Intergenerational transmission of paternal trauma among US Civil War ex-POWs

Dora L. Costa[a,b,1], Noelle Yetter[b], and Heather DeSomer[b]

[a]Department of Economics, University of California, Los Angeles, CA 90095; and [b]Program on the Economics of Aging, National Bureau of Economic Research, Cambridge, MA 02138

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We study whether paternal trauma is transmitted to the children of survivors of Confederate prisoner of war (POW) camps during the US Civil War (1861–1865) to affect their longevity at older ages, the mechanisms behind this transmission, and the reversibility of this transmission. We examine children born after the war who survived to age 45, comparing children whose fathers were non-POW veterans and ex-POWs imprisoned in very different camp conditions. We also compare children born before and after the war within the same family by paternal ex-POW status. The sons of ex-POWs imprisoned when camp conditions were at their worst were 1.11 times more likely to die than the sons of non-POWs and 1.09 times more likely to die than the sons of ex-POWs when camp conditions were better. Paternal ex-POW status had no impact on daughters. Among sons born in the fourth quarter, when maternal in utero nutrition was adequate, there was no impact of paternal ex-POW status. In contrast, among sons born in the second quarter, when maternal nutrition was inadequate, the sons of ex-POWs who experienced severe hardship were 1.2 times more likely to die than the sons of non-POWs and ex-POWs who fared better in captivity. Socioeconomic effects, family structure, father-specific survival traits, and maternal effects, including quality of paternal marriages, cannot explain our findings. While we cannot rule out fully psychological or cultural effects, our findings are most consistent with an epigenetic explanation.

intergenerational | mortality | epigenetics | reversibility | POW

There is growing concern that health can be transmitted across generations, leading to the persistence of poor health and socioeconomic status within families. Maternal exposure to famines, infection, and psychological stress during pregnancy has been linked to poor health of children at birth and in adulthood (1–10), but studies of the intergenerational transmission of paternal health in human populations are few.

We examine whether children born after the US Civil War (1861–1865) to survivors of Confederate prisoner of war (POW) camps were shorter lived because of their fathers' experiences and the mechanisms behind and reversibility of any paternal transmission. Our setting provides a well-defined, severe limited duration paternal shock with clear control groups. Conditions in Confederate POW camps deteriorated sharply when prisoner exchanges stopped, producing starvation, disease, and psychological stress. Thirty-five years after the end of the war, camp survivors faced greater mortality and health risks and had worse socioeconomic outcomes, if they had been imprisoned when camp conditions were at their worst compared with non-POW veterans and ex-POWs imprisoned when conditions were better (11). We use children of non-POW fathers and of former POWs imprisoned when camp conditions were better as controls for children of ex-POW fathers imprisoned when camp conditions were at their worst. We use children born before the war as controls within families for children born after the war.

Mechanisms for the transmission of POW trauma can be biological, cultural or psychological, or socioeconomic. Parents of lower socioeconomic status are less able than higher status parents to protect their children from health shocks (12). Poor health may lead to worse marriage matches (13). Cultural and psychological channels include attitudes and behaviors imparted to children and stresses during childhood from violence, family strife, emotional distance, or anxiety (14–16). Biological pathways include mutations to the DNA of sperm, viruses or prions in seminal fluid, or epigenetic imprinting, which leads to sex-specific effects (17–21). Although in utero and prepuberty are critical times for epigenetic changes, alterations have been observed in the sperm of adult men as a result of age, diet, smoking, alcohol, and exposure to toxins (22). In mice, paternal effects may be transmitted through seminal fluid, with more profound alterations in male rather than female offspring (23) (cf. ref. 24).

Studying paternal mechanisms in human populations requires disentangling socioeconomic, biological, and psychological or cultural effects and maternal and paternal stressors. Research thus has focused on the third generation, but sample sizes are small (20, 25, 26) or lifespans incomplete (27). We have complete lifespans and detailed information on the socioeconomic and family structure for 4,593 children of 1,407 former POWs and 15,310 children of 4,960 non-POW veterans. We are not aware of other large-sample studies in human populations that examine the reversibility of epigenetic transmission as seen in animal studies with maternal dietary supplementation (28, 29), maternal licking of offspring (30), and exercise of mature males

Significance

Understanding whether paternal trauma is transmitted to children to affect their longevity, the mechanisms behind any transmission, and the reversibility of paternal trauma can inform health interventions and increase our understanding of the persistence of health within families. We show that severe paternal hardship as a prisoner of war (POW) led to high mortality among sons, but not daughters, born after the war who survived to the age of 45 but that adequate maternal nutrition countered the effect of paternal POW trauma in a manner most consistent with epigenetic explanations. We are not aware of any large sample studies in human populations that examine the reversibility of paternal trauma nor the long-term impact of paternal ex-POW status on children.

