Long-term Consequences of ADHD Medication Use for Children's Human Capital Development

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Abstract:

This paper estimates effects of ADHD medication use on core human capital outcomes for children diagnosed with ADHD while using rarely available register based data on diagnoses and prescription drug purchases. Our main identification strategy exploits plausible exogenous assignment of children to specialist physicians, while our analysis of health outcomes also allows for an individual level panel data strategy. We find that the behavior of specialist physicians varies considerably across wards and that the prescribing behavior does affect the probability that a given child is treated. Results show that children diagnosed with ADHD in pharmacological treatment have fewer hospital contacts if treated and that treatment to some extent protects against criminal behavior. We do not, on the other hand, find improvements in terms of the likelihood to complete secondary exam.

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"TO date, no study has found any long-term benefit of attention-deficit medication on academic performance, peer relationships or behavior problems, the very things we would most want to improve. Until recently, most studies of these drugs had not been properly randomized, and some of them had other methodological flaws."

- Professor Emeritus of Psychology, L. Alan Sroufe, Minnosota: "Ritalin Gone Wrong", New York Times, January 12, 2012.

I. Introduction

One of the most publicly debated new medical technologies is pharmacological treatment of children diagnosed with Attention-Deficit/Hyperactivity-Disorder (henceforth ADHD). There are concerns, even among some professionals in the field, that children are over-diagnosed with ADHD; that we know too little about effects of ADHD medication and that children are, in fact, over-treated with ADHD medication. This is the first paper to investigate longer-run effects of pharmacological treatment of ADHD on a series of key human capital variables such as health, crime and schooling. More generally, our paper speaks to the literature on the impacts of early health interventions.

ADHD is one of the most common chronic mental health problems among young children. ADHD is estimated to affect about 3-7 % of all children (The Danish Association for Child and Adolescent Psychiatry, 2008; American Psychiatric Association, 2000) or on average one child – more often a boy than a girl – in every classroom. Core symptoms associated with ADHD are attention deficiencies, hyperactivity and impulsiveness and children often simultaneously suffer from other behavioral problems along with depression and anxiety. Hence, ADHD is likely to affect not only one's overall human capital but also one's tendency to engage in risky health behaviors.

Children with ADHD grow up in relatively disadvantaged families and have – even when in pharmacological treatment – much worse long-term outcomes than others who are comparable in terms of age and gender (Dalsgaard et al. (2002); Mannuzza and Klein (2000)) and also when compared to siblings without ADHD (Currie and Stabile (2006); Fletcher and Wolfe (2008)). And although a series of randomized controlled studies show that treatment with central nervous system stimulants[1] is effective in terms of reducing ADHD core symptoms and improving social behavior (van der Oord et al. (2008)), most studies only have very short follow up periods of up to three years after randomization implying that we effectively know very little about the longer-run consequences of treating children pharmacologically. Because these studies rely on surveys with relatively small samples sizes and since attrition has shown to be a real concern in the follow-up studies already carried out, alternative evaluation methods are called for; the seminal Multimodal Treatment Study of Children with ADHD (henceforth MTA), for example, initially included 144 treated children and some studies use as little as ten children in treatment; see van der Oord et al (2008)).

We propose to exploit variation in access to pharmacological treatment generated by variation in specialist physicians' propensity to prescribe to estimate effects of treatment; see for example Duggan (2005) who uses the same type of variation to investigate effects of second-generation antipsychotics on spending on other types of medical care and Doyle (2007, 2008) who uses variation in investigator assignment to estimate causal effects of foster care. Such variation may stem from ward level differences in treatment culture and because of knowledge spillovers; see Coleman, Katz and Menzel (1957) and Soumerai et al. (1998).

A concern with the more standard instrument exploiting treatment tendencies associated with other children at the ward level is so-called treatment by indication: physicians who are observed to treat more might just do so because they meet children who suffer from more severe ADHD. We therefore test the robustness of our main results to using an alternative instrument that attempts to hold fixed the severity of the disorder, namely the share of treated among other children *with less severe ADHD* as approximated by a low number of pre-treatment injuries.

Our analysis of effects on health outcomes allows for a complementary identification strategy: specifically, since a diagnosis is rarely established and treatment initiated before the age of five, we compare outcomes of treated children prior to (age four and three) and after treatment (age 10 +) with untreated diagnosed children before and after. This identification strategy allows for non-

[1] Methylphenidate is the most common pharmacological treatment for ADHD, better known under the brand name Ritalin. A more recent development is Concerta; a once daily extended release formulation of methylphenidate. Another commonly used agent is dexamphetamine, however no extended-release formulations of this are available in Denmark and the use therefore very limited.

random selection into treatment based on, for instance, severity of symptoms or parental backgrounds as long as these mechanisms are constant over time.

We use a combination of Danish registers that apart from rich socio-economic background variables include the following key information: 1) psychiatric history and diagnoses for children and their parents including information about the ward where a diagnosis was first established, 2) the history of prescription drug usage for children and their parents, and 3) health, schooling, and criminal outcomes.

We find that the behavior of specialist physicians varies considerably across wards and that the prescribing behavior does affect the probability that a given child is treated. Results show that children diagnosed with ADHD on the margin of receiving pharmacological treatment have *fewer* hospital contacts if treated, are *less* likely to be charged with crime but *less* likely to complete secondary school exams. That is, children seem to benefit from treatment with regards to outcomes that only depend directly on the child’s own behavior and are within his control, whereas they perform worse in terms of outcomes that may be mediated by others such as school teachers, principals, and parents. This is consistent with a hypothesis of social stigma associated with medication. In fact, results from a teacher survey show that teachers are highly likely to be informed if children in their classroom are treated for ADHD and teachers self-report that they have substantial influence on the decision to let a child enter secondary exams.

In the analysis of health outcomes, we have sufficient data to meaningfully distinguish between birth cohorts. We document that effects are smaller in later cohorts where more children are diagnosed and treated pharmacologically before the age of ten. There are still significant gains from treatment in the later cohorts, but the results support a hypothesis of diminishing returns to broadening the group of treated.

The paper is structured as follows: Section II discusses causes of ADHD and links between ADHD and human capital, Section III presents the empirical framework, and Section III the background for the analysis. Section IV shows the data, Section V the results and Section VI concludes.

II. Causes of ADHD and Links between ADHD and Human Capital

Recently, a series of papers such as Cunha et al. (2006), Currie (2011), and Currie and Almond (2010) have emphasized the importance of investing early in particularly vulnerable children.

Moreover, Cunha and Heckman (2007) show theoretically that early investments not only have a large potential pay-off, they are also efficient in the sense that an equity-efficiency trade-off does not exist, which is the case for later investments. The reasons are that skills acquired in one period persist into future periods and that skills produced at one stage raise the productivity of investment at subsequent stages. Importantly, skills are multidimensional and are likely to complement each other. The group of children with ADHD is a prime example for which we would expect early investments with immediate effects on health capital in general to also have long-term consequences for later health and human capital attainment. This paper investigates investments via pharmacological treatment. Early take-up of pharmacological treatment may have long-term effects on human capital simply because it improves behavior and therefore the likelihood of future treatment but also because of dynamic complementarities: treatment may improve cognitive skills[2] including less impulsive behavior and more awareness of the consequences of one's actions that feed back on outcomes.

To set the scene, we sketch a simple model for the production of skills during childhood. We follow Heckman (2008) and co-authors. The model consists of three periods, $t = 1,2,3$, corresponding to early and late childhood and early youth. Parents invest in their children in period one and two and the investments of interest for this paper are ADHD related interventions in period one, I_1. The technology of skill production for a given child in period t can be summarized in the following way:

$$O_{t+1} = f_t(h, O_t, I_t),$$

where O is a vector of outcomes, f is the production function, h measures initial conditions such as birth weight, mental health (ADHD) and parental abilities, and I indicates parental investments such as pharmacological treatment.[3]

Given the production function, it is clearly critical to be aware of the causes of ADHD since they may be correlated with initial conditions that determine later outcomes. Though not perfectly described, it is well known that genetic factors are very important (Faraone and Doyle (2001)), but also premature birth, birth complications, maternal smoking and alcohol use during pregnancy are associated with ADHD (Linnet et al. (2003)). In our sensitivity section we will therefore investigate

[2] IQ, for example, is malleable up until the age of 10, see Cunha et al. (2006)

[3] Here we ignore the fact that seeking the actual diagnosis may be affected by parental background or the peer group, see e.g. Elder and Lubotsky (2009).

whether effects of treatment vary with health at birth and maternal smoking during pregnancy. Children with ADHD are also more likely than others to have language, cognitive and memory problems (e.g. Jensen et al. (2001) and Frazier et al. (2004)). To address this, we investigate the extent to which results are affected by children with mental retardation (11% in our sample).

The existing literature only considers the direct link between ADHD and measures of human capital (educational outcomes) and not to what degree (or whether at all) pharmacological treatment may serve as a remedy. Currie and Stabile (2006) find that ADHD symptoms at ages 5-12 worsen learning outcomes as measured by short-run educational attainment at ages 9-16.[4] They conclude that mental disorders are much more important for average learning outcomes than physical disorders. Inclusion of siblings fixed effects does not change the results. Fletcher and Wolfe (2008) confirm Currie and Stabiles' findings for short-run educational outcomes and find similarly strong effects on long-run educational outcomes. However, they do find that accounting for family fixed effects makes most of the negative long-run effects disappear. By controlling for ADHD symptoms of siblings they show that rather than reflecting the fact that families learn how to compensate for the ADHD symptoms, a child with ADHD symptoms indeed affects siblings negatively; in other words, negative effects extend beyond those on the individual himself. In fact, for many learning outcomes, the effect of ADHD on siblings' human capital accumulation is as high as the effect of own ADHD on human capital accumulation. For instance, for outcomes such as years of education, school drop-out, college enrolment, and school suspension, the effect of siblings' ADHD on human capital accumulation is significant and of the same order of magnitude for the sibling as for the child him- or herself. On the other hand, for outcomes such as grade repetition, special education and GPA, the effect of ADHD on siblings' outcomes is insignificant and negligible in size.

