Full citation:

Lahav, Y., Avidor, S., Stein, J. Y., Zhou, X., & Solomon, Z. (2020). *Telomere length and depression among ex-prisoners of war: The role of subjective age.* The Journals of Gerontology: Series B, 75(1), 21–29. https://doi.org/10.1093/geronb/gby006

Publisher's version: Available at: https://doi.org/10.1093/geronb/gby006

Telomere Length and Depression among Ex-Prisoners of War: The Role of Subjective Age

Yael Lahav PhD[1], Sharon Avidor PhD[2], Jacob Y. Stein PhD[1,3], Xiao Zhou PhD[1], Zahava Solomon PhD[1,3]

[1] I-Core Research Center for Mass Trauma, Tel-Aviv University, Israel

[2] Ruppin Academic Center, Emek Hefer, Israel

[3] Bob Shapell School of Social Work, Tel-Aviv University, Israel

* Address correspondence to Dr. Yael Lahav, I-Core Research Center for Mass Trauma, Tel-Aviv University, Chaim Levanon 30, Tel-Aviv, 6997801, Israel; E-mail: lahav.yael62@gmail.com; Phone: +972-54-4525084

Abstract

Objectives: Exposure to captivity increases the risk for multiple disturbances that may intensify during old age. In later phases of life, former-prisoners-of-war (ex-POWs) may suffer from depression as well as from accelerated aging, manifested in older subjective age and leukocyte telomere shortening. The current study assesses the link between these varied facets of increased vulnerability during old age and explores (a) the associations between subjective age and telomere length; (b) the mediating role of changes in subjective age over time within the associations between depression and telomere length.

Methods: Eighty eight ex-POWs were assessed prospectively 30 (T1), 35 (T2) and 45 (T3) years after the 1973 Israeli Yom-Kippur War. Depression was assessed at T1; subjective age was assessed at T2 and T3; and telomere length and control variables were assessed at T3.

Results: Older subjective age at T3 was associated with concurrent shorter telomeres, beyond the effect of chronological age. Change in subjective age between T2 and T3 mediated the relations between depression at T1 and shorter telomeres at T3 beyond the effects of control variables.

Discussion: Findings suggest that the detrimental ramifications of accelerated subjective age involve premature cellular senesces, and may explain the relation between depression and accelerated aging processes among trauma victims. Hence, clinical interventions may seek to address accelerated subjective age among trauma survivors who suffer from depression.

Keywords: accelerated aging, trauma, depressive symptoms, telomere shortening, subjective age

Introduction

War captivity is one of the most extreme traumatic events inflicted by men, and its aftermath is not only associated with long term psychological and physiological difficulties, but also with accelerated aging (Avidor, Benyamini, & Solomon, 2014; Solomon et al., 2017). Within this context, the current study assessed the link between the psychological and biological facets of accelerated aging, and the possible mediating role of subjective age within the relations between depression and leukocyte telomere shortening, among former-prisoners-of-war (ex-POWs).

Exposure to war captivity entails prolonged and complex stressors, that increase the risk for multiple psychiatric (Rintamaki, Weaver, Elbaum, Klama, & Miskevics, 2009) and physical disturbances (Lahav, Rodin, & Solomon, 2015), even many years after repatriation. Such detriments may be exacerbated as ex-POWs grow older. The combination of facing age-related losses (e.g., loss of loved ones, declining physical health) and a propensity for engaging in a more introspective stance relating to the life-review in one's later years may result in the reactivation of past traumatic experiences (Gagnon & Hersen, 2000) and thus give rise to elevated distress. Research indicates that later life trauma survivors are at high risk for increases in somatic and psychological distress including deteriorations in health, posttraumatic stress symptoms and depression (Dekel, Solomon, Horesh, & Ein-Dor, 2014; Rintamaki et al., 2009; Solomon et al., 2014).

At the same time, literature suggests that a process of trauma-induced premature aging may take place, wherein both biological and psychological/subjective indicators of aging undergo premature alterations for the worse. Among the insalubrious ramifications of war captivity is the premature manifestation of diseases that typically emerge later in life (e.g., Beebe, 1975). Similarly, entering old age, ex-POWs were found to exhibit higher morbidity (Nice, Garland, Hilton, Baggett, & Mitchell, 1996), dementia (Meziab et al., 2014), and early

mortality (Solomon et al., 2014). It has been argued that the etiological factor underlying these diseases is premature or accelerated ‘‘cellular aging,’’ which may be evident in leukocyte telomere shortening (Zhang et al., 2014).

Trauma and accelerated cellular aging - Telomere shortening

Telomeres are DNA-protein complexes that cap chromosomal ends, promoting chromosomal stability. Whenever cells divide, the telomeres are not fully replicated due to the limitations of the DNA polymerases in completing the replication of the ends of the linear molecules. This process leads to telomere shortening with every replication (Chan & Blackburn, 2004). Telomeres shorten with age and thus telomere length often serves as a biomarker of cellular aging. Shorter telomeres have been empirically linked with age-related conditions and diseases (Serrano & Andrés, 2004), and early mortality (Cawthon, Smith, O'Brien, Sivatchenko, & Kerber, 2003).

Research reveals associations between shorter telomere length and exposure to stressful (Price, Kao, Burgers, Carpenter, & Tyrka, 2013) and traumatic events (Bersani et al., 2016). Findings indicate that survivors of traumatic events such as childhood adversities, combat and war captivity, demonstrate relatively shorter telomeres than comparable samples (e.g., Bersani et al., 2016; O'Donovan et al., 2011). In the same vein, a previous study with the sample presented in the current study revealed that ex-POWs had shorter telomere length compared to equivalent combatants (Solomon et al., 2017).

