**Associations Between Hardiness, C-Reactive Protein and Telomere Length Among Former Prisoners of War**

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**Abstract**

**Background**: War captivity and posttraumatic stress disorder (PTSD) are known to be associated with several poor health outcomes of an accelerated aging process. However, the contribution of personality protective factors to this phenomenon are rarely studied. The present 24-year prospective study examined associations between psychological hardiness and three health outcomes: C-reactive protein (CRP) levels, metabolic syndrome (MetS), and telomere length (TL).

**Methods:** Eighty-eight Israeli former prisoners of war (ex-POWs) were assessed 18 (T1) and 42 (T2) years after repatriation. Data on hardiness was collected at T1 while leukocyte TL, CRP and MetS data was collected 42 years after the war.

**Results**: While adjusting forage, Body-Mass Index (BMI), self-rated health, depressive and PTSD symptoms at T2, higher levels of hardiness at T1 predicted decreased CRP and longer TL at T2.

**Conclusions:** Long-term health vulnerabilities of traumatized ex-POWs are manifested in an accelerated aging process and cellular senescence. Raising awareness of the importance of protective factors such as veterans’ hardiness might be associated with improving their longevity and well-being.

Key words: Captivity; PTSD; Hardiness; CRP, Telomere length, Veterans

**Introduction**

Exposure to traumatic stressful events is known to be associated with both mental and physical health disorders (O’Donovan et al., 2015). At the extreme end of war-related traumatic stress stands the taxing experience of captivity. Prisoners of war (POWs) are often exposed to severe, intentional, repeated man-made traumas. For a prolonged period, they might face brutal physical and psychological torture while being deprived of basic human needs (Basoglu, 2009). It is no surprise, then, that in most studies ex-POWs report high levels ofvaried, debilitating and long-term psychiatric disorders, the most common of which are posttraumatic stress disorder (PTSD; Rintamaki, Weaver, Elbaum, lama, E. & Miskevics, 2009), and depression comorbidity (Ikin, Creamer, Sim, & McKenzie, 2010). Moreover, ex-POWs are also known to suffer from varied physical morbidity (Nice, Garland, Hilton, Baggett, & Mitchell, 1996), and even premature mortality (Solomon et al., 2014).

Accumulated scientific evidence associates PTSD with several expressions of early or accelerated aging among veterans (also termed premature senescence; (Lohr et al., 2015). These manifestations include senescence-related negative health outcomes, such as higher rates of metabolic syndrome (MetS; Wolf et al., 2016) and cardiovascular disease (Edmondson & Cohen, 2013). Moreover, PTSD has been found to associate with biomarkers of senescence such as accelerated leukocyte telomere shortening (LTL; Shalev et al., 2014) and pro-inflammatory markers such as heightened levels of C-reactive protein (CRP; Lindqvist et al., 2017). The accelerated aging process among individuals coping with PTSD has been speculated to result from chronic emotional and physiological reactivity, dysregulation of the hypothalamic-pituitary-adrenal axis (HPA) and risky health-related behaviors that take a biological toll in the form of accelerated cellular aging (Lohr et al., 2015). Specifically, ex-POWs have also been found to suffer from higher rates of medical conditions such as endocrine and metabolic diseases (Jukić, Filaković, Požgain, & Glavina, 2019), cardiovascular disease (Kang, Bullman, & Taylor, 2006), and chronic pain (Amris & de C Williams, 2015). Recently, our team found heightened CRP levels, higher rates of MetS, and shorter telomeres among Israeli ex-POWs compared to control veterans (Solomon et al., 2017; Stein, Levin, Uziel, Abumock, Solomon, 2018). Given the profound implications of war trauma and PTSD for health vulnerabilities, identification of resiliency factors that associate with bio-markers of an accelerated aging process over a veteran's life-cycle is an important task. In this study, we aimed to examine the association between psychological hardiness and long-term health outcomes of accelerated aging.