A series of papers (Ding et al. (2009) and Fletcher and Lehrer (2009, 2011)) instrument for poor mental health (including ADHD) using genetic markers and investigate the effects of poor mental health on academic performance. They find some evidence that inattentiveness is associated with lower academic achievement. The inherent problem, of course, is that there is no knowledge about direct effects of gene composition on educational outcomes. The authors have access to a series of instruments, which do pass conventional F-tests for over-identification.

[4] In their samples from Canada and the US in 1994, only 7-10 % of children with a high hyperactivity score were in drug therapy. These numbers are similar to the 12% reported by Mannuzza and Klein (2000).

As mentioned above, it is well-documented that treatment with central nervous system stimulants is effective in terms of reducing the number and impact of ADHD core symptoms.[5] This evidence is based on a series of randomized controlled trials. In the seminal Multimodal Treatment Study of Children with ADHD (henceforth MTA), 579 children aged 7-9.9 years suffering from ADHD were assigned to different types of treatment for a period of 14 months. Of these, 144 children were assigned to pharmacological treatment. Within the 14-month period careful medication management with or without behavioral treatment was shown to be superior to routine community care or behavioral treatment in terms of reducing core symptoms, see MTA (1999). In follow-up studies considering children three years after randomization, the difference in symptom relief diminishes over time and eventually disappears (see MTA (2004) and Molina et al. (2009)). These studies find no impact of medication management on functioning outcomes such as social skills, relations or reading achievement.

While being informative about symptom relief, these studies cannot stand alone when it comes to determining the long-term consequences of pharmacological treatment of ADHD. Unfortunately, the MTA study, as well as other randomized controlled trials, suffers from serious problems, the most important being selection into (or out of) the experiment; in the case of the MTA study only 13 % of the children initially screened ended up participating. Children were for example excluded if they had low IQ, if they were hospitalized or were otherwise ill, if their primary care-taker was non-English speaking, or if there was no phone in the household. Similarly, a large share of parents refused to let their child enroll into experimental treatment. All of these factors are unlikely to be uncorrelated with gains from treatment. Other problems include Hawthorne effects, attrition and small sample sizes; some studies had as little as ten treated children (see van der Oord et al (2008)). In addition, absent register-based outcome measures, studies rely on test scores or self-reported outcomes collected among non-blinded respondents in follow-up evaluations. Furthermore, random controlled trials only ever measure the intention to treat (ITT) among those who choose to participate, which may be very different from the average treatment effect on the treated (ATET) in the population if persistent individual factors such as own preference for treatment or physician's prescription practices influence the take up of pharmacological treatment. And ultimately, we know

[5] The information is surveyed and incorporated in national treatment guidelines e.g. The Danish Association for Child and Adolescent Psychiatry (2008) for Denmark and Paykina and Greenhill (2008) for the US.

little from the existing randomized controlled trials about the longer-term effects of pharmacological treatment on human capital accumulation in general.

There is some evidence of favorable long-term consequences of pharmacological treatment of ADHD on human capital accumulation based on observational methods. In a survey, Paykina and Greenhill (2008) report less school disruption, anti-social behavior and academic failure following pharmacological treatment, while others raise doubt about such effects (e.g. Mendez et al. (2011)). Regarding accumulation of health capital more specifically, we are aware of only one study by Marcus et al. (2008) that considers the link between pharmacological treatment and health. Their study uses a duration model to investigate the association between compliance in pharmacological treatment and injuries for a group of children in treatment. They find that children treated with high intensity had a non-significantly lower risk of injury than those treated with low intensity. Regarding crime outcomes, a recent study by Lichtenstein et al (2012) uses Swedish register-based data to document the association between treatment receipt in adulthood and crime for individuals with ADHD while exploiting that some individuals periodically drop out of treatment.

III. Empirical Framework

III.A *IV strategies*

The key problem when evaluating the effects of ADHD and its treatment on human capital formation is how to identify relevant counterfactuals. For every child in pharmacological treatment for ADHD, we would ideally like to know his or her outcome in the absence of treatment. Since this is never observed, we instead ask the question: how do we find a non-treated candidate who is similar in terms of observable and unobservable characteristics except for the fact the he is not being treated? In order to credibly identify causal effects, we exploit plausible exogenous variation in access to pharmacological treatment generated by variation in psychiatrists' propensity to prescribe: imagine two children, both diagnosed with ADHD and with the same characteristics; one will be treated but only because he meets a psychiatrist with preferences for using pharmacological treatment while the other does not. This strategy is inspired by Duggan (2005) and Doyle (2007, 2008) as described above.

Let Y indicate a human capital outcome such as criminal behavior. X is a set of observable characteristics that determines both the propensity to receive pharmacological treatment and the outcome. PH is an indicator for receiving pharmacological treatment for ADHD.

Consider now a random coefficient model a la Björklund and Moffit (1987):

(1) $$Y^i = X^i\beta + \alpha^i PH^i + \varepsilon^i$$

or alternatively

(2) $$Y^i = X^i\beta + \overline{\alpha} PH^i + \left[PH^i(\alpha^i - \overline{\alpha}) + \varepsilon^i\right] \quad ,$$

where the term in the squared brackets is the error term. Clearly, PH may be correlated with ε if, for example, an omitted variable such as parental preference leads to an increased likelihood of pharmacological treatment. Also, PH may be correlated with α if pharmacological treatment is based on expected (and foreseeable) gains. To solve this, we implement an instrumental variables strategy that exploits psychiatrists' propensities to prescribe.

Observable and unobservable child and parental characteristics (X and θ) may affect whether the child with ADHD receives pharmacological treatment (PH = 1), yet the prescribing specialist physicians are instrumental in making the decision; see details below. Let Z be a measure of the physician's propensity to prescribe. A model of pharmacological treatment could be:

(3) $$PH^i = 1\left[Z^i\gamma + X^i\delta + \theta^i > 0\right]$$

Had we had a binary instrument (some physicians have a high propensity to prescribe, whereas others have a low propensity), the IV analysis would provide us with the Local Average Treatment Effect (LATE) while assuming that the physician's propensity to prescribe positively affects the likelihood that a given child diagnosed with ADHD receives pharmacological treatment. This is clearly testable (formally, we test the null $\gamma = 0$). A second, un-testable, identifying assumption is that physicians do not affect children's outcomes beyond their choice of treatment. Therefore, it is crucial that individuals are not selective in their choice of physician; see discussion below. A final assumption (already imposed in (3) via the common coefficient γ) is monotonicity: if child i receives pharmacological treatment when met with a physician with a low propensity to prescribe, then child i must also receive treatment if met with a physician with a high propensity to prescribe.

Our instrument is, however, continuous rather than binary. With the IV strategy we therefore identify an Average Marginal Treatment Effect (MTE) rather than the usual LATE. This parameter is the average treatment effect for children on the margin of receiving pharmacological treatment.

A concern with our main instrument that exploits variation in the tendency to treat other children at the ward is so-called treatment by indication: physicians who are observed to treat more just do so because they meet children who suffer from more severe ADHD. We therefore test the robustness of our main results to using an alternative and novel instrument, namely the share of treated among other children *with weak symptoms* as proxied by a low number of pre-treatment injuries. This is precisely an attempt to hold fixed the severity of ADHD.

III.B *Individual level panel data strategy*

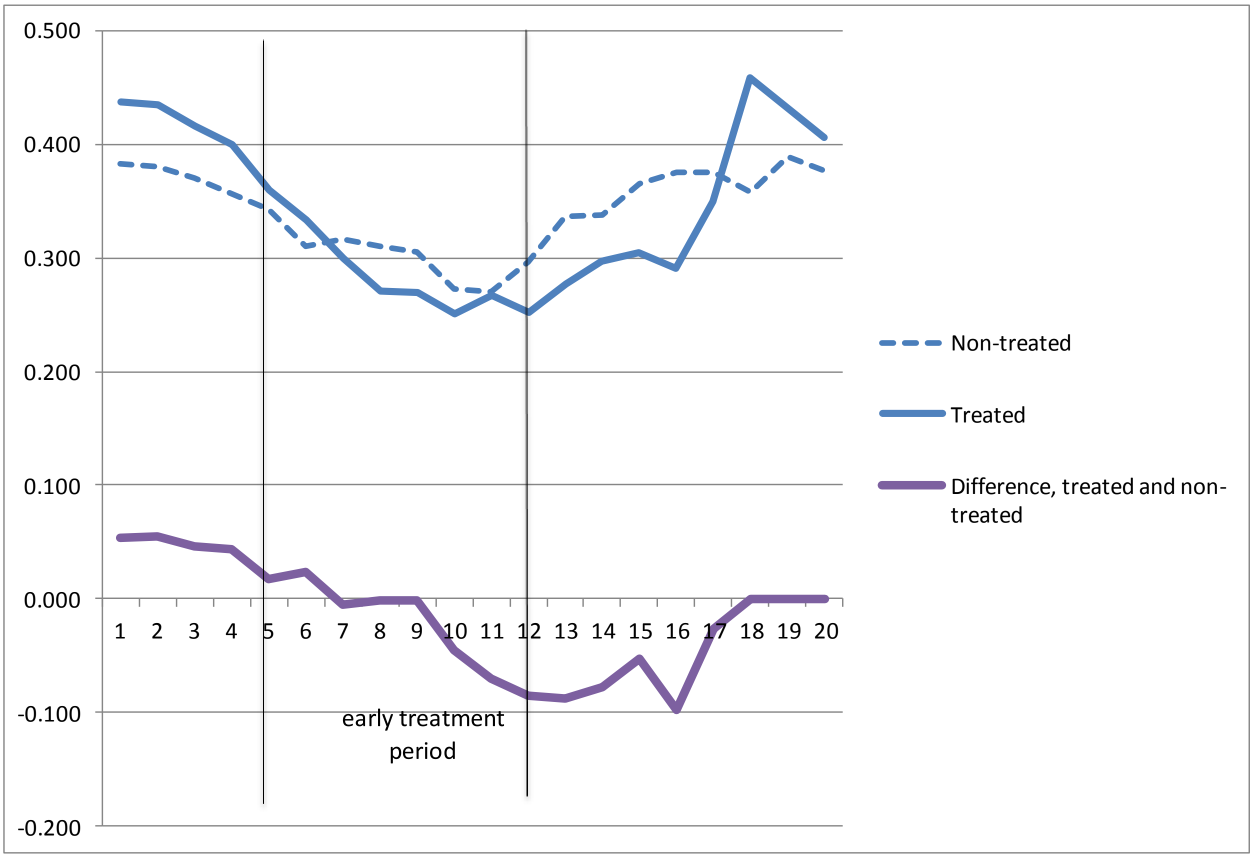
In practice treatment for 6 months or more per year is rarely initiated before the age of five and never before the age of four.[6] For health outcomes, where we have access to individual level panel data, we therefore also employ a difference-in-differences strategy, comparing outcomes for treated children prior to (age 4) and after treatment (in our main analysis age 10 +) with untreated diagnosed children before and after, corresponding to a fixed effects or first difference analysis; see Blundell and Costa Dias (2009). In a world with heterogeneous treatment effects, this will provide estimates of the average treatment effect on the treated (ATET). See Lechner (2011) for an extensive discussion of strengths and weaknesses associated with difference-in-differences strategies.