Trauma and accelerated aging - Subjective age

A similar trend of premature aging among trauma survivors has been found concerning the psychological indicator of subjective age. Subjective age refers to the manner in which individuals perceive and appraise their age. Subjective age does not necessarily serve as an accurate representation of one's calendar age but as an important dimension of an individual’s self-concept. It is associated with one's subjective experience of the physical, psychological,

and social changes associated with age (Wiesmann, Becker, & Hannich, 2017).

Typically, from midlife and onwards, people report feeling younger than their actual age (Kleinspehn-Ammerlahn, Kotter-Grühn, & Smith, 2008). It is argued that this youth-oriented bias serves as a self-enhancing positive illusion (Mirucka, Bielecka, & Kisielewska, 2016) and as such is an identity-related resource (Wiesmann et al., 2017). According to theory, perceiving oneself as younger than one's chronological age, may act as a compensatory strategy that counteracts negative stereotypes and devaluation of old age in Western cultures (Weiss & Lang, 2012). Additionally, a younger age identification reflects a positive conception of the self as competent, valuable, and resourceful (Wiesmann et al., 2017).

In line with this view, previous studies have indicated that individuals in midlife or old age, who reported a younger subjective age exhibited higher levels of life satisfaction (Mirucka et al., 2016) improved physical and cognitive functioning (Stephan, Chalabaev, Kotter-Grühn, & Jaconelli, 2012) and higher longevity (Kotter-Grühn, Kleinspehn-Ammerlahn, Gerstorf, & Smith, 2009).

Nevertheless, this pattern is ostensibly thwarted following trauma exposure. Research consistently indicates that both exposure and psychological reactions to trauma are associated with older rather than younger subjective age (Schafer, 2009). A previous study among ex-POWs of the present sample, has similarly revealed that ex-POWs perceived themselves as older in relation to comparable combat veterans (Avidor et al., 2014).

Arguably, older subjective age and shorter telomere length among trauma survivors could represent different facets of the same phenomenon of accelerated aging. However, the link between them is far from being fully understood. Specifically, one may wonder whether one's subjective evaluation of age might be implicated in the individual's cellular aging (i.e., telomere length)?

Similar to other types of subjective appraisals (e.g., O'Donovan et al., 2012) one's subjective evaluation of age may affect the level of experienced stress, which in turn, may affect biological processes. Maintaining an older subjective age may confront the trauma survivor with negative stereotypes attributed to old age, and hinder the person's personal resources (e.g., Levy, Slade, Chung, & Gill, 2014). These, in turn, might intensify the stress, which will take a toll on biological systems, and eventually lead to increased erosion at the cellular level (Danese & McEwen, 2012).

Depression and accelerated aging

Though depression and posttraumatic stress disorder (PTSD) may coincide in the aftermath of trauma and in a pronounced way after war captivity, the unique features of either form of psychopathology may be discernable within such comorbidity (Dekel et al., 2014). Depressive symptoms may promote stress in and of themselves, and thus predict further vulnerability in later life (Price et al., 2013). This might manifest itself in psychological and biological accelerated aging (Wolkowitz, Reus, & Mellon, 2011).

Research has revealed a link between depression and telomere length (Zhang et al., 2014). Two meta-analyses indicated a significant correlation between depression and shortened telomere length among non-traumatized individuals (Ridout, Ridout, Price, Sen, & Tyrka, 2016; Schutte & Malouff, 2015). Yet, research concerning the association between depression and telomere length among trauma survivors is scarce, and findings are mixed. A study among combat-exposed male veterans found non-significant correlations between depression and telomere length (Bersani et al., 2016), while a recent report based on the present sample of ex-POWs indicated that the chronic trajectory of depression, rather than that of PTSD, was implicated in significantly shorter telomeres (Solomon et al., 2017).

Research has indicated an association between depression and older subjective age. A cross-sectional study found that participants who felt younger had lower adjusted odds for

having a major depressive episode (Keyes & Westerhof, 2012). At the same time, a longitudinal study in a general population sample indicated that increments in depressive symptoms resulted in an accelerated increase in subjective age over time (Ayalon, Palgi, Avidor, & Bodner, 2016).

The link between depression and indicators of premature aging, raises an important question concerning the underlying mechanisms that drive this association. Specifically, one may wonder whether one path through which depression shapes cellular aging includes the accelerated advancement of one's subjective age over time. Suffering from depression often hampers the ability to rely on positive illusions (Moore & Fresco, 2012) and poignantly surfaces negative perceptions of the self and the future (Beck, 2002). These, may foster concurrent older appraisals of age and increase subjective age over time, which, may in turn, burden the physiological system, and lead to accelerated cellular aging.

Maintaining a relatively older subjective age over time may be implicated in an expedited cellular aging also in that it may work as a self-fulfilling prophecy (Wurm, Warner, Ziegelmann, Wolff, & Schüz, 2013). Ex-POWs who perceive themselves as getting older more rapidly may be less inclined to engage in health behaviors, and thus suffer from premature biological aging. Former studies have found that preventive health behaviors (i.e., behaviors that prevent health deterioration such as exercise) are implicated by perceptions of aging (Levy & Myers, 2004), and that the latter may buffer the impact of stress on telomere length (Puterman et al., 2010). At the same time, as old age is often viewed in a negative light in Western culture (Weiss & Lang, 2012), it is also plausible that increased subjective age over time may be experienced as a source of stress on its own right, wearing off biological processes and leading to cellular senescence (Danese & McEwen, 2012).

To the best of our knowledge, the associations between subjective age and concurrent and subsequent telomere length, as well as the role that changes over time in subjective age

play within the link between depression and telomere length have not been assessed to date. The sole study that has explored both indicators of premature aging, assessed the role of telomere length in shaping one's subjective age and not vice versa (Stephan, Sutin, & Terracciano, 2015), and found no relation between subjective and cellular aging in a non-clinical sample.

