Family Ruptures, Stress, and the Mental Health of the Next Generation

ONLINE APPENDIX

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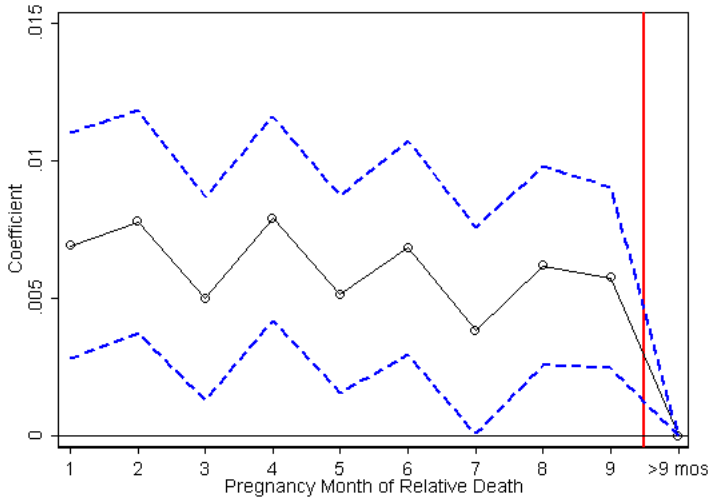
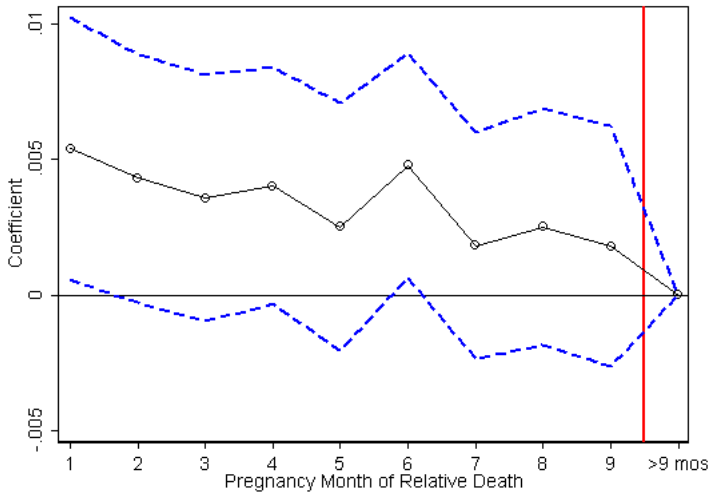
A Additional Results

Figure A1: Effect of Relative Death on the Incidence of the Child Being Born Pre-term



Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st-9th months of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being born pre-term.

Figure A2: Effect of Relative Death on the Incidence of the Child Being Hospitalized for a Perinatal Condition by Age 1



Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. The sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st-9th months of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being ever hospitalized for a condition arising from the perinatal period by age 1.

Table A1: Correlation Between the Timing of Relative Death and Maternal Characteristics

	(1) M.Age	(2) 1st Par.	(3) M.Mar.	(4) M.Div	(5) M.Ed:<HS	(6) M.Ed:HS	(7) M.Ed:SomeColl	(8) M. Wage	(9) M. Foreign
Death During Pregnancy	-0.0103	0.0133***	-0.00201	-0.000280	-0.00111	-0.00205	0.00120	388.3	-0.00156***
	[0.0155]	[0.00188]	[0.00177]	[0.000555]	[0.00137]	[0.00164]	[0.00156]	[489.5]	[0.000482]
Mean, dept. var	27.88	0.496	0.311	0.0303	0.177	0.314	0.202	124317.5	0.0216
Obs.	295678	295678	295678	295678	289087	289087	289087	191074	295678

Note: See table 1for more information on the sample. This table reports the correlation between exposure to relative death during pregnancy and maternal characteristics measured prior to conception. "M." denotes mothers' characteristics. All regressions control for fixed effects for the year and month of conception, the relative's age and age squared, as well as the mother's municipality of residence during the year prior to conception. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A2: Correlation Between the Timing of Relative Death and Paternal Characteristics

	(1) F.Age	(2) F.Mar.	(3) F.Div	(4) F.Ed:<HS	(5) F.Ed:HS	(6) F.Ed:SomeColl	(7) F. Wage
Death During Pregnancy	-0.00854	-0.00161	-0.000448	-0.000751	0.000718	-0.0000391	1022.6
	[0.0203]	[0.00189]	[0.000654]	[0.00154]	[0.00156]	[0.00148]	[666.2]
Mean, dept. var	30.53	0.315	0.0397	0.193	0.351	0.187	208987.8
Obs.	293497	290663	290663	278483	278483	278483	187081

Note: See table 1 for more information on the sample. This table reports the correlation between exposure to relative death during pregnancy and paternal characteristics measured prior to conception. "F." denotes fathers' characteristics. All regressions control for fixed effects for the year and month of conception, the relative's age and age squared, as well as the mother's municipality of residence during the year prior to conception. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A3: Effects of Relative Death *In Utero* on Stillbirths, Perinatal Deaths, and Sex Ratio

	1st Parity			2nd Parity		
	(1) Stillb.	(2) Peri.Death	(3) Male Child	(4) Stillb.	(5) Peri.Death	(6) Male Child
Panel A: All Relative Deaths						
Death During Pregnancy	-0.000132	0.0000845	0.00159	0.0000365	0.000231	0.00151
	[0.000245]	[0.000411]	[0.00262]	[0.000257]	[0.000413]	[0.00313]
Mean, dept. var	0.00156	0.00393	0.514	0.00157	0.00363	0.514
Obs.	143309	143309	143309	99898	99898	99898
Panel B: Close Relative Deaths						
Death During Pregnancy	-0.000171	0.000625	0.00453	-0.000160	0.000132	0.00457
	[0.000483]	[0.000870]	[0.00544]	[0.000427]	[0.000752]	[0.00516]
Mean, dept. var	0.00181	0.00563	0.513	0.00144	0.00461	0.510
Obs.	31442	31442	31442	31241	31241	31241
Panel C: Maternal Parent/Sibling Deaths						
Death During Pregnancy	-0.000190	0.000997	0.00555	-0.000190	0.000280	0.00324
	[0.000498]	[0.000900]	[0.00543]	[0.000448]	[0.000782]	[0.00509]
Mean, dept. var	0.00188	0.00548	0.513	0.00150	0.00440	0.509
Obs.	30304	30304	30304	29999	29999	29999

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A4: Placebo Effects of Relative Death During Pregnancy on *Older* Sibling's Outcomes

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death during younger sib's gestation	0.000838 [0.00236]	-0.00114 [0.00240]	-0.00107 [0.00323]	0.00135 [0.00584]	-0.00110 [0.0139]	-0.00621 [0.0124]
Mean, dept. var	0.0316	0.0502	0.0500	0.0160	0.0632	0.104
Obs.	31582	31678	23905	2443	2437	2437

Note: See table 1 for more information on the sample. In this table we link all of the children in our analysis sample to their older siblings (if they exist). Siblings data is only available for children born in years 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005. The table reports the coefficients on the (placebo) effects of a relative death during the younger child's gestation on the older sibling's birth outcomes. In column (3), the sample is further limited to siblings born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to older siblings of children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E. All regressions control for fixed effects for the younger child's year and month of conception, as well as the mother's municipality of residence during the year prior to conception. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A5: Effects of Relative Death *In Utero* on Additional Birth Outcomes

