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MATERNAL MENTAL HEALTH AND CHILD OUTCOMES: EVIDENCE FROM A NATURAL EXPERIMENT

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Maternal mental health and child outcomes:
Evidence from a natural experiment

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Abstract:

Maternal mental health is often considered a key factor in child development, yet we know little about its importance in adolescence, and almost nothing about its causal effect at this age. My paper addresses this gap, by testing the consequences of experiencing poor maternal health in mid-adolescence (16-19 years old), on three outcomes: the risk of disconnection, mental health and antisocial behavior, measured at age 20. In a first step the paper demonstrates that maternal mental health problems experienced in adolescence are as important as maternal mental health problems experienced at earlier stages in childhood for these three outcomes. In a second step the paper shows that part of the observed relationship represents a causal effect, as mother's mental health problems increase the likelihood that a young person becomes disconnected and that he or she develops mental health problems. In combination, my results contribute to the mounting evidence of adolescence as an impressionable period in life and, more specifically, to our understanding of the causal effects of maternal mental health on outcomes among adolescents.

Introduction

Maternal mental health is often claimed to be a key factor in child development, and numerous studies document how poor maternal mental health correlates with negative child outcomes (for reviews, see e.g. Goodman et al, 2011). These studies show that children of depressed mothers are at heightened risk of developing depression and anxiety disorders (Halligan et al, 2007), experiencing impaired growth (Harpham et al, 2005; Patel et al, 2004), displaying behavioral problems (Brennan et al., 2000; Whitaker et al, 2006) and suffering from poor cognitive skills (Brennan et al, 2000). This literature has, however, mainly focused on poor maternal mental health experienced in utero and early childhood, and we know little about the consequences of experiencing mother's mental health deterioration during adolescence, and almost nothing about the causal nature of this relationship.

Understanding the impact of maternal mental health during adolescence is, however, an important undertaking: While it is now largely accepted that the brain undergoes sensitive periods in utero and during the first two years of life (Knudsen et al, 2006), there is blooming evidence that also adolescence is a sensitive period for brain formation. During such sensitive periods, the brain not only develops dramatically, it is also more susceptible to lasting influences from environmental shocks and influences affecting *how* the brain develops during that period. If the brain is particularly sensitive during adolescence, experiencing poor maternal mental health in the teenage years and early adulthood may have detrimental long-term consequences. And if that is the case, we should pay attention to such at-risk teenagers and young adults and target these groups with interventions aimed at counteracting the negative consequences of being exposed to poor maternal mental health.

To improve our understanding of adolescence as a sensitive period, and to address the gap in the existing literature on maternal mental health, my paper tests the consequences of experiencing poor maternal health in mid-adolescence (16-19 years old), on three outcomes: the risk of disconnection, mental health and antisocial behavior, measured at age 20. The paper does so through two empirical steps: In the first step the paper demonstrates the relevance for the three outcomes of maternal mental health experienced in adolescence relative to maternal mental health experienced at earlier stages in childhood. In the second step I test the degree to which selection drives the observed correlation between maternal mental health problems and the three outcomes. I use Danish administrative data in simple linear models as well as in an Instrumental variables-model (IV-model) where I derive exogeneous variation in mother's risk of developing PTSD from a natural experiment. In combination, my results contribute to the mounting evidence of adolescence as an impressionable period in life and, more specifically, to our understanding of the causal effects of maternal mental health on outcomes among adolescents.

Previous research and suggested mechanisms

Mother's mental health is among the most commonly identified childhood risk factors (Cummings & Davies, 1994; Downey & Coyne, 1990), and growing up with a mother who struggles with poor mental health is a strong predictor of a range of negative short- and long-term outcomes.

We may think of (at least) three reasons why we see this relationship. First, mothers and children may share a genetic disposition of and environments that increase the risk of developing mental health problems and experiencing poor outcomes. According to this explanation mother's mental health does not exert a causal effect on the child outcomes, rather the poor mental health coincides with poor child outcomes because of underlying factors conditioning the life course of both the mother and the child.

Second, mother's poor mental health may causally affect the child's risk of experiencing poor outcomes. This could happen, e.g. because the mental health problems are likely to reduce impulse, attention and emotional control, and therefore reduce mother's ability to engage consistently and dedicatedly in productive activities with the child (Ronda, 2016). For the same reasons, the mental health problems may also reduce or destabilize the mother's labor market affiliation, and through this, the household resources of which numerous studies have demonstrated negative consequences for child outcomes (Altree, 2004; Duncan & Brooks-Gunn, 1999). In addition, mental health problems appear to be contagious which suggests that mother's mental health problems may directly infect the child: Sterley and colleagues (2018) show, using animal experiments, that stress is contagious through a conditioning of the neurotransmittors that control stress. Human studies find similar evidence that stress and other mental states are contagious, as teacher stress causes stress in school students (Oberle & Schonert-Reichl, 2016), and the well-being of a class mate affects you own well-being (King & Datu, 2017, Engert et al., 2014; Fowler & Christakis, 2008; Rosenquist et al, 2013).

A third reason for the observed correlation could be reverse causation, where the poor child outcomes negatively affect mother's mental health.

In sum, various plausible and tested mechanisms suggest that maternal mental health matters for child outcomes and that at least part of the observed correlation reflects causal effects, rather than just shared genetic dispositions of mother and child of experiencing poor mental health and thus negative outcomes. Empirical studies confirm the existence of a causal effect by e.g. demonstrating that also adoptees to mothers with mental health problems experience negative outcomes (Jaffee et al, 2012). Hereby being exposed to mother's mental health problems seem to matter over and above sharing her genes for developing mental disorders.

Mother's mental health during child's adolescence

With this insight, a next important question is whether age at exposure matters: For a little more than a decade, the social sciences have had a strong focus on the early sensitive years, and on the potential for early intervention among high risk children for improving outcomes in later life (Knudsen et al, 2006). In support of this focus existing correlational and causal studies show clear evidence that maternal mental health matters in utero (for reviews see Hartman & Belsky, 2018; Staneva et al, 2015; Suri et al, 2014) and during the first couple of years of childhood (e.g. Allen et al, 2018; Apter-Levi et al, 2016; Côté et al, 2018; Kim-Cohen et al, 2005). i.e. that early exposure to mother's mental health problems matters for later outcomes.

As a supplement to the claim that early childhood represents important and sensitive years is the current quest for also positioning adolescence as a sensitive period. This quest builds on new insights on how the brain develops during the teenage years: Parallel to the social transitions characterizing adolescence, we now know that the brain undergoes significant physiological changes in this period of life. During this period, the brain is particularly plastic, and frequently used neural connections are strengthened, while unused connections are pruned away. The implication of this biological process is that exposure to environmental influences during adolescence are likely to have long term consequences, exactly because these influences will affect what connections are used and what connections remain unused (and thus kept or pruned away). Influences causing stress – i.e. releasing the stress hormone – are found to be particularly damaging, as this hormone is a neurotoxin, that kills brain cells (Blakemore, 2012; Blakemore & Mills, 2013; Curtis, 2015; Fuhrmann et al., 2015).

This biological explanation of why and when conditions experienced in adolescence affect long-term outcomes integrates well with our sociological understanding of what happens in adolescence and of the importance of parental support during this period in life. Adolescence represents one of the most significant and all-encompassing personal and social transition periods in life, where the person goes from being a minor in the family home to becoming a (young) independent adult with rights and responsibilities (Curtis, 2015). The success of this transition relies on personal resources and on resources in the immediate network, in which parents often and still constitute a significant part: Arguably, adolescents spend less time with their parents than infants, toddlers and preschoolers, and they ascribe more value to the inputs from peers than from their parents (Albert et al, 2013). Also, they are now highly susceptible to broader cultural and social influences (Choudhury, 2010; Fiske, 2009; Larson & Richards 1991; Larson et al., 1996; O'Brien & Bierman, 1988). Still, we may expect parents to not only shape life outcomes of their children during childhood; parental support, resources and attention during adolescence is also likely to be the determining factor in securing proper transitions to adulthood and good subsequent outcomes, exactly because the attentive, resourceful parent will act as a stable factor in this period where everything else is revolving around and within the young person (Beardslee, 1986 in Gelfand & Teti, 1990). Here, the stress caused by

poor parental support, resources and attention is a likely threat to the transition and a likely negative environmental influence with likely negative effects on the developing teenage brain.

