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ORIGINAL PAPER

I am a survivor, keep on surviving: early-life exposure to conflict and subjective survival probabilities in adult life

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Abstract
Life-course studies have shown that early-life conditions predict health and socio-economic status in adult life. This study analyzes whether experiencing a traumatic event in childhood, i.e., the Second World War (WW2), affects subjective survival probabilities (SSPs). We rely on a representative sample of European adults who were differentially exposed to WW2 during childhood as a result of their date and place of birth. Results show that exposure to WW2 increases SSPs, with socio-economic and health characteristics not playing a mediating role. War exposure also counterbalances the adverse effects of health impairments on SSPs, but it does not affect health outcomes per se. This fact, jointly with low mortality rates of the cohort under investigation, suggests that selective mortality and post-traumatic stress are not the main channels. Instead, the results support the hypothesis that personal growth and life appreciation emerge after traumatic events, thereby leading to optimistic perceptions of longevity.

Keywords Childhood conditions · Subjective survival probabilities · Second World War · Post-traumatic growth · Life-course approach · PTSD · Europe

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1 Introduction

Subjective probabilities of survival are used in demographic and economic research to predict health outcomes such as actual mortality or longevity (Perozek 2008; Smith et al. 2001) as well as individuals' choices involving uncertainty about the future (Hurd and McGarry 1995 and 2002; Manski 2004; Smith et al. 2001; Elder 2013; Wang 2014), e.g., retirement or financial decisions (Van Solinge and Henkens 2009; Post and Hanewald 2013; Nivakoski 2020) and future health investments (Rappange et al. 2016).

Survival expectations entail individual-specific factors (e.g., perceived health status) driving intertemporal choices that are unobservable to the policy-maker and unpredictable by relying solely on objective data. For instance, individuals who expect to live longer may decide to retire at an age that does not fit the longevity risk estimated in the official life tables (Mirowsky 1999; Hurd et al. 2004; Van Solinge and Henkens 2009). Similarly, subjective survival expectations may influence future risky health behaviors (Viscusi 1990; Gilleskie 1998; Adams et al. 2015). Even if individuals perfectly understand that healthy behaviors reduce morbidity and mortality, they can underestimate the benefits of engaging in those behaviors on the basis of their personal judgments about survival. These decisions may have non-negligible implications for welfare costs regarding, for instance, the sustainability of pensions and healthcare provision.

What are the determinants of subjective survival probabilities? Previous research has deemed past and current health status and health behaviors crucial determinants of the current expectations about future survival (Falba and Busch 2005; Hurd and McGarry 1995 2002; Kutlu-Koc and Kalwij 2017), which, in turn, as mentioned above, may influence future health. Other factors, such as socio-economic status, have also been found to influence subjective survival probabilities (SSPs) (Adams et al. 2014; Arpino et al. 2018). Among the factors entering the formation of survival expectations, childhood characteristics may also play an important role. Life-course research has suggested that the type of childhood experienced predicts quality of life in adulthood (e.g., Giuliano and Spilimbergo 2014; Layard et al. 2014; Elder 2018) and mortality (e.g., Su 2009; Doblhammer et al. 2013; Masters 2018). For instance, growing up in wartime and witnessing episodes of war-related violence in early childhood are largely recognized as key predictors of health and economic performance later in life (e.g., Kesternich et al. 2014; Havari and Peracchi 2017). However, whether exposure to such hardships produces positive or negative consequences is still debated.

On the one hand, a strand of literature documents that exposure to conflict in childhood has negative effects on a variety of human capital dimensions in adult life, such as health (Taylor 2010; Kesternich et al. 2014; Havari and Peracchi 2017; Singhal 2018), mortality (Akbulut-Yuksel 2014), education and labor market outcomes (Ichino and Winter-Ebmer 2004; Akbulut-Yuksel 2014), perceived quality of life (Kesternich et al. 2014), and social and risk attitudes (Conzo and Salustri 2017;

Voigtländer and Voth 2015; Bucciol and Zarri 2015; Bellucci et al. 2020). In addition to physical victimization, lack of familial and social support, psychological distress, and post-traumatic stress disorder (PTSD) emerge as candidate explanations to these negative impacts (Lustig et al. 2004; Pynoos et al. 2001). In fact, PTSD has been shown to persist over time well after the trauma, as documented by medical studies conducted on adults exposed to holocaust in the childhood (Kestenberg 1992; Lev-Wiesel and Amir 2003; Kuwert et al. 2008; Yehuda et al. 2007 and 2008), although other studies documented a reduction in PTSD as time since the traumatic event passes (e.g., Frankenberg et al. 2008).

On the other hand, another strand of literature suggests that war or other traumatic events witnessed in the past do not translate into lower well-being in the future. Research on resilience has shown that children recover rather quickly from trauma since they tend to adapt rapidly (Garmezy 1993; Boyden and Mann 2005). Thus, adverse events in childhood may not have major or long-lasting effects (Masten 2001). Positive psychological changes, instead, emerge because of higher appreciation of life after a traumatic experience. In this regard, it has been shown that exposure to war does not reduce life expectancy (Sagi-Schwartz et al. 2013) or mortality (Todd et al. 2017). Along these lines, some groups exposed to violence—e.g., the Jewish Holocaust survivors (Carmil and Breznitz 1991; Sigal 1998) and the Palestinians exposed to the first Intifada (Barber 2008)—appear resilient and politically engaged (Masten 2001; Werner 2007; Forstmeier et al. 2009), registering positive life changes in struggles with major life crises (Calhoun et al. 2000; Amir and Lev-Wiesel 2001; Lev-Wiesel and Amir 2003). These positive effects are consistent with the "post-traumatic growth" hypothesis: individuals who witnessed serious hardships develop "a significant beneficial change in cognitive and emotional life" (Tedeschi et al. 1998, p.3) that allows them not only to bounce back from trauma but to further develop and grow (Calhoun and Tedeschi 2014).

The previous discussion points to psychological consequences of exposure to war during childhood, which may (or may not) persist over time and have an impact on the formation of expectations about survival even at older ages. Some studies have examined the psychological construction of the determinants of survival expectations. Consistently with the idea that stress and anxiety are likely to create negative perceptions and thoughts about the future, they have been found to negatively impact subjective life expectancy, a close measure to SSP (Joubert 1992; Lester and Abdel-Khalek 2007; Griffin et al. 2013). On the opposite, dispositional optimism, a generalized expectancy that good things will happen in the future and bad things will not, has been found to be positively associated with subjective life expectancy (Griffin et al. 2013). Similarly, Kobayashi et al. (2017) showed a positive effect of an individual's positive psychological state on expectations about survival.

Based on the abovementioned studies on the legacies of childhood traumas and on the psychological determinants of expectations about survival, two competing hypotheses can be formulated. They lead to opposite predictions about the effect of war exposure on subjective survival probabilities. A negative effect can be hypothesized if children exposed to the traumatic events of the Second World War (WW2) manifest signs of PTSD also in the adulthood. A positive effect, on the contrary, is expected if, through the adverse life experience of WW2, individuals learn to appreciate their life to a larger extent and become more resilient and optimistic about their recovery capacity (post-traumatic growth, or PTG).

The current study enters this debate with the aim of providing new causal evidence on whether, and in which direction, exposure to WW2 during childhood affects subjective survival probabilities in adult life. We contribute to both the literature on the long-lasting effects of war exposure during childhood and on the formation of subjective survival probabilities in at least four directions. First, we consider a novel policy-relevant outcome for the literature on war exposure, i.e., subjective survival probabilities. Drawing from the above-mentioned studies, long-lasting psychological distress in the aftermath of a traumatic event would lead individuals to produce pessimistic expectations on their survival chances, whereas PTG and life appreciation would generate more optimistic expectations of survival.

Second, from a methodological point of view, we rely on a representative sample of European adults who happened to be exposed or non-exposed (or exposed to a varying extent) to WW2 early in life. This study exploits WW2 as a large-scale natural experiment, in which individuals from different cultural, economic, and institutional contexts faced a common shock, but were differentially exposed to it as a result of their date and place of birth. This characteristic of WW2 provides our results with higher external validity than those in most of the aforementioned studies, which focus on a single country or on a circumscribed population (Carmil and Breznitz 1991; Kestenberg 1992; Sigal 1998; Lev-Wiesel and Amir 2000; Masten 2001; Barber 2008; Werner 2007; Yehuda et al. 2007 and 2008; Kuwert et al. 2008; Bundervoet et al. 2009; Forstmeier et al. 2009; Sagi-Schwartz et al. 2013; Akbulut-Yuksel 2014; Singhal 2018).

Third, in order to explore the mechanisms behind our results, we use retrospective data and test if the socio-economic status of the family at the time of the war, the absence of a parent, and episodes of dispossession and hunger during WW2 absorb the effect of war on subjective survival probabilities. Such early-life circumstances are shown to be important predictors of human capital outcomes (Kesternich et al. 2014; Havari and Peracchi 2017; Islam et al. 2017) and hence emerge as candidate mediators of the war effects also on subjective survival probabilities.

Fourth, by exploiting the rich set of information on health conditions contained in the Survey on Health, Ageing and Retirement in Europe (SHARE), we also test whether early-life exposure to war moderates the negative effects of health impairments on subjective survival probabilities. If it is found that war exposure reduces the negative influence of bad health conditions on survival probabilities, this would offer support to the PTG hypothesis.

Identification of the causal effect of WW2 in our study rests on two sources of plausibly exogenous variation, i.e., month-place of war events and month-place of individuals' birth. Thus, the estimated impact of war exposure on later life subjective survival probabilities lends itself well to a causal interpretation. We also implement a series of robustness checks that additionally increase the validity of our results. The empirical strategy of this paper allows also to net out confounding effects deriving, for instance, from heterogeneity in the extent of destruction and recovery capacity of the respondents' regions, or in the human capital outcomes and childhood characteristics of the surveyed individuals.

Our results show that individuals exposed to WW2 episodes early in life report, on average, higher subjective survival probabilities than those who were not exposed. The effect of war exposure is larger if the number of war events the respondent has been exposed to is above the median. Current health conditions and behaviors, proxies for socio-economic status both in childhood and adulthood, or other childhood characteristics mediate the effect only marginally. In addition, hunger episodes and socio-economic conditions are the only childhood factors that play a role in subjective survival probabilities, though only through their effect on health and economic status in adulthood. However, even after adjusting for all the aforementioned factors, the effect of war on subjective survival probabilities persists.

Importantly, exposed and non-exposed respondents do not show different health outcomes in later life. This fact, jointly with low mortality rates and scarring effects for the cohort under investigation (e.g., Havari and Peracchi 2017), leads us to exclude selective mortality and post-traumatic stress as possible mechanisms driving our findings. On the other hand, war exposure counterbalances the negative effects of bad health conditions on subjective survival probabilities. This result suggests that personal growth and life appreciation may emerge after traumatic events, thereby leading to higher subjective survival probabilities for individuals exposed to WW2.

2 Data and descriptive statistics

We combine two sources of data. The first dataset is the SHARE. We consider waves 1, 2, 4, 5, and 6 for our variable of interest (*SSP*) as well as socio-demographic and health characteristics of individuals. Respondents' subjective expectations about their survival probabilities are measured by asking the following question: "What are the chances that you will live to be age x or more?," where x is a function of the age of each respondent (i.e., target age).[1]

We merge this information with wave 3 of SHARE (called "SHARELIFE") containing respondents' major life-history events, which have been retrieved retrospectively. We rely on these retrospective data to identify the region where respondents lived during WW2 and to obtain relevant information about their childhood including war-related circumstances such as dispossessions for persecution, parental absence, and hunger periods as well as number of books and of rooms in the house and the main occupation of the breadwinner at age ten, which we use to build a proxy for socio-economic status (SES) of their family of origin (as in Havari and Peracchi 2017).