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced
Panel A: All Relative Deaths						
Death During Pregnancy	0.000603	0.000184	-0.0449***	-0.0352***	0.00388***	-0.00108
	[0.000623]	[0.000708]	[0.00941]	[0.00602]	[0.00125]	[0.00102]
Mean, dept. var	0.0267	0.0336	50.46	34.82	0.128	0.0701
Obs.	288334	288334	286026	278395	289087	289087
Panel B: Close Relative Deaths						
Death During Pregnancy	0.000225	-0.000324	-0.0377**	-0.0352***	0.00542**	0.00132
	[0.00116]	[0.00124]	[0.0162]	[0.0105]	[0.00219]	[0.00155]
Mean, dept. var	0.0348	0.0348	50.40	34.76	0.131	0.0472
Obs.	84584	84584	84016	82300	84817	84817
Panel C: Maternal Parent/Sibling Deaths						
Death During Pregnancy	0.0000839	-0.000228	-0.0408**	-0.0368***	0.00452**	0.00115
	[0.00122]	[0.00129]	[0.0170]	[0.0106]	[0.00221]	[0.00156]
Mean, dept. var	0.0345	0.0348	50.41	34.76	0.130	0.0474
Obs.	80956	80956	80427	78778	81177	81177

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A6: Effects of Relative Death *In Utero* on Birth Outcomes: Results by Trimester

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.
Death in 1st Trimester	-11.93***	0.00382***	0.00131***	-0.00517**	0.00652***
	[3.376]	[0.000939]	[0.000470]	[0.00236]	[0.00144]
Death in 2nd Trimester	-10.69***	0.00450***	0.000854**	-0.00539***	0.00653***
	[2.563]	[0.000902]	[0.000400]	[0.00191]	[0.00122]
Death in 3rd Trimester	-11.79***	0.00349***	0.00154***	-0.00452**	0.00553***
	[2.925]	[0.000965]	[0.000349]	[0.00204]	[0.00117]
Mean, dept. var	3546.3	0.0320	0.00511	0.188	0.0494
Obs.	288337	288337	288337	288337	289087

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A7: Effects of Relative Death *In Utero* on Additional Birth Outcomes: Results by Trimester

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced
Death in 1st Trimester	0.000846	0.00134	-0.0382***	-0.0409***	0.00212	-0.00309**
	[0.000929]	[0.000964]	[0.0142]	[0.0101]	[0.00200]	[0.00143]
Death in 2nd Trimester	0.000675	-0.000291	-0.0325***	-0.0253***	0.00493***	-0.00189
	[0.000930]	[0.000978]	[0.0116]	[0.00845]	[0.00177]	[0.00134]
Death in 3rd Trimester	0.000325	-0.000396	-0.0622***	-0.0394***	0.00445**	0.00143
	[0.000758]	[0.00108]	[0.0131]	[0.00818]	[0.00178]	[0.00162]
Mean, dept. var	0.0267	0.0336	50.46	34.82	0.128	0.0701
Obs.	288334	288334	286026	278395	289087	289087

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A8: Effects of Relative Death *In Utero* on Hospitalizations by Age 1: Results by Trimester

	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp-Peri.	(4) Tot Hosp-Peri.
Death in 1st Trimester	0.00360**	0.00278	0.00467***	0.00436**
	[0.00154]	[0.00319]	[0.00147]	[0.00169]
Death in 2nd Trimester	0.00164	0.00223	0.00335**	0.00301*
	[0.00134]	[0.00247]	[0.00143]	[0.00162]
Death in 3rd Trimester	0.000703	-0.000338	0.00264**	0.00164
	[0.00138]	[0.00249]	[0.00127]	[0.00159]
Mean, dept. var	0.0737	0.102	0.0575	0.0646
Obs.	288606	288606	231398	231398

Note: See tables 1 and 2 for more information on the sample and controls. "Any Hosp-Peri." refers to an indicator for ever being hospitalized for a condition originating in the perinatal period. In columns (3) and (4), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A9: Effects of Relative Death *In Utero* on Hospitalizations by Ages 5, 10, 18, and 27

	Any Hospitalizations By Age			
	(1)	(2)	(3)	(4)
	5	10	18	27
Panel A: All Relative Deaths				
Death During Pregnancy	0.00133	-0.00108	0.00200	0.000583
	[0.00122]	[0.00150]	[0.00200]	[0.00222]
Mean, dept. var	0.113	0.136	0.182	0.191
Obs.	288606	204794	143349	81540
Panel B: Close Relative Deaths				
Death During Pregnancy	0.000831	-0.000814	0.000588	-0.00443
	[0.00223]	[0.00252]	[0.00358]	[0.00403]
Mean, dept. var	0.105	0.137	0.200	0.280
Obs.	84676	72135	60131	39320
Panel C: Maternal Parent/Sibling Deaths				
Death During Pregnancy	0.000645	-0.000783	0.00120	-0.00352
	[0.00224]	[0.00263]	[0.00355]	[0.00413]
Mean, dept. var	0.105	0.137	0.199	0.277
Obs.	81036	69010	57446	37496

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A10: Effects of Relative Death *In Utero* on Prescription Use for Physical Health Conditions (Obesity, Diabetes, Cushing's Syndrome, Hypo- & Hyperthyroidism, Cholesterol, and Beta Blockers) by Age

	Any Physical Health Prescriptions at Ages...						
	(1) 4-6	(2) 9-11	(3) 14-16	(4) 19-21	(5) 24-26	(6) 29-31	(7) 34-36
Panel A: All Relative Deaths							
Death During Pregnancy	0.000122	-0.000310	0.0000419	-0.000335	-0.00278*	-0.000986	0.00571*
	[0.000372]	[0.000533]	[0.000650]	[0.000890]	[0.00144]	[0.00221]	[0.00314]
Mean, dept. var	0.00437	0.00899	0.0154	0.0242	0.0359	0.0514	0.0701
Obs.	112330	114906	114593	101776	70043	47506	27641
Panel B: Close Relative Deaths							
Death During Pregnancy	0.000218	-0.000342	-0.000429	-0.00167	-0.00454**	-0.0000740	0.00554*
	[0.000961]	[0.00134]	[0.00169]	[0.00163]	[0.00203]	[0.00292]	[0.00333]
Mean, dept. var	0.00446	0.00888	0.0152	0.0243	0.0347	0.0504	0.0708
Obs.	17258	20380	25781	30886	31600	32334	22907
Panel C: Maternal Parent/Sibling Deaths							
Death During Pregnancy	0.000397	-0.000525	-0.000274	-0.00134	-0.00400*	0.0000956	0.00611*
	[0.000940]	[0.00142]	[0.00177]	[0.00161]	[0.00212]	[0.00302]	[0.00338]
Mean, dept. var	0.00417	0.00882	0.0154	0.0242	0.0349	0.0502	0.0706
Obs.	16561	19605	24754	29626	30266	30863	21763

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E. Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A11: Effects of Relative Death *In Utero* on ADHD Prescription Use: Differences by Age During 2002-2014

	Ages 4-14			Ages 15-36		
	(1) Any Mental RX	(2) Any ADHD RX	(3) ADHD Avg Dose	(4) Any Mental RX	(5) Any ADHD RX	(6) ADHD Avg Dose
Death During Pregnancy	0.00837**	0.00325**	0.0921*	0.00116	0.00129	0.0226
	[0.00336]	[0.00152]	[0.0474]	[0.00384]	[0.00114]	[0.0398]
Mean, dept. var	0.0824	0.0253	0.513	0.385	0.0247	0.517
Obs.	33126	33126	33126	64854	64854	64854

Note: See tables 1 and 2 for more information on the sample and controls. The sample here is further limited to children of mothers who experience the death of a parent or a sibling. The first three columns consider the outcomes listed at ages 4-14 in our data, while the last three columns consider the outcomes listed at ages 15-36 in our data. Individuals who are at most 14 years old in our data were born in 2005-14=1991 or later. These cohorts were at most 11 years old in 2002, the first year when ADHD prescription drugs became readily available in Sweden. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.

Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A12: Are Effects of Relative Death *In Utero* on Mental Health Prescription Use in Adulthood Driven by "Precipitating Events"?

	Married, 34-36				Not Married, 34-36			
	(1) Any Anx RX	(2) Anx Avg. dose	(3) Any Dep RX	(4) Dep Avg. dose	(5) Any Anx RX	(6) Anx Avg. dose	(7) Any Dep RX	(8) Dep Avg. dose
Death During Pregnancy	0.0160***	0.0634***	0.0136*	0.913**	0.00347	0.0181	0.00467	0.236
	[0.00559]	[0.0203]	[0.00764]	[0.441]	[0.00518]	[0.0378]	[0.00574]	[0.376]
Mean, dept. var	0.0613	0.135	0.104	3.977	0.0702	0.250	0.115	4.923
Obs.	8669	8669	8669	8669	13094	13094	13094	13094

Note: See tables 1 and 2 for more information on the sample and controls. The sample here is further limited to children of mothers who experience the death of a parent or a sibling. The first four columns limit the sample to children who are observed be married at ages 34-36. The last four columns limit the sample to children who are observed to not be married at ages 34-36. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A13: Effects of Relative Death *In Utero* on Maternal Pregnancy Behaviors and Characteristics

	(1) Adeq. PC	(2) Int. PC	(3) Highrisk	(4) Start Smoking	(5) Wgt Gain (kg)	(6) Hosp.∉Muni.	(7) Any Wage Inc.
Panel A: All Relative Deaths							
Death During Pregnancy	-0.00287	-0.00138	-0.00150	0.000225	-0.0155	0.000534	0.0000877
	[0.00228]	[0.00177]	[0.00147]	[0.000242]	[0.0331]	[0.00103]	[0.00129]
Mean, dept. var	0.828	0.914	0.166	0.00370	13.96	0.117	0.927
Obs.	138453	138453	289087	288606	101330	289087	191916
Panel B: Close Relative Deaths							
Death During Pregnancy	-0.0116*	-0.0105**	0.00185	0.000628	-0.0837	0.00101	-0.00101
	[0.00631]	[0.00459]	[0.00277]	[0.000475]	[0.0700]	[0.00218]	[0.00277]
Mean, dept. var	0.814	0.900	0.111	0.00299	13.55	0.107	0.906
Obs.	22208	22208	84817	84676	26752	84817	34873
Panel C: Maternal Parent/Sibling Deaths							
Death During Pregnancy	-0.0126**	-0.0111**	0.00145	0.000502	-0.0715	0.00135	-0.00131
	[0.00632]	[0.00478]	[0.00277]	[0.000483]	[0.0714]	[0.00223]	[0.00287]
Mean, dept. var	0.816	0.902	0.112	0.00279	13.55	0.106	0.911
Obs.	21328	21328	81177	81036	25712	81177	33496

Note: See tables 1 and 2 for more information on the sample and controls. "Adeq. PC" and "Int. PC" are indicators for the mother's prenatal care being adequate and intermediate, respectively. These measures use the Kotelchuk Index (Kotelchuck, 1994), which compares the number of prenatal visits received to the number of expected visits, adjusting for gestational age when care began and gestational age at delivery. Adequate prenatal care means that the ratio of observed to expected visits is at least 80%. Intermediate prenatal care means that the ratio of observed to expected visits is 50-79%. "Highrisk" is an indicator for the mother having any of the following conditions during pregnancy: diabetes, kidney disease, epilepsy, asthma, hypertension, or urinary infection. "Start Smoking" is an indicator for the mother initiating smoking during pregnancy. "Wgt Gain" is the mother's total pregnancy weight gain in kilograms. "Hosp.∉Muni." is an indicator for the mother's hospital at which she gives birth being in a different municipality than her municipality of residence. "Any Wage Inc." is an indicator for the mother having positive wage income in the year of conception or the year after. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A14: Effects of Relative Death *In Utero* on the *Mother's* Prescription Use for Mental Health Conditions

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Panel A: All Relative Deaths							
Death During Pregnancy	-0.000436	0.000434	-0.000589	-0.0000939	0.00571	0.000124	0.0243
	[0.00161]	[0.000292]	[0.00586]	[0.00115]	[0.00540]	[0.00127]	[0.0507]
Mean, dept. var	0.318	0.00560	0.0727	0.102	0.193	0.137	3.223
Obs.	288606	288606	288606	288606	288606	288606	288606
Panel B: Close Relative Deaths							
Death During Pregnancy	0.00146	-0.000304	-0.00316	-0.00364	0.00823	0.00298	0.0667
	[0.00332]	[0.000438]	[0.00961]	[0.00253]	[0.0128]	[0.00205]	[0.0765]
Mean, dept. var	0.337	0.00455	0.0535	0.110	0.234	0.141	2.937
Obs.	84676	84676	84676	84676	84676	84676	84676
Panel C: Maternal Parent/Sibling Deaths							
Death During Pregnancy	0.000164	-0.000272	-0.00161	-0.00363	0.00702	0.00318	0.0662
	[0.00335]	[0.000456]	[0.00951]	[0.00256]	[0.0129]	[0.00209]	[0.0746]
Mean, dept. var	0.335	0.00432	0.0514	0.109	0.230	0.139	2.922
Obs.	81036	81036	81036	81036	81036	81036	81036

Note: See tables 1 and 2 for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E. Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A15: Effects of Relative Death *In Utero* on Main Outcomes: Heterogeneity by Maternal Education

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death During Pregnancy	0.00372***	0.00536***	0.00341***	0.00481	0.00779	0.0103
	[0.000817]	[0.00109]	[0.00120]	[0.00293]	[0.00535]	[0.00827]
Mom Low Ed (HS or less)	0.00853***	0.00759***	0.0114***	0.0101***	0.0152***	0.0138*
	[0.000929]	[0.00118]	[0.00147]	[0.00383]	[0.00432]	[0.00752]
Mom Low Ed*Death During Preg	-0.000135	0.00160	-0.0000795	0.00244	-0.00122	-0.00230
	[0.00126]	[0.00165]	[0.00190]	[0.00505]	[0.00697]	[0.0102]
Mean, dept. var	0.0307	0.0483	0.0577	0.0235	0.0658	0.110
Obs.	272907	273597	221999	18852	20387	20387

Note: See tables 1 and 2 for more information on the sample and controls. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.

Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A16: Effects of Relative Death *In Utero* on the Mother's Subsequent Fertility

	Dep. Var: Mother Has Subsequent Children		
	(1) All Deaths	(2) Close Relative Deaths	(3) Maternal Parent/Sib Deaths
Death During Pregnancy	0.0149***	0.0133*	0.00636
	[0.00356]	[0.00679]	[0.00663]
Mean, dept. var	0.488	0.407	0.408
Obs.	50802	16454	15724

Note: See tables 1 and 2 for more information on the sample and controls. In this table we link all of the children in our analysis sample to their older siblings (if they exist). Siblings data is only available for children born in years 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

B Hypotheses and Related Literature: An Extended Discussion

In Section I, we provide a short description of our hypotheses regarding the impact of exposure to stress on physical health at birth and later in life, differential effects across gestational age at exposure, as well as differential effects with respect to the severity of stress. Here we provide a more extensive discussion of each of these hypotheses, by drawing on the burgeoning literature on early-life shocks (see Almond and Currie, 2011 for a review).

Implications from Evidence on Physical *In Utero* Shocks First, a large number of existing studies point to adverse effects of exposure to *physical* insults during the fetal period on both birth outcomes and later life physical health and economic well-being.[1] The evidence on the consequences of purely *psychological* stressors is more limited, as studies that exploit variation from extreme and rare events like natural disasters and terrorist attacks are limited in their ability to separate the effects of *in utero* stress exposure from any post-natal responses, as well as from the physical health and economic insults associated with these events.[2] Our empirical methodology (described in detail in Section III) and focus on a nearly universal stressor are designed to overcome these limitations.