The empirical evidence on the importance of poor maternal mental health experienced during adolescence is, however, small. There is a substantial amount of research on outcomes of adolescents who have experienced poor maternal mental health at earlier stages in childhood (e.g. Allen et al., 2018; Sanger et al, 2015), but only little research on the consequences of experiencing poor maternal mental health during adolescence (exceptions include Hay et al, 2010). Furthermore, there is limited evidence of whether exposure during adolescence matters vis-à-vis exposure at other periods in life. But because of the persistent and often lifelong nature of mental health problems, it is crucial that we test for exposure at different ages simultaneously because only then will we know if the parameter that we estimate for exposure at any age does not also reflect maternal mental health problems experienced at other ages. A small group of studies test the importance of the timing of maternal depression on child outcomes (see e.g. Bureau et al., 2009; Mikonen et al., 2016; Halligan et al., 2007; Hammen & Brennan, 2003; Hay et al., 2008; Hay et al., 2010; Pawlby et al (2009), but only a handful show results for exposure during adolescence, net of earlier exposure, and they tend to rely on very small samples. Thus, the evidence for deciding whether exposure to poor maternal mental health during adolescence matters is very small. Last, while there are solid causal studies on the impact of mother's mental health during her pregnancy or during the child's early years (see e.g. Ronda, 2016; Torche, 2018), and other studies inform us that the availability of other parental resources during adolescence matters for short and long-term youth outcomes (examples include Wildeman & Andersen, 2017; Chevalier et al, 2013; Dickson, et al., 2016), studies on the causal effect of maternal mental health during adolescence are particularly absent. Without knowledge of the causal nature of the relationship, we are still far from understanding whether we should help and how to help adolescents of mothers with mental health problems.

In combination existing – but lacking - empirical evidence, along with the theoretical/biological claim that adolescence is a sensitive period motivates further empirical studies on the link between mother's mental health experienced in adolescence and later outcomes. My study contributes first, by presenting descriptive evidence that poor maternal mental health experienced during adolescence matters for later outcomes vis-à-vis early exposure, and second, by providing evidence that not all this correlation is driven by selection.

Analytical approach

I provide the contribution through two empirical steps. First, I will demonstrate how mother's mental health measured at different ages throughout childhood is correlated with outcomes reflecting human capital acquisition, mental health and criminal activities, measured at age 20. This demonstration supports my claim,

that we cannot focus exclusively on the early years and early exposure to poor maternal mental health when we wish to understand the full implications of mother's mental health on child outcomes. Second, I will test whether selection bias drives this correlation or if it reflects a causal effect. For this purpose, I first test the implications for the correlation of controlling for observable confounders, and second, I use an IV-setup to get (plausibly) causal estimates of the effect of mother's mental health on three child outcomes.

Sample

This study uses administrative data from Statistics Denmark. All residents of Denmark have a unique personal identification number (equivalent to a U.S. social security number) that identifies the resident in all major transactions with public authorities and private institutions such as banks. Statistics Denmark collects large parts of the information registered by this personal identification number and makes these data available for research purposes. The data are available dating back to 1980, and are suited for my study, because they allow linking of family members. They also contain rich information on health issues, prescription medicine, labor market histories and other characteristics.

Due to different types of data restrictions, the two empirical steps described above rely on two different analytical samples. The two samples are restricted so that they reflect the need for sufficient information on mother's mental health at relevant points in childhood, and the need for sufficient information on outcomes in (early) adulthood. For step one, I select the cohort born in 1992 (N=69,292), which is the cohort in my data, for whom I have information on mother's mental health at most ages, and where I still also have relevant outcome measures measured at (early) adulthood (age 20) in my data: The mental health register starts in 1995, and my data on criminal activities ends in 2012. With the 1992 cohort, I therefore have information on mother's mental health from when the child turns three years old in 1995 and onwards, and I can measure criminal activities (as well as human capital acquisition and mental health) at age 20. This is sample A. I also use sample A for the first part of the second step. For the second part of the second step where I use the IV-model, I focus exclusively on maternal mental health among cohorts born in 1985-1988 (N=236,957), who lived in Denmark in 2004. The natural experiment that I exploit in the IV-setup takes place in late 2004, where these cohorts are between age 16 and 19, and thus in mid-adolescence. This is sample B.

Empirical models

In the first step I use simple OLS models to study the correlation between my three outcomes – human capital acquisition, child mental health and crime - and exposure to poor maternal mental health at four ages, during 1) the preschool years (age 3-6), 2) the first years of school (age 7-12), 3) early adolescence (age 13-15), and 4) mid-adolescence (age 16-19). I present results from 7 models for each of the three outcomes, where the first model includes only indicator a (exposure during the preschool years), the second includes

indicators a and b, etc. This strategy aims first, to show the correlations between exposure to poor maternal mental health at a given age and the outcomes in focus, and second to demonstrate whether and how the correlation between poor maternal mental health at a given age and the outcome in focus, is confounded by poor mental health at another age.

In the second step I first use simple OLS models to demonstrate the degree to which observed/known confounders drive the correlation between poor maternal mental health experienced in adolescence and my three outcomes. I include groups of relevant confounders in a stepwise process to provide evidence of what confounders matter the most. Second, and based on the results from these OLS models that show that selection drives at least part of the link between maternal mental health and child outcomes, I use an IV-model to estimate plausible causal effect of maternal mental health on child outcomes. I choose the IV-model over other models for causal inference such as e.g. sibling models, twin and adoption studies that are often used in studies on maternal mental health on child (for a review, see Gjerde et al, 2017; Jaffee et al, 2013; Tully et al, 2008), because this model solves problems pertaining to reverse causality.

The IV-model

For my IV-model I obtain exogenous variation in maternal mental health from a dramatic event happening on November 3rd 2004 in Seest, a suburb of the Danish city, Kolding. On that day, two employees of N. P Johnsens Fireworks Factory in Seest dropped a box of fireworks, which caught fire and exploded. The fire spread to the rest of the factory, causing two major explosions (of which the largest caused seismic waves comparable to a magnitude 2.2 earthquake, like the seismic waves measured when the south tower of the World Trade Center collapsed on 9/11). The explosion severely damaged 355 houses, of which half were subsequently uninhabitable. 85 people were injured enough that they had to receive medical treatment, and one person – a firefighter – lost his life in the first explosion. The disaster is perceived to be the largest in Denmark since WW2, and both residents of the evacuated area as well as the larger local community surrounding the fireworks factory was affected by the catastrophe.

Aside from the material damage, reports show reduced mental health among individuals living close to the factory, both short (Elklit & Molin, 2006; Elklit, 2007) and longer term (Elklit, et al., 2006). The worsened mental health shows up as a significant rise in the proportion of individuals diagnosed with Post Traumatic Stress Disorder (PTSD) around the factory in the years following the explosion.

Relying on this knowledge on how the fireworks disaster affected the likelihood of developing PTSD, I use a binary indicator for whether mother lived in the local area (as I will define and discuss later) affected by the explosion in November 2004, as an instrument for mother's mental health in an IV-model. Equations 1 and 2 show the model:

$$mother's\ PTSD_i = \alpha_{ih} + \delta_1 x_i + \theta impact\ area_m + r_i \quad (1)$$

$$child\ outcomes_i = \alpha_2 + \delta_2 x_i + \beta \widehat{mother's}\ PTSD_i + u_i \qquad (2)$$

In both equations, *i* represents the child (*i=1,…N*). Equation 1 forms the first stage, in which the endogenous variable (*mother's PTSD*) is regressed on the vector of exogenous controls x_i and the instrument. Observe that this instrument reflects whether the mother, and not the young person, lives in the impact area at the time of the explosion, as indicated by the subscript *m*, which represents the mother (*m=1,…,N*). The second stage uses the predicted value of the endogenous variable to predict the child outcomes in focus, along with the vector of controls, x_i. Random error terms are r_i and u_i.

The validity of the IV-model rests on four key assumptions. First is the *relevance* assumption which implies that the instrument has a causal effect on the endogenous regressor. Second is the *exclusion restriction*, which means that the instrument affects the outcome only through the endogenous regressor. Third is the *exchangeability* assumption which implies that the instrument is random, and thus uncorrelated with individual level characteristics. Fourth is the *monotonicity* assumption, which implies that there are no "defiers", i.e. individuals who respond opposite to what we would expect given the mechanism activated by the instrument (Lousdal, 2018). I shall discuss the validity of these assumptions in my setup later.