The second source of data is an original database we have compiled about WW2 events. It contains information on war episodes including battles, attacks, bombings, invasions, and occupations as reported by Ellis (1993), Davies (2008), and Collier

[1] Based on respondents' age, x for *SSP* is defined as: 75 (age $<$ 65), 80 ($65 \leq$ age $<$ 70), 85 ($70 \leq$ age $<$ 75), 90 ($75 \leq$ age $<$ 80), 95 ($80 \leq$ age $<$ 85), 100 ($85 \leq$ age $<$ 95), 105 ($95 \leq$ age $<$ 100), 110 ($100 \leq$ age $<$ 105), and 120 (age $\geq$ 105).

(2004). Specifically, from these authors, we retrieved information on the region and the month-year of each war episode occurred between September 1939 and April 1945.[2]

We build two measures of exposure to war. The first measure is a dummy variable equal to one if the respondent was exposed to at least one war episode during WW2 (extensive-margin measure). Exposure is calculated by combining month-place of birth with month-place of war events. Individuals born at least 1 month before a war episode are considered as exposed. The second measure of war is a categorical variable equal to zero if the respondent was never exposed to the war, and equal to one or two if she is respectively below or above the median number of war episodes experienced by all sample respondents (ten episodes) (intensive-margin measure).

To show how our war variable has been recorded, we provide an example with Sicily, the largest island of Italy. Sicily has been exposed to 78 war episodes from April to September 1943, when the Allies occupied the region. Most episodes occurred in July and August (59 and 11, respectively), while only one episode occurred in September. Thus, all respondents born in Sicily before September 1943 have experienced at least 11 war episodes. Since this number is higher than the median number of episodes, the war indicator takes value one, while the war categorical variable is equal to two. Respondents born in September 1943 experienced only the war episode occurred in their month of birth, and hence, both the war indicator and the war categorical variable are equal to one. Finally, respondents born after September 1943 are considered as non-exposed since they were born after the last war episode occurred in Sicily.

Our sample is composed by individuals born during the WW2 (Sept 1939–Apr 1945), in the following countries: Austria, Germany, Sweden, The Netherlands, Italy, Spain, France, Denmark, Greece, Switzerland, Belgium, Ireland, Czechoslovakia, and Poland; among these countries, Spain,[3] Switzerland, Ireland, and Sweden did not experience any WW2-related event. We restrict the analysis only to individuals who have never moved to other regions during WW2 to mitigate the bias arising from selective migration. However, in a robustness check, we relax this restriction and also check whether migration acts as a mediator or moderator of war exposure.

We consider only the war cohort since, in comparison with older cohorts, individuals born during WW2 are less subject to mortality and scarring effects of war exposure as documented by Havari and Peracchi (2017), which could produce a selection bias in our estimates. As a direct test for cohort differences in the war effects, we include in the sample also individuals aged 7–12 during the war and find significant effects only for those who experienced a war episode earlier in life (i.e., when they were 0–6 years old). For the older age group (7–12), instead, effects are smaller

[2] WW2 officially began with the German invasion of Poland on September 1, 1939 and conflicts in our sample countries ceased in April 1945.

[3] Since Spain has experienced the Civil War before the beginning of WW2, we have also performed a robustness check excluding Spain from our sample. Results show similar patterns and are available upon request.

in magnitude and statistically insignificant.[4] Older cohorts (age 12 or more) have been excluded also because of their likely involvement into the army (in some cases conscription of girls and boys started at age 12). For all these reasons, our analysis focuses on individuals who were age 0–6 during the war period, which allows us also to rely on the month and place of birth during the war as sources of (within-region) exogenous variation in WW2 exposure. Given that we are considering a homogeneous cohort in terms of age during the war period and that the identification of our effects rests on variations in month and place of birth, our estimates capture the effect of war exposure and not the effect of subsequent conditions experienced by the war cohort (e.g., better labor market opportunities).

Table 1 contains descriptive sample statistics.[5] Respondents are on average 67 years old, have two children and three grandchildren. Half of the sample is composed by female respondents, and the average number of schooling years is 10.6. Most respondents are retired (70%) and married (76%). With respect to health conditions, respondents report on average one chronic disease. Regarding healthy behaviors, 14% of respondents declare to be a smoker at the time of the interview and 19% do not drink alcohol, while the same proportion declares to do so almost every day. Similarly, the share of respondents practicing sport activities more than once a week is 35%, while those who ever or hardly never engage into such activities is 39%. On the basis of their body mass index, most respondents fall into the normal-weight (33%) or overweight (44%) category.

When looking at the distribution of SSP by age (Table 7 in Appendix), the subjective probability of living until a given age or more ("target age") is, as expected, decreasing in respondents' age, starting by an average chance of 70% to survive until age 75 or more for respondents aged less than 65, and then decreasing to a minimum of 56% for respondents aged between 75 and 80 (who were asked to estimate their chances of survival until age 90 or more).

The maps shown in Fig. 1 highlight a substantial variability in the distribution of SSP and war exposure across the NUTS-1 regions contained in our sample. War-exposed respondents have better health behaviors (alcohol consumption, smoking, and sport activities) and labor market outcomes (employment, retirement) than non-exposed ones, while the latter have on average higher income (Table 1).[6] No significant differences between the exposed and the non-exposed are found in terms of marital status and number of chronic diseases, while the exposed have on average fewer children than the non-exposed. As expected, the prevalence of adverse childhood circumstances related to war (dispossessions for persecution, hunger episodes, and absence of biological father) is higher among respondents exposed to WW2. However, SES in childhood—the first factor extracted from a principal component analysis on number of books and rooms in the house (in logarithm) and the main occupation of the breadwinner at age ten—does not statically differ by war exposure.

[4] Results are summarized in Table 17 in Appendix.

[5] See Table 6 in Appendix for variable legend.

[6] SHARE contains for each respondent the imputed household net income in Euros (variable *thinc_m*). We take the logarithm of this variable (see variable legend in Appendix).

Table 1 Descriptive statistics

Variable	Share/mean			*P*-value	Z-stat
	Total (10,668 obs.)	No war (5418 obs.)	War (5250 obs.)		
SSP	65.71	66.4	65.03	0.065	1.846
War	49.2	0	1		
Female	53.3	53.0	53.5	0.019	0.984
Country of birth					
Austria	5.4	2.8	8.2		
Belgium	10.9	1.4	20.7		
Czechoslovakia	5.0	7.3	2.7		
Denmark	8.4	16.2	0.4		
France	6.7	1.4	12.1		
Germany	7.7	0.3	15.4		
Greece	8.7	16.3	0.9		
Italy	7.1	1.4	22.5		
Ireland	13.3	4.4	0.0		
The Netherlands	7.4	2.5	12.5		
Poland	3.1	1.6	4.8		
Spain	7.4	14.6	0.0		
Sweden	10.4	20.4	0.0		
Switzerland	4.8	9.5	0.0		
Wave					
1	24.5	23.1	25.9	0.000	−5.393
2	29.9	31.0	28.8	0.000	4.358
4	0.2	0.1	0.2	0.081	−1.746
5	22.6	20.9	24.4	0.000	−5.953
6	22.8	24.8	20.6	0.000	7.918
Target age					
75	45.5	45.7	45.4		
80	17.7	18.0	17.4		
85	33.9	33.4	34.5		
90	2.8	2.9	2.8		
Age	67.19	67.10	67.29	0.194	−1.300
Childhood SES (High)	49.5	50.5	51.8	0.897	−0.131
Mother at age 10	96.3	96.3	96.3	0.688	0.401
Father at age 10	88.7	91.3	86.1	0.000	5.208
Hunger	5.6	4.2	7.0	0.000	−3.984
Dispossession	2.5	1.4	3.8	0.000	−4.449
Log(Income)	9.96	9.91	10.02	0.000	−6.165
Years of education	10.57	10.54	10.59	0.465	−0.731
No. children	2.20	2.25	2.15	0.005	2.793
No. grandchildren	2.83	2.84	2.82	0.455	0.747

Table 1 (continued)

Variable	Share/mean			*P*-value	Z-stat
	Total (10,668 obs.)	No war (5418 obs.)	War (5250 obs.)		
Job status					
Retired	70.1	65.5	74.8	0.000	−7.719
Employed	11.8	16.7	6.8	0.000	12.414
Unemployed	1.5	1.4	1.6	0.477	−0.711
Sick or disabled	2.2	2.5	2.0	0.097	1.660
Homemaker	12.6	12.5	12.8	0.314	−1.007
Other	1.4	1.0		0.001	−4.044
Marital status					
Married	76.3	76.1	76.5	0.585	−0.546
Never married	4.9	4.9	5.0	0.792	0.264
Divorced	6.9	7.7	6.0	0.194	1.299
Widowed	11.9	11.2	12.6	0.426	−0.796
# chronic diseases	1.18	1.16	1.21	0.531	−0.627
Alcohol					
Never	19.2	18.8	19.6	0.901	−0.125
Less than 1 a month	6.4	6.7	6.1	0.604	0.518
1–2 a month	8.5	9.2	7.8	0.185	1.325
1–2 a week	15.1	17.4	12.8	0.000	4.065
3–4 a week	6.6	7.0	6.3	0.699	0.386
5–6 a week	2.2	1.9	2.5	0.046	−1.955
Almost every day	19.1	14.0	24.3	0.000	−9.838
Smoker	13.6	14.9	12.2	0.000	3.529
BMI class					
Underweight	0.9	0.9	0.9	0.883	−0.148
Normal	33.2	34.7	31.7	0.062	1.867
Overweight	43.6	42.5	44.8	0.100	−1.643
Obese	20.8	19.7	21.8	0.298	−1.041
Sport activities					
More than 1 a week	34.8	6.4	33.2	0.099	1.650
1 a week	15.3	16.3	14.3	0.046	1.955
1–3 a month	11.0	11.9	10.0	0.025	2.241
Hardly ever or never	38.8	35.2	42.4	0.000	−5.782

3 Methods

We estimate the following regression model:

$$SSP_{ijt} = \beta_0 + \beta_1 War_{jt} + \beta_k \sum\nolimits_k X_{ijt,k} + \lambda_j + \pi_t + \varepsilon_{ijt} \qquad (1)$$

