War-torn childhood

A study on the pathways through which war affects self-rated health.

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Abstract

It is well known that early-life exposure to conflict has a negative effect on self-rated health in late life. However, the pathways through which this occurs need to be explored more in depth. Focusing on cohorts brought up during World War II the aim of this study is to shed light on the mechanisms through which early-life exposure to contexts of conflict affect health in later life. We propose two theoretical approaches.The *critical periods* perspective suggests exposure during a developmentally salient period has direct negative effects on health in late. Conversely, the *cumulative risks* perspective suggests exposure in early life will alter baseline characteristics that alter life course trajectories and pose higher risks for health in late life. The results suggest exposure had a direct negative effect on functional limitations in late life, which in turn affected self-rated health in late life. The evidence supports the *critical periods* theoretical scheme.

Introduction

Previous literature on early life exposure to contexts of war and conflict have found a strong causal link with poor health conditions in later life. This research has established experiencing contexts of conflict in early life can lead to higher probabilities of developing cardiovascular diseases, strokes, mental health problems, lower life satisfaction, diabetes, and general lower self-rated health (Kesternich et al. 2015; Lindeboom and Ewijk 2015; Islam et al. 2017; Akbulut-Yuksel 2017).

Parallel to this literature, a wide range of studies have found that early life exposure to such contexts of war and conflict have strong impacts on socioeconomic attainment and on cognitive skills through the interruption of education (Ichino and Winter-Ebmer 2004; Chamarbagwala and Morán 2011; Shemyakina 2011). Although these studies have established there is a strong causal relationship between early-life exposure to conflict, SES, and health in later life, the mechanisms through which this exposure operates need to be explored in more depth. Focusing on cohorts brought up during World War II the aim of this study is to shed light on the mechanisms through which early-life exposure to contexts of conflict affect health in later life.

Theoretical Background

The long arm of childhood: critical periods and cumulative risks.

Literature on early life exposure to crucial events and health disparities draws heavily from life course theory and emphasizes the importance of analyzing the life stages at which the biography intersects with potentially altering events (Elder 1998; Ryder 1965). Within this framework, life course and health literature has provided substantial evidence the childhood period is subject to a higher degree of vulnerability to exposure to events or contexts that may hinder development and create inequalities in later life (Ferraro 2011; Haas 2008; Haas 2006). The plethora of early life influences on adult health has been agglutinated under the concept the *long arm of childhood* (Hayward and Gorman 2004).

Critical periods

Two general theoretical frameworks have been elaborated to explain how the *long arm of childhood* exerts its influence on health throughout the life course. These are the *critical periods* perspective, and the *cumulative risks* perspective. In the *critical periods* theoretical framework, exposures to adversity during a developmentally salient period can have permanent negative effects over the life course, ultimately resulting in a wide variety of negative outcomes in late life (Kuh and Ben-Schlomo 2004). One of the key theoretical elements of the *critical periods* approach is that its effects tend to remain latent during a large part of the life course, and begin manifesting around age 50. In other words, negative exposures during critical periods have direct effects on health in late life, which is to say are largely robust to mediators (Ferraro 2011; Conley and Bennet 2000; Haas 2008).

In a study examining how early life circumstances affect functional health trajectories in health, the results showed these health trajectories are influenced by early life health and socioeconomic circumstances, net of mid-life characteristics (Haas 2008). Another study focusing on the effects of childhood health conditions on lower-body functional limitations in Mexico found poor childhood health predicted lower-body functional limitations in late adulthood -net of mediators such as SES, overall childhood health, risky health behaviors, chronic diseases, and family background (Huang et al. 2011). One of the most well-known proponents of the *critical periods* approach is Barker through the fetal origins hypothesis (Barker 1990). The theory posits that in a context of nutritional deprivation, the fetus adapts its growth to prioritize survival within the womb. This scenario results in higher propensities of developing cardiovascular diseases, cerebrovascular disease, schizophrenia, lower cognitive abilities, lower earnings, and a higher propensity to be unemployed (Schulz 2010; Scholte et al. 2015; Roseboom et al. 2011).

Cumulative risks

An alternative theoretical framework is known as the *cumulative risks* approach. This theoretical view posits that in addition to the insult during a saliently developmental period, this adversity puts the individual at an initial disadvantage that increases the risk of social and behavioral exposures that heighten the likelihood of developing illness and disease in a recursive manner (Kuh and Ben-Schlomo 2004). The alterations of baseline socioeconomic features –especially education- are a common emphasis under this perspective. Individuals with higher SES are more likely to experience good health as they are able to rely on more material resources, tend to engage in healthier behaviors, and have greater access to healthcare and of higher quality (Link and Phelan 2005; Pampel et al. 2010, Luftey and Freese 2005). Conversely, initial disadvantages -—e.g. lower educational attainment, lower cognitive development, poverty- that stem from insults during a critical period acts as a key factor that increases the chance of being exposed to more hazardous situations over the life course, in turn, increasing the probability of developing health problems (Haas 2008).