The impact area

To determine whether the mother of the young person lives in the impact area at the time of the catastrophe (i.e. takes the value 1 on the instrument), I use information on place of residence from the registers. The definition of the impact area is, however, not trivial. One definition could include only the evacuated area, which was severely damaged by the explosions. But from other studies on trauma and mental health issues released by terrorist attacks, earthquakes and exploding landmines, we know that not only individuals who are directly physically affected by or an eyewitness to the incident experience the consequences (see e.g. Hansen et al., 2016; 2017). Thus acts of terrorism and similar traumatizing events may have geographically far-reaching consequences, and a disaster like the exploding fireworks factory in Seest is therefore unlikely to only impact the area immediately surrounding the factory.

While the cited studies may justify considering all of Denmark the impact area, it seems likely that stronger effects are observed close to the locus of the event (see e.g. Newman & Hartman, 2017, Enos, 2017). Therefore, I delimit the indicator to take the value 1 for mothers living in municipalities that a) contain or are geographically adjacent to the evacuated area, and which b) contributed to the disaster relief, according to the Danish Emergency Management Agency (Beredskabsstyrelsen, 2005). This way I focus on mental health responses in mothers who were close to the catastrophe, and who may know someone – e.g. a fireman – who aided the relief, or who was directly affected by the explosion. Thus, my indicator takes the value 1 for mothers who live in the municipalities of Kolding, Fredericia, Lunderskov, Vamdrup and Christiansfeld at

the time of the explosion (N_1=415 (1.45 %); N_2=376 (1.44 %); N_3=231 (1.43 %); N_4=307 (1.41 %)).[1] The distance between the main cities of these municipalities and the fireworks factory is 9-15 kilometers, which plausibly seems short enough that the disaster is directly relevant to the inhabitants and their mental well-being.

While this indicator may be imprecise such imprecision will cause only downward bias in the estimate. If the indicator represents a too narrowly defined treatment area – implying that the control areas were also impacted by the disaster – it will cause a downward bias my estimate, because the control areas would then also have elevated post-disaster PSTD levels. Similarly, if it is too broadly defined – implying that only people living in the area immediately surrounding the impact area were affected by the explosion – it would also "just" downwardly bias my estimate, because the less impacted parts of what I define as my impact area would depress the treatment effect. Such downward bias is unfortunate, but of lesser concern than upward bias.

Robustness tests

From reports we also know of negative consequences of the fireworks disaster among children and youth living in the area. The group suffered from flashbacks of the event, from problems with concentrating, and, as their parents, from PTSD (Duch & Elklit, 2008). Estimated differences in outcomes between exposed and unexposed young people may therefore not only reflect mother's mental health response to the disaster but also – given that many adolescents live with their parents - the direct effect of witnessing the disaster on the young person. Thus, to rule out the possibility that results from the IV-model reflect the direct stress response among the adolescents living in the impact area at the time of the disaster (which is a violation of the exclusion restriction), I test whether my results are robust to the inclusion of an indicator of whether the adolescent lived in the same municipality as the mother (and thus was exposed to the same environmental factors as her). I furthermore run the model on a number of subsamples which include only adolescents who did not live in the same municipality as their mother at the time of the explosion. In the first subsample, I exclude all individuals who did not live in the same municipality as their mother at the time of the explosion, to hereby remove the contamination of the effect of mother's mental health deterioration from any direct effect of the explosion on the mental health of the young person him- or herself (N_{B1}=29,383). Not living with one's mother in mid-adolescence is, however, a marker of social disadvantage, and I further split the sample into groups with identifiable reasons for not living with their mother, to understand why the young people did not live with their mother. First, I exclude all individuals from sample B1 who were placed in foster care at the time of the explosion (N_{B2}=26,823), whereby I exclude the relatively more disadvantaged individuals. Second, I include only individuals from B1 with divorced parents, where the likely reason why

[1] According to the municipal borders as they were defined in 2005.

the individual did not live with the mother is that he or she lived with the father (N_{B3}=16,532). Last, I include only individuals from B1 who were away for educational purposes (N_{B4}=22,259). I shall discuss to the degree to which we may expect mother's mental health to also matter in such family constellations.

Measuring mother's mental health

My indicator of mother's mental health relies on information on individual redemption of psycholeptics and psychoanaleptics, which are the psychopharmaceuticals (PP) that general practitioners (GPs) prescribe to treat symptoms of depression, anxiety, severe stress, sleep disorders and neurosis (ATC codes N06A, N05A, N05BA, N05CD and N05CF). For the first empirical step, I construct a binary indicator of mother's redemption of PPs (i.e. whether she has redeemed at least one dose of PP) at each of the four ages (age 3-6, age 7-12, age 13-15, age 16-19). I use the same indicators for the first part of the second empirical step, where I control for confounders. For the second part of the second empirical step, I focus solely on the use of Selective Serotonin Reuptake Inhibitors (SSRI), which is the medication used to treat PTSD.

Medicine consumption is not necessarily a good proxy of mental health problems, and a key concern is the degree of gatekeeping of the access to the medication and whether there is self-selection into use. Importantly, while access to the different types of medication may vary, they are all prescribed by Danish GPs after establishing certain diagnoses of a mental health problems and importantly the medication cannot just be purchased directly from drugstores. Also, to the degree that there is self-selection into use, the mere self-selection may be an indicator of a mental health problem. On these grounds redemption of PPs prescriptions is arguably a useful indicator of mental health.

Outcome variables

The analyses focus on three different outcomes, human capital acquisition, child mental health and crime, all of which reflect important resources for successful transitions into adulthood. My human capital acquisition indicator takes the value 1 if the young person is not in education or employment at age 19 to 20. This indicator reflects the situation where the young person does not engage in human resource acquisition at a time in life, where this type of activity is considered both normal, expected and beneficial. The second indicator takes the value 1 if the young person redeems PP prescription medication at least once at age 20 and reflects his or her mental health resources for making a successful transition into adulthood. The last indicator takes the value 1 if the young person has been sentenced to prison/jail or probation or has been fined at age 20. This indicator captures a very broad definition of crime and is meant as an indicator of a general propensity to antisocial behavior rather than as a strict measurement of crime. Table A1 in the appendix shows the descriptive statistics for sample A and Table A2 in the appendix shows the descriptive statistics for sample B.

Results

Age at exposure

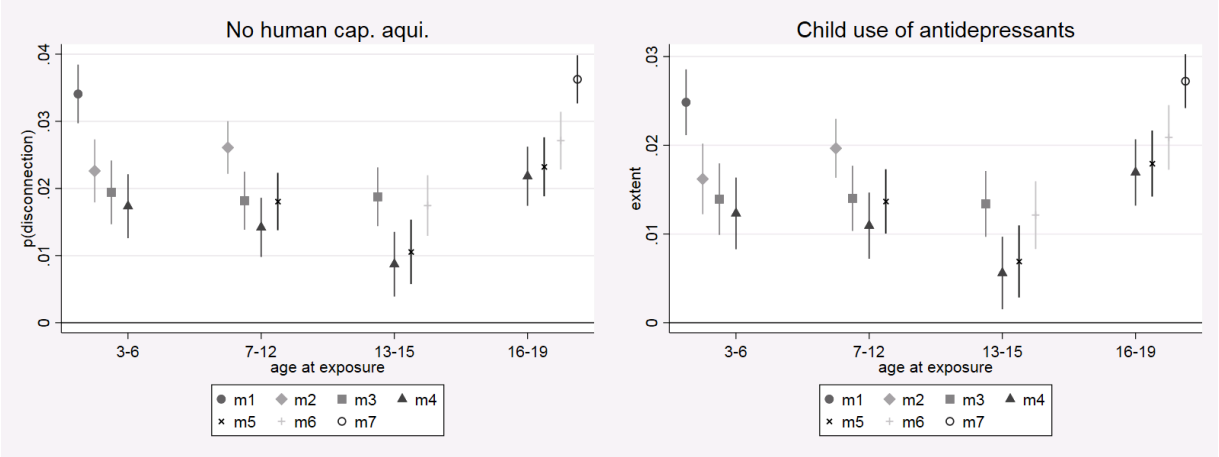
I start by presenting results from the first part of the empirical analysis that addresses the importance of age at exposure to maternal mental health problems. Below is figure 1, with panels A, B and C that show results for each of the three outcomes, by model. Model "m1" includes one indicator, which measures maternal mental health problems age 3-6. Model "m2" includes two indicators, one for maternal mental health problems at age 3-6 and one for problems at age 7-12. Model "m3" includes three indicators, mental health problems at age 3-6, age 7-12 and age 13-15. Model "m4" includes four indicators: mental health problems at age 3-6, age 7-12, age 13-15 and age 16-19. Model "m5" includes three indicators: mental health problems at age 7-12, age 13-15 and age 16-19, model "m6" includes two indicators: maternal mental health problems measured at age 13-15 and age 16-19. The last model, "m7" includes one indicator, that measures maternal mental health problems at age 16-19. By comparing coefficients from each of these 7 models, I show exactly what happens to the impact of exposure to maternal mental health problems at age X, when controlling for mental health problems at age Y. With this strategy it becomes clear that analyzing maternal mental health in a group of children of a specific, narrow age band, may not be very informative on the importance of age at exposure, because an indicator of exposure at that specific age is likely to confound exposure at other ages.