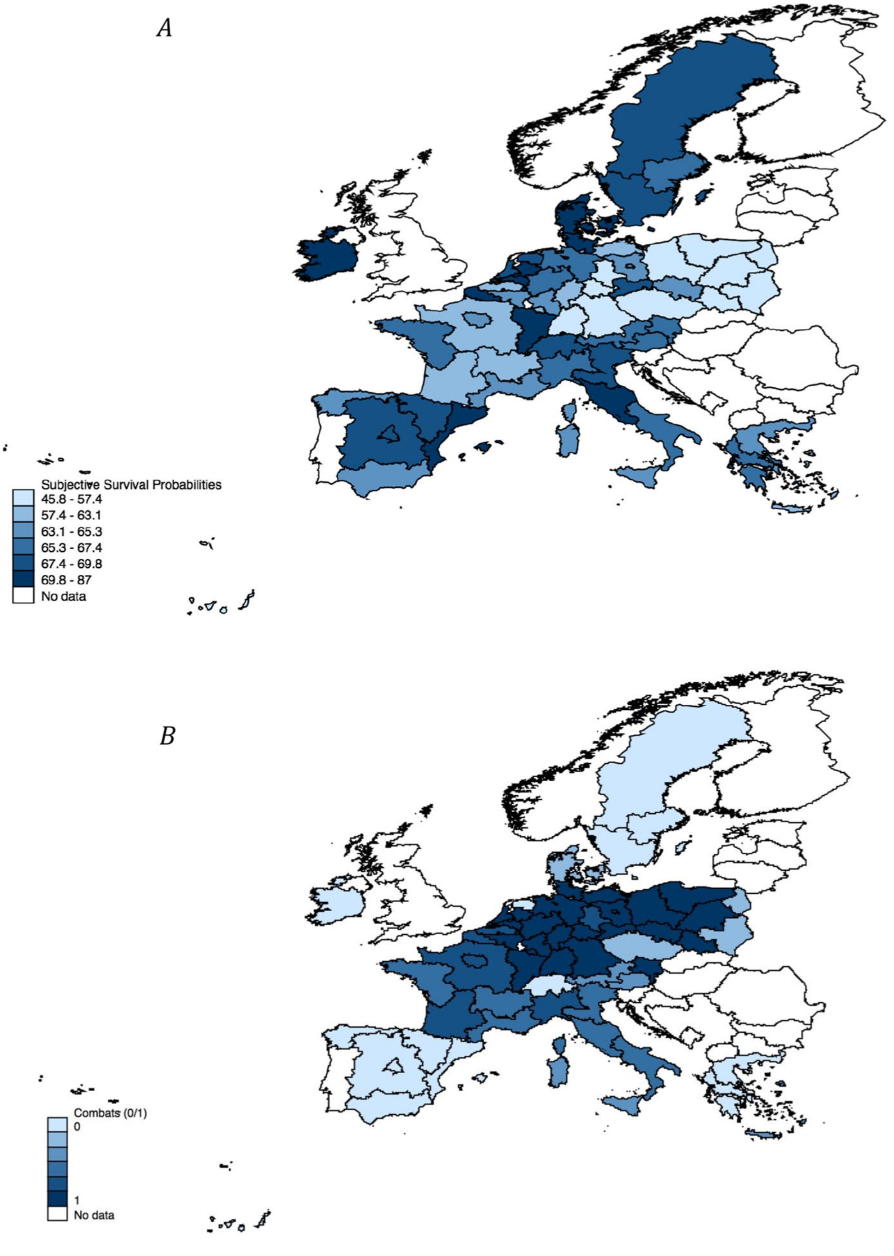

Fig. 1 Subjective survival probabilities and war in Europe. **A** Subjective survival probabilities in Europe. Note: Average subjective survival probabilities by region (NUTS-1 level). **B** Share exposed to WW2. Note: Share of respondents who have been exposed at least to one war event by region (NUTS-1 level)

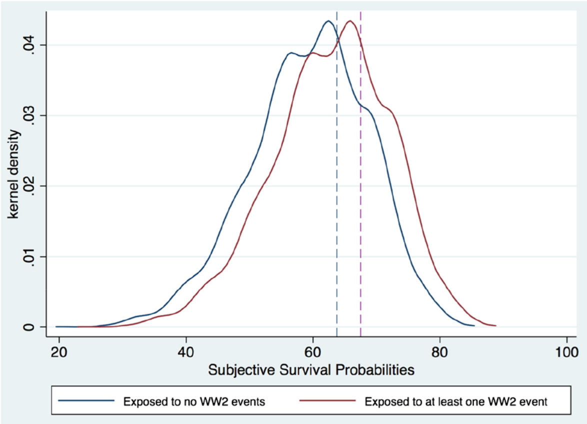

Fig. 2 Subjective survival probabilities and war. Notes: subjective survival probabilities adjusted for gender, month-year of birth, wave of interview, and country of birth. Red and blue dashed lines represent the adjusted means of SSP for respondents never exposed and exposed to WW2, respectively

where SSP_{ijt} is the subjective survival probability of individual i born in month-year t in country j; λ and π are fixed effects for the respondents' country of birth (CoB) and date of birth (month-year, DoB), respectively (estimated using two sets of dummy variables); War_{jt} measures war exposure (at the intensive or extensive margin); and X_{ijt} is a vector of socio-demographic characteristics including socio-economic status (SES) in childhood[7] and in adulthood (i.e., income, years of education, job status, no. of children and grandchildren). More specifically, we implement a stepwise inclusion of (potentially endogenous) controls to assess the robustness of our results to variables that may directly be affected by the war experience.

Additional models also include a proxy for health status (number of chronic diseases) and several measures of health behavior (alcohol consumption, smoking, physical activity, and body mass index) in order to examine the mediating role of these factors. To assess whether war exposure moderates the relationship between current health status and SSP, in additional regressions we also include a measure of health status and the interaction of this measure with war exposure—we repeat this analysis for several health indicators provided by SHARE (see "Sect. 5"). Furthermore, in alternative specifications country fixed effects are replaced by region fixed effects, which circumscribe war exposure at a finer geographical level.

All specifications are estimated through pooled OLS and include controls for gender (female), age, target age, and the interaction between age and target age to net out the age effect as well as the time distance to the target age when assessing survival expectations (see e.g., Arpino et al. 2018). To account for possible period effects, all models also include dummy variables for wave of interview (wave) and, given the (unbalanced) panel structure of the data, standard errors are clustered at individual level.

[7] First extracted factor from a principal component analysis on no. of books and no. of rooms at home at age 10, and breadwinner's occupation as in Havari and Peracchi (2017)—see variable legend in Appendix.

Table 2 The impact of war exposure on subjective survival probabilities

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War	3.655**	3.567**	3.538**	3.546**	3.358**	3.131**
	(1.118)	(1.120)	(1.121)	(1.120)	(1.117)	(1.088)
Female	−0.561	−0.605	−0.603	−0.656	0.461	1.088
	(0.645)	(0.645)	(0.646)	(0.644)	(0.709)	(0.710)
Childhood SES		1.905**	1.964**	1.970**	1.094	0.662
		(0.706)	(0.711)	(0.709)	(0.719)	(0.695)
Mother at age 10			0.429	0.449	0.259	0.0572
			(1.763)	(1.748)	(1.728)	(1.678)
Father at age 10			−0.983	−1.102	−1.017	−1.232
			(1.198)	(1.192)	(1.179)	(1.126)
Hunger				−3.574*	−3.066†	−1.940
				(1.598)	(1.596)	(1.521)
Dispossession				−1.852	−2.476	−2.497
				(2.506)	(2.491)	(2.321)
Adulthood controls	No	No	No	No	Yes	Yes
Health and lifestyle controls	No	No	No	No	No	Yes
Observations	10,674	10,674	10,674	10,668	10,668	10,668
R-squared	0.085	0.087	0.087	0.088	0.097	0.131

Robust standard errors in parentheses clustered at individual level. Adulthood controls include income, years of education, marital status, job status, number of children, and number of grandchildren; health and lifestyle include the number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities. All models include dummies for month-year of birth and country of birth, wave, age, target age, and age * target age. Missing values are flagged for all controls

**$p < 0.01$, *$p < 0.05$, †$p < 0.1$

4 Results

4.1 The effect of WW2 exposure on SSP

Figure 2 shows the distribution of the average SSP by war exposure, adjusting for gender, month-year of birth, wave of interview, and country of birth. Respondents exposed to at least one war episode report on average higher subjective probabilities of living until the target age than non-exposed ones.[8]

Table 2 contains the main results from pooled OLS regressions of SSP on exposure to war (regression coefficients for all controls are in Table 8 of Appendix). In column 1, we estimate our baseline model (without controls) and, consistent with Fig. 2, find a positive effect of exposure to at least one war episode in childhood on SSP at the time of interview. The estimates indicate that individuals exposed to

[8] We checked if results were heterogeneous across gender and we did not find any statistically significant difference between males and females. Similarly, results did not change when controlling for country of residence rather than the country of birth. Results are available upon request.

WW2 reported an average SSP 3.6 percentage points higher than their counterparts not exposed to WW2.

In the second column of Table 2, we include childhood SES to test whether heterogeneity in socio-economic conditions of the family of origin accounts for the estimated effect of war exposure. While SES in childhood positively predicts SSP, the effect of war exposure is unaltered by the inclusion of the new regressor.

Since WW2 caused severe human losses, the impact of war exposure on perceived survival chances could be influenced by the absence of a parent during childhood, which might have shaped cognitive and non-cognitive outcomes of children besides affecting parental investment in their health and education. Our estimates suggest that this is not the case since the war effect does not change when controlling for the presence of biological father and mother at age ten (Table 2, column 3).

Furthermore, during WW2 many civilians witnessed episodes of dispossession due to prosecution for ethnic or political reasons, and of hunger, mainly because of food shortages resulting from war-specific events (e.g., the end of food supply from occupied territories in Germany towards the end of WW2 or the Dutch Famine in 1944–1945). Not only these events might have affected children's mental well-being and resilience capacity, but they could also have produced effects on their physical health, with consequences on subjective survival probabilities in adulthood (Tamis-LeMonda et al. 2004; Glaesmer et al. 2011; Werner 2012; Kesternich et al. 2014, 2015; Havari and Peracchi 2017). We take into account these potential mechanisms by controlling for whether respondents have witnessed any episode of dispossession as well as for whether they suffered from hunger during their life.[9] We find that hunger is negatively associated with SSP in later life, whereas the war effect does not change in magnitude and statistical significance (Table 2, column 4).[10] In addition, the effect of war exposure is robust when adjusting for current differences in socio-demographic characteristics (column 5) and health status and behavior (column 6).

Overall, these results suggest that early-life exposure to war has a positive impact on SSP later in life, regardless of the characteristics of childhood environment and of the socio-economic and health status in adulthood. Adjusting for all childhood and adulthood controls only reduces the gross WW2 effect of about 14% (model 1 vs. model 6, Table 2).

When distinguishing the effect of war exposure depending on the number of war episodes, we find that subjective survival probability increases in the number of WW2 episodes witnessed early in life (Table 3). However, the difference between

[9] We also consider dispossession and hunger episodes occurred during the WW2 only. Results do not change substantially and are available upon request.

[10] A possible concern when dealing with retrospective information is measurement error. In supplementary materials available upon request, we show that information on parental absence and episodes of dispossession and hunger retrieved from respondents' memory are consistent with historical facts regarding WW2. Consider that the reliability of childhood information (and consistency between retrospective data and historical data) has been documented also by other recent studies using SHARELIFE, e.g., Kesternich et al. (2014), Havari and Mazzonna (2015), Havari and Peracchi (2017), and Van den Berg et al. (2016). Moreover, the distribution of these variables by country and year is in line with the results shown by Kesternich et al. (2014) who include also older cohorts, thereby suggesting that lack of significance of these events is not driven imperfect recall by respondents who were too young during WW2.

Table 3 The impact of war exposure on subjective survival probabilities

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War						
Below median (1–9)	3.512**	3.420**	3.385**	3.412**	3.234**	2.754*
	(1.174)	(1.175)	(1.176)	(1.175)	(1.171)	(1.141)
Above median (10+)	3.971**	3.892**	3.876**	3.842**	3.631**	3.957**
	(1.339)	(1.339)	(1.341)	(1.341)	(1.333)	(1.297)
Female	−0.560	−0.604	−0.603	−0.655	0.465	1.101
	(0.645)	(0.645)	(0.646)	(0.644)	(0.709)	(0.710)
Childhood SES		1.907**	1.967**	1.972**	1.096	0.666
		(0.706)	(0.710)	(0.709)	(0.719)	(0.694)
Mother at age 10			0.420	0.442	0.251	0.0355
			(1.762)	(1.748)	(1.728)	(1.674)
Father at age 10			−0.992	−1.111	−1.025	−1.256
			(1.198)	(1.192)	(1.179)	(1.126)
Hunger				−3.573*	−3.065†	−1.930
				(1.598)	(1.596)	(1.522)
Dispossession				−1.854	−2.477	−2.502
				(2.508)	(2.492)	(2.321)
Adulthood controls	No	No	No	No	Yes	Yes
Health and lifestyle controls	No	No	No	No	No	Yes
Observations	10,674	10,674	10,674	10,668	10,668	10,668
R-squared	0.085	0.087	0.087	0.088	0.098	0.131

Robust standard errors in parentheses clustered at individual level. Adulthood controls include income, years of education, marital status, job status, number of children, and number of grandchildren; health and lifestyle include the number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities. All models include dummies for month-year of birth and country of birth, wave, age, target age, and age * target age. Missing values are flagged for all controls

**p < 0.01, *p < 0.05, †p < 0.1

the two coefficients is not statistically significant.[11] Also in these regressions, childhood and adulthood characteristics do not sizeably account for the estimated effect of WW2 on SSP (regression coefficients for all controls are in Table 9 of Appendix). In fact, war coefficient reduces by 21% (14%) when comparing the specification in column 6 with that of column 1 of Table 3 (Table 2), yet remains sizeable and statistically significant.