Hardiness is a multidimensional personality disposition which encompasses three basic facets: commitment, control and challenge. Hardy individuals have a strong belief in the meaningfulness of their activities, they trust their ability to influence the course of events, and they accept change in life as a constructive challenge (Maddi , 2006). Indeed, it has been found that hardiness, also termed "dispositional resilience" (Bartone, 2007), is negatively related to distress and positively related to adaptive coping with stress (Eschleman, Bowling, Alarcon, 2010). Particularly in the military arena, a few studies have found hardiness to be a significant negative predictor of PTSD among combatants (Thomassen, Hystad, Johnsen, Johnsen, & Bartone, 2018) and less deteriorated PTSD trajectories over time among ex-POWs (Zerach, Karstoft, & Solomon, 2017). There are also some indications that hardiness is related to improved functioning of stress-related biological systems. For example, hardiness has been linked to lower cardiovascular activation in response to laboratory-induced stress (Wiebe, 1991), better immune responses (Dolbier, 2001), suppressed pro-inflammatory and increased anti-inflammatory markers (Sandvik, 2013), and greater parasympathetic activation at stress offset (Sandvik et al., 2019).

Notwithstanding the contribution of these studies, their results are based mostly on cross-sectional design studies or manipulated laboratory-induced stress. Moreover, only a few studies have examined psychological resiliency factors- such as hardiness- that may protect trauma-exposed veterans from accelerated aging over time in naturalistic, long-term prospective designs. Given the links between hardiness and biological and psychological coping with stress (Bartone et al., 2015), we hypothesized in this study that high levels of psychological hardiness in 1991 would be associated with lower levels of CRP and MetS, and longer LTL among ex-POWs in 2015. In order to examine the unique role of hardiness in health outcomes, we statistically controlled for age, PTSD, depression, and potentially health related variables (self-rated health (SRH), blood-pressure and BMI), which are known correlates of CRP, MetS and LTL (e.g., Solomon et al., 2017).

**Method**

**Participants and Procedure**

This study was part of a larger prospective longitudinal study of Israeli veterans, with assessments ranging over four decades after the 1973 Yom Kippur War (for details, see (Solomon et al., 2017). Assessment was conducted at two time-points: 1991 (T1) and 2015 (T3). Of the 240 soldiers captured during the war, 164 ex-POWs filled validated self-report questionnaires in T1. Comprehensive medical assessments and questionnaires were completed with 99 randomly selected ex-POWs in T2. Analyses were carried out only among ex-POWs with full data in both T1 and T2 (n=88). At T2, participants underwent a comprehensive physical examination at the Tel Aviv Sourasky Medical Center. Assessments were conducted after participants had received an explanation of the study’s aims and signed an informed consent form. This study was approved by the ethics committees of the Sourasky Medical Centre and Tel-Aviv University. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

**Measurements**

**Hardiness**. Participants completed the Hebrew version (Drory & Florian, 1991) of the third generation Hardiness Scale (Personal View Scale; Maddi, 1987) at T1. This self-report questionnaire is composed of 50 items, measuring the hardiness construct as a composite of three components: commitment, control, and challenge. Using a 6-point Likert scale ranging from 1 (*not at all*) to 6 (*very much*), participants indicated how much they endorsed each item. On this basis, a general hardiness score was computed as the mean of the items, with higher scores reflecting higher levels of hardiness. Previous studies have found adequate internal consistency for the total scale (Drory & Florian, 1991). In the present study, the Hebrew version of this scale had Cronbach’s alphas of .74 for the total scale.

**Depressive symptoms** were assessed using the depression subscale of the Symptom Checklist-90 (SCL-90; Derogatis, 1977)at T2. Participants were asked to indicate how frequently they experienced each symptom during the last two weeks, on a 5-point distress scale. For each participant, we calculated the average frequency of experiencing depressive symptoms at each of the time points. In the current study, the Cronbach α of .92 was high.

**PTSD symptomatology.** A questionnaire based on the PTSD Inventory (PTSD-I; Solomon et al., 1993) was used to assess PTSD symptoms at T2. Four items were added to the initial 17 items of the questionnaire corresponding to the DSM-IV criteria for PTSD, to reflect the newly added symptoms of DSM-5 criteria (Overly negative thoughts and assumptions about oneself or the world; Exaggerated blame of self or others for causing the trauma; Negative affect; and Risky or destructive behavior; American Psychiatric Association, 2013). Participants rated each symptom/item as it was experienced in the previous month on a 4-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). In the current study, the internal consistency was high (α = .90).