From the three panels of figure 1 we learn that the "effect" of early exposure is halved when we control for any subsequent exposure. Thus, a significant part of the relationship between early exposure to maternal mental health problems reflects that early exposure is confounded by later exposure. Similarly, a non-negligent part of the "effect" of exposure at age 16-19 reflects its correlation with early exposure, as is evident from a comparison of the coefficient from m7 to the coefficients from e.g. m4. Last, criminal activities at age 20 seem uncorrelated with exposure between age 3-6 when I control for exposure at later ages.

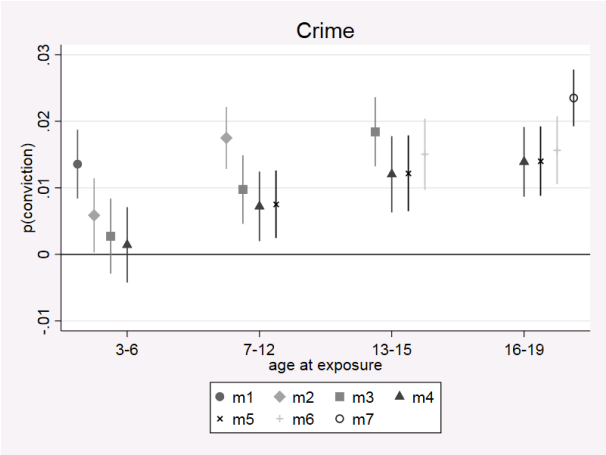
The combined take-home message from the three panels of figure 1 is that age-at-exposure matters, and in fact, early exposure does not seem more important than exposure in early- (13-15) and mid-adolescence (16-19). While these findings do not necessarily suggest that the first years are not important and formative, it is an indication that what happens in adolescence matters, even if it may potentially also do so through its proximity in time to the outcome we study.

Figure 1. The importance of age at exposure to maternal mental health problems

A: Human capital acquisition as outcome B: Child use of PP as outcome



C: Criminal sanctions as outcome



Confounders*?*

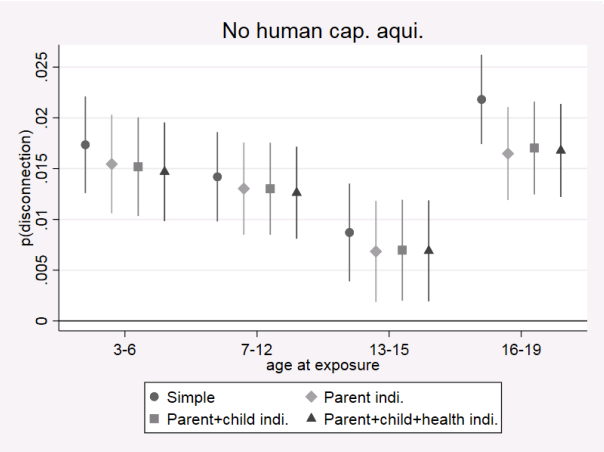
As described, I expect part of the correlation between mother's mental health and child outcomes to reflect mother and child's shared genetic dispositions and shared environment, and another part to reflect a causal effect. In this section I test the degree to which standard confounding variables affect the correlation between mothers' mental health problems and the three child outcomes. I maintain the focus on differences by age of exposure, to show whether different factors or mechanisms drive the correlation between maternal mental health and child outcomes at different ages. The three panels of figure 2 present the results. The first set of coefficients ("Simple") reflects the correlation with no included confounders in the model (similar to model m4 in figure 1). The second set of coefficients ("Parent. indi.) show the correlation when I control for the standard parental indicators, income, unemployment and education (both mother's and father's). The third set of indicators ("Parent.+ child indi.") show the correlation when I include both the parental indicators as well as indicators of child gender and immigration status. With the last set of indicators ("Parent.+ child + health indi."), I control for parental health status by including a measure of how many days of sick leave the

parent has had. I use indicators measured in 1990, e.g. two years before the child is born, and several years before I measure maternal mental health, to reduce the chance that the indicators are the result of maternal mental health problems at the time of measurement.

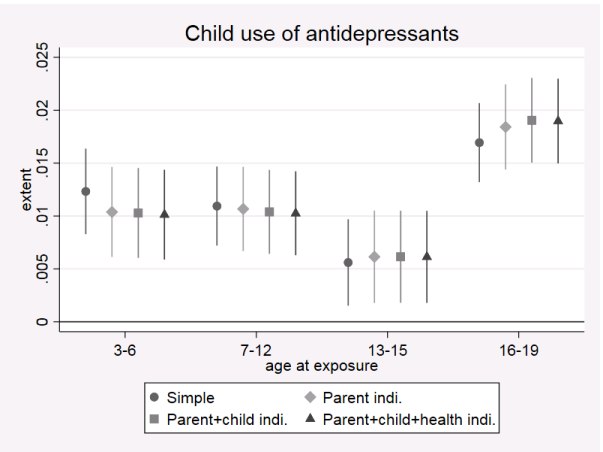
From the figure we learn that the coefficients are susceptible to the inclusion of the confounders, though to a limited degree. Maternal mental health is still positively and significantly correlated with all three outcomes, except for mental health problems observed at age 3-6 and crime (which is expected given the results presented in panel C of figure 1). In addition, we observe small differences in how the correlations change in response the inclusion of the confounders. In particular, the inclusion of confounders appears to have a stronger impact on the correlation between the outcome in focus and exposure to mother's mental health problems experienced at age 16-19, than on the correlation between the outcome in focus and exposure at other ages. However, these patterns are not strong and the observed differences are unlikely to be statistically significant.

Figure 2. Controlling for standard confounding variables

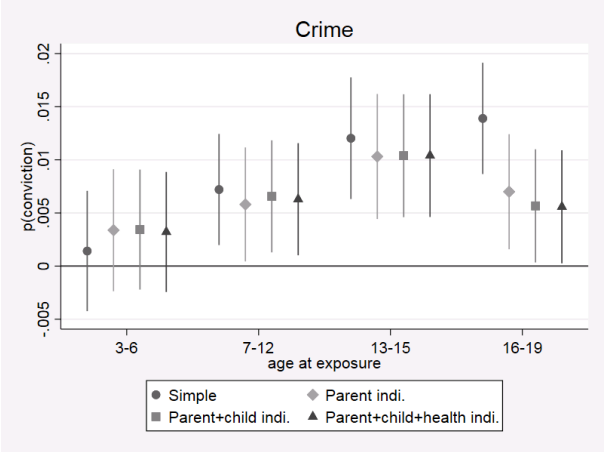
A: Human capital acquisition as outcome



B: Child use of PP as outcome



C: Criminal sanctions as outcome



With the evidence presented in the three panels of figure 2, it becomes clear that we cannot just remove the correlation between maternal mental health and child outcomes using standard controls – at least not for all outcomes. Also, there is no strong evidence that the selection issues differ by the age at which the child gets exposed to poor maternal mental health.

This evidence does not, however, rule out the possibility that unobserved confounders drive the correlation. To address this concern, the next and final analysis of this paper uses an IV-model to get causal inference.