[11] In an alternative specification, we considered a 4-level categorization of war events: no war event and three categories based on the tertiles for positive number of war events. Results confirm that the effect of experiencing war events is positive and statistically significant, with a non-monotonic path. The coefficients of the three categories corresponding to different levels of war exposure are not statistically different among themselves. Results are available upon request.

4.2 WW2 exposure as moderator of the effect of health conditions on SSP

The evidence showing a positive effect of war exposure on SSP provides support to the post-traumatic growth hypothesis discussed in the Introduction. To bring further evidence in support of this hypothesis, we re-estimate the effect of WW2 on subjective survival probabilities (as in the model in column 6 of Table 2) by interacting a number of health indicators measured at the time of interview with the war-exposure dummy. Post-traumatic growth stemming from traumatic early-life exposure to WW2 would increase resilience and self-perceived adaptation skills and make individuals appreciate their life and stay optimistic even in case of negative events (e.g., poor health conditions), thereby counteracting the negative effect of health impairments on subjective survival probabilities. We therefore expect a negative association between poor health and SSP but a positive effect of the interaction between the former and war exposure. This approach is similar to those adopted in previous studies on resilience after a traumatic event (e.g., Lakomý and Kafková 2017; Shrira et al. 2010) in the absence of a direct measure of resilience, which could be obtained using psychometric scales specially developed (see Windle et al. 2011). Results in Table 4 provide evidence of such moderating role of war exposure for several mobility and morbidity indicators including, for instance, having received by a doctor diagnosis of hypertension or having experienced a stroke.[12] Let consider, for example, the case of hypertension. High blood pressure is known (not only among researchers but also among the general population) to be a risk factor for other diseases and for mortality (Prospective study collaboration 2002). Our results indicate that people are aware of this; in fact, they incorporate diagnosis of hypertension in the assessment of their survival probability. Among individuals not exposed to WW2, those who have been diagnosed with hypertension report SSPs that are about 4.8 points lower than their counterparts not being diagnosed with this medical condition.

This negative effect of hypertension on SSP is also found among the WW2-exposed, yet it is weaker (2.5 points). This implies that also the WW2-exposed understand the risks of hypertension and incorporate them in the formation of their own survival expectations, yet they weight these risks lower than individuals who were not exposed to WW2.[13]

These results are consistent with the hypothesis that post-traumatic growth emerging after war exposure increases resilience and leads traumatized individuals to appreciate their lives more, so that they estimate a longer life-duration (in spite of health impairments) than non-traumatized ones.

[12] Regression results and the descriptive statistics for all health proxies contained in the dataset (jointly with the robustness check including migrants) are in Tables 11, 12, and 13in Appendix.

[13] The interaction effect between the war dummy and hypertension can also be interpreted in an alternative way. The effect of WW2 among individuals who have not been diagnosed with hypertension is about 2.4 point. This effect doubles (4.8) among those who suffer high blood pressure, meaning that the war effect emerges especially among people who are in worse health conditions in later life. Similar interactions effects are found for other health indicators.

Table 4 The moderating role of war exposure on health and subjective survival probabilities

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)
War	2.860**	2.546*	3.005**	1.811	2.352†
	(1.097)	(1.152)	(1.100)	(1.231)	(1.215)
Iadla	−15.47***				
	(3.195)				
War * Iadla	13.86**				
	(4.360)				
Lgmuscle		−7.215***			
		(0.846)			
War * Lgmuscle		1.925†			
		(1.155)			
Stroke			−12.56***		
			(2.188)		
War * Stroke			7.649*		
			(3.359)		
# chronic diseases				−3.668***	
				(0.344)	
War * #chronic diseases				1.060*	
				(0.497)	
Hypertension					−4.742***
					(0.823)
War * Hypertension					2.491*
					(1.176)
Observations	10,668	10,668	10,663	10,668	10,663
R-squared	0.117	0.125	0.118	0.131	0.118

Robust standard errors in parentheses clustered at individual level. All models include childhood SES, adulthood controls (income, years of education, marital status, job status, number of children, and number of grandchildren), health and lifestyle (number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities), and dummies for month-year of birth and country of birth, wave, age, target age, and age * target age. Missing values are flagged for all controls. See variable legend in Appendix, Table 6, for definitions of controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

4.3 Substantive importance of the effect of war exposure on SSP

The analysis carried out above documents that the effect of early exposure to war on subjective survival probabilities is statistically significant and robust. The magnitude of the effect ranges between three and four percentage points. Even considering the lower bound of three percentage points, we can argue that the estimated effect is not only statistically but also substantially significant.

In fact, three percentage points is a non-negligible effect if compared to important correlates of subjective and objective survival (Table 8 in Appendix). For example, it is similar in absolute value to the effect of one additional chronic condition or to the effect of a gap of 13 years of education (3/0.236) on subjective survival probabilities. It is also higher than the effect of being a smoker or overweight.

5 Robustness checks

5.1 The role of migration

Our sample is restricted to individuals that never changed their region of residence during WW2. This restriction is likely to deliver a rather homogenous sample of stayers that, while potentially detracting from the external validity of results, allows us to exclude respondents who might have decided to move for unobserved reasons that might be correlated with war exposure and SSP. However, we could still be missing important part of the WW2 impact as most cities were abandoned after massive bombings. Moreover, voluntary or forced relocations were frequent during WW2 because of house destruction, border changes, and ethnic or political prosecution in most European regions. It is therefore possible that relocations influenced perceived survival because, for instance, the new housing, financial, and schooling environment shaped the cognitive and non-cognitive development of the child or produced long-term effects on health. Not accounting for migration could generate an omitted variable bias if, for instance, voluntary relocation was used as a war-coping strategy against war exposure, thereby leading to higher subjective survival probabilities in adult life.

To assess the role of migration, we control for a dummy variable equal to one for respondents who moved to another region in the period 1939–1945[14] and re-estimate the models in Table 2 including also these individuals. Results (Table 10 in Appendix) show that migration does not act as a mediator (columns 1–6) nor as moderator (column 7) of the war effect on SSP, which remains similar numerically and in terms of statistical significance. As an additional test for endogenous migration, we regressed the number of times each respondent changed region of residence from their date of birth up to the interview date on war exposure, as defined in our baseline models. We found no statistically significant correlation between WW2 exposure and migration (including any reported move) among individuals in our sample (results are available upon request).

Another source of sample selection in our estimates can derive from out-of-sample migration, i.e., relocations towards countries outside SHARE. For instance, respondents with high SES—which is positively associated with subjective survival

[14] The third wave of our SHARE dataset (SHARELIFE) asks each respondent the year when she has changed region of residence from the birthdate to the date of interview. Our migration variable is a (0/1) dummy taking value one if the respondent has changed region of residence during the war period at least once. Migrants across regions represent 3.18% of our sample, a fraction consistent with real data considering that the population in our sample countries counted 344 million in 1939 (Lahmeyer 2006, *Populstat* [online]; available at http://www.populstat.info; own elaboration) and the estimated number of refugees in Europe in 1945 is seven million (Barnett 2002). In supplementary materials available upon request, we further document consistency between migration in our data and historical facts.

probabilities—may relocate to non-war places more easily than low-SES respondents through their higher financial resources, influential connections with visa officials and denser personal networks. Consider that this source of bias in our case would lead to the underestimation of the true WW2 effect—the positive effect of war exposure on SSP would have been higher if high-SES migrants were included. However, Kesternich et al. (2014) show that out-migration during and after the war (1939–1947) was a rare phenomenon, and hence, this source of selection is not a major concern in our analysis.

5.2 Selective mortality

An alternative explanation to our finding of a positive effect of WW2 on SSP can be sample selection due to mortality. For instance, healthier individuals could be more likely to survive WW2 events and therefore become more optimistic about their longevity. If this was the case, the estimated effect of war would be biased upwards, due to the unobserved individuals' characteristics connected with both exposure to war and SSP.

We can exclude this type of selection for two reasons. First, Havari and Peracchi (2017) document low mortality rates and scarring effects for the cohort considered in the current study (respondents born during WW2). Second, we run OLS regressions of health indicators on war exposure in order to check for significant health differences by war exposure which could signal a potential sample selection problem due to mortality. In other words, if individuals in poor health conditions were more likely to die during or after exposure to WW2, we should find a positive and significant association between early-life exposure to war and poor health in adulthood. Our estimates document that this is not the case—there are no significant differences by war exposure in most health measures (Table 5),[15] also when we include migrants (Table 14 in Appendix). Hence, selection on mortality does not seem to drive our findings.

Furthermore, our estimates may be biased also because of differential mortality by SES induced by the war. Low-SES respondents have been shown to report lower subjective survival probabilities than the high-SES individuals (Arpino et al. 2018). If mortality was higher among low-SES individuals, this would lead to an overestimation of the war effect. Kesternich et al. (2014) address this issue by comparing the age of death of the SHARE participants' father by SES, war vs. non-war countries, and year of birth (before 1946 vs. after 1945). They find that both low- and high-SES respondents face approximately the same reduction in the age of death of fathers over the two periods, i.e., 0.8 of a year for non-war countries and 0.4 for war countries. These figures suggest that this type of selection is not large enough to drive our findings.

To check econometrically the role of war-induced differential mortality by SES, we regress age of death of respondents' fathers on the SES dummy (*SES Childhood*),

[15] This result remains true when we do not control for health and lifestyle factors as well as hunger and dispossession episodes, which are factors that may be correlated with our health measure of interest.

Table 5 War exposure and health outcomes

Dep. var	War	Std. err	N	R-squared
Adlwa	−0.00687	(0.00956)	10,668	0.091
Iadla	−0.0143*	(0.00645)	10,668	0.053
Mobility	−0.0204	(0.0197)	10,668	0.202
Lgmuscle	$6.70e^{-05}$	(0.0205)	10,668	0.147
Gross motor	−0.0188	(0.0150)	10,668	0.144
Fine motor	−0.00111	(0.0109)	10,668	0.075
Stroke	−0.0110	(0.00763)	10,663	0.031
Cancer	0.00377	(0.00794)	10,663	0.028
# chronic diseases	−0.0525	(0.0553)	10,668	0.134
Hypertension	0.0189	(0.0232)	10,663	0.107
Heart attack	−0.0224	(0.0165)	10,663	0.055
Cholesterol	−0.00600	(0.0222)	10,663	0.051
Diabetes	−0.0237	(0.0183)	10,663	0.088
Parkinson	−0.000406	(0.00242)	10,663	0.028
Stomach ulcer	−0.00225	(0.00825)	10,663	0.032
Cataracts	−0.000494	(0.0102)	10,663	0.038
Lung disease	−0.00679	(0.0116)	10,663	0.033
Hip fracture	−0.00301	(0.00474)	10,663	0.025

Each line summarizes the main results from a regression of the selected health measure on war exposure (controls as in column 6 of Table 2). See variable legend in Appendix, Table 6, for definitions of controls. Robust standard error in parentheses clustered at individual level

*$p < 0.05$

on a dummy equal to one if the respondent is in a war country (*War countries*), on a time-trend variable built using respondents' month-year of birth (*DoB*), and on the interaction among these three variables. Regression results (Table 18 in Appendix) show that high SES is positively correlated with fathers' longevity, while living in a war country reduces age of death of fathers, most likely because they were in the army during WW2. No interaction term is significant, suggesting that war-induced mortality does not differentially change over time by SES (columns 2 and 4). Similar results are obtained when replacing the time-trend variable with dummy variables for respondents born before, during and after WW2 (Table 19 in Appendix).