**Metabolic Syndrome** **and Components**. Among other examinations at T2, measurements relevant to this study were body mass index, fasting blood glucose and diabetes, blood pressure or a diagnosis of hypertension, and HDL-C and triglyceride levels. Medication intake was also recorded. MetS and its components were defined as having at least 3 of the following characteristics: serum triglycerides ≥ 1.7 mmol/L (150 mg/dL); serum HDL-C < 1.036 mmol/L (40 mg/dL); blood pressure ≥ 130/85 mm Hg or taking antihypertensive medication; BMI > 30 kg/m2; and fasting serum glucose ≥ 5.6 mmol/L (100 mg/dL) or a diagnosis of diabetes (Sattar et al., 2003).

**C-reactive protein (CRP).** Blood samples were obtained at 8:00 am at T2 after a fast of at least 8 hours. Venous blood was obtained from all participants from the antecubital vein. White blood cell count and differential were performed using the Coulter STKS (Beckman Coulter, Nyon, Switzerland) electronic analyzer, and wide range CRP level was determined by the Bayer wr-CRP assay (Bayer, Leverkusen, Germany; Rogowski et al., 2005).

**Telomere length.** TL was measured utilizing the Southern blot (Uziel et al., 2007) at T2. Telomere length was measured in total white blood cells obtained from 10ml 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, 5mg, was digested for 16 hours with *RSA*I and *HINF*I, (TTAGGG 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 a 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 50OC, in 1X maleic acid buffer (supplied by the TTAGGG 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 TL, each signal was segmented and its intensity was measured. TL was calculated according to the following equation:

Σ(ODi)/Σ(ODi/Li)

Where ODi is the chemiluminescent signal and Li is the length of the telomere at position i.

**Self-rated Health**: At T2 Participants were asked: ‘*In general, how would you rate your health?*’, and to rate their answer on a 5-point scale (1= *excellent* to 5= *poor*). This question is commonly used in health surveys and has been found to have an independent contribution to the prediction of mortality and future health outcomes (Benyamini & Idler, 1999).

**Data analysis.**

First, descriptive statistics and the associations between the study variables were examined with a series of Pearson correlation analyses. We used the conventional approach for the definitions of correlation magnitude (Neglible=.0-.3, Low=.3-.5, moderate=.5-.7, high=.8-.9, and very high=.9-1; Mukaka, 2012). The main analysis was based on a path analysis model with the three outcome variables, the biological variables, regressed on the seven predictor variables (see Figure 1). As BMI and BP are components of the overall scoring of the MetS variable, the paths from BMI and BP to MetS were constrained to zero. All other paths were estimated. The model parameters were estimated using robust maximum likelihood (MLR: Yuan & Bentler, 2000), using Mplus software (Muthén & Muthén, 2013). The MLR estimator is robust to non-normally distributed data and can produce corrected standard errors under conditions of non-normality (Enders & Bandalos, 2001).

**Results**

Table 1 shows the descriptive statistics and correlations for all study variables. The correlations among the predictive variables were all low to moderate, with the highest correlation found between PTSD and depression (*r* = .480). This indicates that collinearity will not be a problem in the path analysis model. The three biological variables were uncorrelated, but MetS was positively correlated with self-rated health. PTSD was positively correlated with self-rated health. Importantly, hardiness was significantly and negatively correlated with PTSD, depression and Self-rated health, with correlations in the moderate range. Hardiness also correlated significantly and negatively with MetS, and positively with telomere length and BP; the correlation between hardiness and CRP was marginally non-significant (*p* =.052).

The fitted path analysis model was a significant improvement over a null model (χ2(19) = 35.99, *p* = .010), a significant proportion of variance was explained in the outcome variables, and the regression coefficients are reported in Table 2. After controlling for all other variables in the model, hardiness significantly predicted CRP (β = -.259) and telomere length (β = .372), but not MetS (β = -.205). The R-squared estimates showed that for the full model 15.3% and 19.1% of the variation was explained in CRP and telomere length respectively. The percentage explained by hardiness, while controlling for all other variables, was 7.2% for CRP and 14.3% for telomere length.