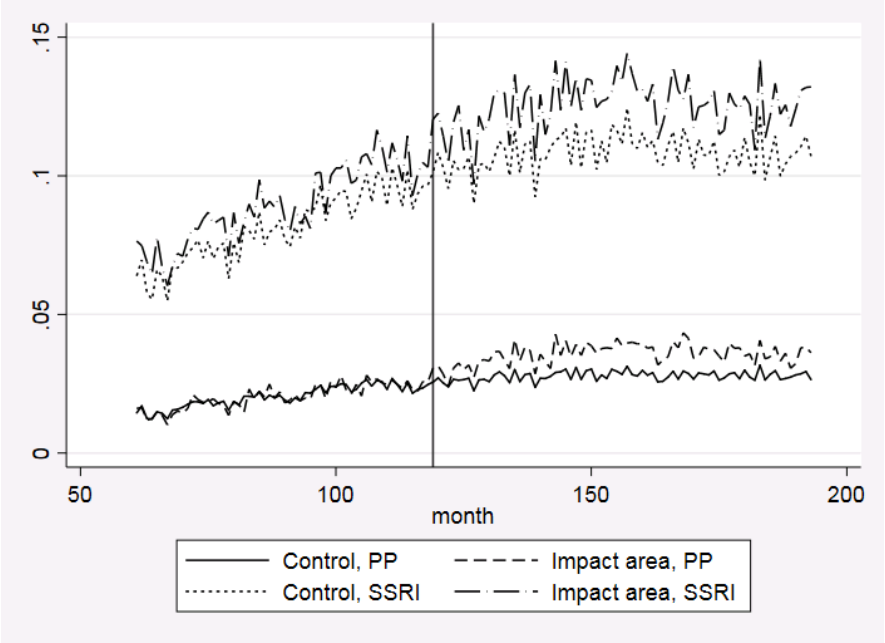
A causal effect?

Before presenting results from my IV-models, I discuss whether my setup fulfills the assumptions of the IV-model.

The relevance assumption

To first demonstrate the relevance of my instrument, Figure 3 below presents the development in the use of PP and SSRI among mothers of 16-19-year-olds who lived in the impact area and the control area, respectively. As shown, trends in use are very similar until month 119 (marked by the vertical line), which is the month of the explosion. From this point onwards, mothers living in the impact area redeem remarkably more prescriptions for these types of medicine, but especially for SSRI, which is the type of medication prescribed for PSTD. The evidence presented in figure 3 thus clearly indicates the effect of the fireworks disaster on mental health in the impact area.

Figure 3: Use of PP and SSRI among mothers in the control group and impact area



I furthermore test the relevance assumption using the F-test of the excluded instruments (table 1). The F-test of all except one of the six first stage specifications is well above the acceptable value of 10 (Stock et al, 2002), and this is evidence of a valid instrument.

Table 1: F-test values

	Extent of SSRI use
B: Full sample	15.54
B+control: Full sample, indicator for whether the adolescent lives in impact area	15.91
B1: Adolescent does not live in same municipality as mother	44.40
B2: B1+ No foster care	34.63
B3: B2+ Broken families	35.78
B4: B1+ In education	18.85

The exclusion restriction

The exclusion restriction is difficult to test empirically, and the validity of the assumption in a specific setup is a matter of conviction. As described, I test the robustness of my results in two ways, first by controlling for whether the adolescent lived with his or her mother at the time of the explosion, and second by also analyzing effects of maternal mental health in four subsamples which exclude adolescents living with their mother.

The exchangeability assumption

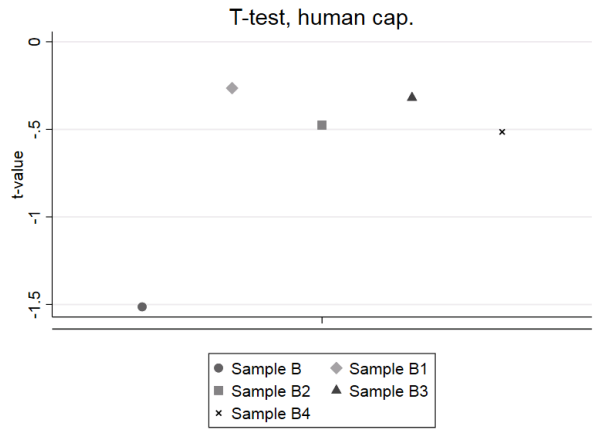
To (partially) verify the *exchangeability* assumption I test the balance of potential confounders across levels of my instrument. This cannot rule out confounding from unmeasured covariates but signals imbalances in the sample, that may drive the results.

I test the balancing properties of my samples with regards to maternal labor market attachment, her health status (general health and mental health) and her age, all measured in 2003, the year prior to the disaster. The relevance of these indicators reflects existing knowledge that vulnerable groups and older women respond more strongly to stressors (Fletcher et al, 2017; Olff, 2017; Xue et al., 2015), implying that imbalances between control and treatment group on such characteristics may bias the results. I furthermore test balancing properties of child age, gender, ethnicity and use of SSRI in 2003, all indicators which may reflect how the young person responds to maternal mental health problems (see table A2 in the appendix for distributions).

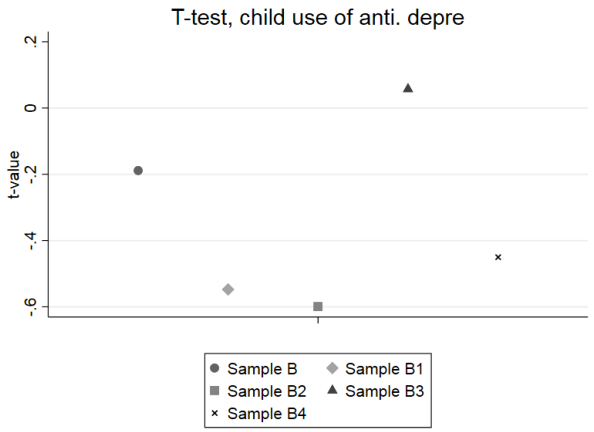
For this purpose, I regress each of my 3 outcome variables on the background characteristics, calculate the predicted values (by background characteristics), and test whether the predictions vary between the treated and the controls. Significant differences in predictions is a signal that the instrument captures and reflects systematic differences between the two groups and is not a pure source of exogeneous variation for maternal mental health. The three panels of Figure 4 present the t-test values derived from these models by (sub)samples and outcomes. As shown, no t-test value crosses the critical value of 1.64 (corresponding to a p-value of 0.1), and this test then supports the validity of the exchangeability assumption.

Figure 4: T-tests for differences in predictions by treatment status

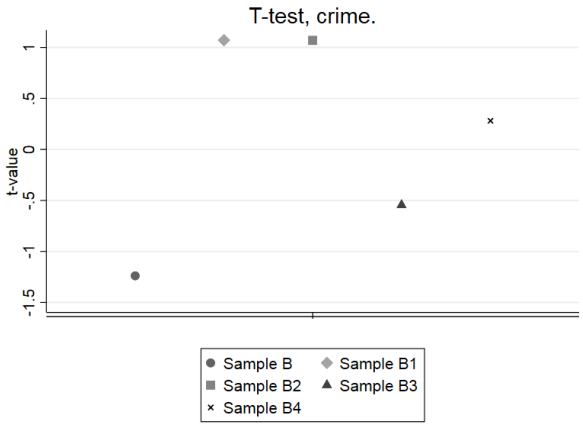
A: Human capital acquisition as outcome



B: Child use of PP as outcome



C: Criminal sanctions as outcome



The monotonicity assumption

Fourth is the *monotonicity* assumption, which implies that no individuals respond opposite to what we would expect given the mechanism installed by the instrument (Lousdal, 2018). This assumption is untestable. Yet, we may calculate differences in the shares of mothers who used SSRI prior to the disaster but stopped after. In the treatment groups, this share should also capture defiers and if shares differ in size between treated and controls, it may signal a violation of the monotonicity assumption. For this purpose, the second row of appendix table A2 reports the share of mothers who stopped using SSRI between 2003 and 2005. The share varies between 3 and 4 percent for both treated and controls, and there are no statistical differences between the two groups. This test does not confirm the monotonicity assumption but indicates that it is valid in my setup.

In sum, the all tests presented in this section support the validity of my instrument, suggesting that my first stage is valid.

Results from the IV-model

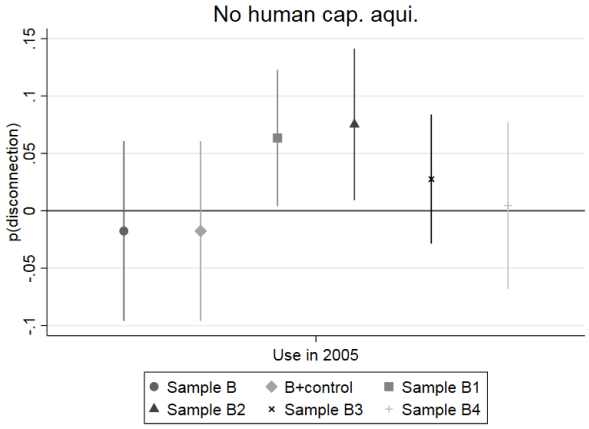
The three panels of Figure 5 show results from the IV-model, by sample and outcome. Recall that I here focus on the effect of exposure to maternal mental health problems in a sample of all adolescents age 16-19, as well as in four subsamples to address the concerns that results from the full sample rely on a model specification that violates the exclusion restriction. At the same time, the use of subsamples where the young person in focus does not live with his or her mother allows me to test the effect among groups that are comparatively more disadvantaged than the average adolescent.