Despite the scarce direct evidence on psychological stressors, the medical and epidemiological literature that tries to identify the mechanisms through which the effects of physical insults operate suggests that maternal stress during pregnancy plays a key role. For example, one hypothesis for why malnutrition during pregnancy harms the unborn child is that nutritional restrictions in the mother inhibit the development of a placental enzyme that is required to convert the stress hormone cortisol into inactive cortisone. Thus, as a consequence of maternal malnutrition, the fetus is exposed to excessive amounts of cortisol *in utero*. Overexposure to cortisol, in turn, is believed to lead to a reprogramming of the hypothalamic-pituitary-adrenal axis (HPA), which could lead to impaired fetal development and worse health in adult age.[3] If stress in fact drives the adverse effects of physical insults such as malnutrition, then a rigorous analysis of the causal effects of *in utero* exposure to stress can provide new insights on the determinants of health and human capital formation more broadly. As such, we expect that exposure to maternal stress due to the death of a relative during the fetal period may have damaging effects on outcomes at birth and in later life.

[1] See, e.g.,G. J. Van den Berg, M. Lindeboom and F. Portrait (2006); Douglas Almond, Lena Edlund, Hongbin Li and Junsen Zhang (2010); Hilary Hoynes, Marianne Page and Ann Huff Stevens (2011); Douglas Almond, Hilary W Hoynes and Diane Whitmore Schanzenbach (2011); Douglas Almond and Bhashkar Mazumder (2012); Hilary W. Hoynes, Diane W. Schanzenbach and Doug Almond (Forthcoming); Robert S Scholte, Gerard J Van Den Berg and Maarten Lindeboom (2015); Maya Rossin-Slater (2013) on malnutrition; Doug Almond (2006); Alan I Barreca (2010) on disease outbreaks; Douglas Almond, Lena Edlund and Mårten Palme (2009); Sandra E Black, Aline Butikofer, Paul J Devereux and Kjell G Salvanes (2013) on radiation; and N.J. Sanders (2012); Adam Isen, Maya Rossin-Slater and Reed Walker (Forthcoming) on air pollution.

[2] See, for example, evidence on hurricanes (Simeonova, 2011; Currie and Rossin-Slater, 2013), earthquakes (Tan et al., 2009; Glynn et al., 2001; Torche, 2011), and the terrorist attacks of September 11 (Berkowitz et al., 2003; Lederman et al., 2004; Lauderdale, 2006; Eskenazi et al., 2007). Another recent paper uses *in utero* exposure to the Superbowl to identify the effects of prenatal stress on birth outcomes (Duncan, Mansour and Rees, 2015).

[3] See Dunkel Schetter (2011) as well as a review of the literature in Jaddoe (2006). Also see Online Appendix F for a more detailed discussion.

Long-Term Effects on Physical Health Second, when it comes to physical health outcomes specifically, the "fetal origins hypothesis," originally put forth by epidemiologist David J. Barker, argues that poor conditions *in-utero* can lead to latent effects on disease much later in life (Barker, 1990). However, while there is ample evidence both from economics and epidemiology supporting Barker's hypothesis, this evidence comes from studies of adults who are older than the individuals in our sample. For example, Almond (2006) documents that individuals exposed to the 1918 influenza pandemic *in utero* are more likely to be disabled in their 50s and 60s, and Hoynes, Schanzenbach and Almond (Forthcoming) show that access to food stamps early in life leads to a significant reduction in the incidence of "metabolic syndrome" in a sample that includes individuals up to age 55.[4] This evidence suggests that—even if *in utero* exposure to psychological stress from family ruptures has a latent effect on physical health that appears in older ages—the time horizon over which we track our sample may not be sufficient for us to measure it, as the oldest individuals that we observe are in their thirties.

Moreover, Sandra E Black, Paul J Devereux and Kjell G. Salvanes (2016)'s analysis of deaths of maternal parents during pregnancy in Norway shows small detrimental impacts on birth outcomes, but no effects on adult BMI. Evidence from this closely related paper also suggests that we may not detect any adverse physical health effects in adulthood.[5]

Differential Effects Across Gestational Age at Time of Shock Third, the existing literature provides some guidance on why we might expect to see differential effects across gestational age due to *physical* shocks such as infections. For example, Robinson (2013) argues that infections in early pregnancy increase the likelihood of symmetric growth restriction of the fetus (proportional growth restriction in the brain and body), while infections in later pregnancy may affect the likelihood of asymmetric growth restriction (brain growth not restricted; only body). While both types exhibit physical health impairments in later life, only the symmetric type shows long-term brain or cognitive impairments. Empirical evidence on the effects of disease outbreaks supports this hypothesis to some extent—for example, Almond (2006)'s seminal study on the 1918 influenza pandemic in the U.S. finds the strongest long-term economic effects for cohorts exposed during their first trimester. On the other hand, follow-up work on *in utero* exposure to the flu in Taiwan does not find differential impacts across the three trimesters (Lin and Liu, 2014). Moreover, studies on the impacts of nutritional and environmental shocks *in utero* offer mixed evidence—some find differential effects across gestational age while others do not.[6]

[4]The "metabolic syndrome" in Hoynes, Schanzenbach and Almond (Forthcoming) is a composite index measure that includes obesity, high blood pressure, and diabetes. Consistent with this evidence, epidemiological studies have documented a correlation between *in utero* exposure to the Dutch famine of 1944 and a higher incidence of obesity and heart disease when the individuals reached middle age (Susser and Lin, 1992).

[5]Another related paper is Li Jiong and Sorensen (2010), who use Danish data to compare the Body Mass Index (BMI) of children of mothers who experienced a death during pregnancy to children of those who did not. However, an important limitation is that this study does not fully account for non-random exposure to death.

[6]For example, Almond, Hoynes and Schanzenbach (2011) demonstrate that the effects of access to Food Stamps on birth weight are most apparent in the third trimester. By contrast, Almond and Mazumder (2011)'s study of Ramadan fasting finds that the effects on birth weight are not statistically different across different months of pregnancy, and the coefficients are individually significant for exposure in months 1, 2, 5, and 7. Unfortunately, Hoynes, Schanzenbach and Almond (Forthcoming)'s

Most relevant to our paper, however, is the literature that attempts to isolate the effects of psychological stress. Here, again, the evidence is quite inconclusive. Studies exploiting various extreme shocks stemming from natural disasters and terrorist attacks offer varying results.[7] Importantly, Sandra E Black, Paul J Devereux and Kjell G. Salvanes (2016)—the only other study to examine the impacts of *in utero* exposure to maternal bereavement—find that the impacts on birth outcomes are very similar across different trimesters of exposure.

Finally, given the relative dearth of evidence on the relationship between *in utero* shocks and later life mental health, it is hard to determine what pattern one should expect. Almond and Mazumder (2011) find that Ramadan fasting in the first month of pregnancy has a statistically significant effect on mental disabilties in older age, while Adhvaryu, Fenske and Nyshadham (2014) do not analyze differences in exposure across gestational age. Malaspina et al. (2008) show some differential impacts of exposure to the Arab-Israeli War on schizophrenia across months of pregnancy (strongest effects in months two and three), but find no statistically significant differences across trimesters.

Thus, we believe that the existing literature does not provide a clear picture of whether we should expect *in utero* exposure to maternal stress to have differential effects across gestational age, and hope that our analysis of this issue can contribute to the current evidence.

Differential Effects With Respect to the Severity of Stress Exposure Fourth, throughout the paper, we explore differential effects of exposure to maternal stress with respect to the intensity of stress exposure, as captured by the distance in the family tree between the mother and the passing relative.