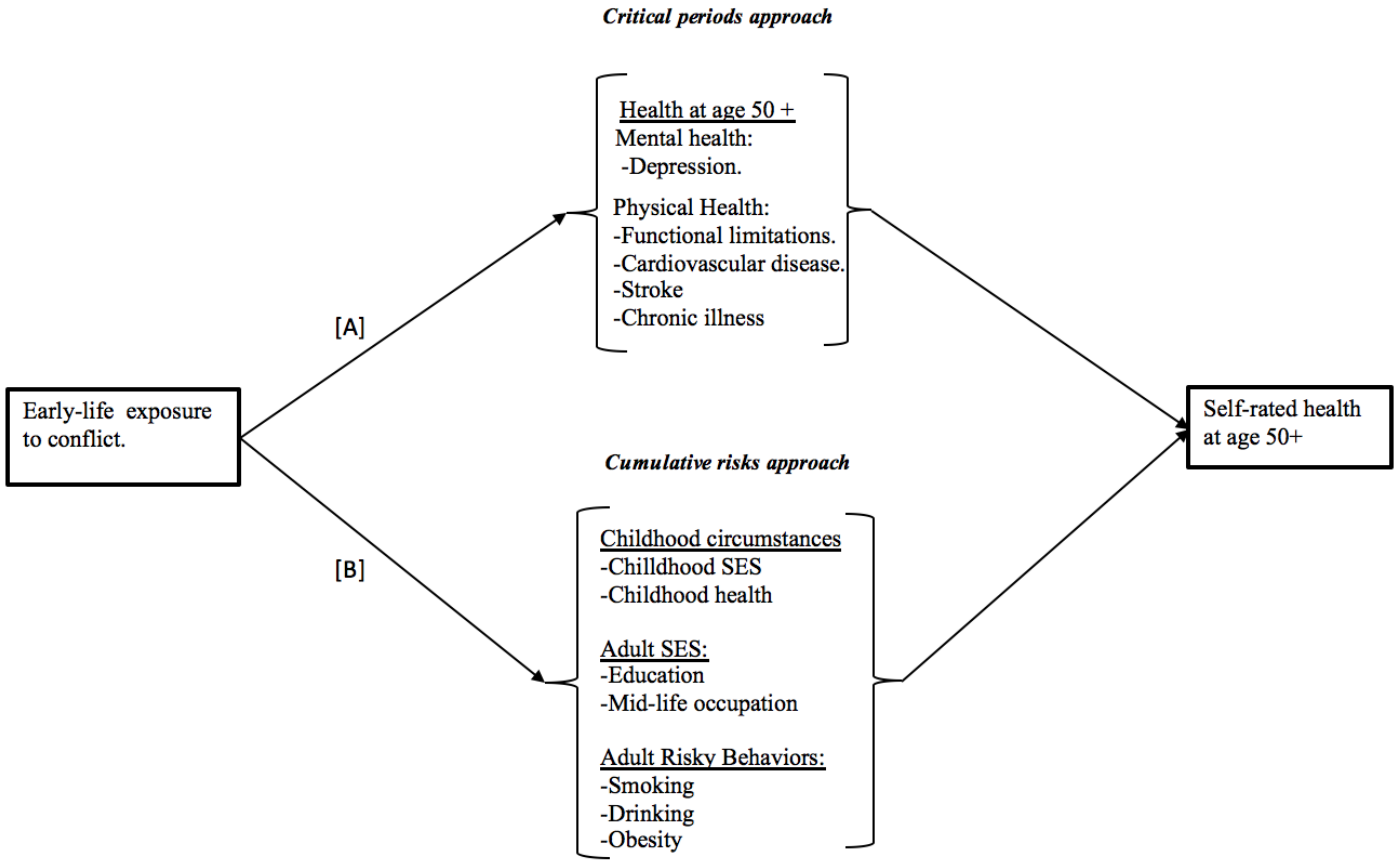
One of the most well-known proponents of this framework is McEwen and the toxic stress model. In his framework, individuals who are subject to an early life insult that may derail their life course trajectory are vulnerable to chronic and heightened episodes that may be hazardous for health throughout the life course (McEwen and McEwen 2017). In McEwen's model, education is a central component. Higher educational attainment enhances coping mechanisms for stress, depression, and anxiety (McEwen and McEwen 2017). Furthermore, education increases learned effectiveness, time-horizons and delayed gratification that lead to healthy behavioral patterns such as exercise or healthy eating (Lauderdale 2001; Lynch 2003). Conversely, those with low education tend to show lower levels of learned effectiveness, which is the human capital enhancement in the form of accumulated knowledge, skills, and resources that enable individuals engage in health-producing behaviors (Elo 2009). As a consequence, people with lower education tend to have unhealthy behaviors that are associated with short time-horizons such as smoking, drinking, or unhealthy diets. Additionally, higher education is associated with higher occupational outcomes, which is positively associated with health and cognitive abilities (Elo 2009; Pampel et al. 2010).

Hypotheses

We propose the two theoretical perspectives outlined above as potential pathways through which early-life exposure to conflict can affect health in late life. Figure 1 summarizes these two potential mechanisms. Pathway [A] depicts the *critical periods* approach which is represented through measures of health in later life – e.g. cardiovascular disease, stroke, functional limitations. If the effects of early-life regional exposure to conflict are directly related to health measures in late life with an absent mediation of pathway [B], this would lend support to the *critical periods* approach. An additional question of interest to our study is through which health conditions does early-life exposure to conflict affect self-rated health. Given these health measures are taken at the same time as the self-rated health measure, this pathway can be interpreted as a decomposition of the effects of early-life exposure to conflict on self-rated health.

Pathway [B] represents the *cumulative risks* approach. This pathway represents disadvantages in early life originated as a consequence of exposure to regional conflict. According to the theoretical framework of the *cumulative risks* approach, we expect to see baseline alterations that can affect the status attainment process and increase the likelihood of poor health -i.e. childhood SES and childhood health, - , alterations of mid-life SES – i.e. educational attainment, and mid-life occupation-, and higher propensity of engaging in risky behaviors -e.g. smoking or drinking excessively. If the effects of exposure were largely mediated through pathway [B], this would lend support to the *cumulative risks* approach, as the alterations in the SES baselines and risky behaviors would entail being exposed to more hazardous situations over the life course. In turn, this would increase the probability of developing health problems in later life (Link and Phelan 2005; Pampel et al. 2010, Luftey and Freese 2005).

Figure 1. Potential pathways for the effects of early-life exposure to conflict on self-rated health.



Nonetheless, it is common for these theoretical frameworks to be at play at the same time. Dalton Conley's work looks at low birth weight babies –a common indicator for exposure to adversity while in the womb. In his studies he has found a direct effect of low birth weight on health outcomes in late life, net of any mediators. This lends support to the critical periods approach. However, this effect is simultaneous with the finding that low birth weight accounts for up to 50% of variation in explaining intelligence, which stunts status attainment and increases the risk of a new insult to health in late life (Conley et al. 2003; Conley and Bennet 2000).

Data

The Survey of Health, Ageing and Retirement in Europe (SHARE) release 6.0 is the source of data for this study. This dataset provides detailed life history characteristics for Europeans ages 50 and above alongside a wide range of health and socioeconomic characteristics in later life. We focus on participants of wave 3 -SHARELIFE- which is particularly appropriate for this study, as it captures detailed residential and living condition histories of individuals since they were born for approximately 30,000 respondents (Börsch-Supan and Schröder 2011). We are interested in individuals who were exposed or had the possibility of being exposed to regional conflict in WWII during their childhood. This entails three important patterns that limit the sample heavily. First, we drop participants in countries that did not participate in World War II. Second, we exclude individuals that were born after 1945, as none of them were potentially exposed to conflict during WWII. Finally, we exclude individuals who were adults during WWII -i.e. respondents above 20 years old. This leaves us with a sample of 6,808 individuals. The percentage of missing data amounts on the dependent and independent models is close to 10%, which is recommended as the acceptable quantity in order to carry out listwise deletion analysis (Johnson and Young 2011; Dong and Peng 2013) .

Variables

Table 1 shows a brief description of variables. A more detailed explanation of the variables that require it are below.

Table 1. Brief description of variables.