From the figure we learn that mother's mental health problems exert a causal effect among some groups of adolescents and for some outcomes. First, while mother's mental health problems do not affect human capital acquisition in the general sample, the problems reduce this type of activities among two of the subsamples of adolescents who do not live with their mother (B1 and B2). Second, poor mental health seems

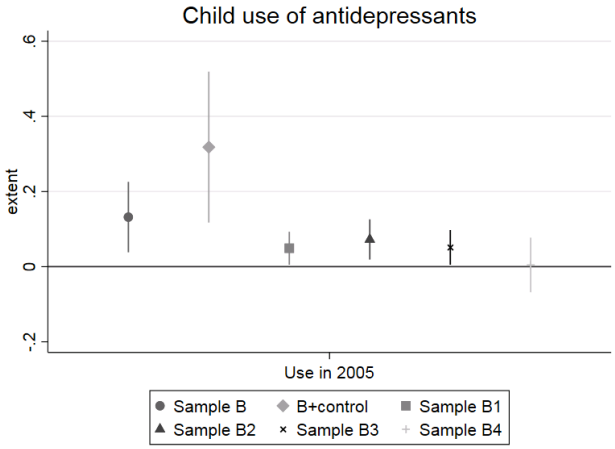
to increase child use of antidepressants in all but one sample (B4). Third, as we could expect, given the results presented in panel C of figure 2, mother's mental health problems do not affect crime among adolescents.

Figure 5: Results from the IV-model

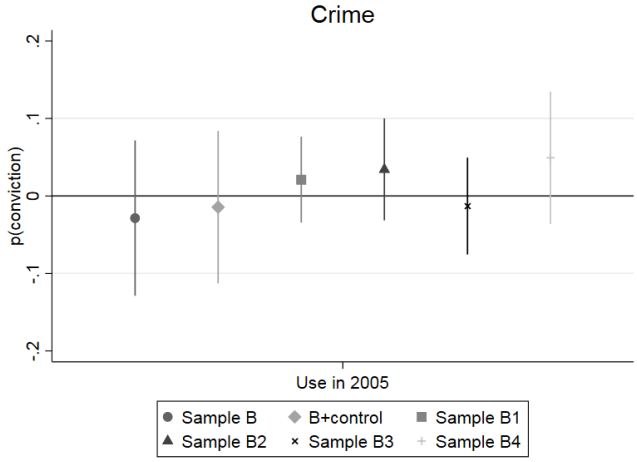
A: Human capital acquisition as outcome



B: Child use of PP as outcome



C: Criminal sanctions as outcome



While we may speculate about the differences found between samples and outcomes, the estimates derived from the IV-models do not rule out the possibility that mother's mental health has a causal effect on adolescents. These findings are likely indications that also adolescence is a sensitive period, similar to the sensitive period of the early years of life. When we see maybe not stronger, but at least more persistent

effects among more disadvantaged adolescents (defined as adolescents that do not live with their mother), this may be a signal that the more vulnerable groups are particularly sensitive, possibly because their disadvantaged situation is a proxy for the internalization of previously experienced stressors. These youth may have fewer resources and less support all in all available to them in the transitional period of adolescence, and the reduction in maternal support caused by the deterioration of mother's mental health may be particularly detrimental among this group.

Discussion and conclusion

The social sciences currently have a strong focus on early childhood interventions, and there is plenty of evidence that nurture, care and inputs received in utero and during the early years matter tremendously for later outcomes. But this should not detract our attention from the existence of other sensitive periods. And not only as periods during which exposure to negative environmental shocks and influences may be particularly detrimental, but also as periods during which the consequences of negative exposure in previous sensitive periods may be altered.

The aim of my study has been to highlight adolescence as a sensitive period during which the young person is susceptible to environmental shocks with lasting consequences. Through two empirical steps, I have demonstrated how exposure to maternal mental health problems in adolescence matters for later outcomes, and I have provided plausible evidence of a causal effect, particularly among the more disadvantaged youth. While the study does not provide conclusive evidence of adolescence as a sensitive period in brain formation – especially since it does not account for the specific mechanisms through which maternal mental health are transmitted to/transformed into the child outcomes in focus – it certainly does not prove the thesis wrong.

A question remains, though, as to whether we, as social scientists, should care about exposure to poor maternal mental health in adolescence, especially when the most persistent effect seems to be on adolescent mental health – maybe the concern for this type of childhood stressor is best left to psychologists? Importantly, what I present here is evidence that age matters for one of the core mechanisms studied in the social sciences, namely the intergenerational transmission of resources. And with the amount of resources put into not only studying the transmission but also counteracting the negative consequences of this transmission in low resource families, it is of vital importance that we understand the underlying mechanism driving it. First, focusing on age at exposure helps understand the importance of age and of how to allocate scarce societal resources for counteracting the negative consequences of growing up in disadvantaged childhood environments: to the degree that age matters for the transmission, we should be keen to help children exposed at the most vulnerable ages. Second, focusing on age also provides plausible reasons why the transmission takes place; when age matters it must reflect certain characteristics affiliated with or processes taking place at particular ages, whether rooted in the social or physiological domain. This

observation helps us understand the underlying mechanism of the transmission and provides important insights into how to counteract a negative transmission.

References

Albert, Dustin, Chein, Jason & Steinberg, Laurence (2013): Peer Influences on Adolescent Decision Making. *Current Directions in Psychological Science.* 22(2): 114-120

Allen, Timothy A., Oshri, Assaf, Rogosch, Fred A, Toth, Sheree L. & Cicchetti, Dante (2018): Offspring Personality Mediates the Association between Maternal Depression and Childhood Psychopathology. *Journal of Abnormal Child Psychology*

Altree, P. (2004): Growing up in disadvantage: a systematic review of the qualitative evidence. *Child: Care, health and development*, 30(6): 679-689

Apter-Levi, Yael, Pratt, Maayan, Vakart, Adam, Feldman, Michal, Zagoory-Sharon, Orna, & Feldman, Ruth (2016): Maternal depression across the first years of life compromises child psychosocial adjustment: relations to child HPA-axis functioning. *Psychoneuroendocrinology*, 64: 47-56

Beredskabsstyrelsen (2005): *Fyrværkeriulykken i Kolding 2004 – En teknisk beredskabsfaglig erfaringsopsamling*. Beredskabsstyrelsen, Birkerød. Found October 9th, 2018 at: https://brs.dk/viden/publikationer/Documents/Fyrv%C3%A6rkeriulykken_Koldingrapport.pdf

Bernard-Bonnin, Anne-Claude (2004): Maternal depression and child development. *Pediatric Child Health*, 9(8): 575-583

Blakemore, Sarah-Jayne (2012): Development of the social brain in adolescence. *Journal of the Royal Society of Medicine*, 105:111-116

Blakemore, Sarah-Jayne & Mills, Kathryn L. (2013): Is Adolescence a Sensitive Period for Sociocultural Processing? *Annual Review of Psychology,* 65(1)

Brennan, Patricia A., Hammen, Constance, Andersen, Margaret J., Bor, William, Najmam, Jake M. & Williams, Gail M. (2000): Chronicity, Severity, and Timing of Maternal Depressive Symptoms: Relationships With Child Outcomes at Age 5. *Developmental Psychology*, 36(6): 759-766

Bureau, Jean-Francois, Easterbrooks, M. Ann & Lyons-Ruth, Karlen (2009): Maternal depressive symptoms in infancy: Unique contribution to children's depressive symptoms in childhood and adolescence? *Development an Psychopathology*, 21: 519-537

Bøe, Tormod, Sivertsen, Børge, Heiervang, Einar, Goodman, Robert, Lundervold, Astri J. & Hysing, Mari (2014): Socioeconomic Status and Child Mental Health: The Role of Parental Emotional WellBeing and Parenting Practices. *Journal of Abnormal Child Psychology*, 42(5): 705-715

Chevalier, Arnaud, Harmon, Colm, O'Sullivan, Vincent & Walker, Ian (2013): The impact of parental income and education on the schooling of their children. *IZA Journal of Labor Economics*, 2(8): 1-22

Choudhury, S. (2010): Culturing the adolescent brain: What can neuroscience learn from anthropology? *Soc. Cogn. Affect. Neurosci.* 5:159-167