**Discussion**

The aim of the present study was to examine the long-term role of psychological hardiness in senescence-related negative health outcomes of MetS, as well as bio-markers of accelerated aging of CRP levels and LTL, over a 24-year period following war captivity. Our main results showed that high levels of hardiness at T1 among ex-POWs significantly predicted lower levels of CRP and longer LTL at T2. These predictions remained significant above and beyond mental health comorbidities of PTSD and depression symptoms, as well as health-related background variables of SRH, age, BMI and BP. To the best of our knowledge, this is the first prospective study to show the long-term implications of hardiness in the bio-psycho derivatives of the accelerated aging process.

Much empirical effort has been invested in the study of risk or vulnerability predictors of negative mental health (Xue et al., 2015) and deteriorated physical health (Jukić et al., 2019) outcomes among veterans. Our results are in line with the few studies that examined the protective role of resiliency factors, such as hardiness (Thomassen et al., 2018), on veterans’ long-term adjustment. While the links between hardiness and adaptive coping with stress are well documented (Eschleman et al., 2010), the biological correlates of these positive effects are still understudied. Therefore, our results validate previous findings about the associations between hardiness and physical health outcomes (Krauss et al., 2018). For example, Connolly et al. (2018) found that the closely related construct of drive toward achievement was positively associated with LTL, and Barthon et al. (2015) found that high hardiness was related to higher HDL, which is a component of the MetS. As these studies were cross-sectional, the direction of these associations is rather questionable. Thus, our prospective results provide more accurate and valid estimation of the hardiness-health outcomes links.

It is important to note that due to the need to keep questionnaires as short as possible for these highly traumatized participants, hardiness was only measured once at T1. Furthermore, no other T1 covariates of psychopathology that might have accounted for our findings, were considered in analyses. Thus, notwithstanding the prospective prediction of hardiness on health outcomes, and although it is rather unlikely that covariates of psychopathology account for the hardiness effect 24 years later, we cannot rule out the possibility that other predictors or critical mediators played a role in this process.

A number of studies have documented the negative long-term sequelae of PTSD among ex-POWs (Jukić et al., 2019). Moreover, studies by our team revealed the close link between exposure to captivity trauma and varied medical morbidity and even mortality among ex-POWs (Solomon et al., 2014). However, our results show that beyond the known contribution of PTSD and depression to the accelerated aging process (Lohr et al., 2015), hardiness still plays a beneficial role in ex-POWs’ medical condition and bio-markers. How can we explain this pattern of results? The answer may direct us to the concept of coping.

Many studies documented the positive role of personality resources, such as optimism (Segovia, Moore, Linnville, & Hoyt, 2015) or positive appraisal of military experiences (King et al., 2015), in predicting long-term positive adjustment among ex-POWs. It may be that hardiness improves ex-POW’s cognitive appraisal of their past, present and even future losses. This positive appraisal might reduce activation of the sympathetic nervous system and the HPA Axis. Indeed, some studies have shown that heightened levels of stress hormones, like glucocorticoids and catecholamines, suppress the production of pro-inflammatory markers such as CRP (Lohr et al., 2015). Eventually, it might also maintain cellular integrity as manifested in longer LTL of ex-POWs (Connolly et al., 2018).

Another way to examine this finding is through the lens of avoidance coping. It has been found that hardiness is negatively related with avoidance coping (Bartone et al., 2015) and that avoidance coping might be a risk factor for PTSD (Thomassen et al., 2018). It is suggested that hardy ex-POWs are less inclined to avoid captivity-related reminders in their body (e.g., the sight of scars) and physical health. Alternatively, they might actively approach these stressful medical conditions, realistically assess their severity and try to tackle this natural course of accelerated aging with healthy behaviors and reduction of health risky behaviors (e.g., alcohol consumption). Thus, more activity and less disengagement might also be reflected in more balanced stress-related biomarkers.

The present study encompasses a number of limitations. First, although this study is longitudinal, the inference of causality should not be drawn due to the lack of pre-war medical and psychological information. Second, we did not include data of hardiness and medical outcomes among the war exposed comparison group, which might have shed light on the effect of captivity, as compared to more normal aging. Third, this study also lacked the analysis of specific pro-inflammatory and anti-inflammatory cytokines, which may have allowed a wider interpretation of the results, especially when coupled with dynamic measurement of cortisol levels. The rather lower percentage of explained variance, although common in long-term studies, still hints at the absence of other relevant predictors of health that should be included in future studies. For example, chronic conditions (e.g., [rheumatoid arthritis](https://medlineplus.gov/rheumatoidarthritis.html)) or use of certain medications might explain CRP levels. Fourth, the path analysis model may not best reflect the associations between the predictor variables. It is possible that that longitudinal mediated processes are present for some of the variables, and the path analysis simply models the cross-sectional correlations. For example, the non-significant direct effect from hardiness to METS may be due to a mediated process through self-reported health. Last, as these tests were taken during a general health survey, we did not focus specifically on immune cells. Future, more detailed studies should elucidate the role of specific cytokines in long-term consequences.