This identification strategy allows for selection into treatment based on, for instance, severity of symptoms or parental characteristics *as long* as these influences are constant over time. Thus, if particularly attentive parents are systematically more (or less) likely to engage in pharmacological treatment *and* more (or less) likely to use health care services at any time, this does not violate the identifying assumptions. However, if attentive parents are more (or less) likely to engage in pharmacological treatment *but only* more (or less) likely to use health care services when children are below 5, this would indeed violate the identifying assumptions.

[6] We exclude one child treated before the age of five.

FIGURE 1

PROBABILITY OF HOSPITAL VISITS BY TREATMENT STATUS*



Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Details about the measurement of the outcome and the sample follow below.

Our main regression model is the following:

$$Y_{ia} = \alpha_i + \beta_1 1(age > 9)_i + \beta_2 treat_i \cdot 1(age > 9)_i + \varepsilon_{it}$$

where Y is the outcome of interest, *treat* indicates that the child belongs to the treatment group (i.e. receives pharmacological treatment before age ten), $1(age > 9)$ indicates post-treatment age, ε is an error term, i indexes individuals, and a indexes age, β_2 is the parameter of interest. Note that since background variables are measured prior to or at childbirth and thus do not vary across time, they will be cancelled out along with the individual level fixed effect.

The key identifying assumption in a difference-in-differences set-up is that there can be no differential trends between the treatment and control group in the absence of treatment. Figure 1 illustrates the relevant identifying variation and main results for the probability of hospital visits in a given year.[7] The figure demonstrates that treated children have a higher probability of interacting with general hospitals prior to diagnosis and treatment than non-treated children, but that (except for the year of birth when children are very rarely exposed to injury) the development is otherwise parallel. After the diagnosis is established and treatment is initiated), this tendency is reversed, however. After the age of seven, treated children – who were initially more disadvantaged – perform better than non-treated children.

IV. Institutional Set-up

This section describes the decision stages and agents involved in diagnosing and treating ADHD. We consider three stages: The first step involves the seeking of a referral for evaluation at the specialist-level, the second step the establishment of a diagnosis, and the final step the treatment decision.

III.A *Seeking of a diagnosis and physician assignment*

Parents – and if not parents then in some cases teachers or school nurses – decide whether to seek a referral for evaluation in the first place. Typically, this involves a visit to the family's general practitioner (GP) who serves as a gatekeeper for specialist treatment. The GP can then – if he agrees with the indications – provide parents with a referral to a specialist, either employed at a child and adolescent psychiatric outpatient clinic at general hospitals or at a private clinic. In the vast majority of cases, relevant specialist physicians are child and adolescent psychiatrists, but pediatricians and neurologists also do assessments and diagnose.

In Denmark, consultations with the GP are free of charge (for the parents) as are those with specialist physicians when equipped with a referral from the GP. Whether patients end up with a specialist employed at general hospitals or at private clinics depends on the available specialist services in the area and whether the child and adolescent psychiatric outpatient clinic at the local general hospital is overbooked. Patients are assigned an available relevant physician at the psychiatric hospital or ward. It *is* possible to consult with a specialist at a private clinic without a GP reference, but parents must then pay the costs themselves.

[7] The picture is similar if we consider other outcomes. Contacts with general hospitals are shown in Figure A2 in the Appendix.

III.B *Diagnoses*

In Denmark, the *International Classification of Diseases* (ICD) diagnostic manual developed by WHO is used for diagnostic purposes.[8] Recently, the Danish Association for Child and Adolescent Psychiatry has published a so-called reference program for ADHD that examines the current evidence for diagnostic tools and treatment practices; see the Danish Association for Child and Adolescent Psychiatry (2008). Measurements of psychopathology such as the Child Behavior Checklist (CBCL) has been standardized in Danish (Bilenberg, 1999) and has been part of the standard clinical assessment in most child and adolescent psychiatric clinics in Denmark since the early 1990'ies. Multi-informants are always used in the assessment of children at hospital-based child and adolescent psychiatric units and a standard assessment often includes a direct observation of the child by a trained psychiatric nurse at the day-care/school and at home with the family is often part of the assessment and also a test of the cognitive level by a psychologist.

III.C *Pharmacological treatment*

Given an ADHD diagnosis, the specialist may recommend pharmacological treatment. This typically implies treatment with Methylphenidate and is the case for 98% of the children in our sample with an ADHD diagnosis established and in treatment before the age of ten. Methylphenidate is almost exclusively used to treat ADHD symptoms.[9] Medications used in the treatment of ADHD all act to increase brain catecholamine level. Although Methylphenidate has been used therapeutically for more than 60 years, the precise prefrontal cortical and subcortical mechanisms of action are poorly understood, but are associated with its ability to block the dopamine and norepinephrine re-uptake transporters (Solanto (1998)). It is well-known, however, that dopamine increases attention, interest and motivation. Common side effects are insomnia, headaches, decreased appetite, increased blood pressure and heart rate, and symptoms of depression and anxiety.

Parents may, of course, refuse pharmacological treatment. Thus, both the specialist a child meets, the severity of early symptoms, and parental preferences may impact on the likelihood of being

[8] ADHD is classified as an F90 diagnosis. This covers hyperkinetic disorders, activity and attention disorders, other hyperkinetic behavioral disorders, and hyperkinetic behavioral disorders without further specification. See WHO (1993).

[9] It may, however, also be used to treat the rare condition of narcolepsy.

treated. Regardless of the choice of pharmacological treatment and the severity of the condition, the reference program advises that children with an ADHD diagnosis are offered social skills training.

V. Data

Our starting point is the population of Danish children born in the period from 1990-1999. The main data stem from the Danish Psychiatric Central Register; see Munk-Jørgensen and Mortensen (1997) for a detailed description. These data include information about psychiatric history and diagnoses for parents and children diagnosed with Danish general hospitals. The data cover the period from 1960-2010 for the adult population but before 1994, information about children's psychiatric diagnoses was not available. Because it is extremely rare that children are diagnosed before age 4, we include children born as early as 1990.

The psychiatric registers are not constructed for research, but for administrative purposes. Diagnoses are therefore clinical diagnoses, not the result of a systematic well-described uniform psychiatric assessment. The validity of the diagnoses of ADHD in the Danish Psychiatric Central Register has previously been shown to be good, however. The agreement percentage on a full diagnosis of ADHD according to the American Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) was 89%, while the remaining 11% lacked only 1 symptom to fulfill the ADHD diagnosis (Linnet et al. (2009)).

Via unique personal identifiers, the information from the Danish Psychiatric Central Register is merged with registers containing rich socio-economic background variables (from 1980-2007), in-patient somatic disease histories (from 1980-2010), and prescription drug usage including data for both parents and children (from 1997-2010). Finally, we have educational information including indicators for whether children take course specific exams after 9th grade and the associated test scores up until 2010 in addition to information about individual level criminal behavior. Our crime data stem from two sources: a) the official crime register that records charges from the age of 15 (the age of criminal responsibility) up until 2011 and b) national police records of all interactions regardless of the age of the involved parties up until 2012.

V.A *Variables and samples*

We define *early pharmacological treatment* as purchases in an amount that corresponds to at least six months of treatment in a given year before the age of ten.[10] Pharmacological treatment of ADHD consists of Amphetamine (N06BA01), Methylphenidate (N06BA04), and Atomoxetine (N06BA09).

To assure that early treatment is relevant for all children in our sample, we select from the original data of children born 1990-1999 the 4,557 children who have been diagnosed with ADHD at Danish general hospitals before the age of ten. These children and their parents are clearly disadvantaged in terms of background characteristics as compared to their non-ADHD counterparts: children suffering from ADHD have worse birth outcomes, their parents have lower levels of education, are more likely to be unemployed and have lower income, are more likely to have a psychiatric diagnosis themselves and have a higher prevalence of both heart disease and respiratory disease and mothers are much more likely to smoke during pregnancy; see Appendix Table A1. Among children with an early diagnosis, we delete one child who was treated before the age of five to make sure that our measures of early injuries used to construct our second instrument and the complementary individual level panel data analysis are not contaminated. This gives us a final sample of 4,556 children.