Name	Brief Description	Range
Dependent variable		
Self-rated Health	Overall self-rated health.	1-5
Key independent variable		
Regional conflict	Number of months residing in combat regions during childhood.	0 - 11
Childhood circumstances		
Childhood SES	Ordinal variable that designates socioeconomic status during childhood -i.e. parental SES background.	1-3
Childhood health	Overall self-rated health for the respondent's childhood years.	1-5
Mid-life socioeconomic status		
Years of education	Total number of completed years of education.	0-25
Mid-life occupation	Occupational scale following the ISCO 88 scheme.	0-9
Health risks		
Never smoked daily	Dummy variable that designates whether the individual never smoked daily	0-1
Past daily smoker	Dummy variable that designates whether the individual was a past daily smoker.	0-1
Current daily smoker	Dummy variable that designates whether the individual is a current daily smoker.	0-1
Excessive drinking	Dummy variable designating whether the respondent ever consumed over 2 alcoholic drinks every day of the week.	0-1
Obese	Dummy variable that designates whether the respondent's BMI falls within the obese category.	0-1
Health conditions in late life		
Functional Limitations	Total number of functional limitations.	0 - 10
Stroke	Dummy variable designating whether the respondent ever had a stroke	0-1
Depression	Dummy variable designating whether the respondent screened positive in the EURO-D scale.	0-1
Chronic illness	Dummy variable designating whether the respondent has a chronic illness.	0-1

Cardiovascular disease	Dummy variable that designates whether the respondent has experienced a heart attack or has a cardiovascular disease.	0-1
Controls		
Year of birth 1941-1945	Dummy variable that designates the belonging to the cohort born between 1941 and 1945.	0 - 1
Year of birth 1936-1940	Dummy variable that designates the belonging to the cohort born between 1936 and 1940.	0 - 1
Year of birth 1931-1935	Dummy variable that designates the belonging to the cohort born between 1931 and 1935.	0 - 1
Year of birth 1926-1930	Dummy variable that designates the belonging to the cohort born between 1926 and 1930.	0 - 1
Male	Dummy variable to designate gender.	0 - 1

Dependent variable: Self-rated health. This ordinal measure is a self-assessed summary statement of the respondent's overall health status that ranges from 1-*poor* to 5-*excellent*. This measure is very commonly used amongst health researchers from a wide range of disciplines. It has been deemed a valid measure of overall health that successfully predicts objectively measured health conditions as well as mortality (Jylhä 2009; Ziebarth 2010). It is important to note that the measure shows a strong association with actual health conditions because these conditions inform and shape the self-assessed ratings. This is true to the point self-rated health can be informed by health conditions that have not necessarily been officially diagnosed (Idler and Yael 1997). Following previous research that compares self-rated health across countries, we dichotomize the variable so that it expresses positive health (excellent/very good = 1) (Haas 2007).

Key independent variable: Regional conflict. Regional conflict is measured as the number of months the respondent has resided in an area that hosted combat during WWII. This variable ranges from no exposure to a maximum of 11 months of exposure. The variable has been taken from previous research from the field of health economics that combines SHARE with in-depth statistical information on warfare during WWII (Kesternich et al. 2014). Similarly to measures used in the literature on natural hazards and human ecology, this measure is best understood as a contextual variable that proxies for local social impact rather than a detailed measure of conflict exposure or economic loss (Elliott 2015; Davenport, Moore, and Poe 2003; Melander and Öberg 2006; Williams 2013).

Childhood circumstances

Childhood SES. This variable is an ordinal measure that captures overall childhood SES and is divided in three categories: Low SES, Mid SES, and High SES. Similar to previous research, the measure is constructed using factor analysis to create a latent variable based on information on parental background provided by both SHARE and SHARELIFE (Kesternich et al. 2014; Mazzonna 2011). The variables used for the construction are parental education, father's occupation following the ISCO 88 scheme, number of books in the household, the ratio of people living in the household to rooms available, and the number of features in the household they lived in during childhood – e.g. central heating, a fixed bath. The Cronbach alpha score was of 0.86, indicating the measure is reliable.

Childhood health. This is an ordinal variable that retrospectively asks the respondents to self-rate their health during childhood years (between ages 0 to 15). The lowest score ranges from 1 – *poor*- to 5 -*excellent.* Previous research has analyzed the validity of retrospective measures of overall childhood health and has concluded that this measure is reliable and is strongly associated with a wide variety of common childhood conditions and activity limitations (Haas 2007; Haas et al. 2017). Additionally, there is little evidence these retrospective reports are influenced by current health reports -i.e. anchoring- (Haas 2007).

Mid-life socioeconomic status:

Years of education. Educational attainment is measured as the total completed years of full-time education. The range goes from 0 to 25 completed years of full-time education.

Mid-life occupation. Occupation at age 35 is measured following the ordinal International Standard Classification of Occupations 1988. This classification provides an ordinal classification of occupational attainment divided in

10 categories, where the higher the number the higher the prestige, skill complexity, and education required (Ganzeboom & Treiman 1996).

Health risks:
As mentioned earlier, previous research has shown individuals with lower levels of learned effectiveness and short time-horizons tend to engage in behaviors that pose risks to health in the long term (Elo 2009; Pampel et al. 2010). In order to capture these behaviors we include indicators that show current and past experiences of health hazardous behaviors.

Smoking habits.
Past daily smoker. This measure is a dummy variable that identifies those who smoked daily in the past.
Current daily smoker. This measure is a dummy variable that designates those that currently smoke daily.
Never smoked daily. This is a dummy variable that identifies those who never smoked daily.

Excessive drinking. This is a measure that stems off of SHARE's item that asks whether the respondents consume over 2 glasses of alcohol every day or 5/6 days of the week. This measure has been shown to be valid and tends to understate actual alcohol consumption on account of social desirability (Allen and Columbus 1995; Vogel-Sprott 1983).

Additionally, we introduce a dummy variable labelled *obese*, which designates whether the respondent shows levels of BMI of 30 or above.

Health conditions:
Functional limitations. This variable is a summary measure of later-life difficulties in undertaking physical activities. The measure is comprised of 10 items that capture whether the individual has difficulties walking 100 meters, sitting for two hours, getting up from a chair, pushing large objects, and climbing up a number of flights of stairs, among others. Is a variable of indicator of summary physical health. Ask STEVEN for help on this.

Stroke. This measure is a dummy variable that designates whether the individual has ever been diagnosed with a stroke or a cerebral vascular disease.

Chronic Illness. This variable gauges whether the respondent has ever been diagnosed with a chronic illness.

Cardiovascular disease. This measure designates whether the despondent has ever been diagnosed with a cardiovascular disease or has had a heart attack.

Depression. This is a dummy variable that designates whether individuals screened positively in the EURO-D depression scale that SHARE provides. This scale is a broadly used indicator of depression that has been validated and shown to be robust to cross-country contexts (Guerra et al. 2015).