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[1]To whom correspondence should be addressed. Email: costa@econ.ucla.edu.

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in the case of metabolic health (31). Data limitations preclude the study of the long-term impact of ex-POW status on children's older age mortality in more recent populations even though the effect of harsh conditions during wartime captivity on the mortality, health, and personality of World War II POW camp survivors has been documented (32–35).

Data and Methods

Identification of the impact of paternal trauma on longevity depends on the comparison of children of non-POW veterans and ex-POWs captured during the prisoner exchange period (before July 1863 or after June 1864) and the nonexchange period (between July 1863 and July 1864). Most POWs were exchanged immediately until mid-1863, when prisoner exchanges stopped as the two sides argued over the terms, particularly the treatment of black soldiers and their white officers. Exchanges began again in December of 1864. Men captured after mid-1863 faced ever-worsening conditions as the crowds of prisoners increased. Among survivors to 1900 in the original Union Army sample, wartime records mention scurvy for 11% of all non-POWs, 14% of ex-POWs captured before July 1863, 23% for ex-POWs captured between July 1863 and July 1864, and 14% for ex-POWs captured after June 1864 (11). Additional identification of the impact of paternal trauma comes from a within-family comparison of children born before and after the war.

Data. The analytical sample consists of the 2,342 children of 732 no-exchange period ex-POWs, 2,416 children of 715 exchange-period ex-POWs, and 15,145 children of 4,920 non-POW veterans, all born after 1866 and surviving to age 45. (*SI Appendix* shows that the data are best suited to examining older age mortality.) For these children, we have information on the longevity of 6,360 fathers and 6,238 mothers. We have two subsets of families with same-sex siblings born before and after the war and surviving to age 45: 855 daughters of 275 fathers and 1,067 sons of 342 fathers. We exclude children born during the war from these subsets.

The records of the veteran fathers were drawn from two different longitudinal samples constructed from handwritten military service and pension records, preserved in the National Archives. The veterans' children are being linked to death records and manuscript census schedules, which provide occupational, residential, and family information. *SI Appendix* describes the sample.

Statistical Analyses. We examine the impact of paternal ex-POW status on childrens' years lived after age 45 by fitting Cox proportional hazard models, clustered at the family level and stratified on enlistment year, to obtain hazard ratios. Our specification is

$$h(t) = h_0(t)\exp(\beta_x X),$$

where t is years lived after age 45, $h_0(t)$ is the baseline hazard, and X is a vector of characteristics specific to each child, and

$$\beta_x X = \beta_0 + \beta_1 P_N + \beta_2 P_E + \beta_3 F + \beta_4 (P_N \times F) + \beta_5 (P_E \times F) + \beta_6 C, \quad [1]$$

where P_N is a dummy indicator equal to 1 if the father was a no-exchange period POW, P_E is a dummy indicator equal to 1 if the father was an exchange-period POW, F is a dummy indicator equal to 1 if the child was female, and C is a vector of control characteristics. We interact sex with paternal POW status because of the observed female survival advantage at all ages even during famines and epidemics (36).

Ex-POWs were not observationally identical to non-POWs (see *SI Appendix*). Ex-POWs had enlisted earlier and at enlistment were less likely to be farmers and more likely to be laborers. Our statistical analyses therefore control for birth year and for preenlistment characteristics: occupational class at enlistment (farmer, artisan, professional or proprietor, laborer, and unknown), a dummy variable equal to 1 if the father enlisted in one of the 13 largest cities in the United States in 1860, a dummy variable equal to 1 if the father was US-born, a dummy variable equal to 1 if the father was Irish-born (a group with high mortality rates), a dummy if wounded in the war, and a dummy variable indicator for the sample.