Table 1 presents means of background characteristics by early pharmacological treatment status. Though some differences in background variables are statistically significant, it is not clear that treated children are either more advantaged or disadvantaged than non-treated children. One exception is that mothers of children in treatment are far more likely to have smoked during pregnancy than mothers of non-treated children and also to have respiratory diseases.[11] Among the advantageous characteristics, children in treatment have slightly higher 5-minute APGAR scores and are more likely to have a birthweight of 2,500 grams or more. Their parents are also slightly more likely to be employed.

[10] This corresponds to 182 defined daily doses (30 mg Methylphenidate) in a calendar year.

[11] See Obel et al. (2011), who use sibling differences to show that if smoking is a causal factor behind hyperkinetic disorders, it only has a minor impact.

TABLE 1

OBSERVABLE CHARACTERISTICS AT CHILDBIRTH, 4,556 CHILDREN BORN 1990-1999 WITH AN ADHD DIAGNOSIS ESTABLISHED BEFORE THE AGE OF TEN

Variable	Treatment before age 10		No treatment before age 10	
	Mean	Std. Dev.	Mean	Std. Dev.
Child:				
Boy (0/1)	0.86	0.35	**0.83**	0.37
5-minute APGAR score	9.78	0.86	**9.71**	1.20
Birthweight less than 1,500 grams (0/1)	0.02	0.12	0.02	0.14
Birthweight, 1,500-2,500 grams (0/1)	0.06	0.23	0.07	0.26
Birthweight, above 2,500 grams (0/1)	0.93	0.3	**0.91**	0.29
Complications at birth (0/1)	0.32	0.47	**0.27**	0.44
Gestation length (weeks)	39.28	2.53	39.17	2.53
Mental retardation (0/1)	0.12	0.33	0.11	0.31
Mother:				
Age at childbirth	27.93	4.85	28.24	5.28
High school or less (0/1)	0.50	0.50	0.53	0.50
Length of education (years)	11.40	2.31	11.41	2.30
Unemployed less than 13 weeks (0/1)	0.82	0.38	**0.79**	0.41
Unemployed 13-26 weeks (0/1)	0.12	0.33	**0.14**	0.35
Unemployed more than 26 weeks (0/1)	0.06	0.24	0.07	0.25
Employed in November (0/1)	0.54	0.50	**0.50**	0.50
Gross income (2004 prices)	176823	69947	172928	76761
Psychiatric diagnosis (0/1)	0.10	0.30	0.10	0.30
Heart disease (0/1)	0.03	0.18	0.03	0.17
Respiratory disease (0/1)	0.21	0.41	**0.18**	0.38
Smoker (0/1)	0.24	0.43	**0.16**	0.36
Father:				
Age at child birth	30.99	6.06	31.34	6.34
High school or less (0/1)	0.44	0.50	0.44	0.50
Length of education (years)	11.29	2.31	11.34	2.39
Unemployed less than 13 weeks (0/1)	0.86	0.35	**0.84**	0.37
Unemployed 13-26 weeks (0/1)	0.08	0.27	0.08	0.27
Unemployed more than 26 weeks (0/1)	0.06	0.24	**0.09**	0.28
Employed in November (0/1)	0.86	0.35	0.84	0.37
Gross income (2004 prices)	264250	128305	256665	152014
Psychiatric diagnosis (0/1)	0.08	0.27	0.08	0.26
Heart disease (0/1)	0.04	0.20	0.04	0.19
Respiratory disease (0/1)	0.14	0.35	0.13	0.34

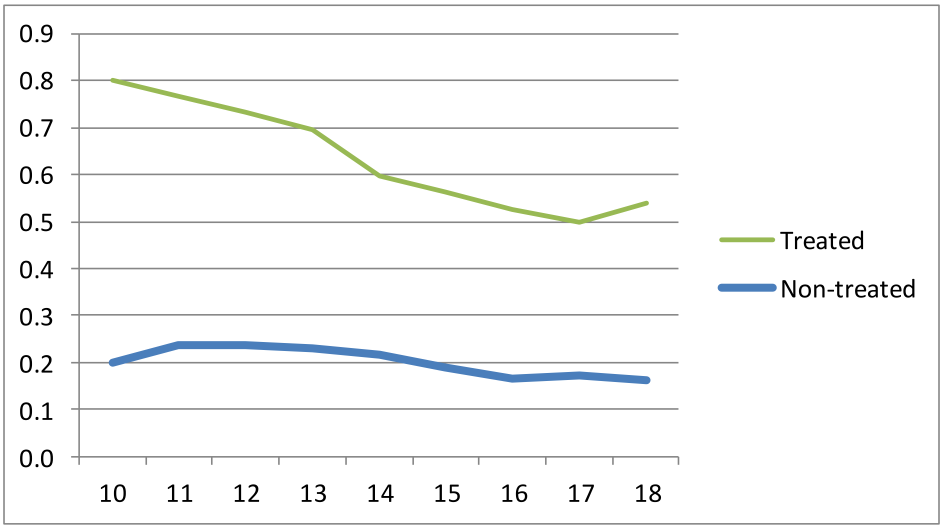
Notes: Bold indicates that mean for treated children is significantly different from the mean for non-treated children at the 5 % level. With the exception of mental retardation, which is diagnosed in connection with the ADHD diagnosis, all variables are measured in the year just prior to the birth of the child or in connection with childbirth. One child is excluded because of treatment before age 5.

Our sample corresponds to about 0.6 % of the children in the relevant cohorts. The share of a cohort diagnosed with ADHD before the age of ten has been increasing from 0.3 % in the 1990 cohort to 1.2 % in the 1999 cohort and boys are almost four times more likely than girls to receive an ADHD diagnosis; see Appendix Table A2. Similarly, among children diagnosed early, the share in early treatment increases across cohorts from 19 % in the 1990 cohort to 43 % in the 1999 cohort; see Appendix table A3.

Treated children may, of course, continue in treatment and also non-treated children may receive treatment later in life. This is important to be aware of when interpreting our formal results below. Figure 2 shows the share of children receiving any pharmacological treatment for ADHD at age ten or later by their early treatment status. We see that most children in early treatment continue after the age of nine but the share is declining with age. Children who are treated for less than six months before the age of ten (our non-treated children) also receive some pharmacological treatment at age ten and later but are much less likely to do so. Again, the share decreases with age. Strictly speaking, therefore, we measure the effects of early treatment initiation *including* the effects of continuing.

FIGURE 2

PROBABILITY OF RECEIVING TREATMENT FOR AT LEAST 6 MONTHS
AT A GIVEN AGE, 4,556 CHILDREN BORN 1990-1999 WITH AN ADHD DIAGNOSIS ESTABLISHED BEFORE THE AGE OF TEN.



Notes: Treatment status is defined as pharmacological treatment for at least six months in a year before the age of ten. One child excluded because of treatment before age 5.

While there were no large differences in background characteristics for the groups of treated and non-treated children, as seen in Figure 1 above treated children do exhibit more risky health behavior prior to treatment than non-treated children in that they have more contacts with general hospitals. Table 2 documents that treated children also have more early injuries than non-treated children.

TABLE 2

RELATIONSHIP BETWEEN PRE-TREATMENT INJURIES AND
EARLY TREATMENT RECEIPT, 4,556 CHILDREN BORN 1990-1999 WITH
AN ADHD DIAGNOSIS ESTABLISHED BEFORE THE AGE OF TEN

	ADHD medication before age ten		No ADHD medication before age ten	
Variable	Mean	Standard error	Mean	Standard error
At least one injury				
- at age 0	0.023	0.004	0.023	0.003
- at age 1	**0.162**	0.010	0.120	0.006
- at age 2	*0.218*	0.012	0.192	0.008
- at age 3	**0.238**	0.012	0.209	0.008
- at age 4	**0.231**	0.012	0.203	0.008

Notes: Bold (italic) indicates that means are significantly different at the 5 % (10 %) level.

We interpret this type of early health behavior as a proxy for the severity of ADHD in a child and we exploit this directly in the construction of the instrument based on psychiatrists' propensity to treat other children with less severe ADHD in the formal analysis below. Section VI.A will explain the instrument in details. Of course, it will also be important to control for early symptoms at the individual level.

Our estimations will consider three types of outcomes: health outcomes (one or more contacts with general hospitals at age 10, number of contacts with general hospitals at age 10; one or more contacts with the emergency ward at age 10; number of contacts with the emergency ward at age 10), [12] crime outcomes (one or more interactions and number of interactions with the police at ages 12-15 15; at least one charge at ages 16 and 17), and schooling outcomes (whether the child took 9th grade exam in Danish, the associated standardized exam grade, and the standardized end-of-year

[12] Excluding hospital visits associated with the treatment of ADHD

teacher assessment).[13] For the health outcomes that are measured at age 10, we will rely on all birth cohorts from 1990-1999 while analyses of crime and schooling outcomes will use only subsets of these cohorts. Information about charges at age 16, for example, is available until 2011 allowing us to use the 1990-1995 cohorts.

VI. Results

As discussed above, our main estimation strategy uses variation in physicians' propensity to prescribe pharmacological treatment to identify causal effects of treatment. This section first details the instrument and discusses ward assignment. After this, we present our analysis of the effects of pharmacological treatment. The last part of the section shows results on health outcomes from our complementary identification strategy that exploits individual level panel data.

VI.A *Ward assignment and variation*

Formally, we define our instrument as: for child *i* the share of treated among other children born in the same cohort and diagnosed at the same ward. Below, we also investigate whether results are robust to instead using: for child *i* the share of treated among other children born in the same cohort and diagnosed at the same ward *with less severe ADHD as approximated by a low number of early injuries and poisoning*. We define the number of early injuries and poisoning to be low if the sum of these before the age of five lies below the median. Table 2 above provides the rationale for using this measure as a proxy for severity.

To be included in our IV analysis, the ward must diagnose at least one other child born in the same cohort (at least one other child with less severe ADHD from the same cohort) when exploiting the propensity to treat other children (other children with less severe ADHD). In the *health sample* we rely on variation in the treatment propensity among 422 ward-cohort combinations with on average just above nine children per ward-cohort combination. Importantly, our analysis below includes regional and cohort fixed effects. Therefore, we essentially exploit variation in physician behavior (towards children born at the same point in time) relative to the behavior of other physicians in the broader region.