Controls:
Cohort variables. To test for potential cohort effects, we include a set of dummy variables that identify 5-year age groups. This set of dummy variables acts as an indicator of the stage of childhood the respondents lived through WWII. Yet, it is important to notice these variables have a dual interpretation. They also identify the age at which the respondent took the survey. Given health is linearly and negatively correlated with age, if we were to identify a linear pattern in the cohorts, it is likely the coefficients of these variables are identifying an age effect and not a cohort effect.

Analytical approach

The analysis is carried in two stages. The first stage consists in establishing an association between early-life exposure with the potential mediators outlined above. In order to gauge this association, we estimate a set of regressions with the following specification:

$$Y_{mi} = \alpha + \beta_1(\text{Regional conflict})_i + \beta_2(\text{Controls}) + \varepsilon_i$$

Where Y_{mi} is the outcome of interest *m* for the individual *i*, α is the constant, β_{1_i} is the key independent variable *regional conflict*, β_{2_i} represents a series of controls -i.e. cohorts, and gender, and ε_i is the error term. The first set of mediators we test as outcomes are those relative to pathway [B] in figure 1 -i.e. childhood circumstances, adult SES, and adult risky behaviors. The second set of mediators we test as outcomes are those relative to pathway [A] in figure 1- i.e. health conditions at age 50+. Significant associations with any of these outcomes would provide partial evidence as potential mediators between early life exposure to conflict and self-rated health in late life.
The second stage of the analysis focuses on mediation analysis. In order to clarify the mechanisms through which early-life exposure to conflict affects health in late life, we set a series of step-wise ordered logistic regressions with self-rated health as the outcome of interest. We estimate a series of models that include all variables of interest independently to see whether the effects of regional conflict on self-rated health may be mediated through pathways [A] or [B]. The series of ordered logistic regressions follow the specification below:

$$Y_i = \alpha + \beta_1(\text{Regional conflict})_i + \beta_2(\text{Controls}) + \beta_3(\text{Childhood circumstances})_i + 4(\text{Mid} - \text{life SES})_i + \beta_5(\text{Health risks}) + \beta_6(\text{Health conditions}) + \varepsilon_i$$

The baseline model is comprised of Y_{mi} as the outcome of interest *self-rated health*, β_{1_i} as the key independent variable *regional conflict* , and β_{2_i} which represents a series of controls -i.e. cohorts, and gender. In subsequent models we independently introduce the mediators of interest to gauge whether they mediate the effect of our key independent variable on *self-rated health.* Pathway [B] is represented by β_3, β_4 and β_5, which are a set of indicators for childhood circumstances, mid-life socioeconomic status and adult health risks for health.

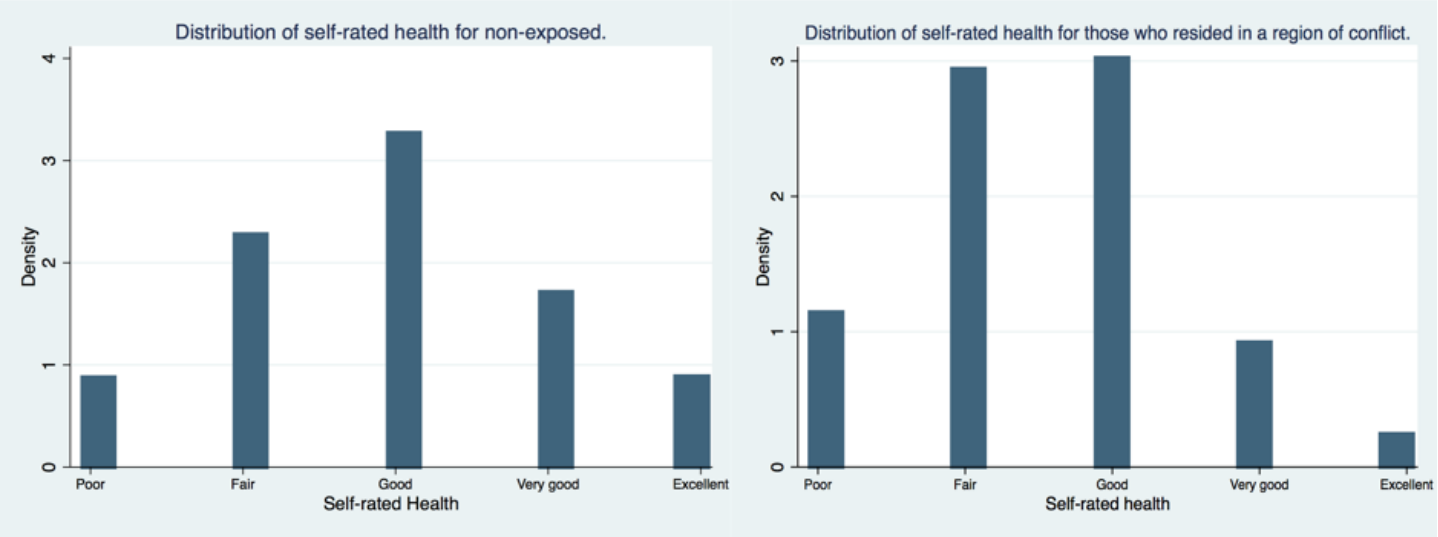
While this approach may provide insight when it comes to the statistical significance of the mediating pathways, it relays inaccurate results in terms of how much mediation occurs. This renders it impossible to gauge what pathway is of most importance. In linear models, the effect of a predictor *x* on an outcome *y*, mediated through a variable *z*, can be easily gauged using stepwise approaches and comparing coefficient sizes across models. The issue with discrete outcomes and probabilistic models is that mediation cannot be conducted in the same way as one would test mediation in an OLS model. Hence, if a coefficient size drops in half from the reduced model to the model which includes mediators in an ordered logistic regression, one cannot say 50% of the effect is accounted by the mediator. This is because, in nonlinear binary probability models -which can be extended to ordered probability models as well-, the regression returns coefficient estimates equal to the ratio of the true regression coefficient divided by a scale parameter, which is a function of the error standard deviation (Breen, Karlson, and Holm 2013). Because the error variance may differ across models, the initial effect of *x* does not decompose into direct and indirect effects in the same way a linear model would.

There is a special procedure called KHB that accounts the differentials in error variance across models in a discrete outcome setting (Breen, Karlson, and Holm 2013). This method allows to appropriately gauge the proportion mediated of a specific pathway in discreet outcome models. In other words, with the KHB method we are able to see which of the pathways shows stronger mediation of the effect of early life exposure to regional conflict on self-rated health.

Results

Figure 2 shows the distribution of the self-rated health categories comparing those who were exposed to combat during childhood to those who were not exposed. As can be seen, those who were exposed (right), show a higher preponderance of individuals in the low categories of self-rated health. In comparison, those who were not exposed show a distribution that resembles a normal distribution and is much less skewed towards negative values of self-rated health. This is consistent with the information displayed in the descriptive statistics in table 2. The binary version of self-rated health shows approximately 30% of respondents who were not exposed to combat in early-life claim to have a positive self-rated health. Conversely, only approximately half of the respondents in the exposed group show positive ratings of health -i.e. 16%.