We examine whether the impact of ex-POW status operates through postwar socioeconomic characteristics by including postwar controls for socioeconomic status. We run specifications in which we add controls for birth order, the total number of siblings, and postwar paternal characteristics: a dummy variable equal to 1 if the father had more than $100 in personal property wealth in 1870, nonfarmer paternal occupational class in 1880, and county population density in 1880. We add paternal farmer occupation in 1880 as an additional stratifying variable because adding full occupational dummies for paternal occupation in 1880 led to the proportionality assumption not being satisfied. We then run separate specifications for sons at least 21 y of age in 1910 and for married daughters in 1910, controlling for paternal occupational class in 1880. For sons, we also control for own nonfarmer occupational and marital status in 1910 and also stratify on own farmer occupation. For daughters, we control for husband's nonfarm occupational class in 1910 and additionally stratify on husband's farm occupation in 1910. *SI Appendix* investigates the direct impact of paternal ex-POW status on a wider set of socioeconomic status measures and on family structure.

We test whether our results are driven by maternal quality by adding to our specifications maternal lifespan and maternal grandfather's real estate wealth and additionally stratifying on the mother living to age 80 and unknown real estate wealth (unknown real estate wealth is set equal to 0.1) to satisfy the proportional hazards assumption. *SI Appendix* investigates the relationship between paternal ex-POW status and different measures of maternal grandfather's wealth and examines the direct impact of paternal ex-POW status on maternal mortality.

We then examine the two subsets of families with same-sex siblings born before and after the war. We estimate Cox regression models, stratified on the family and with standard errors clustered on the family, where

$$\beta_x X = \beta_1 W + \beta_2 (P_N \times W) + \beta_3 (P_E \times W) + \beta_4 B, \quad [2]$$

where W is a dummy variable indicating that the daughter or son was born after 1865 and B is the birth year.

SI Appendix compares our results with the estimated impact of ex-POW status on veterans' own mortality and assesses magnitudes. We examine excess mortality for the 26% of children with this information in *SI Appendix*.

We investigate the reversibility of paternal POW trauma among sons by interacting paternal POW status with quarter of birth. That is, in a Cox proportional model, clustered at the family level and stratified on year of enlistment,

$$\beta_x X = \beta_0 + \beta_1 P_N + \beta_2 P_E + \beta_3 Q + \beta_4 (P_N \times Q) + \beta_5 (P_E \times Q) + \beta_6 C, \quad [3]$$

where Q is a vector of quarter of birth dummies and the vector of controls C are birth year and paternal characteristics at enlistment. *SI Appendix* details the use of quarter of birth in historical populations.

Results

Among children born after the war and surviving to age 45, the sons of no-exchange period ex-POWs were a statistically

significant 1.11 times more likely to die compared with the sons of non-POWs and 1.09 times more likely to die compared with the sons of exchange-period ex-POWs, controlling for birth year and paternal enlistment characteristics (specification 1 in Table 1). Sons of exchange-period ex-POWs were statistically indistinguishable from non-POWs. There was no statistically significant impact of paternal POW status on daughters. Adding controls for postwar paternal characteristics, including paternal socioeconomic status, did not change the results (specification 2 in Table 1). The results remain unchanged when we examined sons who were at least age 21 in 1910 and add controls for sons' marital status and occupational category in 1910 or when we look at married daughters controlling for their husbands' occupational category in 1910 (specification 3 in Table 1). Adding controls for maternal lifespan and maternal grandfather's real estate wealth to specification 2 left the results unchanged (specification 4 in Table 1).

We find the same sex-specific pattern within families: Among families with same-sex children born before and after the war, there is no discernible impact of paternal POW status for daughters born after the war, but the sons of no-exchange ex-POW fathers born after the war died at 2.23 times the rate of those born before the war (Table 2). Because of the sample size, the magnitude should be interpreted with caution.

Paternal ex-POW status had little impact on sons' socioeconomic outcomes and family structure (*SI Appendix*, Table S11). The sons of no-exchange period ex-POWs were less likely to be farmers compared with the sons of non-POWs or exchange-period ex-POWs, but the effect becomes small and statistically insignificant once we control for paternal occupation in 1880. The sons of no-exchange period ex-POWs were marginally statistically significantly more likely to be homeowners in 1920 (but not in 1910), and those surviving to 1940 had a statistically insignificant 0.3 more years of education compared with the sons of non-POWs. They were as likely to be living in the same city as their father in 1900 if 21 y of age or older. If ever married, the sons of no-exchange period ex-POWs were less likely ever to be divorced than sons of non-POWs or exchange-period POWs.

Veterans' ex-POW status was uncorrelated with their wives' family wealth, but wounded veterans married poorer wives (*SI Appendix*, Table S12). The longevity of the children's mothers in 1900 was unaffected by their husbands' ex-POW status (*SI Appendix*, Table S13).