In contrast with the abundance of studies estimating differential effects across gestational age at the time of shock, the existing literature provides relatively little guidance on whether we might expect to see heterogeneous effects with respect to the intensity of the shock. To the best of our knowledge, only a few existing studies analyze a range of shocks of the same type but of differential intensity.[8] Most closely related to our paper, Aizer, Stroud and Buka (Forthcoming) explore potential non-linearities in the effect of stress

work on the long-term effects of early-life access to Food Stamps does not explore differences in effects across gestational age. When it comes to the literature on environmental shocks, studies on the impacts of radiation exposure consistently find the largest damaging effects on cognitive ability in months 3 and 4 of pregnancy, during a particularly sensitive period of fetal brain development (Almond, Edlund and Palme, 2009; Black et al., 2013). On the other hand, Bharadwaj et al. (2014)'s work on the effects of air pollution on fourth grade test scores finds statistically significant effects of similar magnitudes in both the first and third trimesters in a disadvantaged sub-sample. Due to data constraints, Isen, Rossin-Slater and Walker (Forthcoming) are unable to explore differential effects across gestational age in their analysis of the impacts of air pollution on long-run earnings.

[7] For instance, Currie and Rossin-Slater (2013)'s analysis of hurricanes does not find any statistically different effects across trimesters of exposure. Similarly, Mansour and Rees (2012) show that the impacts of exposure to the Arab-Israeli war are similar across the different months of pregnancy. On the other hand, Eskenazi et al. (2007), Camacho (2008), and Torche (2011) find the strongest effects in the first trimester when analyzing the September 11th terrorist attacks, landmine explosions, and a large earthquake, respectively.

[8] There is more evidence if we compare *across* studies from different contexts. For example, when it comes to malnutrition, *in utero* exposure to the 1959-1961 Chinese famine (Almond et al., 2010) is likely associated with a more severe level of nutritional deprivation than exposure to regular fasting under Ramadan (Almond and Mazumder, 2011). However, differences in effects across these two studies cannot be entirely attributed to heterogeneous treatment effects with respect to the intensity of the *in utero* shock; there are many other factors that are different across the two contexts. In light of this issue, we view the fact that our methodology permits a detailed exploration of differential effects with respect to the intensity of shock *in the same context* as a contribution.

by separately analyzing different quartile ranges of the maternal cortisol distribution. Interestingly, the effects on birth outcomes do not vary with the severity of stress exposure. By contrast, the adverse impacts on cognition—captured by child IQ at age 7 and educational attainment—are the largest for the most severe stress; in fact, the effects on cognitive outcomes are not statistically significant in the linear specifications, but are instead driven entirely by the highest quartile of the maternal cortisol distribution. This evidence suggests that mental health and cognition outcomes may be more sensitive to the severity of stress exposure than birth outcomes.[9] Medical research supports the conjecture that adverse impacts on mental health require a very high exposure to the stress hormone cortisol. The relationship between cortisol and cognitive function is believed to be non-linear: while exposure to lower levels of the stress hormone is not deemed harmful, a range of adverse mental conditions have been associated with excessive exposure to the stress hormone.[10]

C Analyzing the Correlation between Treatment and First Parity

We explored the correlation between treatment and first parity births in detail, and conclude that it is mechanically driven by differential seasonality in conceptions by parity that coincides with a seasonal pattern in relative deaths. In particular, Appendix Figure C1a plots the distribution of months of conception by parity. We see that first parity births are more likely than second parity births to be conceived during October-April (i.e., the winter months in Sweden). By contrast, second parity births are more likely than first parity births to be conceived in May-September (i.e., the summer months). Appendix Figure C1b plots the distribution of the relatives' months of death in our sample, showing that relatives are more likely to die in the winter months than in the summer months. Put differently, relatives are more likely to die in the same months when first parity births are more likely to be conceived, which leads to a mechanical correlation between treatment—death during pregnancy—and first parity. Appendix Figures C1c and C1d show that the same seasonal patterns of birth by parity and of death are present in the entire Swedish population (using all births and deaths between 1969 and 2009).[11]

Appendix Figure C2 plots histograms of the distribution of the distance in days between the relative's death date and the child's conception date for the whole sample and separately by first and second parity. The graphs show that the distribution of this distance is relatively uniform for first parity births in our

[9] Aizer, Stroud and Buka (Forthcoming)'s finding that the impacts on birth outcomes do not vary with the severity of stress exposure is broadly consistent with Currie and Rossin-Slater (2013)'s analysis of hurricanes. For a range of close distances to the path of the hurricane, they find that the estimated impacts are relatively flat; the impacts only fade at larger distances with mild exposure.

[10] In humans, excessive cortisol exposure *in utero* is associated with impairment of brain development (see e.g., Yu et al. (2004)) and with poor mental and motor development (see e.g., Huizink et al. (2003)).

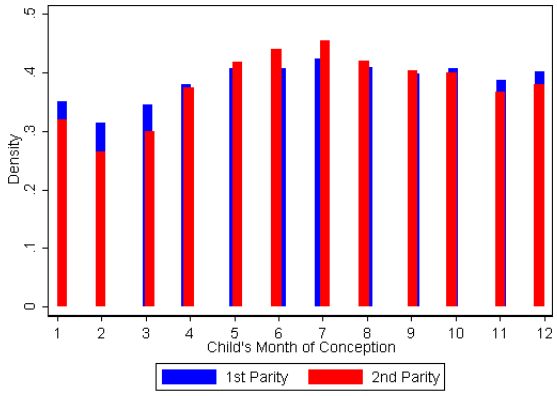
[11] The differential seasonality of births by parity arises from a financial incentive for tight child spacing in Sweden, which is often referred to as the "30 months rule" (Sundström and Stafford, 1992). This incentive stems from the structure of parental leave benefits: a mother who has a second child within 30 months of the birth of her first child is eligible to receive a parental leave benefit that is determined based on her earnings before the birth of her *first* (and not second) child. Since many mothers reduce labor force participation and earnings after the birth of their first child, having a second child within the 30 month window usually leads to a higher benefit. The seasonal pattern of deaths is attributed to exposure to cold weather in the winter months.

sample. However, there are "missing" observations during the first half of the pregnancy among second parity births, consistent with the fact that second parity births are less likely to experience the death of a relative during early pregnancy due to the seasonal patterns discussed above.[12] To address this issue, all of our analyses include month of conception and parity fixed effects, and we show that our results are also robust to the inclusion of parity×month of conception interactions in Online Appendix D. Moreover, we demonstrate that our results remain strong when we limit our sample to first parity births only, which, as noted above, exhibit a relatively uniform distribution of the distance between the relative death date and the child's date of conception.

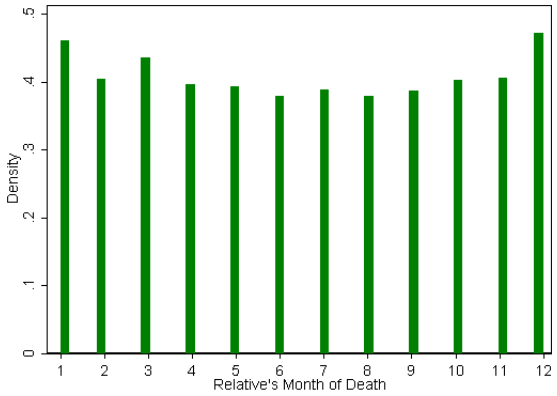
[12]Distributions for third and higher parity births are similar to the distribution for first parity births. Only second parity births exhibit the "missing observations" pattern.