Figure 2. Ordinal self-rated health measure for those exposed to regional conflict and those non-exposed.



In looking at the differences in the mediators in exposed and non-exposed groups there are variables that show substantial differences. There are important differences in educational attainment, where the non-exposed group shows a mean of 9.72 completed years of education, whereas the exposed group shows a lower mean with 9.1 years of education. We find a similar pattern with mid-life occupation, where the non-exposed shows a higher average in terms of the occupational scale. In terms of risky behaviors, the exposed group shows higher prevalence in recent daily smoking and excessive drinking behaviors. Similarly, the prevalence of obesity is slightly higher amongst those who have been exposed to regional conflict. Overall, it seems those exposed to combat show lower levels of socioeconomic status, as well as higher prevalence in smoking, drinking, and obesity.

In looking at health conditions at age 50 and above, we find there are important differences as well. Those exposed show an average of 1.53 functional limitations, whereas those not exposed show an average of 1.06 functional limitations. Similar patterns can be found in whether respondents were diagnosed with a stroke, a heart disease, and whether they screened positive in depression. The prevalence of chronic illnesses are almost identical for both groups. Hence, the descriptive statistics show both the critical periods approach as well as the cumulative risks approach are plausible.

Table 2. Descriptive statistics

Variable	Full sample (N = 6808)					Exposed (N = 4212)					Not exposed (N = 2596)				
	%	Mean	SD	Min	Max	%	Mean	SD	Min	Max	%	Mean	SD	Min	Max
Self-rated health (1=positive)	0.21			0	1	0.16			0	1	0.29			0	1
Regional conflict		2.27	2.08	0	11		-	-	-	-		-	-	-	-
Male	45.91			0	1	45.04			0	1	46.01			0	1

Year of birth 1941-1945	33.9			0	1	34.15			0	1	53.7			0	1
Year of birth 1936-1940	28.08			0	1	24.86			0	1	22.95			0	1
Year of birth 1931-1935	21.29			0	1	24.70			0	1	14.2			0	1
Year of birth 1926-1930	16.25			0	1	17.30			0	1	10.15			0	1
Year of birth 1921-1925	8.41			0	1	9.40			0	1	6.67			0	1
Childhood SES (Low)	51.2			0	1	50.44			0	1	47.81			0	1
Childhood SES (Mid)	37.21			0	1	38.44			0	1	36.94			0	1
Childhood SES (High)	11.6			0	1	11.12			0	1	15.25			0	1
Childhood health		3.93	1.00	1	5		3.90	1.00	1	5		4.07	0.99	1	5
Years of education		9.62	4.10	0	25		9.10	4.06	0	25		9.72	4.25	0	25
Mid-life occupation		4.03	0.04	0	9		3.72	0.05	0	9		4.55	0.06	0	9
Never smoked daily	56.24			0	1	50.83			0	1	59.27			0	1
Past daily smoker	29.36			0	1	27.81			0	1	32.12			0	1
Recent daily smoker	14.4			0	1	17.05			0	1	12.92			0	1
Excessive drinking	14.6			0	1	16.3			0	1	11.7			0	1
Obese	16.7			0	1	17.4			0	1	15.5			0	1
Functional Limitations		1.29	1.85	0	10		1.53	1.97	0	10		1.06	1.67	0	10
Stroke	4.0			0	1	4.4			0	1	3.0			0	1
Chronic illness	61.71			0	1	61.64			0	1	61.66			0	1
Cardiovascular disease	13.4			0	1	15			0	1	12.1			0	1
Depression	22.3			0	1	23.7			0	1	19.7			0	1

Table 3 shows regression estimates for indicators of childhood circumstances, mid-life SES, and adult health risks. The effects of exposure to combat in early life -as given by the *regional conflict* coefficient- are net of gender and birth cohorts. The first set of outcomes in table 3 are indicators of baseline circumstances -i.e. childhood SES and childhood health. The first model provides ordered logistic regression estimates for childhood SES. Although combat exposure has an expected negative effect on the likelihood of being in a high childhood SES group, the coefficient is not statistically significant. The lack of results regarding SES are probably due to the way this variable is measured. This variable is information that is based on the status attainment and wealth of their parents. The next model shows ordered logistic regression estimates for childhood health. The effects of early life exposure to conflict on childhood health are negative. Every month of exposure to combat decreases the likelihood of having a positive childhood self-rated health by 2.5%. However, the results are marginally significant at a level of $p<0.10$. Overall, it seems exposure to combat in early life did not alter childhood socioeconomic status and had a relatively small effect on childhood health.

The second set of outcomes are indicators of adult socioeconomic status. The firs model provides OLS estimates for years of education. Every month of exposure to combat decreases 0.078 years of completed education, holding all else constant. Hence, those exposed to 10 months of combat show approximately one year less in the educational attainment measure. Furthermore, this estimate is statistically significant at a $p<0.01$ level. We find similar patterns for the ordered logistic estimates of mid-life occupation, provided in the following column. Every month of exposure to conflict decreased the odds of being in a higher occupational category by 5.7%. Coupled with the findings in the previous column regarding educational attainment, it seems exposure to conflict did affect the status attainment process. This indicates alterations in status attainment is a potential mediator for the effect of early-life exposure on self-rated health in later life.

Table 3 . Regression estimates for childhood circumstances, mid-life SES, and health risks.

	Childhood circumstances		Mid-life SES		Health risks			
	Childhood SES (Ordered logistic)[1]	Childhood health (Ordered logistic)[1]	Years of education (OLS)	Mid-life occupation (Ordered logistic)[1]	Obese (Logistic)[1]	Past daily smoker (Logistic)[1]	Recent daily smoker (Logistic)[1]	Excessive drinking (Logistic)[1]
Regional conflict	0.996	0.975+	-0.078**	0.943**	1.018	1.038*	1	1.019
	(0.09)	(0.01)	(0.03)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Year of birth 1936-1940[2]	0.692***	0.989	-0.306*	0.943**	0.820*	1.171*	0.624***	0.831+
	(0.05)	(0.06)	(0.12)	(0.07)	(0.08)	(0.09)	(0.06)	(0.08)
Year of birth 1931-1935[2]	0.501***	1.02	-0.899***	0.943**	0.935	1.049	0.478***	0.804*