Côté, Sylvana M., Ahun, Marilyn N., Herba, Catherine M., Brendgen, Mara, Geoffroy, Marie-Claude, Ori, Massimiliano, Liu, Xuecheng, Vitaro, Frank, Melchior, Maria, Boivin, Miochel & Tremblay, Richard E. (2018): Why is Maternal Depression related to Adolescent Internalizinv Problems? A 15-Year Population-Based Study. *Journal of the American Academy of Child & Adolescent Psychiatry*, in press

Cummings, E. Mark & Davies, Patrick T. (1994): Maternal Depression and Child Development. *The Journal of Child Psychology and Psychiatry*¸ 35(1): 73-122

Curtis, Alexa C., (2015): Defining adolescence. *Journal of Adolescent and Family Health*, 7(2):

Dickson, Matt, Gregg, Paul & Robinson, Harriet (2016): Early, Late or Never? When Does Parental Education Impact Child Outcomes? *The Economic Journal*, 126(596): 184-231

Downey, Geraldine & Coyne, James (1990). Children of Depressed Parents: An Integrative Review. *Psychological Bullletin*, 108(1): 50-76

Duch, Christina & Elklit, Ask (2008): Børneperspektiv på en katastrofe. *Psykolognyt*, 4:8-12

Duncan, Greg J. & Brooks-Gunn, Jeanne (1999): *Consequences of Growing Up Poor*. Russell Sage Foundation. NYC

Elklit, Ask, Petersen, Tóra & Molin, Keld (2008): *De Psykosociale følger af fyrværkerikatastrofen i Seest. Del 3 – Beboerne 15 måneder efter*. Psykologisk Institut, Aarhus Universitet

Elklit, Ask (2007): Psychological consequences of a firework factory disaster in a local community. *Social Psychiatry and Psychiatric Epidemiology*, 42(8): 664-668

Elklit, Ask & Molin, Keld (2006): *De Psykosociale følger af fyrværkerikatastrofen i Seest. Del 1 – Beboerne 3 måneder efter*. Psykologisk Institut, Aarhus Universitet

Engert, Veronika, Plessow, Franziska, Miller, Robert, Kirschbaum, Clemens & Singer, Tania (2014): Cortisol increase in emphathic stress is modulated by emotional closeness and observation modality. *Psychoneuroendocrinology*. 45: 192-201.

Enos, Ryan D. (2017): *The Space Between Us*. Cambridge University Press, Cambridge

Fiske, S. T. (2009): *Handbook of Neuroscience for the Behavioral Sciences*, New York: Wiley

Fletcher, Shelley, Elklit, Ask, Shevlin, Mark & Armour, Cherie (2017): Predictors of PTSD Treatment Response Trajectories in a s Sample of Childhood Sexual Abuse Survivors: The Roles of Social Support, Coping and PTSD Symptom Clusters. *Journal of Interpersonal Violence*,

Fowler, J. H. & Christakis, N. A. (2008): Dynamic spread of happiness in a large social network: longitudinal analysis over 20 years in the Framingham Heart Study. *British Medical Journal*, 337

Furhmann, Delia, Knoll, Lisa J. & Blakemore, Sarah-Jayne (2015): Adolescence as a Sensitive Period of Brain Development. *Trends in Cognitive Sciences*, 19(10): 558-566

Gelfand, Donna M. & Teti, Douglas M. (1990): The effects of maternal depression on children. *Clinical Psychology Review*, 10(3): 329-353

Gervai, Judit (2009): Environmental and genetic influences on early attachment. *Child and Adolescent Psychiatry and Mental Health*, 3:25

Gjerde Line C., Eilertsen, Espen Moen, Reichborn-Kjennerud, Ted, McAdams, Tom A, Zachrisson, Henrik Daae, Zambrana, Imac Maria, Roysamb, Espen, Kendler, Kenneth S. & Ystron, Eivind)2017): Maternal perinatal and concurrent depressive symptoms and child behavior problems: a sibling comparison study. *Journal of Child Psychology and Psychiatry*, 58(7): 779-786

Goodman, Sherryl H, Rouse, Matthew H., Connell, Arin M., Broth, Michelle Robbins, Hall, Christine M. & Heyward, Devin (2011): Maternal Depresssion and Child Psychopathology: A Meta-Analytic Review. *Clinical Child and Family Psychology Review*¸ 14(1): 1-27

Halligan, Sarah L., Murray, Lynne, Martins, Carla & Cooper, Peter J. (2007): Maternal depression and psychiatric outcomes in adolescent offspring: A 13-year longitudinal study. *Journal of Affective Disorders*, 97: 145-154

Hammen, Constance & Brennan, Patricia A. (2003): Severity, Chronicity, and Timing of Maternal Depression and Risk for Adolescent Offspring Diagnoses in a Community Sample. *Arch Gen Psychiatry*, 60: 253-258

Hansen, Bertel T., Østergaard, Søren D., Sønderskov, Kim M. & Dinesen, Peter T. (2016): Increased Incidence Rate of Trauma – and Stressor – related Disorders in Denmark After the September 11, 2001, Terrorist Attacks in the United States. *American Journal of Epidemiology*, 184(7): 494-500

Hansen, Bertel T., Dinesen, Peter T. & Østergaard, Søren D. (2017): Increased Incidence Rate of Trauma- and Stressor-related Disorders in Denmark After the Breivik Attacks in Norway. *Epidemiology*, 28(6): 906-909

Harpham, Trudy, Huttly, Sharon, De Silva, Mary J. & Abramsky, Tanya (2005): Maternal mental health and child nutritional status in four developing countries. *Journal of Epidemiology & Community Health*, 59: 1060-1064

Hartman, Sarah & Belsky, Jay (2018): Prenatal programming of postnatal plasticity revisited – And extended. *Development and Psychopathology*, 30: 825-842

Hay, Dale F., Pawlby, Susan, Waters, Cerith S. & Sharp, Deborah (2008):Antepartum and postpartum exposure to maternal depression: different effects on different adolescent outcomes. *Child Psychology and Psychiatry*, 49(10): 1079-1088

Hay, Dale F., Waters, Cerith S., Perra, Oliver, Pawlby, Susan & Sharp, Deborah (2010): Mothers' Antenatal Depression and Their Children's Antisocial Outcomes. *Child Development*, 81(1): 149-165

Kessler, Ronald C., Amminger, G. Paul, Aguilar-Gaxiola, Sergio, Alonso, Jordi, Lee, Sing, & Üstün, T. Bedirhan (2007): Age of onset of mental disorders: a review of recent literature. *Current Opinion in Psychiatry*, 20(4): 359-364

Kieling, Christian, Baker-Henningham, Helen, Belfer, Myron, Conti, Gabriella, Ertern, Ilgi, Omigbodun, Olayinka, Augusto Rohde, Luis Augusto, Srinath, Shoba, Ulkuer, Nurper & Rahman, Atif (2011): Child and adolescent mental health worldwide: evidence for action. *The Lancet*, 378: 1515-1525

Kim-Cohen, Julia, Moffitt, Terrie E., & Taylor, Alan (2005): Maternal Depression and Children's Antisocial Behavior. Nature and Nurture Effects. *Arch Gen Psychiatry*, 62(2): 173-181

King, Ronnel B. & Datu, Jesus Alfonso (2017): Happy classes make happy students: Classmates' well-being predicts individual student well-being. *Journal of School Psychology*, 65: 116-128

Knudsen, Eric I., Heckman, James J., Cameron, Judy L. & Shonkoff, Jack P. (2006): Economic, neurobiological, and behavioral perspectives on building America's future workforce. *PNAS*, 103(27): 10155-10162

Larson, R. & Richards M. H. (1991): Daily companionship in late childhood and early adolescence: changing developmental contexts. *Child Development*, 62: 284-300

Larson, R. W., Richards, M. H., Moneta, G. Holmbeck, G., Duckett, E. (1996): Changes in adolescents' daily interactions with their families from ages 10 to 18: disengagement and transformation. *Developmental Psychology*, 32: 744-754

Leszczensky, Lars & Wolbring, Tobias (2018): How to Deal with Reverse Causality Using Panel Data? Recommendations for Researchers Based on a Simulation Study. SocArXiv, 30. Aug. 2018. Web. Doi: 10.31235/osf.io/8xb4z

Lousdal, Mette Lise (2018): An introduction to instrumental variable assumptions, validation and estimation. *Emerging Themes in Epidemiology*, 15(1): 1-7