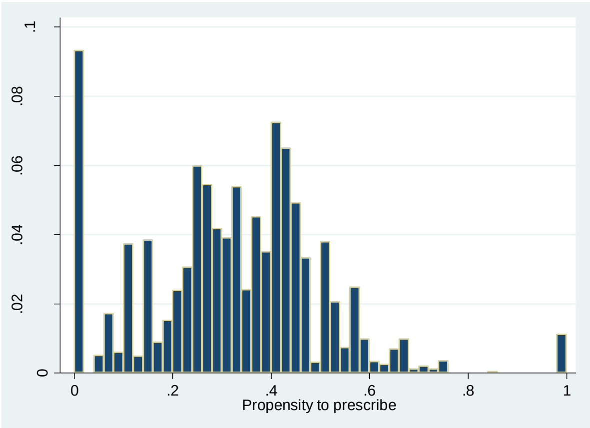
[13] When standardizing grades, we allow for cohort specific means and standard deviations.

In this section, we conservatively present the results for the instruments while relying on the larger *health sample.* Our conclusions do not change when considering the smaller estimation samples; in fact because of the smaller sample size, the set of tests that we apply to our instruments to render probable that the identifying assumptions are not violated obviously have less power with fewer observations.

Figure 3 shows the distributions of the propensity to prescribe. Clearly, there is very little support above values of 0.6. In practice, we will rely on variation in the instrument that lies strictly within the area of support and the results should be interpreted accordingly.

FIGURE 3

DISTRIBUTION OF PHYSICIANS' PROPENSITY TO PRESCRIBE (HOSPITAL LEVEL)



Notes: The propensity to prescribe is defined as the share of other children born in the same cohort and diagnosed at the same ward.

Table 4 shows how the instrument relates to background characteristics of the child and his family (and simple hospital characteristics). We simply regress the treatment propensity (at the child level) on the set of background variables and report the coefficient estimates. Standard errors are clustered at the cohort-ward level to allow for correlation between children diagnosed at the same ward and born in the same cohort. We find that although a few coefficients are statistically significant, most are small in size.

TABLE 4

OLS, CHILD AND PARENTAL BACKGROUND CHARACTERISTICS AND PHYSICIANS' PROPENSITY TO PRESCRIBE

	IV: Propensity to treat at ward	
	Coefficient Estimate	Standard Error
Child variables:		
Injury and poisoning* (DS00-DT98), age 0 (0/1)	0.004	0.016
Injury and poisoning* (DS00-DT98), age 1 (0/1)	-0.006	0.007
Injury and poisoning* (DS00-DT98), age 2 (0/1)	0.008	0.006
Injury and poisoning* (DS00-DT98), age 3 (0/1)	-0.004	0.006
Injury and poisoning* (DS00-DT98), age 4 (0/1)	-0.001	0.006
Boy (0/1)	-0.003	0.007
5-minute APGAR score	-0.020	0.023
Birth weight less than 1,500 grams (0/1)	*0.018*	0.025
Birth weight, 1,500-2,500 grams (0/1)	-0.016	0.011
Complications at birth (0/1)	0.007	0.007
Gestation length (weeks)	0.000	0.001
Mental retardation diagnosis (0/1)	0.003	0.009
Mother variables:		
Age at child birth	0.000	0.001
Length of education (years)	-0.002	0.001
Unemployed less than 13 weeks (0/1)	-0.003	0.007
Employed in November (0/1)	0.000	0.007
Gross income, kr. 100.000 (2004 prices)	**0.013**	0.005
Psychiatric diagnosis (0/1)	0.007	0.009
Heart disease (0/1)	-0.011	0.013
Respiratory disease (0/1)	-0.004	0.007
Smoker (0/1)	0.008	0.008
Father variables:		
Age at child birth	0.000	0.001
Length of education (years)	0.001	0.001
Unemployed less than 13 weeks (0/1)	-0.003	0.008
Employed in November (0/1)	-0.014	0.009
Gross income, kr. 100.000 (2004 prices)	0.001	0.002
Psychiatric diagnosis (0/1)	0.012	0.009
Heart disease (0/1)	0.020	0.013
Respiratory disease (0/1)	0.009	0.008
Sample size	4556	
R-squared	0.222	

Notes: Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Cohort and regional dummies included. Standard errors clustered at cohortXhospital level. One child excluded because of treatment before age 5.

TABLE 5

PROBIT, PROPENSITY TO DIAGNOSE AND PROPENSITY TO PRESCRIBE (COUNTY LEVEL)

Variable	Marginal effect	Standard error
Propensity to prescribe	0.0014	0.0011
Controls	YES	
Sample size	714677	

Notes: Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Conditioning set corresponds to the variables in Table 4 above. Cohort and regional dummies included. Standard errors clustered at cohortXcounty level.

As discussed above, one issue is treatment by indication. An indirect test of this phenomenon is to consider the relationship between the instrument and the propensity to diagnose at the population level: if living in an area where wards treat more heavily also implies that a child from the overall population is, for example more (or less) likely to be diagnosed in the first place, we would worry that high treatment wards see different types of children. Table 5 investigates this relationship. We estimate a probit for diagnosis receipt and condition on the full set of background variables in addition to the propensity to prescribe at the wards within the county (i.e. the wards a child living within the county would have a risk of meeting) and conclude that there is little relationship between the risk of receiving a diagnosis and the original instrument. The results imply that if the propensity to treat in county increases by 10 percentage points (relative to a mean of 0.32), this will increase the number of diagnoses relative to the mean with 0.00017/0.006= 2.8 %.

Another issue is whether wards that treat more early also differ in terms of other practices. We saw above that children who are treated early are also more likely to receive treatment later in their life. Table 6 documents that wards with a high propensity to prescribe also continue treatment longer. Table 7, on the other hand, documents that the instruments are not strongly associated with the use of the five most common other nervous system drugs. One coefficient is significant at the 5 % level and all are small size.[14] In the same way, we also investigate whether our instruments are associated with age at diagnosis and find no significant effects: an increase in age at diagnosis of one year is associated with a reduction in the overall propensity to prescribe of 0.002. Strictly speaking, therefore, our instruments measure the propensity to prescribe more early and to continue longer but

[14] Results are robust to excluding children who are treated with these five other types of nervous system drugs.

it is informative about the ADHD medication tendency alone. This is the treatment regime to have in mind when interpreting our results.

TABLE 6

OLS, PROPENSITY TO PRESCRIBE AND FUTURE TREATMENT

(SEQUENCES OF TREATMENT)

	Treatment age 13		Treatment age 14		Treatment age 15	
	Coefficient Estimate	Standard Error	Coefficient Estimate	Standard Error	Coefficient Estimate	Standard Error
Propensity to treat	**0.687**	0.052	**0.638**	0.060	**0.589**	0.069
Sample size	2544		1971		1480	

Notes: Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. One child excluded because of treatment before age 5.

TABLE 7

OLS, PROPENSITY TO PRESCRIBE AND THE FIVE MOST COMMON OTHER TYPES OF NERVOUS SYSTEM DRUGS

	IV: Propensity to treat at ward	
	Coefficient Estimate	Standard Error
Other types of nervous system drugs:		
Valproic acid (N03AG01), antiepileptic/migrain	**-0.039**	0.016
Lamotrigine (N03AX09), anti-epileptic	0.015	0.017
Risperidone (N05AX08), antipsychotic	-0.006	0.011
Clopenthixol (N05AF02), antipsychotic	0.030	0.019
Diazepam (N05BA01), anxiety/insomnia	-0.005	0.011
Sample size	4556	

Notes: Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Conditioning set corresponds to the variables in Table 4 above. Cohort and regional dummies included. Standard errors clustered at cohortXcounty level. One child excluded because of treatment before age 5.

We finally investigate strategic settlement*:* it is possible that some parents move to get access to pharmacological treatment via a ward with a higher propensity to prescribe if they believe strongly in a positive effect. This would be problematic for our estimation strategy because it would imply a potential correlation between the instrument and gains from treatment. First remember that our instrument is largely uncorrelated with parental and child characteristics suggesting that this issue is of minor importance. Second, while we cannot test strategic settlement directly, we can investigate the extent of movement across counties. Table 8 describes the moving patters in our sample. We order physicians' propensity to prescribe *at the county level*[15] from lowest to highest and allocate children to the lowest one-third, middle one-third, and highest one-third both according to where they were born and where they live at age nine (the last year of the treatment period). We see that the vast majority of children stay within the same county-type between birth and the age of nine. Furthermore, movement patterns are essentially symmetric and there is no pronounced tendency for families to move from counties where physicians prescribe less often to counties where physicians prescribe often. In our robustness analysis below we show that a) constructing our instrument at the county level instead in order to minimize local strategic choices and b) basing our instrument on place of birth instead of diagnosis do not change our results.

TABLE 8

MOVEMENT BETWEEN COUNTIES WITH LOW, MIDDLE AND HIGH PROPENSITIES TO TREAT

		County at age 9		
		Lowest one-third	Middle one-third	Highest one-third
County at birth	Lowest one-third	79.6	16.3	4.1
	Middle one-third	16.6	60.8	22.5
	Highest one-third	3.5	24.2	72.5

[15]It is not possible to do this on hospital level since we cannot allocate children to hospitals within counties in case of movements.