The present longitudinal study aims to fill these aforementioned gaps. The current investigation capitalizes on data collected from ex-POWs and comparable combat veterans, 30 (T1), 35 (T2), and 42 (T3) years after the Israeli Yom-Kippur war. Based on the literature reviewed above, we hypothesize that (a) higher subjective age will be associated with concurrent and subsequent shorter telomere length, controlling for the effect of chronological age; (b) that the relations between depression and telomere length will be mediated by the change in subjective age over time, controlling for the effect of chronological age, health related measures and PTSD.

Methods

Participants and procedure

This study is part of a prospective study of the biopsychosocial implications of war captivity. The larger study included four waves of assessment, ranging over four decades after participants' repatriation (Solomon et al., 2017). In the current study we used only the three most recent points of assessment: 2003 (T1), 2008 (T2) and 2015 (T3), in which data concerning the study's variable were included.

Of the 240 infantry soldiers captured during the 1973 Yom-Kippur War, 123,176 and 158 ex-POWs participated in T1,T2 and T3, respectively (see Solomon et al., 2017, for further details). At T3, the mean age of ex-POWs was 63.8 (*SD*=3.4; range=60-77). The mean years of education was 14 (SD=3.7; range= 6-25 years). Of the ex-POWs, 29.9% were

working in full-time jobs, 21.8% had part-time jobs, and 48.3% were unemployed. 75.5% reported an average monthly income or above.

Handling missing data

Participant attrition and addition, are inherent to longitudinal designs (Collins, Schafer, & Kam, 2001). In the present study, we used an anchor wherein ex-POWs were included in the sample only if they had data regarding telomere length as well as data concerning the other study's variables (i.e., depression, subjective age) (n=88). Overall, 1% to 33% of data was missing across waves. To determine whether the missing data was random or biased, analyses of differences were conducted for all of the variables, using Little's Missing Completely at Random (MCAR) test (Collins et al., 2001).

The analysis revealed that the data were not missing completely at random, $\chi 2(88)=153.901$, $p<.001$. Supplementary analyses revealed that the ex-POWs with missing data regarding subjective age at T2, endorsed significantly shorter telomere lengths at T3 ($t=5.8$, $p<.001$) than ex-POWs without missing data. As the mechanism of missingness was unknown and there were indications that the missingness was related to the observed data, we assumed that the data were missing at random (MAR) rather than completely at random. We used SPSS 24, employing a Maximum-likelihood (ML) estimation procedure for handling missing data. ML is considered to be the optimal method for both addition and attrition of participants over time (Collins et al., 2001). The final sample comprised of 88 ex-POWs.

Measures

Depressive symptoms were assessed at T1, subjective age was assessed at T2 and T3, and telomere length was assessed only at T3. Control variables were assessed at T3.

Depressive Symptoms (T1). Depressive symptoms were assessed using the 6-item depression subscale of The Symptoms Checklist-90 (SCL-90; Derogatis, 1977). SCL-90 is a widely used, well-validated, 90-item, self-report questionnaire measuring a range of

psychological issues. Items are rated on a scale from 0 (*not at all*) to 4 (*extremely*), referring to the two-week period prior to completing the questionnaire. In the current study the SCL-90 demonstrated high internal consistency (Cronbach's α=.94).

Subjective age (T2, T3). Drawing on subjective age measure (Barak & Schiffman, 1981), and adapted from a previous study, subjective age was assessed by five statements concerning subjective perceptions of age: (a) Felt age: "I feel as though I am … ," (b) Age appearance: "I look as though I am … ," (c) Behavior age: "I act as though I am … ," (d) Age-related interests: "My interests are mostly those of a person … ," and (e) Vitality age: "I feel vital as though I am … ," compared with one's age group. Answers were given on a three-point scale (1–3) ranging from younger than, same age as, to older than one's age. The average score was used as a composite subjective age score, with a higher score reflecting an older subjective age compared to one's chronological age. The measure demonstrated high internal consistency in the current study (Cronbach's α T1=.83; Cronbach's α T2=.89).

Telomere length (T3). Average telomere length was measured by Southern blot (Uziel et al., 2007). Genomic DNA was extracted (ArchivePure; 5-prime) according to the manufacturer's instructions and quantified (NanoDrop; Thermo). DNA, 5mg, was digested for 16 hours with *RSA*I and *HINF*I, (TeloTAGGG length assay; Roche). 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 hours. The DNA was then UV-cross-linked (120mJ) to the membrane and incubated for 16 hours 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 minutes at RT, twice in Stringent wash buffer II (0.2X SSC, 0.1% SDS) for 15 minutes at 50^{O}C, in 1X maleic acid buffer (supplied by the TeloTAGGG length

assay kit; Roche) for 5 minutes, in blocking solution (kit) for 30 minutes at RT, in Anti-DIG-AP solution for 30 minutes at RT, twice in washing buffer (kit) for 15 minutes at RT and, finally, in detection solution (kit) for 5 minutes at RT. The membrane was then applied with ~40 drops of CSPD substrate and exposed to a sensitive film for 1.5 hours. After development, the film was scanned and quantified by the Quantity One software (Versadoc; BioRad). To calculate telomere length, each signal was segmented and its intensity was measured. Telomere length 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.

Control variables (T3). Participants were asked about their chronological age and substance use (smoking and drug use habits; yes/no). As part of a medical examination, participants' BMI was calculated. Additionally, participants' PTSD symptoms were assessed via the PTSD Inventory (PTSD-I; Solomon et al., 1993). Ex-POWs indicated the frequency of items reflecting the DSM-5 symptoms of PTSD (American Psychiatric Association, 2013). In the current study, the internal consistency was high (Cronbach's α=.90).