There was little attenuation in the impact of ex-POW status from fathers to sons. No-exchange period ex-POWs alive in 1900 were 1.14 times more likely to die than non-POWs (*SI Appendix*, Table S13). Controlling for paternal life span in examining children's mortality has only a modest impact on the magnitude of hazard rates (*SI Appendix*, Table S14).

The impact of own or paternal ex-POW status during the no-exchange period was comparable to the effect of childhood low socioeconomic status and urban residence, at a time when cities were ravaged by infectious disease (*SI Appendix*, Table S15).

Excess mortality among the sons (but not the daughters) of ex-POWs was largely from cerebral hemorrhage and, marginally statistically significantly, from cancer. There were no excess deaths from suicides and accidents (*SI Appendix*, Table S16). A caveat is that the results are only for children dying in the small set of states that provide cause of death in online indexes.

Birth in the second quarter magnifies the impact of paternal no-exchange ex-POW status, whereas birth in the fourth quarter eliminates the impact of paternal no-exchange POW status (see Table 3 and *SI Appendix*, Fig. S6). Among sons born in the second quarter, sons of no-exchange period ex-POWs were 1.2 times likelier to die than the sons of non-POWs and of ex-POWs during the no-exchange period. Sons of no-exchange POWs born during the second quarter were 1.1 times more likely to die than sons of non-POWs born during the fourth quarter. Among sons born in the fourth quarter, there was no statistically significant impact of paternal ex-POW status.

Discussion

Mechanisms. We use a proof by elimination to elucidate the mechanisms behind the transmission of paternal POW trauma. We find no evidence that paternal ex-POW status was transmitted to children through socioeconomic channels (Table 1 and *SI Appendix*, Table S11). The negative effect of ex-POW status on paternal (11) but not sons' socioeconomic status is in keeping with evidence that sons of land lottery winners in 19th-century Georgia did not have better socioeconomic outcomes than sons of nonwinners (37) and may be specific to the United States in this period. The impact of paternal ex-POW status on sons' older

Table 1. Paternal ex-POW status and death among children born after the war

	1			2			3			4		
Hazard ratios and tests	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p
Sons												
Father ex-POW, no exchange	1.110	0.045	0.009	1.104	0.043	0.012	1.115	0.052	0.021	1.106	0.044	0.011
Father ex-POW, exchange	1.016	0.046	0.663	1.012	0.038	0.733	1.021	0.043	0.626	1.024	0.036	0.493
Father non-POW	1.000			1.000			1.000			1.000		
Difference between no-exchange and exchange ex-POWs												
$\chi^2(1)$, $\Pr > \chi^2$		4.06	0.044		4.00	0.046		3.48	0.062		3.14	0.077
Daughters	1.000											
Father ex-POW, no exchange	0.997	0.039	0.929	0.990	0.038	0.799	0.967	0.049	0.517	0.992	0.038	0.848
Father ex-POW, exchange	0.963	0.034	0.294	0.959	0.035	0.259	0.989	0.047	0.824	0.970	0.035	0.404
Father non-POW	1.000			1.000			1.000			1.000		
Global test of proportional hazards assumption												
$\chi^2(n)$, $\Pr > \chi^2$	$\chi^2(15)$	15.79	0.396	$\chi^2(24)$	29.44	0.204				$\chi^2(26)$	35.49	0.127
Sons, column 3							$\chi^2(25)$	33.54	0.118			
Daughters, column 3							$\chi^2(25)$	32.43	0.146			

The sample is all children born after the war and survived to age 45. Hazard ratios are relative to a non-POW father and are estimated from Eq. 1, with standard errors clustered on the family and stratification on paternal enlistment. Specification 1 controls for birth year and paternal enlistment characteristics. Specification 2 adds controls for paternal socioeconomic status and family demographic and residential characteristics. Specification 3 examines, separately, sons age 21 or over and married daughters, both of whom survived to 1910, and adds controls for either son's or son-in-law's occupational class in 1910. Specification 4 adds to specification 2 controls for maternal lifespan and maternal grandfather's real estate wealth. Specifications 1, 2, and 4: 19,903 children of 6,367 fathers. Specification 3: 8,427 sons of 4,665 fathers and 5,982 daughters of 3,766 fathers. See *SI Appendix*, Table S10 for additional proportionality assumption tests.