	(0.04)	(0.07)	(0.13)	(0.05)	(0.09)	(0.09)	(0.05)	(0.09)
Year of birth 1926-1930[2]	0.571***	0.926	-1.015***	0.943**	0.768*	1.446***	0.354***	0.772*
	(0.05)	(0.07)	(0.15)	(0.06)	(0.09)	(0.14)	(0.05)	(0.10)
Year of birth 1921-1925[2]	0.478***	0.975	-1.325***	0.943**	0.376***	1.175	0.244***	0.570***
	(0.05)	(0.09)	(0.18)	(0.06)	(0.06)	(0.14)	(0.05)	(0.09)
Male	0.914+	1.211***	1.013***	0.943**	0.816**	4.192***	1.717***	4.321***
	(0.05)	(0.06)	(0.09)	(0.21)	(0.06)	(0.27)	(0.13)	(0.39)

Notes: N(6,808) Standard Errors in Parenthesis; 1. Odds Ratios; 2.Reference group: Year of birth 1941-1945; all models include country fixed effects † p<0.1, * p<0.05, ** p<0.01, *** p<0.001

The next set of outcomes are indicators of risky behaviors towards health and the condition of obesity. As can be seen in the results, the effects of regional conflict on the likelihood of being obese in late life are positive, however; they are not statistically significant at a $p < 0.05$ level. The same can be said for the effects of exposure to conflict on the likelihood of being a recent daily smoker and on the likelihood of excessive drinking. However, we do find statistically significant effects of *regional conflict* on the likelihood of being a past daily smoker. Every month of exposure to combat in early-life increases the likelihood of being a past daily smoker by 3.8%, holding all else constant. Overall, the results show the cumulative risk pathway is a viable explanation through the alteration of the status attainment process. Yet, there is little evidence exposure to conflict in early life increased the likelihood of engaging in risky behaviors towards health and being obese.

Now we focus on the health conditions as potential mediators. Table 4 shows regression estimates for health conditions at age 50 and above. The first of the columns shows estimates for functional limitations in late life. We find every month of exposure to regional conflict increases the incidence rate of functional limitations by a factor of 1.029, and it is statistically significant at a $p<0.05$ level. Additionally, every month of exposure to combat increases the likelihood of screening positively in the depression EURO-D scale by 5.7%. We find a similar pattern for cardiovascular diseases. Every month of exposure to conflict increases the likelihood of developing a cardiovascular disease or having a heart attack by 5.2%. In other words, those exposed to 10 months of combat are 52% more likely to develop a cardiovascular disease. However, we find no significant effects on having a chronic illness or experiencing a stroke. Overall, the results indicate functional limitations, depression, and cardiovascular disease are potential mediators through which early life exposure to regional conflict can affect health ratings in late life.

Table 4. Regression estimates for health conditions at age 50+.

	Functional limitations (Negative Binomial)[2]	Depression (Logistic)[1]	Chronic illness (Logistic)[1]	Stroke (Logistic)[1]	Cardiovascular disease (Logistic)[1]
Regional exposure	1.029*	1.057**	1.002	1.059	1.052*
	(0.01)	(0.02)	(0.02)	(0.04)	(0.02)
Year of birth 1936-1940[3]	1.144*	1.048	1.141+	1.406	1.439**
	(0.06)	(0.08)	(0.08)	(0.30)	(0.16)
Year of birth 1931-1935[3]	1.351***	0.968	1.507***	2.027**	2.154***
	(0.08)	(0.08)	(0.13)	(0.44)	(0.25)
Year of birth 1926-1930[3]	1.934***	1.342**	1.564***	2.459***	2.732***
	(0.12)	(0.13)	(0.15)	(0.55)	(0.33)
Year of birth 1921-1925[3]	2.176***	1.435**	1.589***	2.882***	3.224***
	(0.16)	(0.16)	(0.18)	(0.71)	(0.44)
Male	0.529***	0.399***	0.704***	1.15	1.910***
	(0.02)	(0.02)	(0.04)	(0.16)	(0.15)

Notes: N(6,808) Standard Errors in Parenthesis; 1. Odds Ratios; 2. Incidence Rate Ratios; 3.Reference group: Year of birth 1941-1945; all models include country fixed effects †p<0.1, * p<0.05, ** p<0.01, *** p<0.001

Table 5 shows logistic regression estimates for self-rated health, where the value of one indicates whether respondents rate their health positively. The first column shows a reduced model with controls for birth cohorts and gender for self-rated health. As can be seen, every month of exposure to conflict decreases the likelihood of having a positive self-rated health by 6.1%. This means that those who were exposed 10 months are 61% less likely to have a positive self-rated health. This result falls in line with previous research (Kesternich et al. 2014). This baseline model is important for comparison as we introduce mediators in subsequent models.

Models 2 to 5 introduce mediators that provide support for the cumulative risks perspective. Model 2 introduces the variables that indicate childhood circumstances. The coefficients for all three variables are statistically significant and show substantially high odds ratios. However, the interest lies in how much the coefficient for *regional conflict* drops and how much the Akaike information criterion and the Bayesian information criterion change when we introduce these variables in the model. As can be seen in model 2, the odds ratios for *regional conflict* barely differ from those in model 1. Yet, the AIC and BIC do experience a decrease, which indicates this model is of better fit than model 1. The same can be said for model 3, which adds adult socioeconomic status as mediators. Model 4 introduces indicators for health risks. None of them are statistically significant, the coefficients of *regional conflict*, the AIC and the BIC are almost identical to model 1. Similar patterns are found in model 5 with obesity as a mediator. Overall, the evidence in models 2 to 5 does not support the cumulative risks perspective, as the mediation that occurs seems to be of little importance. However, this needs to be formally tested using the KHB method in table 6.

Table 5. Logistic regression estimates for self-rated health (1= Positive Self-rated health, Odds ratios).