Meinck, Franziska, Cluver, Lucie Dale, Orkin, Frederick Mark, Kuo, Caroline, Sharma, Amogh Dhar, Hensels, Imca Sifra & Sherr, Lorraine (2017): Pathways From Family Disadvantage via Abusive Parenting and Caregiver Mental Health to Adolescent Health risks in South Africa. *Journal of Adolescent Health*, 60(1): 57-64

Meyer, Alexandria, Bress, Jennifer H., Hajcak, Greg & Gibb, Brandon (2018): Maternal Depression Is Related to Reduces Error-Related Brain Activity in Child and Adolescent Offspring. *Journal of Clinical Child & Adolescent Psychology*, 47(2): 324-335

Mikkonen, Janne, Moustgaard, Heta, Remes, Hanna & Martikainen, Pekka (2016): Intergenerational transmission of depressive symptoms – The role of gender, socioeconomic circumstances, and the accumulation of parental symptoms. *Journal of Affective Disorders*, 204: 74-82

Newman, Benjamin J. & Hartman, Todd K. (2017): Mass Shootings and Public Support for Gun Control. *British Journal of Political Science*

Oberle, Eva & Schonert-Reichl, Kimberly A. (2016): Stress contagion in the classroom? The link between classroom teacher burnout and morning cortisol in elementary school students. *Social Science & Medicine*, 159: 30-37

O'Brien, S. F. & Bierman, K. L. (1988): Conceptions and perceived influence of peer groups: interviews with preadolescents and adolescents. *Child Development*, 59: 1360-1365

Olff, Miranda (2017): Sec and gender differences in post-traumatic stress disorder: an update. *European Journal of Psychotraumatology*, 8(4): 1351204

Patel, Vikram, Rahman, Atif, Jacob, K. S. & Hughes, Marcus (2004): Effect of maternal mental health on infant growth in low income countries: new evidence from South Asia. *British Medical Journal*, 328(7443): 820-823

Pawlby, Susan, Hay, Dale F., Sharp, Deborah, Waters, Cerith S. & Keane, Veronica (2009): Antenatal depression predicts depression in adolescent offspring: Prospective longitudinal community-based study. *Journal of Affective Disorders*, 113: 236-243

Phil, Franziska Meinck D., Suri, Rita, Lin, Alex, Cohen, Lee S., & Altshuler, Lori L. (2014): Acute and Long-Term Behavioral Outcomes of Infants and Children Exposed in Utero to Either Maternal Depression or Antidepressants: A review of the Literature. *Journal of Clinical Psychiatry*, 75(10): 1142-1152

Rosenquist, J. N, Fowler, J. J & Christakis, N. A. (2013): Social network determinants of depression. *Molecular Psychiatry*, 16(3)

Sanger, Camilla, Iles, Jane E, Andrew, Catharina S. 6 Ramchandani, Paul G. (2015): Association s between postnatal maternal depression and psychologica outcomes in adolescent offspring: a systematic review. *Archieves of Women's Mental Health*, 18(2): 147-162

Staneva, Aleksandra, Bogossian, Fiona, Pritchard, Margo & Wittkowski, Anja (2015): The effects of maternal depression, anxiety, and perceived stress during pregnancy on preterm birth: A systematic review. *Women and Birth*, 28: 197-193

Sterley, Toni-Lee, Baimoukhametova, Dinara, Füzesi, Tamás, Zurek, Agnieszka A., Daviu, Nuria, Rasiah, Neilen P. Rosenegger, David & Bains, Jaideep S. (2018): Social transmission and buffering of synaptic changes after stress. *Nature Neuroscience*, 21: 393-403

Stock, James H, Wright, Jonathan H. & Yogo, Motohiro (2002): A Survey of Weak Instruments and Weak Identification in Generalized Method of Moments. *Journal of Business & Economic Statistics*, 20(4): 518-529

Tully, Erin C., Iacono, William G. & McGue, Matt (2008): An Adoption Study of Parental Depression as an Environmental Liability for Adolescent Depression and Childhood Disruptive Disorders. *American Journal of Psychiatry*, 165: 1148-1154

Whitaker, Robert C., Orzol, Sean M., & Kahn, Robert S. (2006): Maternal mental health, Substance Use, and Domestic Violence in the Year after Delivery and Subsequent Behavior Problems in Children at Age 3 years. *Arch Gen Psychiatry*, 63(5): 551-560

Wildeman, Christopher & Andersen, Signe Hald (2016): Paternal incarceration and children's risk of being charged by early adulthood: Evidence from a Danish policy shock. *Criminology*, 55(1): 32-58

Xue, Chen, Ge, Yang, Tang, Bihan, Liu, Yan, Kang, Peng, Wang, Meng & Zhang, Lulu (2015): A Meta-Analysis of Risk Factors for Combat-Related PTSD among Military Personnel and Veterans. *PLoS ONE*,

Yehuda, Rachel, Engels, Stephanie Mulherin, Brand, Sarah R., Seckl, Jonathan, Marcus, Sue M., & Berkowitz, Gertrud S. (2005): Transgenerational Effects of Posttraumatic Stress Disorder in Babies of Mothers Exposed to the World Trade Center Attacks during Pregnancy. *The Journal of Clinical Endocrinology & Metabolism*, 90(7): 4115-4118

APPENDIX

Table A1: Descriptive statistics, sample A

Variable name	Mean (std.)
Outcome variables (measured at age 20)	
No human capital acquisition	0.05 (0.22)
Using antidepressants	0.04 (0.19)
Crime	0.07 (0.26)
Exposure to maternal mental health problems (no. of doses)	
Age 3-6	0.16 (0.37)
Age 7-12	0.27 (0.44)
Age 13-15	0.24 (0.43)
Age 16-19	0.28 (0.45)
Mother's characteristics (measured in 1990)	
Income (1000 DKK)	132.71 (75.41)
Unemployment	0.11 (0.31)
Education (>highschool)	0.56 (0.50)
Sick leave (days)	2.99 (19.40)
Father's characteristics (measured in 1990)	
Income (1000 DKK)	197.01 (130.79)
Unemployment	0.08 (0.27)
Education (>highschool)	0.65 (0.48)
Sick leave (days)	3.61 (21.02)
Child characteristics	
Gender	0.49 (0.50)
Immigrant	0.09 (0.28)

Table A2: Descriptive statistics, sample B

	Sample B		Sample B1		Sample B2		Sample B3		Sample B4	
	Con.	Treat.	Con.	Treat.	Con.	Treat.	Con.	Treat.	Con.	Treat.
Mother's SSRI, 2005	0.31	0,38***	0,38	0,80***	0,36	0,71***	0,40	0,92***	0,36	0,68***
Potential defiers	0.03	0,03	0,04	0,03	0,04	0,03	0,04	0,03	0,04	0,03
Mother's characteristics (measured in 2003)										
SSRI (extent)	0.29	0,29	0,38	0,42	0,36	0,32	0,40	0,36	0,37	0,39
Unemp. (days)	48.06	48,23	69,01	72,39	67,00	78,49	79,13	94,57	65,37	80,59
Log (Income, DKK)	12.26	12,24*	12,14	12,15	12,16	12,14	12,16	12,10	12,17	12,18
Sick leave (weeks)	0.07	0,08***	0,09	0,10	0,09	0,09	0,10	0,12	0,09	0,11
Age (>45)	0.37	0,34***	0,30	0,32	0,31	0,31	0,24	0,24	0,31	0,31
Child characteristics										
SSRI 2003	0.01	0,01	0,02	0,02	0,02	0,02	0,02	0,02	0,01	0,02
Girl	1.49	1,49	1,53	1,58†	1,54	1,59†	1,50	1,49	1,54	1,57†

Immigrant	0.9	0,08	0,06	0,06	0,06	0,05	0,05	0,03	0,06	0,04
Age (>17)	0.76	0,77	0,57	0,57	0,54	0,52	0,62	0,64	0,60	0,58
In foster care, Nov. 2004	0.02	0,02	0,09	0,09	0	0	0	0	0,08	0,09
Outcome variables										
No hum. C.	0.04	0.04	0.09	0.12**	0.07	0.10**	0.07	0.09	0.06	0.06
Antidepre.	0.03	0.04*	0.05	0.07*	0.04	0.07**	0.04	0.07**	0.04	0.05
Crime	0.07	0.06	0.09	0.09	0.08	0.09	0.10	0.09	0.08	0.09
N	230,985	5,972	28,855	528	26,342	481	16,237	295	21,869	390

Note: *:p<0.001*:p<0.01; *:p<0.05; †:p<0.10