Table 2. Paternal ex-POW status and death among same-sex siblings born before and after the war

	Daughters				Sons			
	Prewar	Postwar			Prewar	Postwar		
Hazard ratios and tests	e^{β}	e^{β}	$\hat{\sigma}$	p	e^{β}	e^{β}	$\hat{\sigma}$	p
Father no-exchange ex-POW	1.000	0.961	0.309	0.903	1.000	2.225	0.932	0.056
Father exchange ex-POW	1.000	1.113	0.434	0.784	1.000	0.812	0.239	0.480
Father non-POW	1.000	0.865	0.170	0.746	1.000	1.073	0.179	0.675
Difference between no-exchange and exchange ex-POW								
$\chi^2(1)$, $\Pr > \chi^2$			0.10	0.753			4.71	0.030
Difference between no-exchange ex-POW and non-POW								
$\chi^2(1)$, $\Pr > \chi^2$			0.37	0.543			2.20	0.138
Global test of proportional hazards assumption								
$\chi^2(4)$, $\Pr > \chi^2$			3.53	0.473			3.50	0.478

The samples are same-sex siblings born before and after the war who survived to age 45. Children born during the war are excluded. Results are estimated from Eq. **2**, with stratification and SE clustered on the family. Hazard ratios are relative to a daughter or son born before the war and are by paternal ex-POW status. The samples are of 855 daughters of 275 fathers and 1,067 sons of 342 fathers.

age mortality but not on their socioeconomic status suggests that some health effects may manifest only at older ages, after socioeconomic outcomes have been determined, and is consistent with the lack of a mediating association between own socioeconomic status and in utero exposure to the Dutch Hunger Winter on later mortality (38). In contrast, the 1918 Influenza Pandemic and the Chinese Famine affected educational attainment of children in utero during these events (6, 13). Differential effects of malnutrition and viral illnesses or differences in exposure duration and in sample size (e.g., increased schizophrenia rates may not be noticed in smaller samples) may explain the contrasting impact of these experiences.

The comparison of sons born before and after the war within families in Table 2 allows us to reject the hypothesis that traits that permitted fathers to survive in POW camps may have hurt their own and their sons' survival chances after the war or that permanent family effects, such as family wealth, account for the transmission.

Ex-POW status had a statistically insignificant impact on marriage quality, perhaps because the war, in which over 80% of 18 y olds in 1861 served (39) and in which 14% of the men in the main Union Army sample died, had reduced the supply of eligible men. The negative impact of war injuries on marriage match quality (*SI Appendix*, Table S12) is in keeping with the labor demands of a nonmechanized society.

Our finding that sons born in the second quarter were the ones affected by paternal ex-POW status suggests that psychological transmission to boys only is an unlikely explanation. We also found no direct evidence of pathopsychological effects from paternal ex-POW status: Paternal ex-POW status had no impact on family structure or proximity to family members (*SI Appendix*, Table S11), and the number of deaths from accidents and suicides was not correlated with paternal ex-POW status (*SI Appendix*, Table S16). While there might be indirect effects through mothers, these effects were not large enough to affect mothers' own mortality (*SI Appendix*, Table S13).

The sex-specific transmission we observe is consistent with an epigenetic effect in which transmission occurs on the Y chromosome, as hypothesized in the Överkalix studies, which found that food availability during the male slow-growth period affects the longevity of sons, but not of daughters, and of grandsons, but not granddaughters. Among 146 male probands in these studies, the estimated mortality hazard ratios on paternal over- and undernutrition, respectively, were a statistically significant 1.70 and a statistically insignificant 1.11 (a magnitude similar to ours) (20). A follow-up of eight descendants of the Överkalix study found that different geneontology pathways were implicated in paternal and maternal line transmission of nutritional stress (40). Although sexual dimorphism in developmental origins of health and disease is little understood, it is considered an outcome of both genetics and epigenetics (41). Sex-specific effects may differ by species and type and timing of the insult. In some mouse studies, a paternal diet, stress, or toxin exposure affects female (30, 42, 43) or male (23, 44, 45) offspring only, whereas in others both are affected (24) or the only robust results are for one sex (46).