TABLE 9

FIRST STAGE RESULTS, HEALTH SAMPLE

	Coef. Est.	Std. Err.	Coef. Est.	Std. Err.
Instruments:				
Propensity to treat	**0.485**	0.054	**0.329**	0.067
Hospital variables:				
Big hospital (0/1)			0.011	0.014
A hospital treating only mild cases of ADHD (0/1)			0.016	0.029
Child variables:				
Injury and poisoning* (DS00-DT98), age 0 (0/1)			-0.028	0.045
Injury and poisoning* (DS00-DT98), age 1 (0/1)			**0.047**	0.021
Injury and poisoning* (DS00-DT98), age 2 (0/1)			0.000	0.018
Injury and poisoning* (DS00-DT98), age 3 (0/1)			0.007	0.017
Injury and poisoning* (DS00-DT98), age 4 (0/1)			*0.033*	0.018
Boy (0/1)			**0.053**	0.019
5-minute APGAR score			-0.013	0.059
Birth weight less than 1,500 grams (0/1)			**-0.128**	0.061
Birth weight, 1,500-2,500 grams (0/1)			-0.060	0.033
Complications at birth (0/1)			0.009	0.017
Gestation length (weeks)			-0.006	0.004
Mental retardation diagnosis (0/1)			0.034	0.022
Mother variables:				
Age at child birth			-0.003	0.002
Length of education (years)			-0.002	0.004
Unemployed less than 13 weeks (0/1)			-0.003	0.032
Unemployed 13-26 weeks (0/1)			-0.028	0.036
Employed in November (0/1)			0.025	0.021
Gross income, kr. 100.000 (2004 prices)			0.008	0.013
Psychiatric diagnosis (0/1)			-0.002	0.022
Heart disease (0/1)			0.034	0.037
Respiratory disease (0/1)			0.029	0.017
Smoker (0/1)			*0.041*	0.023
Father variables:				
Age at child birth			-0.002	0.001
Length of education (years)			-0.002	0.004
Unemployed less than 13 weeks (0/1)			0.019	0.029
Unemployed 13-26 weeks (0/1)			0.015	0.038
Employed in November (0/1)			0.020	0.026
Gross income, kr. 100.000 (2004 prices)			0.007	0.006
Psychiatric diagnosis (0/1)			0.013	0.027
Heart disease (0/1)			0.017	0.041
Respiratory disease (0/1)			0.005	0.024
Sample size	4287			
R-squared	0.027		0.052	

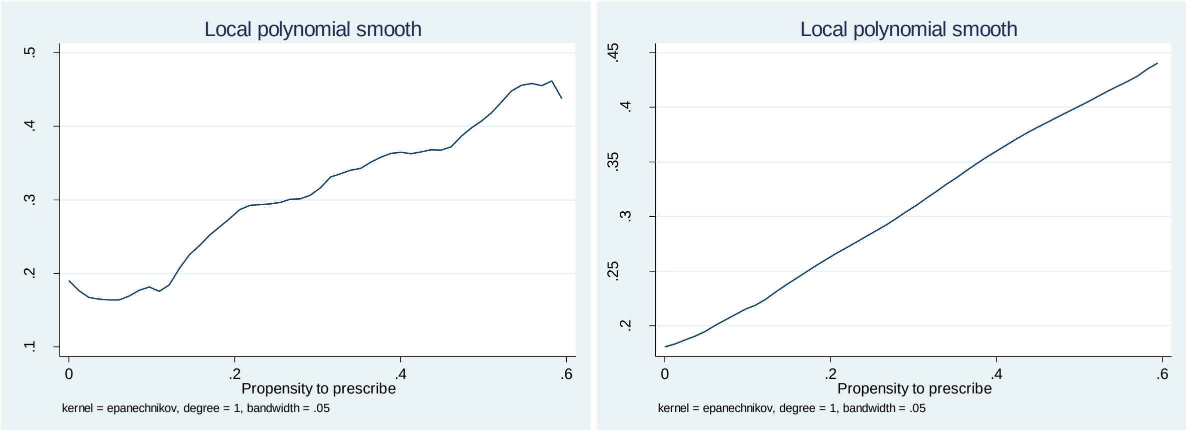
Notes: Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Cohort and regional dummies included. Instrument lies in 0-0.6 interval and wards are only included if they diagnose at least two children born in the same cohort. Standard errors clustered at cohortXcounty level. One child excluded because of treatment before age 5.

VI.B *First stage results*

Table 9 shows the first stage results for the *health sample*. We see that the relationship between the probability of treatment receipt and the instrument is positive: being exposed to a high-intensity treatment regime increases the probability that a given child is treated. Results are robust to conditioning on background characteristics. Figure 3 documents that the relationship between the probability of treatment receipt (actual and predicted) and the instrument is in fact monotonous over the support of the instruments. As argued by Doyle (2007), this provides some support for the monotonicity assumption discussed above.

FIGURE 3

PHYSICIANS' PROPENSITY TO PRESCRIBE AND THE PROBABILITY OF EARLY TREATMENT



Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Left graph shows results without control variables, right panel results with control variables (same as Table 9). One child excluded because of treatment before age 5.

VI.C *Effects of treatment on health, crime and schooling outcomes: OLS and IV Results*

We first investigate the effects of treatment on health outcomes. The top panel of Table 10 shows the main results from the OLS and IV analyses. We see that treatment is effective in reducing interactions with general hospitals at age 10 and that emergency ward visits significantly contribute to this. As is common, the OLS results are much smaller in size than the IV results.

TABLE 10

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10

		OLS		IV	
	Mean	(1)	(2)	(3)	(4)
		Health outcomes, age 10:			
First stage				**0.485**	**0.329**
				0.054	0.066
# Hospital contacts, age 10	0.498	*-0.026*	*-0.027*	**-0.651**	**-0.800**
		0.014	0.014	0.290	0.441
Hospital contacts, age 10 (0/1)	0.278	*-0.070*	**-0.074**	-0.134	-0.166
		0.034	0.033	0.091	0.156
# Emergency ward visits, age 10	0.233	*-0.034*	*-0.030*	**-0.285**	-0.280
		0.018	0.018	0.119	0.203
Emergency ward visits, age 10 (0/1)	0.181	-0.016	-0.014	*-0.150*	-0.109
		0.013	0.013	0.077	0.131
Control variables		NO	YES	NO	YES
Sample size		4556		4287	
		Crime outcomes, age 12:			
First stage				**0.485**	**0.329**
				0.054	0.066
# Police interactions age 12 (0/1)	0.041	0.006	0.004	-0.048	**-0.171**
		0.009	0.009	0.056	0.083
Police interactions age 12 (0/1)	0.033	0.002	0.001	-0.054	*-0.122*
		0.006	0.006	0.038	0.065
Control variables		NO	YES	NO	YES
Sample size		4556		4287	
		Crime outcomes, age 13:			
First stage				**0.485**	**0.329**
				0.054	0.066
# Police interactions age 13 (0/1)	0.080	0.030	0.029	-0.032	*-0.322*
		0.030	0.029	0.146	0.172
Police interactions age 13 (0/1)	0.045	0.003	0.002	-0.021	**-0.144**
		0.008	0.008	0.041	0.072
Control variables		NO	YES	NO	YES
Sample size		4556		4287	

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Standard errors reported below coefficient estimates. Cohort and regional dummies included. Instrument lies in 0-0.6 interval. Standard errors clustered at cohortXcounty level. One child excluded because of treatment before age 5.

TABLE 10 CONTINUED

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10

		OLS		IV	
	Mean	(1)	(2)	(3)	(4)
		Crime outcomes, age 14:			
First stage				**0.454**	**0.297**
				0.062	0.071
# Police interactions age 14 (0/1)	0.156	0.017	0.023	-0.105	-0.164
		0.038	0.036	0.218	0.372
Police interactions age 14 (0/1)	0.077	-0.009	-0.008	-0.051	-0.048
		0.009	0.009	0.060	0.106
Control variables		NO	YES	NO	YES
Sample size		3719		3554	
		Crime outcomes, age 15:			
First stage				**0.391**	**0.249**
				0.071	0.077
# Police interactions age 15 (0/1)	0.201	*0.084*	**0.093**	*-0.500*	-0.600
		0.044	0.042	0.278	0.504
Police interactions age 15 (0/1)	0.101	0.018	*0.020*	-0.171	-0.127
		0.012	0.012	0.111	0.179
Control variables		NO	YES	NO	YES
Sample size		3055		2914	
		Crime outcome, age 16:			
First stage				**0.356**	**0.214**
				0.097	0.101
Convicted age 16 (0/1)	0.040	-0.001	0.002	-0.114	-0.158
		0.010	0.010	0.090	0.175
Control variables		NO	YES	NO	YES
Sample size		1938		1826	

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Standard errors reported below coefficient estimates. Cohort and regional dummies included. Instrument lies in 0-0.6 interval. Standard errors clustered at cohortXcounty level. Grades and teacher evaluations standardized at cohort level. One child excluded because of treatment before age 5.

TABLE 10 CONTINUED

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10

		OLS		IV	
	Mean	(1)	(2)	(3)	(4)
		School outcomes:			
First stage				**0.373**	**0.229**
				0.097	0.103
Exam 9th grade, Danish (0/1)	0.379	**-0.101**	**-0.081**	**-0.601**	-0.293
		0.028	0.027	0.240	0.374
Control variables		NO	YES	NO	YES
Sample size		1735		1638	
Exam grade, 9th grade, Danish	0.017	0.126	0.160		
		0.113	0.123		
				N/A	
Teacher evaluation, 9th grade, Danish	0.026	-0.017	-0.026		
		0.104	0.107		
Control variables		NO	YES	NO	YES
Sample size		670			

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Standard errors reported below coefficient estimates. Cohort and regional dummies included. Instrument lies in 0-0.6 interval. Standard errors clustered at cohortXcounty level. Grades and teacher evaluations standardized at cohort level. One child excluded because of treatment before age 5.

The next set of results concern criminal convictions. The OLS results suggest no or even increases in criminal behavior due to treatment. This pattern is reversed in the IV analyses; here we find large and significant gains from treatment at ages 12-13. Parameter estimates are similar at older ages though, possibly because of smaller samples, primarily insignificant.