Notwithstanding these limitations, our study makes an important contribution to the field. Our results emphasize the importance of the hardiness personality construct in long-term adaptation to captivity. Thus, the identification and screening of psychological resilience among soldiers during their training, and the integration of hardiness as part of combatants' fundamental military education, might be beneficial even after harsh experiences such as captivity for the future health of its survivors. Given the wide-spectrum of psychopathological outcomes and medical morbidities that reflect the accelerated aging process of ex-POWs, our results highlight the importance of hardiness for improved medical condition.

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Table 1. Descriptive statistics and correlations for study variables.

|  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Age | BMI | BP | PTSD 2015 | Depression 2015 | Self-rated health 2015 | Hardiness 1991 | CRP | METS | Telomere length |
| Age | 1.00 |  |  |  |  |  |  |  |  |  |
| BMI | -.088 | 1.00 |  |  |  |  |  |  |  |  |
| BP  | .134 | -.031 | 1.00 |  |  |  |  |  |  |  |
| PTSD 2015 | -.139 | .035 | .121 | 1.00 |  |  |  |  |  |  |
| Depression 2015 | -.216\* | -.004 | .058 | .480\*\* | 1.00 |  |  |  |  |  |
| Self-rated health 2015 | -.081 | -.151 | .066 | .315\*\* | .432\*\* | 1.00 |  |  |  |  |
| Hardiness 1991 | -.157 | .161 | -.268\* | -.304\*\* | -.335\*\* | -.359\*\* | 1.00 |  |  |  |
| CRP | -.154 | .188 | .135 | .047 | .125 | .058 | -.207 | 1.00 |  |  |
| METS | -.141 | .287\*\* | .348\*\* | .155 | .160 | .306\*\* | -.253\* | .164 | 1.00 |  |
| Telomere length | -.090 | -.061 | -.154 | -.026 | -.089 | -.146 | .359\*\* | -.130 | -.207 | 1.00 |
|  |  |  |  |  |  |  |  |  |  |  |
| Mean | 63.645 | 29.086 | .799 | 2.505 | 1.374 | 3.431 | 4.095 | 4.056 | 2.205 | 5.323 |
| (SD) | (3.634) | (6.799) | (.446) | (.839) | (1.003) | (1.026) | (.590) | (4.904) | (1.215) | (1.568) |
| \*Correlation significant at the .05 level, \*\* .01 level (2-tailed). *Note*. BMI=Body-mass index; BP=Blood pressure; PTSD=Posttraumatic stress disorder; C-reactive protein (CRP) levels; and metabolic syndrome (MetS). |

Table 2. Standardized regression coefficients for model predicting biological variables.

|  |  |  |  |
| --- | --- | --- | --- |
|  | CRP | METS | Telomere length |
| Age | -.194\* | -.162\* | -.022 |
| BMI | .225\*\* | - | -.112 |
| BP  | .114 | - | -.041 |
| PTSD 2015 | -.086 | .009 | .143 |
| Depression 2015 | .038 | -.047 | -.005 |
| Self-rated health 2015 | -.014 | .234\* | -.069 |
| Hardiness 1991 | -.259\*\* | -.205 | .372\*\*\* |
|  |  |  |  |
| R-squared | .153\* | .155\* | .191\* |

*Note*. \**p* <.05; \*\**p* < .01; \*\*\**p* < .001; ‘–‘= path not estimated; BMI=Body-mass index; BP=Blood pressure; PTSD=Posttraumatic stress disorder; C-reactive protein (CRP) levels; metabolic syndrome (MetS).

Figure 1. Path analysis model predicting biological variables.



*Note*. BMI=Body-mass index; BP=Blood pressure; PTSD=Posttraumatic stress disorder; C-reactive protein (CRP) levels; metabolic syndrome (MetS).