Analytic strategy

To examine if subjective age is associated with adjusted age telomere length, we used linear regression model including age in the first step and subjective age at T2 and T3 in the second step of the model. To assess the mediating role of subjective age within the association between depression and telomere length over time, we used multiple step mediation methodology (Hayes, Preacher, & Myers, 2011). Specifically, we used Sequential mediation analysis using PROCESS computational tool procedure (Model 6), to examine whether subjective age at T2 and T3 mediated the link between depression at T1 and telomere length at T3. Age, BMI, substance use and PTSD symptoms at T3 and were treated

as control variables. All predictors were standardized and mean-centered prior to entering them into the regression analyses.

Results

Table 1 presents the sample characteristics for the study's variable. Analyses indicated a significant negative correlation between subjective age at T3 and adjusted age telomere length at T3 (β=-.56, p=.03, 95% CI [-1.02, -.02]), indicating that higher subjective age were associated with shorter telomeres at that time point, beyond the effect of chronological age. However, the correlation between subjective age at T2 and adjusted age telomere length at T3 was non-significant (β=.11, p=.76, 95% CI [-.65, .88]).

Multiple step mediation analyses (see table 2) revealed that depression had a significant direct effect on telomere length. Additionally, depression at T1 indirectly predicted telomere length via subjective age at both T2 and T3 (See Figure 1). Higher levels of depression at T1 were associated with older subjective age at T2, which, in turn, predicted an increase in subjective age between T2 and T3. Concomitantly, higher (i.e., older) rates of subjective age at T3 were associated with shorter telomeres at T3. The effects of chronological age (β=-.11, t= -.93, p=.35), BMI (β=.13, t=.95, p=.34), substance use (β=-.01, t=-.06, p=.95), and PTSD symptoms at T3 (β=.24, t=1.85, p=.07) in predicting of telomere length were non-significant.

Discussion

This study was designed to assess the role that subjective age plays in ex-POWs' premature aging, as manifested in telomere length. Two questions were investigated. The first was whether subjective age is linked with concurrent and subsequent telomere length. The second was whether change in subjective age over time serves as a mechanism underlying the relation between depression and telomere length. The findings indicate that ex-POWs who evince older subjective age also exhibit concurrent shorter telomeres. Furthermore, the

change in subjective age over time mediated the relation between ex-POWs' depressive symptoms and the length of their telomeres.

The current results indicated a significant association between ex-POWs' subjective age and their concurrent adjusted age telomere length. These findings are consisted with former evidence regarding the association between subjective age and physical health (e.g., Stephan et al., 2012), and further this body of knowledge suggesting that the negative implications of older subjective age include cellular aging as well.

Two main explanations might be offered for the present results. The first plausible explanation is that the relation between subjective age and telomeres might be rooted in the effects of subjective age on one's health processes during aging. While holding perceptions of a young subjective age might serve as an adaptive self-enhancing positive illusion (Mirucka et al., 2016) or an identity-related resource (Wiesmann et al., 2017) that may promote psychological and physical well-being (Stephan et al., 2012), older subjective age may be related to intensified distress. Perceiving oneself as older may confront ex-POWs with the negative stereotypes attributed to old age, and hinder their personal resources (Wiesmann et al., 2017), leading to heightened stress. Such elevated stress may burden the biological systems and increase erosion at the cellular level (Danese & McEwen, 2012).

An alternative explanation suggests that the link between subjective age and telomere length reflects the effects of one's physical condition on his or her subjective appraisal of age. Previous studies have shown that health conditions such as illnesses and physical functioning often account for the greatest portion of differences in subjective age (Hubley & Hultsch, 1994), and that compromised health relates to an older age identity (Kleinspehn-Ammerlahn et al., 2008). The process wherein the individual's perceives "internal" signals from the body that provide a sense of his or her physical condition and underlie mood and emotional states is known as introception (Craig, 2003) .It might be that ex-POWs exhibiting accelerated

cellular erosion, "detect" this process via the introception of these negative changes in health, and that this information, in turn, contributes to their perception of their age. Given that the association between subjective age and telomere length found in the current study is cross-sectional, one cannot determine the direction of the effect. Furthermore, a bidirectional association is also plausible, wherein subjective age and telomere length reciprocally shape one another.

Notably, though the findings indicated the cross-sectional relation between subjective age and telomere length, we did not find a significant link between ex-POWs' subjective age and subsequent telomere length. These findings are inconsistent with former results indicating that subjective age is related with one's physical health and longevity (e.g., Kotter-Grühn et al., 2009) and are therefore highly surprising. One explanation for the present results suggests that since telomere length mirrors one's current pace of cellular aging, it is shaped partly by one's present perceptions regarding age. Hence, telomere length is related to concurrent subjective age and not with one's past age perceptions. A second explanation suggests ex-POWs' older subjective age might increase tendencies for negative attitudes regarding aging and acts as a source of stress (Bellingtier, & Neupert, 2016). This is particularly so during old age, when the experience of being old as well as the multiple losses accompanying old age may lead to the reactivation of past trauma (Gagnon & Hersen, 2000). Thus, ex-POWs' older subjective age at T3 and not at T2, had a physical toll and was significantly associated with shorter telomeres. Lastly, the third explanation suggests that the present results are the product of methodological limitations and specifically the big time gap between the study's measurement points. It is plausible that during the period of eight years between the last two measurements, as many participants entered their mid-sixties, central processes that are related to aging, such as declines in health or retiring from work, have occurred, leading to prominent changes in ex-POWs' subjective age. Having no additional

assessments during this time, this study could not detect these modifications in subjective age and thus could not uncover their effects on cellular aging

Considering our second objective, as hypothesized, the results indicated a direct link between depressive symptoms and telomere length, and further revealed that this link was indeed mediated by increases in subjective age over time. That is, depression predicted an increase in subjective age, which, in turn, predicted relatively shorter telomeres.