5.3 Within- and between-country heterogeneity

All estimates presented so far include dummies for respondents' country of birth, which account for the geographic variation in exposure to war. To account for the within-country differences in exposure to war and SSP as shown in Fig. 1A and B, we replace country fixed effects with region of residence fixed effects at NUTS-1 level (as in the EU official nomenclature). The use of region instead of country dummies reduces potential unobserved differences between the treatment

(WW2-exposed) and control (not WW2-exposed) group, allowing to compare individuals living in the same region who share similar political institutions, health systems/policies, intensity of war episodes and post-war recovery paths. Moreover, since data on the exact location of respondents' residence during WW2 are not available in SHARE, relying on within-region variation in war exposure would allow us to mitigate measurement error in the exposure measures. In addition, while respondents living miles away from a war episode are considered exposed in the same manner as those living in the place where that event occurred, our results can be nevertheless considered as lower-bound estimates of the real war-exposure effect.

In Table 16 of Appendix, we replicate the estimates in Table 2 with region instead of country dummies. Our main results are robust to this check.[16]

5.4 Endogenous fertility

Fertility decisions during WW2 could have been affected by war events. For instance, mothers who anticipated war episodes could have postponed childbirth to non-war periods. If fertility control in Europe during WW2 was, reasonably, more frequent among high-status classes, respondents whose health would have been better in any case are underrepresented in our sample, thereby generating a downward bias to our estimates. Given that previous studies have consistently shown that good health conditions are positively correlated with subjective survival probabilities (and this is also empirically supported by our estimates in Table 8, column 6 in Appendix), the estimated effect of WW2 exposure should be considered as a lower-bound of true war effect. However, to assess the extent of war-induced differential fertility by SES, we rely on the fertility statistics calculated for SHARE respondents by Kesternich et al. (2014). The authors compare the number of children per mother by SES across three periods, i.e., pre-war (before 1939), during war (1939–1945), and post-war (after 1945). As expected, higher fertility is found for the low-SES group, while the fertility time trend is similar for high- and low-SES respondents. For both groups before and during war, there has been a fertility increase of about 0.14 children per woman and of about one child when looking at during war and after-war periods.

As an additional check for endogenous fertility, we compare SSP across groups of war-exposed respondents conceived before the first war-episode (*C_Bef*) with those respondents conceived afterwards (*C_After*). More specifically, we implement two sets of analyses, where the group of children conceived before the first war episode is defined as those conceived up to 3 and 5 months before the event, respectively (results are displayed in Fig. 3A and B in Appendix). For parents of the *C_Bef* groups the first WW2 event was presumably more unpredictable than for parents of the *C_After* group, which includes respondents conceived when

[16] To account for heterogeneity between war and non-war countries, we also re-estimate the specifications in Table 2 excluding non-war countries. This check allows us to avoid overestimation of the war effect resulting from the inclusion of a large control group of respondents who experienced no war events. Results are very similar to those in Table 2 (Table 15 in Appendix).

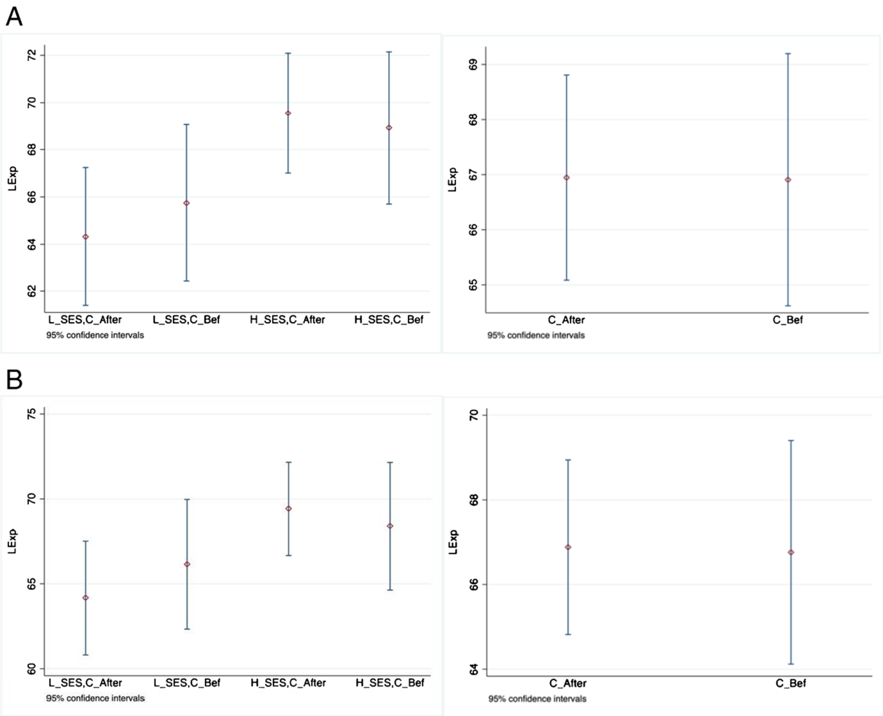

Fig. 3 **A** Average SSP for respondents conceived up to 3 months before and after the first war episode in the region. **B** Average SSP for respondents conceived up to 5 months before and after the first war episode in the region

WW2 already reached the region. If there was a war-induced fertility adjustment, we might have observed a significant difference in SSP across these two groups. However, we find no significant differences in SSP between those conceived before and those conceived after the first war event (right-hand side panels of Fig. 3A and B in Appendix), neither within the high-SES group nor within the low-SES group (left-hand side panels of Fig. 3A and B in Appendix).

By comparing the *C_After* and the *C_Bef* groups we can draw two additional conclusions. First, selective out-of-sample migration, potentially due to high-SES parents relocating in response to WW2 episodes, would not entirely explain our results. This type of migration would have implied significantly different SSP among *C_After* and *C_Bef* respondents, especially in the high-SES group. Figure 3A and B in Appendix suggests that this is not the case.

The second consideration refers to the "fetal origin" hypothesis (Barker 1990), which posits that adverse environmental circumstances before and immediately after the birth negatively affect mental and physical health (de Rooij et al. 2010; Van den Berg et al. 2016; Oskorouchi 2019) as well as socio-economic indicators (Almond and Currie 2011) during childhood and adult life. The childhood and adulthood proxies for health and socio-economic status we have included in the previous regressions should account for the indirect effects of "in utero" exposure to war. However, mothers' stress or health problems during pregnancy do not seem to be the key explanation to our findings. Pregnant mothers of the *C_After* group were exposed to WW2 episodes for a longer period than their counterparts in the *C_Bef* group. If WW2 affected respondents' survival expectations when they were "in utero," a significant difference in SSP between the *C_After* and *C_Bef* group should be observed. Figure 3A and B in Appendix shows that this is not the case, both when comparing the *C_After* and *C_Bef* group (right-hand panels) and when considering also SES differences between these two groups (left-hand panels).

6 Discussion and concluding remarks

This paper examined whether early-life exposure to a large-scale conflict predicts subjective survival probabilities in later life. By focusing on an underexplored, though relevant, aspect of aging, the current study bridges two main literatures. One literature documents that childhood conditions, such as exposure to war, influence several dimensions of life in the adulthood, whereas the other uses subjective survival probabilities as an important aging measure alternative to other health measures. Exploiting variation in the time and place of conflict episodes, this study provides empirical evidence on whether, and in which direction, early-life exposure to the Second World War influences subjective judgments about longevity.

Based on previous studies on the legacies of childhood traumas and on the psychological determinants of expectations about survival, two competing hypotheses can be formulated which lead to opposite predictions about the effect of war exposure on subjective survival probabilities. A negative effect can be hypothesized if children exposed to the traumatic events of WW2 manifest signs of PTSD also in the adulthood. A positive effect, on the contrary, is expected if, through the adverse life experience of WW2, individuals learn to appreciate their life to a larger extent and become more resilient and optimistic about their recovery capacity (post-traumatic growth, PTG).

Our evidence provides support to this last hypothesis. Other things equal, respondents exposed to WW2 events during childhood tend to report higher subjective probabilities of survival than those who were not exposed. The estimated effect is driven neither by systematic differences in health conditions (which would have signaled war-induced differential mortality) nor by a large set of childhood and adulthood characteristics. Furthermore, the adverse effect of health impairments on subjective survival probabilities is positively moderated by early-life exposure to WW2, meaning that the negative effect of bad health conditions on subjective assessments of survival are reduced or even eliminated for those who were exposed to WW2 during childhood. This suggests that the war experience buffers the individuals' judgments about their own longevity against several health conditions (e.g., hypertension and stroke) and mobility difficulties they face in adult life.

Since exposed and non-exposed respondents do not systematically differ in terms of health, a possible interpretation to our results is that the higher life appreciation and resilience acquired through the war experience make affected individuals that are overly optimistic about their longevity. This conclusion is also based on the fact that, as other authors (Kesternich et al. 2014; Havari and Peracchi 2017), we did not find evidence of selective mortality for the cohorts under investigation. Using data from the Human Mortality Database, Havari and Peracchi (2017) document that death rates rose in the period between the two world wars, but they did not find any scarring effects at later ages among the survivors of the cohorts born during the WW2 period. This means that if one considers individuals who survived until older ages, no substantive difference in (objective) mortality risks should be found between those exposed and those not exposed to WW2. Similar conclusions have been reached by Sagi-Schwartz et al. (2013) and Todd et al. (2017).

Because of a low number of death events observed across the survey waves (about 300 cases), we could not directly compare subjective and objective survival probabilities. However, our finding that individuals exposed to WW2 report higher subjective survival probabilities, combined with the fact that WW2 was not found to impact health conditions and that we did not find evidence of selective mortality, suggests that war-exposed individuals tend to overestimate their survival chances. This is also consistent with the Griffin et al.'s (2013) finding that, even after controlling for health conditions, those who scored high on their optimism index were significantly more likely to report a subjective life expectancy higher than actuarial estimates.

However, our results may seem contradictory with an opposing view of optimism as a positive determinant of subjective well-being and hence of health. The beneficial health effects of optimism could have nevertheless mattered before the date of interview. Put differently, the lack of significant health differences between the exposed and the non-exposed to war might signal the positive role of life appreciation in improving the health status of the former, whose health would have been worse otherwise. This does not imply that overly optimistic evaluations of survival chances would have negligible consequences on future health. Quite on the contrary, it has been argued, and empirically proven, that optimism has positive effects on subjective well-being and health only if it is not excessive and unrealistic (for a review see Diener and Chan 2011). A recent study (Chipperfield et al. 2018), for example, found that it is maladaptive to be unrealistically optimistic when health subsequently declines in reality. In fact, the risk of death was considerably higher for individuals with optimistic expectations that were unrealistic than for individuals with realistic expectations. Given that health declines are stronger and more rapid at older ages, it can be speculated that especially at older ages negative effects of (overly) optimistic expectations may prevail over positive effects.

Against this background, our estimates suggest that individuals exposed to negative early-life circumstances such as WW2 may have generated optimistic views about their capabilities and resistance to negative external events. While this overly optimistic view might have produced null or positive consequences on subjective well-being and health in their childhood and adulthood, it might be nonetheless harmful in subsequent phases of life. This could also explain, in part, why longer lives do not seem to be also satisfied lives, as observed in recent studies (e.g. Nemitz, 2021).

This study does not come without limitations. Future research should focus, for instance, on the effect of the scale of each war event, which could not be estimated here due to lack of information. It would be worth to investigate if an increase in the number of casualties occurred in each event monotonically raises later-life subjective survival probabilities, or if what matters, instead, is just exposure to any casualty, regardless of the number.