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Regional conflict	0.939**	0.936**	0.938**	0.939**	0.941**	0.950*	0.947*	0.954*
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Year of birth 1936-1940[1]	0.749***	0.784**	0.787**	0.741***	0.734***	0.791**	0.752***	0.811*
	(0.06)	(0.07)	(0.07)	(0.06)	(0.06)	(0.07)	(0.06)	(0.08)
Year of birth 1931-1935[1]	0.542***	0.584***	0.620***	0.535***	0.537***	0.658***	0.536***	0.718**
	(0.05)	(0.06)	(0.06)	(0.05)	(0.05)	(0.07)	(0.05)	(0.08)
Year of birth 1926-1930[1]	0.455***	0.488***	0.517***	0.445***	0.442***	0.631***	0.471***	0.663**
	(0.05)	(0.06)	(0.06)	(0.05)	(0.05)	(0.08)	(0.06)	(0.09)
Year of birth 1921-1925[1]	0.375***	0.408***	0.436***	0.368***	0.348***	0.538***	0.393***	0.575***
	(0.06)	(0.06)	(0.07)	(0.06)	(0.05)	(0.09)	(0.06)	(0.10)
Male	1.359***	1.325***	1.210**	1.334***	1.346***	1.101	1.186*	0.953
	(0.09)	(0.09)	(0.09)	(0.10)	(0.09)	(0.08)	(0.08)	(0.08)
Childhood SES (Mid)[2]		1.259**						1.095
		(0.10)						(0.10)
Childhood SES (High)[2]		1.969***						1.526***
		(0.19)						(0.18)
Childhood health		1.691***						1.669***
		(0.07)						(0.07)
Years of education			1.038***					0.474**
			(0.01)					(0.13)
Mid-life occupation			1.086***					0.383***
			(0.02)					(0.03)
Past daily smoker				1.096				1.201*
				(0.09)				(0.10)
Recent daily smoker				0.882				0.855
				(0.09)				(0.09)
Excessive drinking				1.019				0.888
				(0.10)				(0.09)
Obese					0.514***			0.658***
					(0.05)			(0.07)
Functional Limitations						0.667***		1.019+
						(0.02)		(0.01)
Stroke						0.454**		1.066***
						(0.13)		(0.02)
Chronic illness						0.386***		0.736***
						(0.03)		(0.07)
Cardiovascular disease						0.520***		0.525***
						(0.07)		(0.07)
Depression							0.476***	0.721***
							(0.04)	(0.03)
AIC	5566.29	5330.94	5418.12	5567.64	5522.08	5037.66	5481.56	4730.59
BIC	5672.96	5457.61	5537.91	5694.31	5635.42	5171.00	5594.90	4930.21

*Notes: N(6,808); Standard Errors in Parenthesis; 1. Reference group: Year of birth 1941-1945; 2. Child's SES (Low); all models include country fixed effects †p<0.1, * p<0.05, ** p<0.01, *** p<0.001*

Models 6 and 7 introduce health conditions in late life as mediators of the effect of exposure to combat in early life on self-rated health in late life. Model 6 introduces indicators of physical health conditions, whereas model 7 introduces depression as an indicator for mental health.
The effects of physical health conditions as mediators seem to be substantial, as the coefficient for *regional conflict* drops from 0.939 to 0.950 (note that the closer the value is to 1, the smaller the effects, since these are odds ratios). Furthermore, the AIC and the BIC are the lowest for all models -with the exception of model 8, which includes all covariates. In other words, health conditions as mediators are those that fit the data best. This finding, coupled with the lack of evidence for mediation through childhood circumstances, adult SES, and health risks, suggest support for the critical periods perspective. However, this cannot be formally tested with these estimates. It is necessary to employ the KHB method in order to gauge the correct percentage of mediation that occurs through these variables.

Table 6 shows the KHB decomposition of the effects of regional conflict on self-rated health. This table is comprised of two sections. The first section shows a comparison of coefficients between the reduced model without any mediators -which would be equivalent to model 1 in table 5- and the full models. Additionally, it provides an estimate of the difference in coefficients and tests whether these differences are statistically significant. The second section of the table shows the KHB calculations of the percentage mediated attributed to each mediator. Similarly to table 5, we first introduce the indicators that show alterations of childhood circumstances, adult SES, and health risks as potential mediators. If the KHB were to reveal substantial mediation through these pathways, this would lend support to the cumulative risks perspectives.

Table 6. KHB decomposition of regional conflict effects on self-rated health (1 = Positive Self-rated Health, Odds ratios).

Models	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Reduced (without mediator)	0.939**	0.939**	0.939**	0.939**	0.939**	0.939**	0.939**
Full (with mediators)	0.936**	0.938**	0.939**	0.941**	0.950*	0.947*	0.954*
Difference	0.997	0.998*	1	0.998	0.989***	0.992***	0.943***
Paths of Coefficient reduction (%)	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**
Childhood SES (Mid)	-2.87						-0.79
Childhood SES (High)	-6.66						-2.24
Childhood health	9.34						6.29
Years of education		1.93					1.34
Mid-life occupation		1.09					-4.27
Past daily smoker			-1.48				-1.43
Recent daily smoker			0.08				-0.22
Excessive drinking			-0.02				0.24
Obese				0.18			0.1
Functional Limitations					26.52		19.61
Chronic illness					2.01		1.73
Stroke					2.32		1.93
Cardiovascular disease					4.81		4.83
Depression						13.96	3.73

Notes: N(6,808) Standard Errors in Parenthesis; the KHB decomposition is specified in the same way as table 5. 1. All models include country fixed effects † p<0.1, * p<0.05, ** p<0.01, *** p<0.001

As can be seen in model 1 of table 6, the differences in the coefficients of *regional conflict* in the reduced model and the full model are not statistically significant. This indicates that the mediation of early life exposure to conflict on self-rated health in late life through childhood circumstances is not supported by the evidence. In model 2 we test the mediation through mid-life SES. The difference is statistically significant, however; the coefficient reduction is extremely low at a 1.93 % for education and 1.09% for mid-life occupation. Furthermore, in model 3 and 4 we introduce health risks as potential mediators. The KHB calculations show the differences in the coefficient of *regional conflict* between the reduced model and the full model are not statistically significant. In sum, the evidence does not support the cumulative risks hypothesis.

Models 5 and 6 introduce health conditions in late life as mediators of the effects of *regional conflict* on self-rated health. In both models we see statistically significant differences between the *regional conflict* coefficient in the reduced model and the full model. Furthermore, when we look at the KHB calculations of how much the coefficient drops we find that functional limitations has a strong mediating effect. When functional limitations is introduced in the model, 26.52% of the *regional conflict* coefficient is reduced. Cardiovascular disease mediates 4.81% of the effect, which is a mild mediation compared to functional limitations. Stroke and chronic illnesses mediate very small portions of the coefficient. Additionally, we find a very strong mediating effect in model 6 through depression, as the coefficient for *regional conflict* decreases by 13.96% when depression is included as a mediator in the model.

However, it is crucial to test these mediation effects by including them in the model at the same time, as they are correlated and the exact percentage drop in the coefficient of *regional conflict* may be unprecise. It is important to note that this model is not affected by multicollinearity (we implement variance inflation and tolerance tests for multicollinearity using a linear probability model in the appendix). Model 7 introduces all the potential mediators in the KHB calculations. As can be seen in the second portion of the table, functional limitations is the strongest mediator with a reduction of the *regional conflict* coefficient of 19.61%. All other mediators reduce the coefficient in much smaller dimensions. Additionally, the percentage reduced through depression is of only 3.73% in this model. This drop in percentage mediated is likely due to the correlation between functional limitations and depression. Overall, the results show support for the critical periods perspective.