The finding that depression predicted an increase in advanced subjective age over time, are consistent with previous studies (e.g., Ayalon et al., 2016). This trend might be rooted in the physiological as well as the psychological outcomes of depression. Suffering from depression might pose a toll on one's physical state. Depression has been found to be related to an impeded immune system, to a high risk for morbidity, and altogether poorer prognoses in the face of various diseases (Wolkowitz et al., 2011). These negative physical conditions, thus, might have shaped appraisals of age (Kleinspehn-Ammerlahn et al., 2008), thus fostering an expedited advancement of subjective age over time.

Additional characteristics of depression could also facilitate accelerated subjective age. Decrease in energy, lack of vitality, fatigue, motor retardation, negative perceptions of the self, pessimistic approach concerning the future, and preoccupation with death, all of which are features of depression (APA, 2013), are also characteristics of old age, and might further the more rapid perception of oneself as growing older. At the same time, depression often hinders the ability to relay on positive illusory perceptions when making evaluations regarding the self (Moore & Fresco, 2012). Such self-illusions may be required in order to maintain a young subjective age in late life (Mirucka et al., 2016), and thus their absence might increase tendencies for elevated subjective age over time.

In the present study, this course of accelerated increase in subjective age, in turn, was linked with cellular aging, manifested in shorter telomere, beyond the effects of

chronological age, substance use, BMI and PTSD symptoms. How can one explain the unique effects of negative changes in subjective age in predicting cellular aging? It is possible that an acceleration in subjective age acts as a source of stress particularly in the Western culture which typically undervalues older age and older adults (Weiss & Lang, 2012). Individuals who perceived themselves as growing older more rapidly may encounter negative stereotypes related to the elderly, as well as worries regarding physical or cognitive decline or concerns about anticipated losses in interpersonal and vocational domains. Additionally, old age is typically characterized in excess reflection and reminiscence about one's past (e.g., Erikson, 1959). Accelerated subjective age might increase such tendencies, which may in turn be painful for survivors of harsh traumatic events, such as captivity, and give rise to negative feelings of guilt, sorrow and despair (Gagnon & Hersen, 2000). Facing these multifarious strains could increase the levels of perceived stress, which over time take their toll on biological systems, and eventually lead to an exacerbated erosion at the cellular level (Danese & McEwen, 2012).

Finally, it may be suggested that accelerated subjective aging might influence biological aging through a behavioral pathway. Former studies indicated that people with older self-perceptions were less inclined to engage in preventive health behaviors such as an appropriate diet, exercise, and medication compliance (Levy & Myers, 2004). Such behaviors may lead to premature biological aging (Puterman et al., 2010). In this manner, an accelerated subjective age serves as a self-fulfilling prophecy, gradually leading from the subjective to the objective, from perceived age to telomere length.

The current findings should be considered in the context of several limitations. First, the current study relied on self-report measures of subjective age and depression, which may be subjected to response biases and shared method variance. Future studies should include additional methods of data collection, such as clinical interviews. Second, although the

Southern Blot method utilized for the assessment of telomere length in the current study is considered the gold standard, there are other methods that are commonly reported in the literature. Thus, one should consider that variability in measurement methods might have affected the results. Third, due to the small sample size, we controlled only for a limited number of health related variables. Future studies should employ a prospective design with a larger sample while controlling for additional factors that may potentially affect biological aging, such as preventive health behaviors. Finally, the present study explored the role of subjective age among Israeli ex-POWs. Since perceptions regarding old age might be shaped by cultural norms, there is need for future studies that will investigate the contribution of subjective age to telomere length among other cultures.

Bearing these limitations in mind, the present findings have both theoretical and clinical importance. The current results are indicative of potential pathways that have not been previously examined, and offer new directions for research on accelerated aging. The present findings imply that subjective age has detrimental effects on biological aging, and that this is one pathway through which symptoms of depression generate greater vulnerability and perhaps increase early aging among ex-POWs. The results of the present investigation may be relevant not only for ex-POWs, but also to older veteran populations, and are in line with recent findings regarding minority older adults residing in communities where they are at risk for adversity such as trauma and discrimination (Chae, Nuru-Jeter, Adler, Brody, Lin, Blackburn, Epel, 2014).

The present results imply that therapeutic work with elderly trauma survivors such as ex-POWs would benefit from acknowledging the high rates of depression among this population, while also accounting for their perceptions of aging as these are closely intertwined. By doing so, such interventions should strive to rehabilitate ex-POWs' ability to maintain a more youthful personal age, in hope that it may be associated with limiting

premature cellular aging. That said, the findings reported above are a preliminary step in a much needed direction, and considerable research is necessary in order to understand the relation between subjective age and cellular aging in the aftermath of extreme trauma.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Avidor, S., Benyamini, Y., & Solomon, Z. (2014). Subjective age and health in later life: the role of posttraumatic symptoms. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 71, 415–424. http://dx.doi.org/10.1093/geronb/gbu150

Ayalon, L., Palgi, Y., Avidor, S., & Bodner, E. (2016). Accelerated increase and decrease in subjective age as a function of changes in loneliness and objective social indicators over a four-year period: results from the health and retirement study. *Aging & mental health, 20*(7), 743-751. http://dx.doi.org/10.1080/13607863.2015.1035696

Barak, B., & Schiffman, L. G. (1981). Cognitive age: A nonchronological age variable. *ACR North American Advances*.