We finally investigate effects on school outcomes. Less than 40 % sit the exit exam in Danish, which is extremely low compared to the population mean of about 95 %. Importantly, both the OLS and IV analyses suggest that children are *harmed* from being treated; treated children are much less likely to enter the 9th grade exit exams, though the coefficient estimates from the specification that includes control variables are not significant. When considering exam results conditional on taking the exam, however, treated children fare at least as well as untreated children. Teachers, on the other hand, grade treated children slightly lower than untreated children. Of course, the differences between the coefficients associated with exam grades and teacher evaluations are not significantly different. Unfortunately, since a very low share of children diagnosed with ADHD take 9th grade exams, our IV strategies fail in these cases. Despite this, our results at least suggest that more treated children could take a 9th grade exam while still keeping the average test score among the treated at least as high as that for the untreated children.

To sum up, children seem to benefit from treatment with regards to outcomes such as hospital visits and crime that depend directly on the child's own behavior and are within his control, whereas they perform worse in terms of outcomes that are likely to be mediated by others such as school teachers and principals.

TABLE 11

SELF-REPORTED TEACHER INFORMATION AND INFLUENCE

Are there children in your classroom with ADHD?(N=46)	
Yes	0.370
No	0.630
Do you expect to be infored if one of your pupils has an ADHD diagnosis? (N=46)	
Yes, definitely	0.717
Yes, that would be natural	0.283
Maybe	0.000
No, not necessarily	0.000
No, definitely not	0.000
Do you expect to be informed if one of your pupils is in pharmacological treatment for his ADHD? (N=45)	
Yes, definitely	0.622
Yes, that would be natural	0.378
Maybe	0.000
No, not necessarily	0.000
No, definitely not	0.000
Is your recommendation important for whether a child takes exam after 9th grade? (N=44)	
Yes	0.409
Yes, partly	0.455
No	0.136

Notes: Teacher survey sent out to 99 schools in our local area (all school in the municipalities: Aalborg, Aarhus, Viborg, and Hedensted).

To learn more about teachers' level of information and their potential influence on our particular school outcome, we have carried out a teacher survey. As documented by Table 11, teachers self-report that they would usually be informed about ADHD diagnoses and pharmacological treatment and that they would have considerable influence on the decision of whether a given child should take the exam after 9th grade.

Threats to validity in the IV strategy

We documented above that our instrument measures propensities to prescribe more early and to continue longer but it is informative about the ADHD medication tendency alone. We also saw that there is little tendency for families to move to counties where wards treat more intensively. We formally address issues of treatment by indication and strategic settlement in this section. Due to space considerations, we restrict ourselves to the main health outcomes. The full set of results is available on request. First, we use our alternative instrument based on the propensity to treat children with less severe ADHD. Results are shown in column 3 in Table 12. Second, we address potential selection of wards within a county by defining our instrument at the county level instead of at the ward level. In this analysis, county is defined at the time of diagnosis (column 4). Finally, we investigate strategic settlement in the time between the birth of the child and the actual diagnosis by using variation in treatment propensities in the birth county (column 5). Results vary a little between the main specification and the three additional robustness analyses but the overall conclusion is the same.

TABLE 12

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10

SELECTED OUTCOMES, VARYING INSTRUMENTAL VARIABLE DEFINITIONS

		OLS	IV	IV less severe ADHD	IV county at diagnosis	IV county at birth
	Mean	(1)	(2)		(3)	(4)
			Health outcomes, age 10:			
First stage			**0.329**	**0.358**	**0.332**	**0.453**
			0.066	0.060	0.082	0.076
# Hospital contacts, age 10	0.498	*-0.027*	**-0.800**	*-0.601*	**-1.354**	**-0.704**
		0.014	0.441	0.354	0.641	0.436
Hospital contacts, age 10 (0/1)	0.278	**-0.074**	-0.166	-0.194	-0.278	-0.024
		0.033	0.156	0.122	0.200	0.144
Control variables		YES	YES	YES	YES	YES
Sample size		4556	4287	4256	4464	4522

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Bold indicates that coefficient is significant at the 5 % level and italic indicates significance at the 10 % level. Cohort and regional dummies included. Instruments lie in 0-0.6 interval. Standard errors clustered at cohortXcounty level. One child excluded because of treatment before age 5.

VI.D *Effects of treatment on health outcomes: Individual level panel data results*

As discussed above, our individual level panel data information on health outcomes allows for a difference-in-difference analysis. Table 13 shows the formal results. Again, due to space considerations, we conservatively show results for the indicator for interactions with general hospitals and emergency wards but other results are available on request. In line with the IV analysis, we find that early pharmacological treatment is effective in significantly improving health outcomes: the probability of being in contact with a general hospital at least once is reduced with 7 percentage points, corresponding to just below 30% of the mean at age 10.

TABLE 13

INDIVIDUAL LEVEL PANEL DATA RESULTS, EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10 ON HEALTH OUTCOMES

	OLS, no control vars.	OLS control vars.	Age 4 comparisons	Differential trends	Std. Errors clustered at countyXcohort	Placebo test age 4 vs. age 3
	(1)	(2)	(3)	(4)	(5)	(6)
			Health outcomes, age 10:			
# Hospital contacts, age 10	*-0.070*	**-0.074**	**-0.140**	**-0.149**	**-0.140**	
	0.034	0.033	0.050	0.050	0.048	
Hospital contacts, age 10 (0/1)	*-0.026*	*-0.027*	**-0.062**	**0.061**	**-0.062**	
	0.014	0.014	0.020	0.020	0.020	
# Emergency visits, age 10	*-0.034*	*-0.030*	**-0.070**	*-0.061*	**-0.070**	
	0.018	0.018	0.035	0.035	0.028	
Emergency visits, age 10 (0/1)	-0.016	-0.014	**-0.047**	**-0.041**	**-0.047**	
	0.013	0.013	0.018	0.018	0.018	
			Health outcomes, age 4:			
# Hospital contacts, age 4)						0.002
						0.068
Hospital contacts, age 4 (0/1)						-0.001
						0.029
# Emergency visits, age 4						0.019
						0.047
Emergency visits, age 4 (0/1)						0.009
						0.032
Sample size				4556		

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Age 4 comparisons in columns 4, 5, and 6. Differential time trends specification allows for different trends for boys, children with a birthweight above 2,500 grams, children whose mothers were employed prior to birth and who were smoking during pregnancy, children with at least one injury at age three, and counties. Bold (italic) indicates significance at the 5% (10%) level. Standard errors assume homoscedasticity unless otherwise noted. One child excluded because of treatment before age 5.

We perform a range of sensitivity checks of the main panel data results in column 3 in Table 13: We first investigate the issue of differential trends according to pre-treatment characteristics predictive of future treatment receipt (column 4). We next cluster standard errors at the cohort-county level to account for common shocks regarding hospital, pharmacological, pedagogical, or schooling practices for children born into the same cohort and county (column 5). Finally, calculating the differences in the estimated effects from regressions using age three and age four comparisons corresponds to performing a naturalistic and non-randomized placebo test of the effect of treatment on age four outcomes using age three outcomes as pre-treatment comparison (column 6). In addition to the sensitivity tests shown in Table 13, we have also investigated more thoroughly treatment initiation prior to the age of ten. In particular, we considered the effects of treatment initiation prior to the age of 7 (8 and 9) on outcomes measured at age 7 (8 and 9). This sensitivity analysis naturally leads to further placebo-type analyses where we exploit that treatment initiation after the age of 7 (8 and 9) must not affect previous outcomes. All sensitivity checks support our main results and the latter additional analysis is available on request.

V. Heterogeneity in Results

We finally investigate whether results vary across subgroups. We have performed analyses for boys, children born to mothers with more than a high school degree, children without a mental retardation diagnosis, children with a birthweight above 3,000 grams, children born to mothers who did not smoke during pregnancy, and children who are not in treatment with the five most common other types of nervous system drugs. Table A4 in Appendix shows the results for the main health outcomes. Our main conclusions are robust to these exercises and the full set of results is available on request. As pointed out above, many more children are diagnosed early and treated pharmacologically in the later cohorts. Two competing hypotheses may explain this development. First of all, it is possible that diagnostic tools have improved in recent years and that diagnosed cases born in later cohorts suffer from ADHD to the same extent as children born in earlier cohorts. In this case we will expect the effects of pharmacological treatment to be the same in early and late cohorts. Secondly, it is possible that the group of treated has been broadened to include children with less severe symptoms. In this case we will expect the effects of pharmacological treatment to decline in late cohorts. Table 14 investigates this for the larger health sample. We distinguish between the 1990-1994 cohorts and the 1995-1999 cohorts from the original sample. We see that estimated effects are smaller among later cohorts and significantly so in the panel data analysis. There are still significant gains from treatment in the later cohorts, but the results are at least in line with diminishing returns to broadening the group of treated.

TABLE 14

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE

AGE 10 ON HEALTH OUTCOMES IN EARLY AND LATER BIRTH COHORTS

		OLS	IV	Panel data analysis
	Mean	(1)	(2)	(3)
		Health outcomes, age 10, 1990-1994 birth cohorts:		
First stage			**0.214**	
			0.101	
# Hospital contacts, age 10	0.499	**-0.213**	-3.965	**-0.254**
		0.060	2.761	0.091
Hospital contacts, age 10 (0/1)	0.277	**-0.067**	-0.531	**-0.122**
		0.027	0.571	0.040
Control variables		YES	YES	YES
Sample size		1470	1385	1470
		Health outcomes, age 10, 1995-1999 birth cohorts:		
First stage			**0.366**	
			0.096	
# Hospital contacts, age 10	0.497	-0.021	-0.272	**-0.112**
		0.040	0.404	0.057
Hospital contacts, age 10 (0/1)	0.278	-0.010	-0.045	**-0.046**
		0.016	0.175	0.024
Control variables		YES	YES	YES
Sample size		3086	2902	3086

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Age 4 comparisons in column 3. Bold (italic) indicates significance at the 5% (10%) level. Standard errors clustered at cohortXcounty. One child excluded because of treatment before age 5.