Figure C1: Distributions of Month of Conception by Parity and Relatives' Months of Death

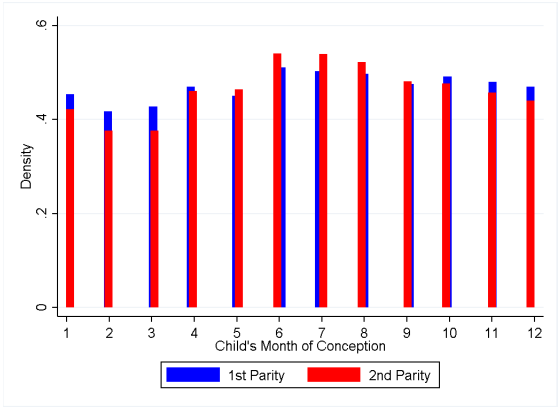
(a) Our Sample: Child's Month of Conception



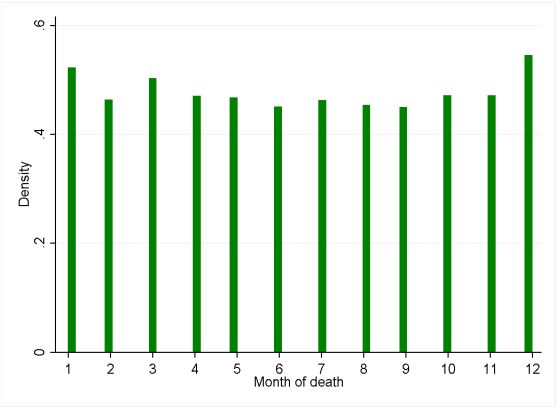
(b) Our Sample: Relative's Month of Death



(c) Population: Child's Month of Conception



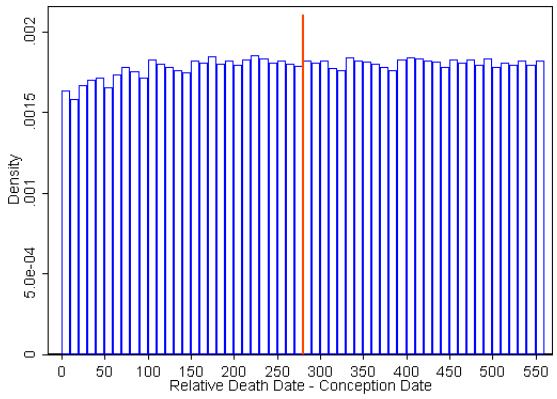
(d) Population: Month of Death



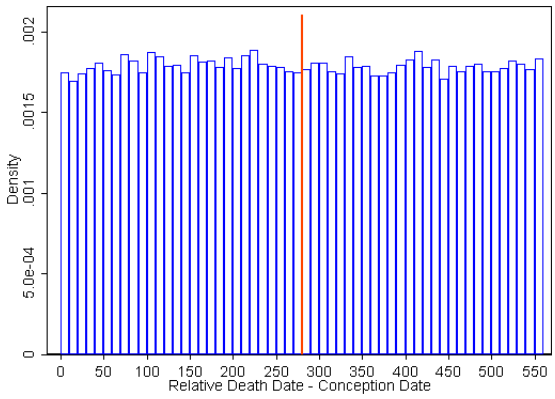
Notes: Sub-figure (a) plots the distributions of the month of conception by parity. Sub-figure (b) plots the distribution of the relative month of death in our sample. Sub-figure (c) plots the distributions of month of conceptions by parity in the entire population. Note that, because we only have information on the date of birth, but not the date of conception, for the entire population, this graph is made assuming that the date of conception is 9 months before the date of birth. The sample includes all births in Sweden between 1969 and 2009. Sub-figure (d) plots the distribution of months of death in the entire population. The sample includes all deaths in Sweden between 1969 and 2009.

Figure C2: Distribution of Relative Death Dates Around Child's Expected Birth Date

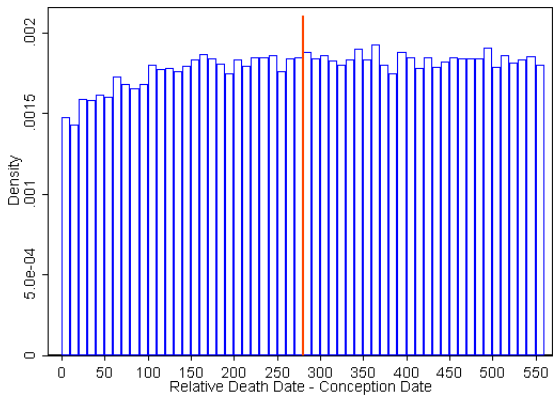
(a) All Observations



(b) 1st Parity



(c) 2nd Parity



Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. The graphs plot histograms of the distribution of the distance in days between the relative death date and the child's conception date. The vertical red line in each graph depicts the expected birth date at 280 days post-conception.

D Supplemental Results

Two-Stage Least Squares Models As described in Section III, our key treatment variable is an indicator for a relative's death occurring between the child's date of conception and the *expected* date of birth at 280 days after conception. However, we can also use this variable to instrument for exposure to death before the child's *actual* date of birth. Appendix Table D1 presents results from two-stage least squares (2SLS) specifications for our main outcomes of interest. As the instrument (relative death before expected birth date) is different from the actual exposure variable (relative death before actual birth date) for only about 1 percent of the individuals in our data, the first stage is very strong with a coefficient of around 0.97. The 2SLS results are quite similar to the main ones we present above.

"Exogenous" and Unexpected Deaths The reliability of our results rests on the assumption that the timing of relative death within a narrow time frame surrounding the expected date of birth is uncorrelated with other factors that may affect child outcomes. We have already shown that this timing is generally uncorrelated with a variety of observable parental characteristics, and that there are no placebo effects on older siblings' birth outcomes. Now, we also explore the sensitivity of our findings to sample limitations based on causes of death that are determined to be more exogenous than others.

More specifically, we turn to the work of Jérôme Adda, Anders Björklund and Helena Holmlund (2011), who study the effect of parental death around age 18 on children's educational and labor market outcomes in Sweden. To find plausibly exogenous causes of deaths, Jérôme Adda, Anders Björklund and Helena Holmlund (2011) test for a placebo correlation between a death occurring after an outcome is determined. So, for example, a death occurring shortly after age 18 cannot affect scores on a cognitive test taken at a younger age. They determine that the following causes of death pass this exogeneity test: endocrine and metabolic diseases, accidents, and other causes.[13] Appendix Table D2 presents results for our main outcomes where we limit the sample to only these three causes of death. Although we lose some power with the sample size reductions, the results are qualitatively similar to the main ones presented above.[14]

We also study plausibly unexpected causes of death by focusing on relative deaths from cardiovascular conditions (i.e., heart attacks) and instantaneous deaths from accidents in Appendix Table D3. Again, results remain qualitatively similar to our main ones (although both the point estimate and the standard errors are larger), suggesting that anticipation of relative deaths is unlikely to substantially bias our estimates.

Heterogeneity by Proximity of Mother to the Relative So far, we have used the closeness of the deceased relative to the mother on the family tree as a proxy for the severity of stress. Alternatively, one could imagine using the geographical distance between the relative's home and the mother's home to measure

[13] Other causes are all causes except infectious and parasitic disease, neoplasms, endocrine and metabolic diseases, mental and behavioral disorders, circulatory system, respiratory system, digestive system, accidents, suicides and homicides.

[14] We unfortunately cannot replicate the method used by Adda, Björklund and Holmlund (2011) to determine which causes of death are exogenous in our sample. To do this, we would need to have a comparison group of children who do not experience a relative death surrounding the time of their birth. However, our sample contains only individuals who experience a relative death within a limited time frame of childbirth.