Beck, A. T. (2002). Cognitive models of depression. *Clinical advances in cognitive psychotherapy: Theory and application*, *14*(1), 29-61.

Beebe, G. W. (1975). Follow-up studies of World War II and Korean War prisoners II. Morbidity, disability and maladjustments. *American journal of epidemiology*, *101*(5), 400-422.

Bellingtier, J. A., & Neupert, S. D. (2016). Negative aging attitudes predict greater reactivity to daily stressors in older adults. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 00(00), 1–5. http://dx.doi.org/10.1093/geronb/gbw086

Bersani, F. S., Lindqvist, D., Mellon, S. H., Epel, E. S., Yehuda, R., Flory, J., . . . Abu-Amara, D. (2016). Association of dimensional psychological health measures with telomere length in male war veterans. *Journal of affective disorders, 190*, 537-542. http://dx.doi.org/10.1016/j.jad.2015.10.037

Cawthon, R. M., Smith, K. R., O'Brien, E., Sivatchenko, A., & Kerber, R. A. (2003). Association between telomere length in blood and mortality in people aged 60 years or older. *The Lancet, 361*(9355), 393-395.

Chae, D. H., Nuru-Jeter, A. M., Adler, N. E., Brody, G. H., Lin, J., Blackburn, E. H., & Epel, E. S. (2014). Discrimination, racial bias, and telomere length in African-American men. *American journal of preventive medicine, 46*(2), 103-111. http://dx.doi.org/10.1016/j.amepre.2013.10.020

Chan, S. R., & Blackburn, E. H. (2004). Telomeres and telomerase. *Philosophical Transactions of the Royal Society B: Biological Sciences, 359*(1441), 109-122. http://doi.org/10.1098/rstb.2003.1370

Collins, L. M., Schafer, J. L., & Kam, C.-M. (2001). A comparison of inclusive and restrictive strategies in modern missing data procedures. *Psychological Methods, 6*(4), 330-351. https://doi.org/10.1037/1082-989X.6.4.330

Craig, A. D. (2003). Interoception: the sense of the physiological condition of the body. *Current opinion in neurobiology, 13*(4), 500-505. https://doi.org/10.1016/S0959-4388(03)00090-4

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & behavior, 106*(1), 29-39. http://dx.doi.org/10.1016/j.physbeh.2011.08.019

Dekel, S., Solomon, Z., Horesh, D., & Ein-Dor, T. (2014). Posttraumatic stress disorder and depressive symptoms: joined or independent sequelae of trauma? *Journal of psychiatric research, 54*, 64-69. http://dx.doi.org/10.1016/j.jpsychires.2014.03.003

Derogatis, L. R. (1977). *The SCL-90 Manual I: Scoring, administration and procedures for the SCL-90*. Baltimore: Johns Hopkins University School of Medicine, Clinical Psychometrics Unit.

Erikson, E. H. (1959). Identity and the life cycle: Selected papers by Erik H. *Erikson. Psychological Issues, 1*, 1-171.

Gagnon, M., & Hersen, M. (2000). Unresolved childhood sexual abuse and older adults: Late-life vulnerabilities. *Journal of Clinical Geropsychology, 6*(3), 187-198. https://doi.org/10.1023/A:1009589115942

Hayes, A. F., Preacher, K. J., & Myers, T. A. (2011). "Mediation and the estimation of indirect effects in political communication research", *In Sourcebook for political communication research: Methods, measures, and analytical techniques*, eds E. P. Bucy and R. Lance Holbert (New York, NY:Routledge), 434–465.

Hubley, A. M., & Hultsch, D. F. (1994). The relationship of personality trait variables to subjective age identity in older adults. *Research on Aging, 16*(4), 415-439.

Keyes, C. L., & Westerhof, G. J. (2012). Chronological and subjective age differences in flourishing mental health and major depressive episode. *Aging & mental health, 16*(1), 67-74. http://dx.doi.org/10.1080/13607863.2011.596811

Kleinspehn-Ammerlahn, A., Kotter-Grühn, D., & Smith, J. (2008). Self-perceptions of aging: Do subjective age and satisfaction with aging change during old age? *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 63*(6), P377-P385. http://dx.doi.org/10.1093/geronb/63.6.P377

Kotter-Grühn, D., Kleinspehn-Ammerlahn, A., Gerstorf, D., & Smith, J. (2009). Self-perceptions of aging predict mortality and change with approaching death: 16-year longitudinal results from the Berlin Aging Study. *Psychology and aging, 24*(3), 654-667. http://dx.doi.org/10.1037/a0016510

Lahav, Y., Rodin, R., & Solomon, Z. (2015). Somatic complaints and attachment in former prisoners of war: A longitudinal study. *Psychiatry: Interpersonal and Biological Processes, 78*, 354-366. https://doi.org/10.1080/00332747.2015.1061311

Levy, B. R., & Myers, L. M. (2004). Preventive health behaviors influenced by self-perceptions of aging. *Preventive medicine, 39*(3), 625-629. https://doi.org/10.1016/j.ypmed.2004.02.029

Levy, B. R., Slade, M. D., Chung, P. H., & Gill, T. M. (2014). Resiliency over time of elders' age stereotypes after encountering stressful events. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *70*(6), 886-890. http://dx.doi.org/10.1093/geronb/gbu082

Lohr, J. B., Palmer, B. W., Eidt, C. A., Aailaboyina, S., Mausbach, B. T., Wolkowitz, O. M., . . . Jeste, D. V. (2015). Is post-traumatic stress disorder associated with premature senescence? A review of the literature. *The American Journal of Geriatric Psychiatry, 23*(7), 709-725. http://dx.doi.org/10.1016/j.jagp.2015.04.001