Another limitation of this study stems from the limited data available about respondents' parents. One mechanism through which pre-school children could develop PTG is through interaction with parents. Pre-school years is deemed a very sensitive period and an age where war exposure may persist throughout life (Leon 2012; Currie and Vogl 2013; Arroyo and Eth 1996; Pynoos and Nader 1993), because of—among other mechanisms—psychological distress (Kijewski and Freitag 2016), confusion, and self-blame (Dorresteijn et al. 2019). These reactions might be magnified by children's perception that caregivers where stressed or extremely worried during traumatic events. Moreover, anxiety caused by war episodes may harness emotional stability of parents, thereby setting the grounds for poor caregiving (Punamäki et al. 1997). For instance, food or job scarcity during the war might have required high parental effort in terms of time, physical, and cognitive resources, with coping activities carried out often outside the household. Regarding the WW2, the seminal work by Burlingham and Freud (1942) suggested that children under 5 years were little affected by bombing if they were not injured, and if they were in their mother's care and their mother showed no signs of panic. Furthermore, pre-school children are also less exposed to social interactions outside the family than their older peers, and hence, they might have known traumatic events through the "filter" of their parents; therefore, they might have transformed such events—consciously/deliberatively or unconsciously/unintentionally (e.g., Zoellner and Maercker 2006)—into a positive or negative experiences over time, depending not only on the type of interaction developed with their parents during the war, but also on the valence of the memories and emotions about the events transmitted by their caregivers. Thus, an interesting avenue for future research is to examine how parental attitudes towards the WW2 affected the development of PTG of children, and how this in turn affected children's survival expectations.

Furthermore, the focus on children aged 0 to 6 during the war period in this paper allowed us to rely on month and place of birth as sources of exogenous variation. However, we did not find evidence of any effect for older children. The finding that the effect of WW2 exposure on SSP was limited to younger children indeed deserves further research. The role of age at experiencing a trauma and subsequent response is complex, as different mechanisms may theoretically operate in opposite directions (Masten and Narayan 2012). Some studies on traumas at early ages found higher resilience and a lower probability of developing negative outcomes the younger the age at the trauma (e.g., Jensen & Shaw 1993; Pine et al. 2005), because of young children cognitive immaturity, higher plasticity of personality at younger ages, and more compensatory care provided to the youngest children (Masten and Narayan 2012). However, other studies found a negative relation between age at trauma and PTG (e.g., Kimhi et al. 2009a,b; Yu et al. 2010), or no relation at all (Hafstad et al. 2010, 2011; Kilmer & Gil-Rivas 2010). Hence, future studies should further explore the theoretical and empirical effects of early childhood exposure to war on the development of post-traumatic growth.

Notwithstanding these limitations, our results have non-negligible implications for both the individual and the society. Studies on the subjective dimension of health and well-being emphasize the importance of individuals' perceptions about their current status and expectations about its future dynamic. In particular, coherently with the theory of life-cycle behavior (Hurd et al. 2004), subjective assessment of survival probabilities influences decisions in different spheres of life such as retirement, investments, and healthy behaviors (Adams et al. 2015; Carbone et al. 2005; Salm 2010; Scott-Sheldon et al. 2010). An upward bias in predicting the probability of survival might have detrimental consequences both for the individuals' well-being and the costs of the welfare state (especially in aging societies). For instance, it might increase the chances of taking risky financial decisions and of delaying retirement. Individuals who are overly optimistic about their future longevity might also be more likely to undertake unhealthy behaviors, such as smoking or drinking (Adams et al. 2015), and less likely to go to doctors and take medications (Pressman and Cohen 2005).

Concluding, our study showed that having experienced WW2-related events in childhood increased subjective survival probabilities in later life. Individuals who witnessed such adversities early in life might develop the belief that, in spite of the health impairments associated with aging, they are survivors and keep on surviving.

Appendix

Tables 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18 and 19

Table 6 Variable legend

Variable	Description
SSP	0–100 scale variable for the respondent's expectation to live for at least 10 years. It answers the question "What are the chances that you will live to be age *x* or more?," where *x* is the target age belonging to the set {75, 80, 85, 90, 95, 100, 105, 110}. Based on respondents' age, *x* for *SSP* is defined as: 75 (age < 65), 80 (65 ≤ age < 70), 85 (70 ≤ age < 75), 90 (75 ≤ age < 80), 95 (80 ≤ age < 85), 100 (85 ≤ age < 95), 105 (95 ≤ age < 100), 110 (100 ≤ age < 105), and 120 (age ≥ 105)
Target age	Age targeted for the SSP question (75, 80, 85, 90, 95, 100, 105, 110)
War	(0/1) dummy for the exposure to at least 1 war episode during the WW2
War (above and below the median)	Categorical variable measuring the exposure to war events during the WW2 with respect to the median number of war events to which respondents have been exposed (i.e., 10). 0 = never exposed; 1 = exposed to 1–9 war episodes; 2 = exposed to 10 + war episodes
Female	(0/1) dummy for females
Age	Age of respondents at the time of interview

Table 6 (continued)

Variable	Description
Childhood SES (High)	(0/1) dummy for respondent with childhood SES above the median. Childhood SES has been computed with the first extracted factor from a principal component analysis (PCA) on childhood socio-economic characteristics (natural logarithm of the no. of rooms at age 10, natural logarithm of the no. of books at age 10, occupation of the breadwinner at age 10)
Mother at age 10	(0/1) dummy for biological mother alive when the respondent was 10
Father at age 10	(0/1) dummy for biological father alive when the respondent was 10
Hunger	(0/1) dummy for experience of hunger episodes during the whole life
Dispossession	(0/1) dummy for experience of dispossession episodes during the whole life
Log(Income)	Logarithm of net household income. In wave 1, SHARE generates the variable from the respondents' gross household income that is before taxes and subsidies, and then adjusts it throughout the EU tax-benefit micro-simulation model EUROMOD; in the subsequent waves SHARE imputes net household income by aggregating all individual net incomes at household level
Years of education	Years of education of respondent
Children	No. of children of respondent
Grandchildren	No. of grandchildren of respondent
Job (Ref = Retired)	Categorical variable for job status (retired, employed or self-employed, unemployed, sick or disabled, homemaker)
Marital status (Ref = Married)	Categorical variable for the marital status of respondents (1 = married, 2 = never married, 3 = divorced, 4 = widowed)
# chronic diseases	Number of chronic diseases that respondent has declared as diagnosed by doctors (among the following 12: heart attack, high blood pressure or hypertension, high blood cholesterol, stroke or cerebral vascular disease, diabetes or high blood sugar, chronic lung disease, cancer or malignant tumor, stomach or duodenal ulcer, peptic ulcer, Parkinson, cataracts, hip fracture or femoral fracture)
Alcohol consumption (Ref = Never)	Categorical variable for the days of alcoholic drinks consumption (1 = never, 2 = less than 1 a month, 3 = 1–2 a month, 4 = 1–2 a week, 5 = 3–4 a week, 6 = 5–6 a week, 7 = almost every day)
Smoker	(0/1) dummy for the smokers
BMI class (Ref = Normal)	Categorical variable for the body mass index (BMI) class (1 = underweight, 2 = normal, 3 = overweight, 4 = obese)
Sport (Ref = More than once a week)	Categorical variable for the days of sport activities (1 = more than 1 a week, 2 = 1 a week, 3 = 1–3 a month, 4 = hardly ever of never)
DoB	Date of birth of respondent (month-year)
Country of birth	Country of birth of respondent
Wave	Wave of interview (1, 2, 4, 5, 6)
Migrated	(0/1) dummy for respondent who has changed region of residence during the WW2 period

Table 6 (continued)

Variable	Description
Mobility measures	(0/1) dummy for the respondent with difficulties in performing at least one of the following tasks:
Adlwa	Dressing, bathing or showering, and eating or cutting up food (Wallace and Herzog)
Iadla	Telephone calls, taking medications, and managing money
Mobility	Walking 100 m, walking across a room, climbing several flights of stairs, and climbing one flight of stairs
Lgmuscle	Sitting 2 h, getting up from chair, stooping, kneeling, crouching, and pulling or pushing large objects
Gross motor	Walking 100 m, walking across a room, climbing one flight of stairs, and bathing or showering
Fine motor	Picking up a small coin, eating or cutting up food, and dressing
Disease	(0/1) dummy for the respondent having being told by a doctor to have one of the following diseases (i.e., being treated or bothered by the disease at the time of interview):
Stroke	Stroke or cerebral vascular disease
Cancer	Cancer or malignant tumor
Hypertension	High blood pressure or hypertension
Heart attack	Heart attack
Parkinson	Parkinson disease
Stomach ulcer	Stomach or duodenal or peptic ulcer
Cholesterol	High blood cholesterol
Diabetes	Diabetes or high blood sugar
Cataracts	Cataracts
Lung disease	Chronic lung disease
Hip fracture	Hip fracture or femoral fracture

Table 7 Subjective probabilities of living until target age or more

Target age	Obs	Mean	Std. dev	Min	Max
75	4856	70.04	24.574	0	100
80	1889	64.92	27.002	0	100
85	3621	61.10	26.524	0	100
90	302	56.26	27.643	0	100

Note: There are no individuals aged 80 or more in our sample. Hence, the highest target age considered is 90

Table 8 The impact of war exposure on subjective survival probabilities (extensive margin)

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War	3.655**	3.567**	3.538**	3.546**	3.358**	3.131**
	(1.118)	(1.120)	(1.121)	(1.120)	(1.117)	(1.088)
Female	−0.561	−0.605	−0.603	−0.656	0.461	1.088
	(0.645)	(0.645)	(0.646)	(0.644)	(0.709)	(0.710)
Age	1.674	1.627	1.618	1.562	2.088†	1.857†
	(1.102)	(1.101)	(1.101)	(1.100)	(1.102)	(1.072)
Target age (Ref = 75)						
80	−66.75	−67.09	−65.74	−69.21	−57.66	−65.17
	(76.49)	(76.46)	(76.49)	(76.49)	(76.90)	(76.32)
85	−31.08	−31.44	−31.32	−32.60	−29.86	−37.35
	(27.53)	(27.50)	(27.50)	(27.51)	(27.78)	(27.67)
90	−161.6	−163.7	−166.4	−185.9	−161.9	−153.0
	(494.7)	(494.5)	(494.9)	(492.3)	(496.8)	(498.1)
Age * SSP target age (Ref = 75)						
80	0.890	0.896	0.876	0.928	0.747	0.860
	(1.148)	(1.147)	(1.148)	(1.148)	(1.154)	(1.145)
85	0.275	0.281	0.279	0.299	0.245	0.353
	(0.400)	(0.399)	(0.399)	(0.400)	(0.404)	(0.402)
90	1.909	1.939	1.973	2.235	1.899	1.782
	(6.566)	(6.564)	(6.569)	(6.535)	(6.594)	(6.611)
High childhood SES		1.905**	1.964**	1.970**	1.094	0.662
		(0.706)	(0.711)	(0.709)	(0.719)	(0.695)
Mother at age 10			0.429	0.449	0.259	0.0572
			(1.763)	(1.748)	(1.728)	(1.678)
Father at age 10			−0.983	−1.102	−1.017	−1.232
			(1.198)	(1.192)	(1.179)	(1.126)
Hunger				−3.574*	−3.066†	−1.940
				(1.598)	(1.596)	(1.521)
Dispossession				−1.852	−2.476	−2.497
				(2.506)	(2.491)	(2.321)
Log(Income)					0.605†	0.442
					(0.337)	(0.332)
Years of education					0.295**	0.236**
					(0.0918)	(0.0888)
Children					−0.224	−0.168
					(0.320)	(0.311)
Grandchildren					0.233†	0.217†
					(0.134)	(0.129)
Job (Ref = Retired)						
Employed					2.904**	2.137*
					(0.906)	(0.886)

Table 8 (continued)