Conclusions

It is well known that early-life exposure to conflict has a negative effect on self-rated health in late life (Kesternich et al. 2015; Lindeboom and Ewijk 2015; Islam et al. 2017; Akbulut-Yuksel 2017). Yet, the pathways through which this occurs need to be explored more in depth. Focusing on cohorts brought up during World War II the aim of this study is to shed light on the mechanisms through which early-life exposure to contexts of conflict affect health in later life. We propose two broad theoretical approaches to explain how this phenomenon unfolds: the *critical periods* approach and the *cumulative risk* approach. The *critical periods* perspective suggests exposure during a developmentally salient period has direct negative effects on health in late life and tend to remain latent during a large part of the life course -symptoms begin manifesting around age 50. Conversely, the *cumulative risks* perspective suggests exposure during a developmentally salient period will alter baseline characteristics that create life course trajectories that pose higher risks for health in late life.

The results suggest exposure to conflict affected self-rated health in late life following the *critical periods* theoretical scheme. First of all, the lack of evidence supporting mediators related to childhood circumstances, adult SES, and health risks suggests the cumulative risks pattern is not the most common pathway for those exposed to conflict in early life. This finding, coupled with the findings related to mediation through health conditions in late life, support the critical periods perspective. Overall, being exposed to conflict in early life affected self-rated health through negative health conditions in late life. Furthermore, it seems these negative effects tend to remain latent until late life.

STEVEN HAAS ASK:
The importance of FUNCTIONAL LIMITATIONS

That being said, there are important limitations to this study. First of all, the dataset does not provide an item that relates the respondents to direct exposure to a conflict. As a proxy, we use Kesternich's measure of exposure to combat (Kesternich et al. 2014). This measure is based on the respondents' region of residence which is then cross-referenced with military data. Although this is a widely accepted measure amongst other social scientists who study armed conflict, it is not a direct measure of exposure. Furthermore, the region of residence is measured following the NUTS2 geographic unit, which shows substantial variation in terms of size across Europe.
Another important limitation to this study is the selection effects. SHARELIFE only captures respondents who were aged 50+ in Europe and alive in 2009. It is possible those individuals who fled Europe during the war are not in the sample. But more importantly, it is likely those individuals who were most exposed to combat and hardship during the war did not survive until 2009. Hence, it is possible those individuals that comprise the SHARELIFE sample today are those who were less exposed to conflict. Yet, if such selection were true, we believe the effects highlighted in this study would be understated and the estimates are biased downward.

Future research should be….

Robustness checks: differential cohort effects. Interaction .

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Appendix

Multicollinearity check

In order to address potential concerns on multicollinearity between the variables we have used in our analysis, we estimate a linear probability model using the same specification as model 8 in table 5. Subsequently, we implement variance inflation and tolerance tests for multicollinearity. As can be seen in table 1A of the appendix, none of the variables are beyond the accepted thresholds. Multicollinearity is an issue when the variance inflation factor approximates 10, and when the tolerance factor is near 0.1 or 0.2 (Long & Freese 2005). It is important to note that we are not interested in the estimates of the linear probability model itself, we are only interested in the subsequent multicollinearity checks, which is why the estimates of the LPM are not shown.

Table 1 A. Variance inflation factor and tolerance for linear probability model estimates on self-rated health

Variable	VIF	1/VIF
Regional conflict	1.7	0.588835
Year of birth 1936-1940	1.42	0.703549
Year of birth 1931-1935	1.42	0.705189

Year of birth 1926-1930	1.36	0.734093
Year of birth 1921-1925	1.28	0.779932
Male	1.36	0.737846
Childhood SES (Mid)	1.35	0.739155
Childhood SES (High)	1.61	0.621237
Childhood health	1.1	0.911676
Years of education	1.8	0.555022
Mid-life occupation	1.58	0.631227
Functional Limitations	1.38	0.723972
Stroke	1.04	0.962371
Chronic illness	1.2	0.8321
Cardiovascular disease	1.09	0.91413
Depression	1.18	0.84477
Past daily smoker	1.31	0.765335
Recent daily smoker	1.2	0.831994
Excessive drinking	1.14	0.878407
Obese	1.06	0.942681
Germany	2.49	0.401459
Sweden	3.15	0.317022
Netherlands	2.49	0.401854
Italy	3.11	0.321479
France	2.71	0.369129
Denmark	2.38	0.421052
Greece	3.34	0.29901
Switzerland	1.86	0.536947
Belgium	3.69	0.271295
Mean VIF	1.79	

Differential effects by cohort

Another potential scenario is that there are differential effects of war by cohort. In others, experiencing combat at different ages may render differential effects. According to the outlined theory in the main text, we expect those individuals that experienced a war context in earlier stages of childhood to be most vulnerable (Ferraro 2011; Haas 2008; Haas 2006). However, there is another body of literature that suggests it is individuals at the verge of transitioning to adulthood that are most vulnerable to life altering events (Elder (Elder 1974, 1998; Ryder 1965). In Elder's seminal work *Children of the Great Depression*, those individuals who were at the verge of transitioning to adulthood while entering the labor market during the depression of 1929 were the most vulnerable to alter their career expectations and life plans. These cohorts abandoned the delaying of gratification and long-term plans in favor of shorter more immediate plans. Previous research has shown that individuals with low discount factors and shorter time horizons show lower overall health in late life (McEwen and McEwen 2017; Seeman et al. 2004).

Table 2A shows logistic regression estimates for self-rated health. Model 1 shows the likelihood of different birth cohorts on having a positive self-rated health. The odds ratios decrease in a linear pattern following the older birth cohorts. In other words, it seems the older the birth cohort is, the less likely they are to signal they have a positive health. Another key to this table is that it introduces a series of interactions between birth cohorts and regional

conflict exposure. As can be seen in model 2, none of the interactions are statistically significant, which indicates there is no evidence supporting differential effects by birth cohort.

Table 5. Logistic regression estimates for self-rated health (1= Positive Self-rated health, Odds ratios).

	(1)	(2)
Regional conflict	0.939**	0.918*
	(0.02)	(0.03)
Year of birth 1936-1940	0.749***	0.711**
	(0.06)	(0.07)
Year of birth 1931-1935	0.542***	0.504***
	(0.05)	(0.06)
Year of birth 1926-1930	0.455***	0.440***
	(0.05)	(0.07)
Year of birth 1921-1925	0.375***	0.412***
	(0.06)	(0.08)
Male	1.359***	1.358***
	(0.09)	(0.09)
Year of birth 1936-1940 X Regional conflict		1.039
		(0.04)
Year of birth 1931-1935 X Regional conflict		1.052
		(0.05)
Year of birth 1926-1930 X Regional conflict		1.028
		(0.06)
Year of birth 1921-1925 X Regional conflict		0.947
		(0.08)
AIC	5566.29	5571.74
BIC	5672.96	5705.08

*Notes: N(6,808); Standard Errors in Parenthesis; 1. Reference group: Year of birth 1941-1945; 2. Child's SES (Low); 2. Reference group: Never smoked daily; all models include country fixed effects †p<0.1, * p<0.05, ** p<0.01, *** p<0.001*