Meziab, O., Kirby, K. A., Williams, B., Yaffe, K., Byers, A. L., & Barnes, D. E. (2014). Prisoner of war status, posttraumatic stress disorder, and dementia in older veterans. *Alzheimer's & Dementia, 10*(3), S236-S241. http://dx.doi.org/10.1016/j.jalz.2014.04.004

Mirucka, B., Bielecka, U., & Kisielewska, M. (2016). Positive orientation, self-esteem, and satisfaction with life in the context of subjective age in older adults. *Personality and Individual Differences, 99*, 206-210. https://doi.org/10.1016/j.paid.2016.05.010

Moore, M. T., & Fresco, D. M. (2012). Depressive realism: A meta-analytic review. *Clinical psychology review, 32*(6), 496-509. https://doi.org/10.1016/j.cpr.2012.05.004

Nice, D. S., Garland, C. F., Hilton, S. M., Baggett, J. C., & Mitchell, R. E. (1996). Long-term health outcomes and medical effects of torture among US Navy prisoners of war in Vietnam. *Journal of the American Medical Association, 276*, 375-381. https://doi.org/10.1001/jama.1996.03540050035020

O'Donovan, A., Epel, E., Lin, J., Wolkowitz, O., Cohen, B., Maguen, S., . . . Neylan, T. C. (2011). Childhood trauma associated with short leukocyte telomere length in posttraumatic stress disorder. *Biological psychiatry, 70*(5), 465-471. https://doi.org/10.1016/j.biopsych.2011.01.035

O'Donovan, A., Tomiyama, A. J., Lin, J., Puterman, E., Adler, N. E., Kemeny, M., . . . Epel, E. S. (2012). Stress appraisals and cellular aging: A key role for anticipatory threat in the relationship between psychological stress and telomere length. *Brain, behavior, and immunity, 26*(4), 573-579. https://doi.org/10.1016/j.bbi.2012.01.007

Price, L. H., Kao, H.-T., Burgers, D. E., Carpenter, L. L., & Tyrka, A. R. (2013). Telomeres and early-life stress: an overview. *Biological psychiatry, 73*(1), 15-23. http://dx.doi.org/10.1016/j.biopsych.2012.06.025

Puterman, E., Lin, J., Blackburn, E., O'Donovan, A., Adler, N., & Epel, E. (2010). The power of exercise: buffering the effect of chronic stress on telomere length. *PLoS One, 5*(5), e10837. https://doi.org/10.1371/journal.pone.0010837

Ridout, K. K., Ridout, S. J., Price, L. H., Sen, S., & Tyrka, A. R. (2016). Depression and telomere length: A meta-analysis. *Journal of affective disorders, 191*, 237-247. http://dx.doi.org/10.1016/j.jad.2015.11.052

Rintamaki, L. S., Weaver, F. M., Elbaum, P. L., Klama, E. N., & Miskevics, S. A. (2009). Persistence of traumatic memories in World War II prisoners of war. *Journal of the*

American Geriatrics Society, 57(12), 2257-2262. https://doi.org/10.1111/j.1532-5415.2009.02608.x

Schafer, M. H. (2009). Parental death and subjective age: Indelible imprints from early in the life course? *Sociological Inquiry, 79*(1), 75-97. http://dx.doi.org/10.1111/j.1475-682X.2008.00270.x

Schutte, N. S., & Malouff, J. M. (2015). The association between depression and leukocyte telomere length: a meta-analysis. *Depression and anxiety, 32*(4), 229-238. http://dx.doi.org/10.1002/da.22351

Serrano, A. L., & Andrés, V. (2004). Telomeres and Cardiovascular Disease Does Size Matter? *Circulation Research, 94*(5), 575-584. http://dx.doi.org/10.1161/01.RES.0000122141.18795.9C

Solomon, Z., Benbenishty, R., Neria, Y., Abramowitz, M., Ginzburg, K., & Ohry, A. (1993). Assessment of PTSD: Validation of the revised PTSD Inventory. *The Israel Journal of Psychiatry and Related Sciences, 30*(2), 110-115.

Solomon, Z., Greene, T., Ein-Dor, T., Zerach, G., Benyamini, Y., & Ohry, A. (2014). The long-term implications of war captivity for mortality and health. *Journal of behavioral medicine, 37*, 849-859. doi:10.1007/s10865-013-9544-3

Solomon, Z., Tsur, N., Levin, Y., Uziel, O., Lahav, M., & Ohry, A. (2017). The implications of war captivity and long-term psychopathology trajectories for telomere length. *Psychoneuroendocrinology, 81*, 122-128. https://doi.org/10.1016/j.psyneuen.2017.04.004

Stephan, Y., Chalabaev, A., Kotter-Grühn, D., & Jaconelli, A. (2012). "Feeling younger, being stronger": An experimental study of subjective age and physical functioning among older adults. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 68*(1), 1-7. https://doi.org/10.1093/geronb/gbs037

Stephan, Y., Sutin, A. R., & Terracciano, A. (2015). How old do you feel? The role of age discrimination and biological aging in subjective age. *PLoS One, 10*(3), e0119293. https://doi.org/10.1371/journal.pone.0119293

Uziel, O., Singer, J. A., Danicek, V., Sahar, G., Berkov, E., Luchansky, M., . . . Lahav, M. (2007). Telomere dynamics in arteries and mononuclear cells of diabetic patients: effect of diabetes and of glycemic control. *Experimental gerontology, 42*(10), 971-978. https://doi.org/10.1016/j.exger.2007.07.005

Weiss, D., & Lang, F. R. (2012). "They" are old but "I" feel younger: Age-group dissociation as a self-protective strategy in old age. *Psychology and aging, 27(1),* 153-163. https://doi.org/ 10.1037/a0024887

Wiesmann, U., Becker, M.-L., & Hannich, H.-J. (2017). Positive Aging in Nursing Home Residents. *The Journal of Gerontopsychology and Geriatric Psychiatry, 30(2),* 71-78. http://dx.doi.org/10.1024/1662-9647/a000166

Wolkowitz, O. M., Reus, V. I., & Mellon, S. H. (2011). Of sound mind and body: depression, disease, and accelerated aging. *Dialogues Clin Neurosci, 13*(1), 25-39.