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
Unemployed					−2.699	−2.905
					(2.023)	(2.002)
Sick or disabled					−7.689***	−4.446*
					(1.959)	(1.904)
Homemaker					−1.664†	−1.668†
					(0.997)	(0.977)
Other					−0.873	−0.0504
					(2.137)	(2.043)
Marital status (Ref = Married)						
Never married					−3.875*	−3.221*
					(1.585)	(1.535)
Divorced					−1.361	−0.743
					(1.390)	(1.346)
Widowed					−2.647*	−1.926†
					(1.048)	(1.030)
# chronic diseases						−3.135***
						(0.258)
Alcohol consumption (Ref = Never)						
Less than once a month						0.579
						(1.184)
1/2 days a month						1.509
						(1.094)
1/2 a week						1.623†
						(0.949)
3/4 days a week						4.561***
						(1.133)
5/6 days a week						2.888
						(1.776)
Almost everyday						1.746†
						(0.927)
Smoker						−1.897*
						(0.892)
BMI class (Ref = Normal)						
Underweight						−1.360
						(2.911)
Overweight						1.153†
						(0.669)
Obese						0.453
						(0.877)
Sport (Ref = More than once a week)						
Once a week						−3.199***
						(0.736)

Table 8 (continued)

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
1–3 times a month						−3.344***
						(0.827)
Hardly ever or never						−5.631***
						(0.658)
DoB	Yes	Yes	Yes	Yes	Yes	Yes
Country of birth	Yes	Yes	Yes	Yes	Yes	Yes
Wave	Yes	Yes	Yes	Yes	Yes	Yes
Observations	10,674	10,674	10,674	10,668	10,668	10,668
R-squared	0.085	0.087	0.087	0.088	0.097	0.131

Robust standard errors in parentheses clustered at individual level. Missing values are flagged for all controls

***$p<0.001$, **$p<0.01$, *$p<0.05$, †$p<0.1$

Table 9 The impact of war exposure on subjective survival probabilities (intensive margin)

Dep. Var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War						
Below median (1-9)	3.512**	3.420**	3.385**	3.412**	3.234**	2.754*
	(1.174)	(1.175)	(1.176)	(1.175)	(1.171)	(1.141)
Above median (10+)	3.971**	3.892**	3.876**	3.842**	3.631**	3.957**
	(1.339)	(1.339)	(1.341)	(1.341)	(1.333)	(1.297)
Female	-0.560	-0.604	-0.603	-0.655	0.465	1.101
	(0.645)	(0.645)	(0.646)	(0.644)	(0.709)	(0.710)
Age	1.697	1.650	1.642	1.584	2.108†	1.915†
	(1.104)	(1.102)	(1.103)	(1.102)	(1.103)	(1.074)
Target age (Ref = 75)						
80	-66.32	-66.65	-65.29	-68.80	-57.27	-63.99
	(76.47)	(76.44)	(76.47)	(76.46)	(76.88)	(76.27)
85	-31.15	-31.51	-31.39	-32.66	-29.92	-37.54
	(27.53)	(27.51)	(27.50)	(27.51)	(27.79)	(27.68)
90	-165.0	-167.2	-170.0	-189.1	-164.9	-162.3
	(494.8)	(494.7)	(495.0)	(492.5)	(497.0)	(498.4)
Age * Target age (Ref = 75)						
80	0.884	0.889	0.869	0.922	0.741	0.842
	(1.147)	(1.147)	(1.147)	(1.147)	(1.153)	(1.144)
85	0.276	0.282	0.279	0.300	0.245	0.355
	(0.400)	(0.399)	(0.399)	(0.400)	(0.404)	(0.402)
90	1.954	1.985	2.021	2.277	1.938	1.905
	(6.568)	(6.566)	(6.570)	(6.537)	(6.596)	(6.615)
High childhood SES		1.907**	1.967**	1.972**	1.096	0.666
		(0.706)	(0.710)	(0.709)	(0.719)	(0.694)

Table 9 (continued)

Dep. Var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
Mother at age 10			0.420	0.442	0.251	0.0355
			(1.762)	(1.748)	(1.728)	(1.674)
Father at age 10			-0.992	-1.111	-1.025	-1.256
			(1.198)	(1.192)	(1.179)	(1.126)
Hunger				-3.573*	-3.065†	-1.930
				(1.598)	(1.596)	(1.522)
Dispossession				-1.854	-2.477	-2.502
				(2.508)	(2.492)	(2.321)
Log(Income)					0.605†	0.440
					(0.337)	(0.332)
Years of education					0.295**	0.235**
					(0.0918)	(0.0887)
Children					-0.221	-0.161
					(0.320)	(0.311)
Grandchildren					0.231†	0.212
					(0.134)	(0.129)
Job (Ref = Retired)						
Employed					2.902**	2.126*
					(0.907)	(0.887)
Unemployed					-2.682	-2.855
					(2.024)	(2.007)
Sick or disabled					-7.697***	-4.457*
					(1.958)	(1.903)
Homemaker					-1.669†	-1.680†
					(0.997)	(0.977)
Other					-0.886	-0.0887
					(2.138)	(2.045)
Marital status (Ref = Married)						
Never married					-3.861*	-3.175*
					(1.583)	(1.531)
Divorced					-1.359	-0.731
					(1.391)	(1.346)
Widowed					-2.654*	-1.943†
					(1.049)	(1.030)
# chronic diseases						-3.145***
						(0.259)
Alcohol consumption (Ref = Never)						
Less than once a month						0.577
						(1.183)
1/2 days a month						1.529
						(1.094)

Table 9 (continued)

Dep. Var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
1/2 a week						1.617†
						(0.949)
3/4 days a week						4.581***
						(1.135)
5/6 days a week						2.951†
						(1.778)
Almost everyday						1.767†
						(0.926)
Smoker						-1.919*
						(0.892)
Bmi class (Ref = Normal)						
Underweight						-1.396
						(2.906)
Overweight						1.164†
						(0.669)
Obese						0.449
						(0.877)
Sport (Ref = More than once a week)						
Once a week						-3.192***
						(0.736)
1—3 times a month						-3.350***
						(0.827)
Hardly ever or never						-5.650***
						(0.657)
DoB	Yes	Yes	Yes	Yes	Yes	Yes
Country of birth	Yes	Yes	Yes	Yes	Yes	Yes
Wave	Yes	Yes	Yes	Yes	Yes	Yes
Observations	10,674	10,674	10,674	10,668	10,668	10,668
R-squared	0.085	0.087	0.087	0.088	0.098	0.131

Robust standard errors in parentheses clustered at individual level. Missing values are flagged for all controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, $^{\dagger}p < 0.1$

Table 10 The role of migration

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)	(7)
War	3.690***	3.583***	3.557***	3.572***	3.411***	3.153***	3.266**
	(1.111)	(1.111)	(1.112)	(1.112)	(1.107)	(1.076)	(1.078)
Migrated	− 2.198	− 2.426	− 2.453	− 2.360	− 2.496	− 2.225	− 0.269
	(1.953)	(1.958)	(1.962)	(1.967)	(1.938)	(1.867)	(3.012)
War * Migrated							− 2.992
							(3.743)
Female	− 0.402	− 0.449	− 0.450	− 0.488	0.649	1.227†	1.221†
	(0.636)	(0.636)	(0.637)	(0.636)	(0.699)	(0.700)	(0.700)
Childhood SES		1.813**	1.866**	1.875**	0.958	0.507	0.517
		(0.691)	(0.696)	(0.695)	(0.706)	(0.681)	(0.681)
Mother at age 10			0.389	0.424	0.229	− 0.0578	− 0.0486
			(1.714)	(1.703)	(1.679)	(1.624)	(1.626)
Father at age 10			− 0.842	− 0.960	− 0.898	− 1.223	− 1.227
			(1.177)	(1.175)	(1.160)	(1.106)	(1.107)
Hunger				− 3.031*	− 2.577†	− 1.446	− 1.429
				(1.539)	(1.531)	(1.459)	(1.459)
Dispossession				− 1.136	− 1.709	− 1.959	− 1.982
				(2.388)	(2.373)	(2.218)	(2.215)
Adulthood controls	No	No	No	No	Yes	Yes	Yes
Health and lifestyle controls	No	No	No	No	No	Yes	Yes
Observations	11,007	11,007	11,007	11,001	11,001	11,001	11,001
R-squared	0.085	0.086	0.086	0.087	0.097	0.131	0.131

Robust standard errors in parentheses clustered at individual level. Adulthood controls include income, years of education, marital status, job status, number of children, and number of grandchildren; health and lifestyle include the number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities. All models include dummies for month-year of birth and country of birth, wave, age, target age, and age * target age; missing values are flagged for all controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 11 Descriptive statistics of diseases and health functionalities

Health variable	With migrated				Without migrated			
	% yes	No. no	No. yes	N	% yes	No. no	No. yes	N
Mobility indicators								
Adlwa	7.2	10,207	794	11,001	7.2	9906	762	10,668
Iadla	2.0	10,776	225	11,001	2.0	10,451	217	10,668
Mobility	28.1	7906	3095	11,001	28.1	7668	3000	10,668
Lgmuscle	37.2	6911	4090	11,001	37.2	6701	3967	10,668
Gross motor	13.3	9539	1462	11,001	13.3	9254	1414	10,668
Fine motor	7.9	10,137	864	11,001	7.8	9836	832	10,668
Diseases								
At least one chronic disease	66.1	3730	7266	10,996	66.1	3610	7053	10,663
Stroke	3.1	10,658	338	10,996	3.1	10,334	329	10,663
Cancer	4.6	10,494	502	10,996	4.5	10,182	481	10,663
Hypertension	40.9	6501	4495	10,996	40.8	6311	4352	10,663
Heart attack	11.2	9768	1228	10,996	11.2	9472	1191	10,663
Cholesterol	26.7	8056	2940	10,996	26.8	7810	2853	10,663
Diabetes	13.0	9571	1425	10,996	13.1	9268	1395	10,663
Parkinson	0.8	10,910	86	10,996	0.8	10,580	83	10,663
Stomach ulcer	4.3	10,519	477	10,996	4.3	10,203	460	10,663
Cataracts	6.8	10,248	748	10,996	6.8	9938	725	10,663
Lung disease	5.9	648	10,348	10,996	5.9	10,038	625	10,663
Hip fracture	1.3	138	10,858	10,996	1.3	10,528	135	10,663

See variable legend in Table 6 for details on the health variables

Table 12 The moderating role of war exposure on health and subjective survival probabilities (excluding migrants)

	War	Std. err	Health impairment	Std. err	War * Health impairment	Std. err	Obs	R-squared
Mobility indices								
Adlwa	3.119**	(1.109)	−9.638***	(1.832)	1.776	(2.342)	10,668	0.120
Iadla	2.860**	(1.097)	−15.47***	(3.195)	13.86**	(4.360)	10,668	0.117
Mobility	2.550*	(1.141)	−9.103***	(0.957)	1.748	(1.289)	10,668	0.130
Lgmuscle	2.546*	(1.152)	−7.215***	(0.845)	1.933†	(1.155)	10,668	0.125
Gross motor	2.833**	(1.095)	−8.974***	(1.349)	2.084	(1.782)	10,668	0.122
Fine motor	3.079**	(1.107)	−10.93***	(1.685)	2.652	(2.182)	10,668	0.122
Diseases								
# chronic diseases	1.811	(1.231)	−3.668***	(0.344)	1.060*	(0.497)	10,668	0.131
Stroke	3.005**	(1.100)	−12.56***	(2.188)	7.649*	(3.359)	10,663	0.117
Cancer	3.359**	(1.106)	−5.681**	(2.105)	−0.148	(2.793)	10,663	0.115
Hypertension	2.352†	(1.215)	−4.742***	(0.823)	2.491*	(1.176)	10,663	0.118
Heart attack	3.229**	(1.129)	−7.499***	(1.365)	−0.446	(1.933)	10,663	0.121
Parkinson	3.304**	(1.105)	−9.221†	(5.410)	8.451	(6.721)	10,663	0.114
Stomach ulcer	3.293**	(1.103)	−3.355†	(2.004)	0.984	(2.756)	10,663	0.114
Cholesterol	2.858*	(1.159)	−3.626***	(0.910)	1.697	(1.278)	10,663	0.116
Diabetes	2.964**	(1.137)	−7.363***	(1.243)	1.467	(1.885)	10,663	0.120
Cataracts	3.448**	(1.116)	−0.094	(1.495)	−1.650	(2.199)	10,663	0.114
Lung disease	3.197**	(1.103)	−6.536***	(1.750)	1.719	(2.491)	10,663	0.116
Hip fracture	3.384**	(1.106)	0.360	(3.159)	−3.857	(4.799)	10,663	0.113