"closeness". However, physical proximity to a relative may not only capture the closeness between the mother and the relative, but also the closeness of the *child's* relationship with the relative. As a consequence, post-natal stress from bereavement experienced by the child may be greater when the relative lives nearby (e.g., the death of a frequently-visiting grandmother who lived close to the child may be a bigger shock if it happens after birth than before). In this case, comparing *in utero* with post-natal deaths would lead to an underestimate of the effect of pre-natal stress. Consistent with this story, when we explore the heterogeneity in effects by the physical proximity of the mother to the deceased relative in Appendix Table D4, we see somewhat stronger effects for deaths of relatives who lived in different municipalities than the mothers.

Inheritances and the Severity of Stress We find that some of the adverse mental health effects arise when the deceased is a close relative of the expectant mother (such as her parent or sibling), but not when we consider deaths of other more distant relatives (namely, grandparents). As discussed above, we interpret this difference as resulting from varying degrees of emotional stress associated with the relative's passing. An alternative interpretation is that the adverse effects are equal, but that a grandparent's death entails a larger income transfer to the family than the death of other closer relatives. Such an income effect could assuage any adverse effects of stress associated with the passing of a grandparent.

To shed light on this alternative interpretation, three sources of income are relevant: bequests, generation-skipping transfers, and life insurance payouts. Appendix Table D5 displays these three sources of income following the death of a parent and grandparent, respectively, for the universe of deaths in Sweden occurring from 2002 to 2005.[15] The three leftmost columns display the average amount in SEK in each class of recipients, i.e., *not* the average amount conditional on the amount received being greater than zero. The rightmost column displays the sum of the three income classes.

Column 1 shows the average amount received as inheritance following the death of a relative: SEK 30,000 ($4,560) from a parent and SEK 7,000 ($1,064) from a grandparent.[16] The second relevant possibility to receive income in conjunction with a grandparent's passing is through a generation-skipping transfer. Column 2 shows that the unconditional mean of the generation-skipping transfer to grandchildren is SEK 32,000 ($4,864), an amount roughly similar to the unconditional average inheritance from a parent. While these numbers are averages based on the entire population rather than our sample alone, and while inheritances and generation-skipping transfers only occur for a strict subset of all deaths, these statistics indicate that inheritances and generation-skipping transfers together are likely not much larger when a grandparent dies than when a parent dies. Finally, column 3 shows that insurance payouts are small and uncommon. Together

[15] We display average amounts for the universe of deaths in Sweden—and not only for our sample—because the bequest data are not linked to our dataset. Moreover, bequests data exist for the years 2002 to 2005 only. We do not observe bequests or life insurance payouts from sibling deaths.

[16] Inheritance from a parent is far more common than inheritance from a grandparent. This is understandable in light of the fact that, in the absence of a will, an individual only inherits from her grandparent if her own parents are deceased. Moreover, less than 20 percent of all deceased in Sweden write a will; further, writing a will only enables transfer of 50% of the assets, while the remainder must be allocated according to the above-mentioned inheritance rules. These amounts presented in the table, however, represent averages across all spouses, children, or grandchildren of all deceased individuals, i.e., the table displays the unconditional amounts.

these facts suggest that losing a grandparent does not entail a larger positive income effect than losing other (closer) relatives.

Addressing the Correlation Between Treatment, Parity, and Foreign-Born Mothers As discussed in Section III and in detail in Online Appendix C, we find that our treatment variable—death during pregnancy—is statistically significantly correlated with two characteristics, child parity and the mother's place of origin. We conduct several analyses to show that these correlations are not driving our main results.

First, Appendix Table D6 presents the results for our main outcomes of interest separately by first and second parity births. Given that second parity births exhibit "missing" observations in the distribution of the distance between the relative's death date and the child's conception date, it is reassuring that our results remain strong when we only focus on first parity births in Panel A.

Second, to account for the differential seasonality in births by parity, we estimate specifications that control for parity×month-of-conception fixed effects in Appendix Table D7, with results similar to the main ones presented above.

Third, in Appendix Table D8, we drop foreign-born mothers as this group exhibits a highly skewed distribution of the distance between the relative's death date and the child's conception date. Our results remain largely unchanged.

Table D1: 2SLS Effects of Relative Death *In Utero* on Main Outcomes

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death Before Childbirth	0.00404*** [0.000651]	0.00635*** [0.000862]	0.00361*** [0.000917]	0.00667*** [0.00213]	0.00888** [0.00372]	0.00940** [0.00447]
Mean, dept. var	0.0320	0.0494	0.0575	0.0238	0.0666	0.111
First Stage Coef.	0.971	0.971	0.971	0.972	0.973	0.973
First Stage F-Stat	4732830.8	4745576.4	3688443.6	321520.3	358656.9	358656.9
Obs.	288294	289044	231398	19604	21715	21715

Note: See tables 1 and 2 for more information on the sample and controls. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. In these regressions, the explanatory variable is an indicator for the death of a relative occurring between a child's date of conception and date of birth. It is instrumented by an indicator for the death of a relative occurring between a child's date of conception and his *expected* date of birth (at 280 days post-conception). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.

Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D2: Effects of Relative Death *In Utero* on Main Outcomes: "Exogenous Deaths"

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death During Pregnancy	0.00176	0.00687**	0.00457*	0.0185**	0.0159	0.0188
	[0.00207]	[0.00270]	[0.00276]	[0.00739]	[0.0121]	[0.0145]
Mean, dept. var	0.0323	0.0506	0.0564	0.0288	0.0680	0.111
Obs.	34349	34447	28560	2502	2352	2352

Note: See tables 1 and 2 for more information on the sample and controls. The sample is further limited to children of mothers who experience a relative death from causes determined to be exogenous in Adda, Björklund and Holmlund (2011). These are deaths from endocrine and metabolic causes, accidents, and other causes. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D3: Effects of Relative Death *In Utero* on Main Outcomes: "Sudden Deaths"

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death During Pregnancy	0.00341***	0.00692***	0.00362***	0.0117***	0.00898*	0.00779
	[0.000881]	[0.00131]	[0.00132]	[0.00359]	[0.00514]	[0.00679]
Mean, dept. var	0.0328	0.0502	0.0580	0.0247	0.0685	0.111
Obs.	148477	148836	117919	7419	10791	10791

Note: See tables 1 and 2 for more information on the sample and controls. The sample is further limited to children mothers who experience a relative death from "sudden" causes—cardiovascular causes (i.e., heart attacks) and instantaneous deaths from accidents. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.

Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D4: Effects of Relative Death *In Utero* on Main Outcomes: By Whether Relative Lived in Same Muni. as Mother

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Panel A: Same Muni as Mother						
Death During Pregnancy	0.00404***	0.00687***	0.00219	0.00746**	-0.0000843	0.00119
	[0.00107]	[0.00146]	[0.00167]	[0.00310]	[0.00530]	[0.00714]
Mean, dept. var	0.0343	0.0519	0.0600	0.0233	0.0681	0.110
Obs.	113033	113338	86790	9103	9891	9891
Panel B: Different Muni than Mother						
Death During Pregnancy	0.00400***	0.00577***	0.00453***	0.00620**	0.0159***	0.0149**
	[0.000796]	[0.000987]	[0.00110]	[0.00291]	[0.00533]	[0.00645]
Mean, dept. var	0.0305	0.0478	0.0560	0.0242	0.0654	0.111
Obs.	175299	175744	144605	10502	11872	11872

Note: See tables 1 and 2 for more information on the sample and controls. In Panel A, the sample is limited to children of mothers whose relatives lived in the same municipalities as them. In Panel B, the sample is limited to children of mothers whose relatives lived in different municipalities than they did. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D5: Inheritances, Generation-Skipping Transfers, and Life Insurance Payouts

	Average amount (SEK), specific transfer class			Total amount (SEK)
Deceased relative	Inheritance	Generation-skipping transfer	Life Insurance Payout	All classes
Parent	30000	7000	1500	38500
Grandparent	7000	32000	500	39500

Note: The table presents average amounts of the three sources of income following the death of a relative—inheritances, generation-skipping transfers and life insurance payouts—from a deceased parent and grandparent, respectively. For each income type, the three leftmost columns displays the average amount in Swedish Krona (SEK) in each class of recipients, i.e., *not* the average amount conditional on the amount received being greater than zero. The rightmost column displays the sum of the three income classes.