Wurm, S., Warner, L. M., Ziegelmann, J. P., Wolff, J. K., & Schüz, B. (2013). How do negative self-perceptions of aging become a self-fulfilling prophecy? *Psychology and aging, 28*(4), 1088 .http://dx.doi.org/ 10.1037/a0032845

Zhang, L., Hu, X., Benedek, D., Fullerton, C., Forsten, R., Naifeh, J., . . . Smerin, S. (2014). The interaction between stressful life events and leukocyte telomere length is associated with PTSD. *Molecular psychiatry, 19*(8), 855-856. http://dx.doi.org/10.1038/mp.2013.141

Table 1

Sample characteristics for the study variables (n=88)

Measure	
Depressive Symptoms T1 (M, SD)	1.76, .97 (range: 0–5)
Subjective age T2 (M, SD)	1.78, .48 (range: 1–3)
Subjective age T3 (M, SD)	1.23, .62 (range: 1–3)
Telomere length T3 (M, SD)	5.32, 1.57 (range: 2.5–9.9)
Age T3 (M, SD)	63.80, 3.43 (range: 60–77)
BMI T3 (M, SD)	29.25, 6.57 (range: 21.4–75)
Substance use T3	
yes	10 (11.4%)
no	78 (88.6%)
PTSD symptoms T3 (M, SD)	8.94, 5.04 (range: 0–16)

Table 2

Standardized Regression coefficients and Bootstrap 95% Confidence Intervals for predicting telomere length

Measure	β	*Bootstrap 95% Confidence Intervals*
Direct	-.39	{-.7072, -.0727}*
Indirect through T2 Subjective Age	.08	{-.0197, .2684}
Indirect through T3 Subjective Age	-.04	{-.1975, .0260}
Indirect through T2 and T3 Subjective Age	-.03	{-.0935, -.0060}*

Note. 95% Confidence Intervals are presented in brackets. Confidence intervals that do not include 0 (null association) are significant. * Significant at .05.

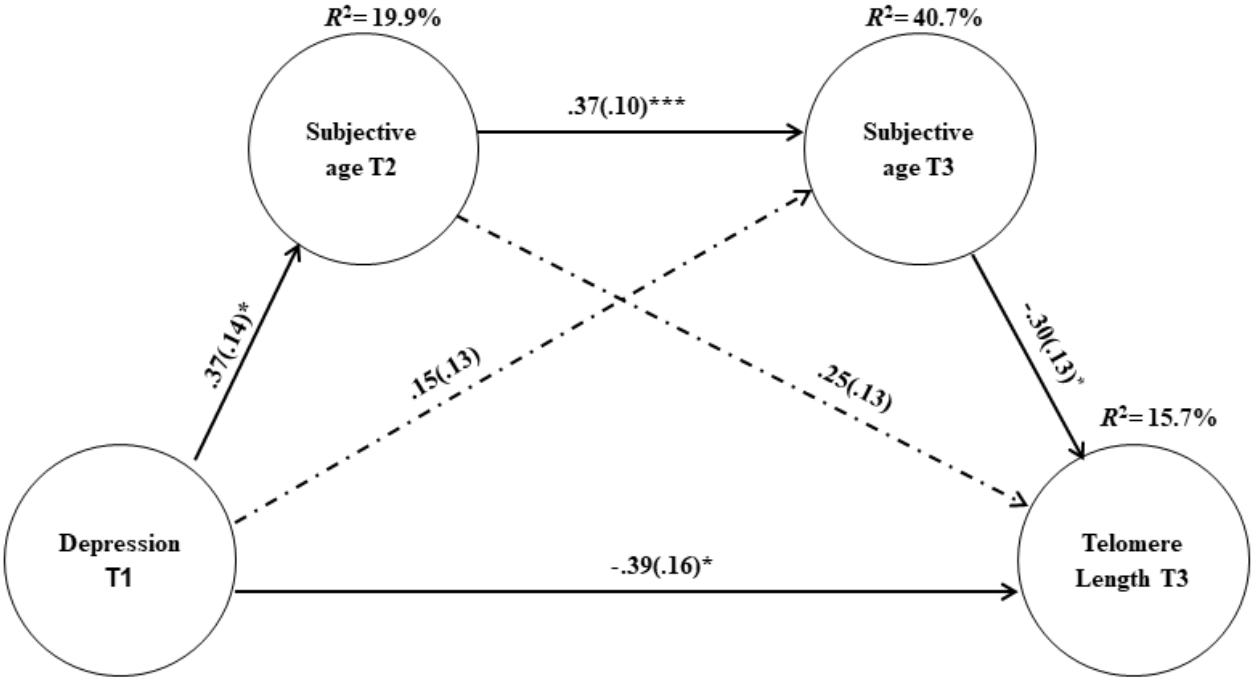

Figure 1. Standardized coefficients β(SE) for the association between Depression and Telomere Length through subjective age at T3 as well as through the sequential mediation of subjective age at T2 and T3. Explained variance is located above all dependent variables. PTSD symptoms at T1 as well as Age, BMI, substance use at T3 were treated as control variables. * $p<0.05$, ** $p<0.01$, *** $p<0.001$.