Each row displays regression coefficients from the full specification model in Table 2, column 6, with SSP as dependent variable and war and the interaction between war and the listed health impairment as additional controls. The variable "# chronic diseases" is excluded from each specification

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 13 The moderating role of war exposure on health and subjective survival probabilities (including migrants)

	War	Std. err	Health impairment	Std. err	War * Health impairment	Std. err	Obs	R-squared
Mobility indices								
Adlwa	3.162**	(1.099)	−9.896***	(1.810)	2.072	(2.309)	11,001	0.120
Iadla	2.902**	(1.087)	−15.42***	(3.151)	13.83**	(4.261)	11,001	0.117
Mobility	2.545*	(1.128)	−9.228***	(0.944)	1.886	(1.267)	11,001	0.130
Lgmuscle	2.405*	(1.138)	−7.576***	(0.840)	2.468*	(1.141)	11,001	0.125
Gross motor	2.840**	(1.085)	−9.046***	(1.321)	2.282	(1.743)	11,001	0.122
Fine motor	3.111**	(1.098)	−11.21***	(1.656)	2.892	(2.144)	11,001	0.122
Diseases								
# chronic diseases	1.855	(1.213)	−3.691***	(0.339)	1.034*	(0.486)	11,001	0.131
Stroke	3.077**	(1.089)	−12.19***	(2.187)	7.463*	(3.313)	10,996	0.117
Cancer	3.414**	(1.095)	−5.575**	(2.093)	−0.103	(2.754)	10,996	0.115
Hypertension	2.551*	(1.200)	−4.599***	(0.814)	2.074†	(1.157)	10,996	0.118
Heart attack	3.237**	(1.117)	−7.598***	(1.350)	−0.137	(1.901)	10,996	0.121
Parkinson	3.354**	(1.094)	−10.12†	(5.237)	8.534	(6.612)	10,996	0.114
Stomach ulcer	3.33913**	(1.093)	−3.733†	(1.951)	1.277	(2.686)	10,996	0.114
Cholesterol	2.836*	(1.143)	−3.764***	(0.900)	1.957	(1.259)	10,996	0.116
Diabetes	2.998**	(1.126)	−7.425***	(1.227)	1.605	(1.860)	10,996	0.120
Cataracts	3.502**	(1.104)	−0.588	(1.479)	−1.701	(2.171)	10,996	0.114
Lung disease	3.259**	(1.090)	−6.431***	(1.731)	1.303	(2.454)	10,996	0.116
Hip fracture	3.435**	(1.095)	0.398	(3.172)	−3.604	(4.744)	10,996	0.113

Each row displays regression coefficients from the full specification model in Table 2, column 6, with SSP as dependent variable and war and the interaction between war and the listed health impairment as additional controls. The variable "# chronic diseases" is excluded from each specification

***$p<0.001$, **$p<0.01$, *$p<0.05$, †$p<0.1$

Table 14 The impact of war exposure on health (including migrants)

Dep. var	War	Std. err	Observations	R-squared
Adlwa	– 0.00529	(0.00971)	11,001	0.090
Iadla	– 0.0137*	(0.00646)	11,001	0.052
Mobility	– 0.0214	(0.0191)	11,001	0.202
Lgmuscle	0.00255	(0.0200)	11,001	0.147
Gross motor	– 0.0201	(0.0147)	11,001	0.146
Fine motor	– 0.000807	(0.0108)	11,001	0.076
Stroke	– 0.00843	(0.00774)	10,996	0.034
Cancer	0.00239	(0.00770)	10,996	0.029
# chronic diseases	– 0.0620	(0.0540)	11,001	0.135
Hypertension	0.0128	(0.0226)	10,996	0.108
Heart attack	– 0.0232	(0.0160)	10,996	0.056
Cholesterol	– 0.00753	(0.0216)	10,996	0.051
Diabetes	– 0.0235	(0.0178)	10,996	0.087
Parkinson	– 0.000627	(0.00235)	10,996	0.027
Stomach ulcer	0.000621	(0.00846)	10,996	0.031
Cataracts	– 0.00324	(0.0100)	10,996	0.039
Lung disease	– 0.00935	(0.0114)	10,996	0.034
Hip fracture	– 0.00295	(0.00461)	10,996	0.024

Each line summarizes the main results from a regression of the selected health measure on war exposure (controls as in column 6 of Table 2). Robust standard error in parentheses, clustered at individual level

$^{***}p<0.001$, $^{**}p<0.01$, $^{*}p<0.05$, $^{\dagger}p<0.1$

Table 15 The impact of war exposure on subjective survival probabilities (excluding non-war countries)

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War	3.369**	3.296**	3.298**	3.300**	3.183**	2.914**
	(1.121)	(1.122)	(1.123)	(1.122)	(1.122)	(1.094)
Female	−0.414	−0.442	−0.435	−0.504	0.845	1.448†
	(0.716)	(0.717)	(0.718)	(0.716)	(0.787)	(0.792)
Childhood SES		1.613*	1.601*	1.648*	0.768	0.244
		(0.765)	(0.768)	(0.765)	(0.790)	(0.769)
Mother at age 10			0.701	0.694	0.776	0.425
			(2.040)	(2.011)	(1.991)	(1.922)
Father at age 10			−0.070	−0.195	−0.214	−0.500
			(1.336)	(1.332)	(1.321)	(1.255)
Hunger				−3.776*	−3.338†	−2.314
				(1.747)	(1.744)	(1.675)
Dispossession				−1.931	−2.398	−2.622
				(2.529)	(2.509)	(2.365)
Adulthood	No	No	No	No	Yes	Yes
Health and lifestyle	No	No	No	No	No	Yes
Observations	8282	8282	8282	8276	8276	8276
R-squared	0.097	0.097	0.097	0.099	0.108	0.139

Robust standard errors in parentheses clustered at individual level. Adulthood include income, years of education, marital status, job status, number of children, and number of grandchildren; health and lifestyle include the number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities. All models include dummies for month-year of birth and country of birth, wave, age, target age, and age * target age. Missing values are flagged for all controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 16 The impact of war exposure on subjective survival probabilities (with region fixed effects)

Dep. var.: *SSP*	(1)	(2)	(3)	(4)	(5)	(6)
War	3.681**	3.588**	3.575**	3.507*	3.414*	3.297*
	(1.370)	(1.365)	(1.368)	(1.370)	(1.364)	(1.339)
Female	−0.597	−0.641	−0.639	−0.683	0.329	0.985
	(0.641)	(0.641)	(0.641)	(0.640)	(0.706)	(0.708)
Childhood SES		1.912**	1.965**	1.951**	1.159	0.761
		(0.712)	(0.716)	(0.715)	(0.728)	(0.703)
Mother at age 10			0.445	0.469	0.305	0.108
			(1.755)	(1.738)	(1.726)	(1.687)
Father at age 10			−0.883	−1.024	−0.964	−1.203
			(1.194)	(1.190)	(1.180)	(1.129)
Hunger				−3.556*	−3.101†	−1.991
				(1.597)	(1.594)	(1.520)
Dispossession				−2.173	−2.740	−2.741
				(2.374)	(2.369)	(2.212)
Adulthood controls	No	No	No	No	Yes	Yes
Health and lifestyle controls	No	No	No	No	No	Yes
Observations	10,674	10,674	10,674	10,668	10,668	10,668
R-squared	0.098	0.099	0.099	0.100	0.108	0.140

Robust standard errors in parentheses clustered at individual level. Adulthood include income, years of education, marital status, job status, number of children, and number of grandchildren; health and lifestyle include the number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities. All models include dummies for month-year of birth and region (NUTS-1 level), wave, age, target age, and age * target age. Missing values are flagged for all controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 17 The impact of war exposure on subjective survival probabilities (including children aged 7–12)

Dep. var.: *SSP*	(1)	(2)
At least 1 event when 0–6	1.988*	1.790*
	(0.807)	(0.790)
At least 1 event when 7–12	0.733	0.980
	(0.918)	(0.899)
Female	1.231*	1.166*
	(0.560)	(0.551)
Childhood SES	0.352	0.257
	(0.545)	(0.535)
Mother at age 10	0.564	0.127
	(1.395)	(1.347)
Father at age 10	−0.949	−1.003
	(0.867)	(0.850)
Hunger	−0.804	−0.699
	(0.961)	(0.932)
Dispossession	−4.371**	−3.490*
	(1.484)	(1.445)
Migrated		0.796
		(1.410)
Observations	18,105	20,454
R-squared	0.153	0.152

Robust standard errors in parentheses clustered at individual level. All models include adulthood (income, years of education, marital status, job status, number of children and number of grandchildren), health and lifestyle (number of chronic diseases, alcohol consumption, smoking, BMI class, and sport activities), and dummies for month-year of birth and country of birth, wave, age, target age, and age * target age. Missing values are flagged for all controls

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 18 The role of war-induced differential mortality by SES

Dep. var.: *Age of death of father*	(1)	(2)	(3)	(4)
Childhood SES	1.557***	2.015	1.573***	1.661
	(0.394)	(1.217)	(0.273)	(1.048)
War countries	−2.457**	−1.649	−4.822***	−4.181*
	(0.793)	(2.308)	(0.0782)	(1.716)
DoB	0.00101	0.00199	0.00106	0.00232
	(0.00139)	(0.00428)	(0.00132)	(0.00435)
Childhood SES * War countries		−0.643		−0.328
		(1.612)		(1.443)
Childhood SES * DoB		−0.000179		−0.000445
		(0.00266)		(0.00277)
War countries * DoB		−0.00139		−0.00195
		(0.00460)		(0.00472)
Childhood SES * War countries * DoB		0.000305		0.00115
		(0.00351)		(0.00364)
Country dummies	No	No	Yes	Yes
Observations	15,768	15,768	15,768	15,768
R-squared	0.010	0.010	0.020	0.020

Robust standard errors in parentheses clustered at country level. Only native-born respondents from SHARE waves 1, 2, 4, and 6 who are present also in wave 3 (SHARELIFE) are included

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

Table 19 The role of war-induced differential mortality by SES

Dep. var.: *Age of death of father*	(1)	(2)	(3)	(4)
Childhood SES	1.522***	2.080**	1.528***	1.698**
	(0.390)	(0.683)	(0.273)	(0.612)
War countries	−2.481***	−1.473	−4.808***	−4.151***
	(0.803)	(1.393)	(0.0801)	(0.638)
Born during WW2	0.105	1.266	0.133	1.171
	(0.408)	(1.310)	(0.366)	(1.290)
Born after WW2	0.727†	1.356*	0.755*	1.436*
	(0.413)	(0.658)	(0.394)	(0.646)
Childhood SES * War countries		−0.904		−0.422
		(0.996)		(0.889)
Childhood SES * Born during WW2		0.0491		−0.0506
		(0.809)		(0.854)
Childhood SES * Born after WW2		−0.572		−0.667
		(0.903)		(0.858)
War countries * Born during WW2		−2.216		−2.028
		(1.430)		(1.391)
War countries * Born after WW2		−0.632		−0.732
		(0.807)		(0.813)
Childhood SES * War countries * Born during WW2		1.318		1.445
		(1.352)		(1.369)
Childhood SES * War countries * Born after WW2		0.360		0.587
		(1.091)		(1.067)
Country dummies	NO	NO	YES	YES
Observations	15,768	15,768	15,768	15,768
R-squared	0.011	0.011	0.021	0.021

Robust standard errors in parentheses clustered at country level. Only native-born respondents from SHARE waves 1, 2, 4, and 6 who are present also in wave 3 (SHARELIFE) are included. Omitted category: *Born before WW2*

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, †$p < 0.1$

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Data availability Data and programs are available upon request.

Declarations

Conflict of interest The authors declare no competing interests.

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