Table D6: Effects of Relative Death *In Utero* on Main Outcomes: By Parity

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Panel A: 1st Parity						
Death During Pregnancy	0.00504***	0.00753***	0.00488***	0.0101***	0.00982	0.0123*
	[0.000993]	[0.00131]	[0.00144]	[0.00351]	[0.00697]	[0.00739]
Mean, dept. var	0.0396	0.0585	0.0713	0.0259	0.0702	0.112
Obs.	142902	143309	117411	7910	7651	7651
Panel B: 2nd Parity						
Death During Pregnancy	0.00191**	0.00474***	0.00120	-0.00125	0.00787	0.0128*
	[0.000912]	[0.00115]	[0.00137]	[0.00356]	[0.00557]	[0.00750]
Mean, dept. var	0.0224	0.0373	0.0417	0.0205	0.0622	0.105
Obs.	99669	99898	79834	7020	8667	8667

Note: See tables 1 and 2 for more information on the sample and controls. In Panel A, the sample is limited to 1st parity children. In Panel B, the sample is limited to 2nd parity children. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D7: Effects of Relative Death *In Utero* on Main Outcomes: Control for Parity by Month of Conception FE

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death During Pregnancy	0.00393***	0.00618***	0.00352***	0.00654***	0.00863**	0.00919**
	[0.000632]	[0.000839]	[0.000890]	[0.00208]	[0.00369]	[0.00441]
Mean, dept. var	0.0320	0.0494	0.0575	0.0238	0.0666	0.111
Obs.	288337	289087	231398	19605	21763	21763

Note: See tables 1 and 2 for more information on the sample and controls. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. These regressions also control for a full set of interactions between parity indicators and month of conception indicators. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.

Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table D8: Effects of Relative Death *In Utero* on Main Outcomes: Drop Foreign-Born Mothers

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any ADHD 9-11	(5) Any Anx 34-36	(6) Any Dep 34-36
Death During Pregnancy	0.00393***	0.00627***	0.00347***	0.00678***	0.00869**	0.00921**
	[0.000630]	[0.000822]	[0.000914]	[0.00215]	[0.00376]	[0.00452]
Mean, dept. var	0.0317	0.0492	0.0574	0.0240	0.0661	0.111
Obs.	282581	283307	226674	18579	21297	21297

Note: See tables 1 and 2 for more information on the sample and controls. In column (3), the sample is further limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). In columns (4)-(6), the sample is further limited to children of mothers who experience the death of a parent or sibling. The sample drops children of mothers who are foreign-born. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in Online Appendix E.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

E Definitions of Health-Related Outcomes

Diagnosis (ICD) codes For all children and siblings, we get obtain comprehensive inpatient medical records for all visits associated with the following diagnosis codes (ICD-10):

- Psychological disease (F00-F99)
- Suicide (X60-X84)
- Type II diabetes (E10-E14)
- Obesity (E65-E68)
- Heart disease (I20-I25, I30-I52)
- Neoplasms (C00-D48)
- Cushing's syndrome (E24)
- Perinatal (P00-P96)
- Deformations at birth (Q00-Q99)
- Drug and alcohol abuse (Z72)
- Thyroid-related issues (E00-E07)
- External cause (S00-T98, V01-Y98)
- Sexually transmitted disease (A50-A64)
- Stroke (I61-I64)

For earlier years, the analogous ICD-9 and ICD-8 codes are applied.

Prescription drug (ATC) codes Prescription drugs are classified according to the Anatomical Therapeutic Chemical Classification System (ATC). To associate certain prescription drugs to mental health diagnoses, we use the classification system below, employed by the National Board of Health and Welfare in Sweden (Socialstyrelsen, 2012):

- Mental health (all): ATC-code begins by "N."
- ADHD: ATC-code begins by "N06BA"
- Bipolar disease: ATC-code begins by "N05AN01"
- Psychotic conditions: ATC-code begins by "N05A," but excluding "N05AN01"

- Depression: ATC-code begins by “N06A”
- Anxiety: ATC-code begins by “N05B”
- Sleeping disorders: ATC-code begins by “N05C”
- Addiction: ATC-code begins by “N07”
- Parkinson: ATC-code begins by “N04”

- Diabetes: ATC-code begins by “A10.”
- Obesity: ATC-code begins by “A08AB01" or "A08AA10.”
- Cushing’s syndrome: ATC-code begins by “J02AB0.”
- Neoplasm: ATC-code begins by “L01.”
- Thyroid: ATC-code begins by “L01.”

F Stress *In Utero*: More References

While it is well established that malnutrition in pregnant women affects the unborn child, the mechanism through which maternal adversity impacts the child is not well understood. One prominent theory proposes a neuro-scientific mechanism in which stress plays a key role (Jaddoe, 2006). It is hypothesized that nutritional restrictions inhibit the development of a placental enzyme that is required to convert the stress hormone cortisol into inactive cortisone. As a consequence of maternal malnutrition, the fetus is thus exposed to excessive amounts of cortisol in utero. Overexposure to cortisol, in turn, is believed to lead to a reprogramming of the hypothalamic-pituitary-adrenal axis (HPA), which could lead to impaired fetal development and worse health in adult age (Jaddoe, 2006).

Substantial evidence from preclinical laboratory studies show that the offspring of prenatally stressed animals displays over activity and impaired negative feedback regulation of the HPA, alternations which have been linked to a diverse spectrum of psychopathology, including schizophrenia and depression (M., 2001; Huizink AC, 2004; Kofman, 2002). Nevertheless, in humans, evidence of an explicit link between maternal stress and long-term disturbance in the HPA is scarce (Kapoor A and Matthews, 2006). A significant association between measures of prenatal anxiety and individual differences in salivary cortisol has been established in a sample of 10-year-old children from the Avon Longitudinal Study of Parents and Children (ALSPAC)(O’Connor TG, 2005). In another sample, young children whose mothers exhibited higher levels of morning cortisol during pregnancy were found to show higher levels of salivary cortisol (Gutteling BM, 2004, 2005). These results suggest that prenatal anxiety can have lasting effects on HPA functioning in the child, and are consistent with the hypothesis that that prenatal anxiety might constitute a mechanism for an increased vulnerability to psychopathology in children and adolescents.

In humans, researchers have also documented an association between antenatal maternal stress and an increased risk of obstetric complications such as preterm birth, low birth weight, and fetal distress (Crandon, 1979; Lou HC, 1994; Wadhwa PD, 1993), negative reactivity to novelty (Davis EP, 2004), an increase in neonatal crying (Rieger M, 2004), behavioral and/or emotional abnormalities at young ages (O'Connor TG, 2002), a depressed Apgar score (Crandon, 1979; Ponirakis A, 1998), and a higher incidence of ADHD during childhood (Van den Bergh BRH, 2004, 2005). Moreover, in a rare study of the association between maternal stress and non-health related outcomes, researchers established that maternal depression at mid-gestation was associated with a small but significant increase in violent crime in Finland (MakiP, 2003). While these studies establish correlations between antenatal maternal stress and outcomes later in life, the causal link is not clear. The studies assess the level of maternal anxiety and stress using the mother's own rating of symptoms, and some studies also included cortisol measures or an appraisal of recently experienced adverse life events such as divorce, job loss, or marital discord. Because these measures may not be independent of unobserved factors that affect child outcomes, maternal stress may be endogenous.

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