VI. Conclusion

This paper investigates the effect of early pharmacological treatment of ADHD on children's human capital development while relying on register-based data on diagnoses and treatment for children born in 1990-1999. Our main analysis exploits variation in psychiatrists' propensities to prescribe treatment and a particular innovation of our paper is that we are to some extent able to control the severity of ADHD for the purposes of constructing the instrumental variable. Our analysis of effects on health outcomes allows for a complementary difference-in-difference analysis where we compare outcomes of treated children prior to (age four) and after treatment (age 10 +) with untreated diagnosed children before and after.

We find that the behavior of specialist physicians varies considerably across wards and that the prescribing behavior does affect the probability that a given child is treated. Results show that children diagnosed with ADHD on the margin of receiving pharmacological treatment have fewer hospital contacts if treated and that treatment to some extent protects against criminal behavior. We do not, on the other hand, find improvements in terms of the likelihood to complete secondary exam. In fact, treated children are significantly *less* likely to complete secondary school exams even though there is some evidence that they actually perform better when they take an exam when compared to children who similar in terms of observable characteristics.[16] That is, children seem to benefit from treatment with regards to outcomes that only depend directly on the child's own behavior and are within his control, whereas they perform worse in terms of outcomes that are likely to be mediated by others such as school teachers and principals. This is consistent with a hypothesis of social stigma associated with medication. In fact, our teacher survey confirms that teachers (self-report that they) know whether children diagnosed with ADHD receive pharmacological treatment and that they have substantial influence on the decision for a child to complete secondary exams. Of course, this does not preclude parents – or the children themselves – from affecting the decision.

We do find that estimated health effects are significantly smaller in later cohorts where more children are diagnosed and treated pharmacologically before the age of ten. There are still significant gains from treatment in the later cohorts, but these results are consistent with a hypothesis of diminishing returns to broadening the group of treated.

From the point of view of the individual, the family and the society, the long-term benefits of pharmacological treatment of ADHD extend beyond the relief of the symptoms related to the syndrome. These benefits should be traded off against the (low) financial costs of the drugs combined with the potential detrimental short- and long-term side effects of medication such as insomnia, decreased appetite and increased blood pressure.

[16] Unfortunately, since only 36 % of the children in our sample end up taking an exit exam, our IV strategy fails in this case due to small sample sizes and we have to rely on OLS.

This paper is the first to document the longer-term consequences of pharmacological treatment of ADHD on socioeconomic outcomes based on nationwide population registers for Denmark. Future work will investigate whether not only the individual suffering from ADHD is affected by its treatment; it may be that also parents, siblings, and peers in the classroom benefit.

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Appendix

TABLE A1

OBSERVABLE CHARACTERISTICS AT CHILDBIRTH, CHILDREN WITH AND WITHOUT AN ADHD DIAGNOSIS BEFORE THE AGE OF TEN

Variable	No ADHD diagnosis			ADHD diagnosis before age 10		
	# obs	Mean	Std. Dev.	# obs	Mean	Std. Dev.
Child:						
Boy (0/1)	710120	0.51	0.50	4557	**0.84**	0.37
5-minute APGAR score	654512	9.83	0.86	4331	**9.73**	1.10
Birthweight less than 1,500 gran	656115	0.01	0.08	4353	**0.02**	0.14
Birthweight, 1,500-2,500 grams	656115	0.04	0.20	4353	**0.07**	0.25
Birthweight, above 2,500 grams	656115	0.95	0.21	4353	**0.91**	0.28
Complications at birth (0/1)	710120	0.21	0.40	4557	**0.29**	0.45
Gestation length (weeks)	654637	39.59	1.92	4346	**39.21**	2.53
Mental retardation (0/1)	710120	0.00	0.06	4557	**0.11**	0.32
Mother:						
Age at childbirth	707357	29.05	4.81	4553	**28.14**	5.15
High school or less (0/1)	648886	0.39	0.49	4358	**0.52**	0.50
Length of education (years)	648886	12.25	2.52	4358	**11.41**	2.30
Unemployed less than 13 weeks	664613	0.83	0.38	4431	**0.80**	0.40
Unemployed 13-26 weeks (0/1)	664613	0.11	0.32	4431	**0.14**	0.34
Unemployed more than 26 week	664613	0.06	0.23	4431	**0.06**	0.24
Employed in November (0/1)	664576	0.63	0.48	4431	**0.51**	0.50
Gross income (2004 prices)	664576	188327	109197	4431	**174186**	74643
Psychiatric diagnosis (0/1)	707736	0.04	0.19	4553	**0.10**	0.30
Heart disease (0/1)	707736	0.03	0.16	4553	**0.03**	0.17
Respiratory disease (0/1)	707736	0.11	0.31	4553	**0.19**	0.39
Smoker (0/1)	562081	0.08	0.27	3945	**0.18**	0.39
Father:						
Age at child birth	684806	31.89	5.76	4277	**31.23**	6.25
High school or less (0/1)	632113	0.32	0.47	4064	**0.44**	0.50
Length of education (years)	632113	12.21	2.58	4064	**11.33**	2.37
Unemployed less than 13 weeks	651266	0.88	0.32	4197	**0.84**	0.36
Unemployed 13-26 weeks (0/1)	651266	0.06	0.23	4197	**0.08**	0.27
Unemployed more than 26 week	651266	0.06	0.24	4197	**0.08**	0.27
Employed in November (0/1)	651237	0.89	0.32	4197	**0.84**	0.36
Gross income (2004 prices)	651237	296103	199811	4197	**259101**	144851
Psychiatric diagnosis (0/1)	685964	0.03	0.18	4281	**0.08**	0.27
Heart disease (0/1)	685964	0.03	0.17	4281	**0.04**	0.20
Respiratory disease (0/1)	685964	0.09	0.29	4281	**0.14**	0.34

Notes: Bold indicates that mean for children with an ADHD diagnosis before the age of ten is significantly different from the mean for children without an ADHD diagnosis at the 5% level. With the exception of mental retardation, which is diagnosed in connection with the ADHD diagnosis, all variables are measured in the year just prior to the birth of the child or in connection with childbirth. Unemployed less than 13 weeks includes no unemployment.

TABLE A2

PERCENTAGE OF BIRTH COHORT DIAGNOSED WITH ADHD BEFORE AGE TEN

Birth Cohort	Cohort size	Percent with ADHD among all	Average age at diagnosis	Percent with ADHD among boys	Percent with ADHD among girls
1990	69,026	0.29	7.01	0.48	0.08
1991	69,667	0.37	6.85	0.63	0.10
1992	72,869	0.42	6.93	0.70	0.12
1993	72,227	0.44	7.03	0.73	0.14
1994	74,766	0.53	7.11	0.88	0.16
1995	74,342	0.63	7.24	1.04	0.19
1996	71,700	0.72	7.20	1.17	0.25
1997	71,342	0.84	7.22	1.42	0.23
1998	69,549	0.96	7.22	1.52	0.36
1999	69,189	1.21	7.17	1.90	0.49
All cohorts	714,677	0.64	7.14	1.04	0.21

TABLE A3

PERCENTAGE OF CHILDREN DIAGNOSED WITH ADHD IN PHARMACOLOGICAL TREATMENT FOR ADHD BEFORE THE AGE OF TEN

Birth Cohort	No. diagnosed	Percentage treated	Average age first treatment (in treatment before age 10)	Percentage treated among boys	Percentage treated among girls
1990	197	18.78	8.16	19.88	11.54
1991	257	19.84	8.14	21.43	9.09
1992	304	21.71	8.12	22.52	16.67
1993	318	22.33	8.14	22.30	22.45
1994	394	24.87	7.97	25.82	19.30
1995	468	28.21	8.19	30.08	17.39
1996	518	33.20	8.26	33.56	31.40
1997	599	34.39	7.93	35.07	30.00
1998	665	38.95	8.03	38.79	39.67
1999	837	43.61	7.93	45.54	35.76
All cohorts	4557	31.97	8.04	32.70	28.16

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten.

TABLE A4

EFFECTS OF PHARMACOLOGICAL TREATMENT BEFORE AGE 10 ON HEALTH OUTCOMES, BY OBSERVABLE CHARACTERISTICS

	Boys	Mentally retarded excluded	Mothers more than high school	Birthweight > 3000 g	Non-smoking mothers	No injuries at age 3	Not treated with other types of nervous system drug
IV analysis:							
First stage	**0.314**	**0.297**	**0.349**	**0.261**	**0.277**		**0.234**
	0.074	0.073	0.084	0.075	0.073		0.071
# Hospital contacts, age 10	**-1.286**	**-1.024**	-0.169	**-1.167**	*-1.154*		**-1.405**
	0.568	0.537	0.483	0.588	0.630		0.741
Hospital contacts, age 10 (0/1)	-0.258	-0.231	-0.162	-0.275	-0.152		-0.258
	0.187	0.190	0.209	0.223	0.205		0.241
Control variables	YES	YES	YES	YES	YES	YES	YES
Sample size	3606	3804	2173	3346	3620		3751
Panel data analysis:							
# Hospital contacts, age 10	**-0.109**	**-0.122**	**-0.159**	**-0.125**	**-0.149**	**-0.141**	**-0.128**
	0.044	0.057	0.055	0.051	0.046	0.064	0.052
Hospital contacts, age 10 (0/1)	**-0.052**	**-0.060**	-0.050	**-0.048**	**-0.066**	**-0.065**	**-0.067**
	0.021	0.023	0.032	0.020	0.025	0.020	0.026
Control variables	YES	YES	YES	YES	YES	YES	YES
Sample size	3829	4039	2300	3560	3831	3681	3998

Notes: Treatment is defined as pharmacological treatment for at least six months in a year before the age of ten. Age 4 comparisons in panel data analysis. Bold (italic) indicates significance at the 5% (10%) level. Standard errors clustered at cohortXcounty. One child excluded because of treatment before age 5.