigating possible reciprocities between social factors, traumatic stress and cellular damage should therefore set an agenda for studies in health psychology.

Clinical Implications

Given the aforementioned relations between protracted traumatic stress and impaired health (e.g., Schnurr & Green, 2004) and

Table 2
Predicting Telomeres Length at 2015 by Loneliness and Social Support at 1991

Variable	*b*	*SE*	β	*t*	*p*
		Step 1			
Age	−.05	.05	−.11	−.97	.34
BMI	.03	.03	.13	1.16	.25
Smoking	−.88	.53	−.23	−1.65	.10
Substance abuse	.59	.69	.12	.85	.40
PTSD symptoms	.10	.05	.29	1.83	.07
Depression	−.57	.26	−.36	−2.15	.03
$R^2\Lambda$ = 8.9%, *F* change = 1.29, $p = .27$					
		Step 2			
Age	−.06	.04	−.13	−1.32	.19
BMI	.02	.02	.10	1.07	.29
Smoking	−1.05†	.47	−.28†	−2.25	.03
Substance abuse	.78	.62	.16	1.27	.21
PTSD symptoms	.08	.04	.25	1.78	.08
Depression	−.28	.24	−.18	−1.19	.24
Loneliness	−1.18**	.33	−.38***	−3.60	.001
Lack of PSS	−1.98†	.88	−.24†	–2.25	.027
$R^2\Lambda$ = 23.6%, *F* change = 13.65, $p < .000$					

Note. PTSD = posttraumatic stress disorder; BMI = body mass index; PSS = perceived social support.
† $p \leq .05$. ** $p < .01$. *** $p < .001$.

the worse health outcomes and high mortality rates found among ex-POWS (Solomon et al., 2014), mitigating premature aging and enhancing longevity seem to be an imperative for clinical interventions. The present findings suggest that interventions that alleviate loneliness (Mann et al., 2017; Masi, Chen, Hawkley, & Cacioppo, 2011) and foster social resilience (e.g., J. T. Cacioppo, Reis, & Zautra, 2011) may be vital in this respect because they may potentially of facilitate TL maintenance.

Specifically, in the case of ex-POWs, wherein the traumatic stress was interpersonal and therefore implicated in several interpersonal deficits (e.g., insecure attachment, distrust, marital dissatisfaction; Stein et al., 2015), it may be imperative to reinstate trust in a supportive network. This may be done by fostering communal coping (Lyons, Mickelson, Sullivan, & Coyne, 1998), "the pooling of resources and efforts of several individuals (e.g., couples, families or communities) to confront adversity" (p. 580). As people increasingly share their distress, it is plausible that the individual impact of each stressor decreases. As Helgeson, Jakubiak, Van Vleet, and Zajdel (2018) suggested, as partners "share" the management of an illness by appraising it as "our" problem rather than that of one of the partners they attenuate the effects on the individual. Diminishing veterans' conviction that they must cope alone with their traumatic pasts (Stein, 2017), such coping may promote a sense of connection and support and thus assuage the allostatic toll on TL (Zalli et al., 2014).

Limitations

The current findings must be interpreted in the context of several limitations. First, the investigation included a relatively small sample consisting solely of male participants. The small sample restricted the amount of variables that could be examined and sets certain constraints for the generalization of the findings. Second, the study design that was employed, albeit longitudinal, was not experimental and lacked prospective data (e.g., pre- and postmeasures of loneliness, perceived social support and TL). Furthermore, the single assessment of each variable and the amount of time elapsed between the assessment of loneliness and perceived social support and that of TL may introduce a number of confounds that the study was unable to address. The present study is, therefore, limited in its ability to determine causality, as are most studies in the field (Holt-Lunstad, 2018; Uchino, Bowen, Carlisle, & Birmingham, 2012a). Finally, TL was measured in this study using total white blood cells, and thus cell composition was not measured. Given that TL may differ between leukocyte subsets (Baerlocher & Lansdorp, 2003), total blood TL may potentially be confounded. Future research should readdress the question targeted in the current study prospectively, with larger and more gender heterogeneous samples, and examine TL in distinct leukocyte subsets. Moreover, as the relation between social support and TL may too be nuanced (e.g., depending on the source of support or age of support recipient; Barger & Cribbet, 2016; Carroll et al., 2013), such nuances should be accounted for in the future.

Notwithstanding the limitations, the current study is the first to identify a long-term association between loneliness and perceived social support and cellular aging in a sample that has been exposed to extreme stress such as wartime captivity. These findings may guide intervention as well as inspire new research that may drive forward the burgeoning field of social genomics (Slavich & Cole, 2013). In the timely endeavor of bridging relationship science and health (Uchino, 2013; Thoits, 2011), the current findings are an important step.

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