Table 3. Paternal ex-POW status and death by own quarter of birth among sons born after the war

	First quarter			Second quarter			Third quarter			Fourth quarter		
Hazard ratios and tests	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p	e^{β}	$\hat{\sigma}$	p
Father no-exchange ex-POW	1.090	0.073	0.198	1.204	0.085	0.008	1.101	0.071	0.134	1.013	0.068	0.849
Father exchange ex-POW	1.036	0.065	0.571	0.942	0.057	0.322	1.092	0.069	0.159	0.972	0.060	0.642
Father non-POW	1.017	0.032	0.602	1.000			0.986	0.030	0.636	0.933	0.030	0.031
Within-quarter difference												
No-exchange and exchange ex-POW												
$\chi^2(1)$, $\Pr > \chi^2$		0.42	0.518		8.94	0.003		0.01	0.915		0.28	0.598
No-exchange ex-POW and non-POW												
$\chi^2(1)$, $\Pr > \chi^2$		1.09	0.296					3.04	0.081		1.50	0.221
Difference with second quarter												
No-exchange ex-POWs, $\chi^2(1)$, $\Pr > \chi^2$		1.82	0.178					1.17	0.280		4.28	0.039

Hazard ratios are relative to a non-POW father born in the second quarter and are for years lived after age 45. Estimated from Eq. **3**, with standard errors clustered on the family and with stratification on the father's enlistment year. Global test of the proportional hazards assumption: $\chi^2(21) = 21.29$, $\Pr > \chi^2 = 0.441$. The sample is of 10,313 sons of 5,615 fathers.

The increase in death rates from cerebral hemorrhage and cancer (*SI Appendix*, Table S16) is in agreement with animal studies of epigenetic transmission of paternal diet. Preconceptional paternal fasting in mice is linked in male offspring to elevated serum glucose, which is associated with hypertension, a risk factor for cerebral hemorrhage (46). Folate deficiency in male mouse diets alters the sperm epigenome in a way that might put the offspring at greater risk for cancer (47).

Results on quarter of birth imply that the in utero environment and paternal experience contribute to programing children's phenotypes through epigenetic modifications. Researchers have interpreted quarter of birth effects as reflecting inadequate nutrition in past populations (e.g., ref. 48; see also *SI Appendix*). Maternal dietary supplements of folic acid or genistein counteract early development exposure to bisphenol A in mice (28). In late postnatal life, male primordial germ cells (PGCs) undergo extensive epigenetic reprogramming. Inadequate maternal nutrition during late gestation, when more demands are made on maternal nutrition, may delay or impair remethylation (49). Studies of the Dutch Hunger Winter found that although early gestation was a particularly vulnerable time and girls were more affected than boys overall, famine in late gestation led to changes in methylation at three genes related to cardiovascular health in boys but not girls (50–52).

The transmission of shocks in the availability of paternal food on longevity through the male line in the Överkalix studies hints at a nutritional explanation. The psychological loss of a parent during boys' slow growth period predicts their children's prematurity and lower birth weight, but the effects are evident for both sexes (53).

Strengths and Limitations. The study design provides two control groups for the children of former Union Army POWs when camp conditions were at their worst: the children of ex-POWs when camp conditions were better, and the children of non-POW veterans. Within families, children born before the war provide control groups for children born after the war. The rich, high-quality data permit detailed examinations of the socioeconomic status, family arrangements, and residential locations of fathers, mothers, and children, thus enabling us to investigate several mechanisms for the transmission of intergenerational trauma.

There are some limitations of the study. While there is information on mortality, the data are best suited to study older age mortality, cause of death data are sparse, and there is no information on children's physical or mental health. Second, there are no data on interactions between family members and on paternal posttraumatic stress disorder (PTSD) because it was not a recognized condition in this time period. We can use a process of elimination to argue in favor of an epigenetic explanation, but we cannot reject definitively cultural or psychological explanations. Third, even if the mechanism is epigenetic, we cannot disentangle the impact of the stress of captivity from that of starvation.

Conclusion

Our findings point to the importance of paternal stress on sons' longevity at older ages, a transmission that may have occurred through epigenetic channels. Whether the findings can be generalized to more recent populations is unknown. Improvements in the adequacy of maternal nutrition in developed countries today should reduce epigenetic transmission. Psychological trauma may be more common among children of Japanese-, Korean-, or Vietnamese-held ex-POWs or genocide survivors because Civil War POWs faced neither torture nor murder and returned home to a grateful nation. Their status as heroes may have given their experiences meaning. Nonetheless, the findings provide "proof of concept" for the transmission and reversibility of paternal trauma in a human population.

Materials and Methods

Data and STATA programs for both the main paper and *SI Appendix* are available online at doi.org/10.3886/E104880V1. Users interested in obtaining additional data on the fathers, collected from military and pension records, can do so at uadata.org.

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