Medical Augmentation of Labor and the Risk of ADHD in Offspring: A Population-Based Study

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abstract

BACKGROUND AND OBJECTIVE: Oxytocin for labor augmentation is widely used in obstetric care in Western countries. Two recent, smaller studies found opposing results regarding the association between prenatal exposure to oxytocin for labor augmentation and attention-deficit/hyperactivity disorder (ADHD). In Denmark, oxytocin is the medication used for nearly all medical augmentations of labor, and we examined the association between medical augmentation of labor and ADHD in a large cohort study based on national register data.

METHODS: All singletons born after spontaneous onset of labor in Denmark between 2000 and 2008 (N = 546 146) were included in the study. Data from the Danish Medical Birth Registry on medical augmentation of labor (yes/no) were used to identify exposed children. ADHD was defined based on the diagnostic codes of *International Classification of Diseases, 10th Revision*, for hyperkinetic disorder and information on dispensed ADHD medication. A multivariate proportional hazards regression model was used to test the association.

RESULTS: Among 546 146 deliveries, 26% included medical augmentation of labor, and 0.9% of the children were identified as having ADHD (n = 4617). We found no association between augmentation of labor and ADHD in the offspring (hazard ratio: 1.05 [95% confidence interval: 0.98–1.13]).

CONCLUSIONS: Our study does not support an association between medical augmentation of labor and ADHD in the child.

WHAT'S KNOWN ON THIS SUBJECT: Almost one-half of Danish first-time mothers with expected uncomplicated delivery receive oxytocin for labor augmentation. Oxytocin is listed as a drug with potential harmful effects, and recent studies suggest a possible association with subsequent attention-deficit/hyperactivity disorder in the child.

WHAT THIS STUDY ADDS: Using large nationwide registers, we were unable to detect any association between augmentation of labor and ADHD in offspring. Our findings do not support a causal role of perinatal exposure to oxytocin during delivery on the development of ADHD.

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Ms Henriksen contributed to the conception and design of the study, participated in the analysis and interpretation of data, and drafted the initial manuscript; Mr Wu was responsible for data management, register linkages, and statistical analyses and participated in the interpretation of data; Mr Secher conceptualized and designed the study, and reviewed the manuscript; Mr Obel contributed to the conception and design of the study, provided critical input and advice regarding the study design, and reviewed the manuscript; and Ms Juhl conceptualized and designed the study, participated in the analysis and interpretation of results, reviewed the manuscript, and supervised the junior first author. All authors approved the final manuscript as submitted, and all authors agree to be accountable for all aspects of the work.

The current study was approved by the Danish Data Protection Board (7-505-29-1056/2) and the Danish Serum Institute (6-8011-203/3).

Augmentation of labor with oxytocin, an artificially produced hormone, is a frequent intervention in Western obstetrics when progression of labor is considered insufficient (ie, dystocia). When used incorrectly, oxytocin in childbirth can lead to harm of both mother and child. Oxytocin is labeled as a high-alert drug by the US Institute for Safe Medication Practices,[1,2] which means that birth-attending midwives and physicians must demonstrate diligence before using this intervention. The majority of all deliveries in the United States are medically augmented[3]; in Denmark, 26% of all deliveries during the 2000–2008 period included medical augmentation of labor, and the proportion was 43% among nulliparous, low-risk women.[4] According to clinical guidelines, oxytocin is the choice of medication used for labor augmentation in Denmark.[5] In Sweden, 48% of all first-time deliveries included medical augmentation (1995–2002).[6] Oxytocin has an almost immediate and short-lasting effect on the uterine myometrium; recently, longer lasting effects have been suggested, such as a nonreversible influence on fetal brain development.[7,8] Kurth and Haussmann[7] found a doubled risk of attention-deficit/hyperactivity disorder (ADHD) in children exposed to maternal oxytocin labor augmentation in 88 ADHD cases compared with 84 control subjects. If confirmed, this finding may lead to a re-evaluation of the appropriateness of such widespread oxytocin use in obstetric practice.

In contrast, Silva et al[8] found a reduced risk of ADHD among girls, but not boys, who were prenatally exposed to labor augmentation with oxytocin. Given these conflicting results and the extensive use of oxytocin for medical augmentation, the goal of the present study was to examine the association between maternal medical labor augmentation and ADHD in offspring in Denmark. This large cohort was based on national register data with complete follow-up.

METHODS

Study Data

Using the Danish Medical Birth Registry (DMBR),[9] we identified all live-born singletons born in Denmark between 2000 and 2008 after spontaneous onset of labor (N = 546 146). Children exposed to medical labor augmentation were identified from the DMBR. Using the mother's and child's unique person identification numbers,[10] the DMBR data were linked to the Danish National Patient Register,[11] the Danish Psychiatric Central Register,[12] and the Danish Register of Medicinal Products Statistics[13] to determine subsequent ADHD in the child. The 546 146 live-born singletons constituted 97% of all singletons born after spontaneous onset of labor in Denmark between January 1, 2000, and December 31, 2008. Approximately 3% were excluded due to missing information on gestational age, maternal education, income, or cohabiting status or if the child was adopted or could not be linked to his or her biological mother.

Measurement of Exposure

Information on labor augmentation came from the DMBR, which contains information on all deliveries and newborns. Thus, the dichotomous variable "medical augmentation of labor" (yes/no) was used to determine exposure status (ie, whether a woman had been treated with any medication for labor augmentation during delivery). In theory, this variable may include medications other than oxytocin. However, in Denmark, oxytocin is used for nearly all medically augmented deliveries in line with the recommendations of the Danish Society of Obstetrics and Gynecology,[5] and this variable can therefore be considered a valid measure of prenatal oxytocin exposure.

Measurement of Outcome

ADHD is a diagnosis from the *Diagnostic and Statistical Manual of Mental Disorders*, an official diagnostic system for mental disorders in the United States. It is used in clinical practice in Denmark, but in the registers, we use *International Classification of Diseases, 10th Revision* (ICD-10) codes. Furthermore, some children in Denmark are treated outside the hospitals by specialists in child and adolescent psychiatry. We therefore used a combination of the ICD-10 diagnosis hyperkinetic disorder (F90) and dispensed ADHD-specific medication to identify children likely to have ADHD. From the Danish National Patient Register, which contains information on all hospitalizations for inpatients and outpatients, as well as emergency department visits, and from the Danish Psychiatric Central Register, which contains diagnose codes, date of onset and end of any treatment, and place of treatment for all patients treated in a psychiatric department in Denmark, we used information related to diagnoses and treatment of ADHD.

From the Danish Register of Medicinal Products Statistics, which contains information on the total sale of medical products classified according to the Anatomical Therapeutic Chemical (ATC) Classification System,[14] information was gathered on dispensed medication for ADHD. All children were identified for whom ADHD medication had been dispensed: ATC code N06BA04 (central nervous system–stimulating drug only, methylphenidate), ATC code N06BA07 (modafinil), or ATC code N06BA09 (noradrenaline reuptake inhibitors, atomoxetine).

Danish children with ADHD were defined as those having either an ICD-10 diagnostic code (F90) or having been dispensed at least ADHD-specific medication, or both. Date of first hospitalization, date of first outpatient visit, or date of first dispensed medication was used to define time of ADHD onset.

Statistical Analysis

A Cox proportional hazards model was used to calculate hazard ratios with 95% confidence intervals for offspring with ADHD according to maternal medical labor augmentation. Analyses were stratified according to child gender and by age at ADHD onset. The children were followed up from the day of birth until onset of ADHD, emigration, death, or end of follow-up by December 31, 2008, whichever came first. For multivariate analyses, the following prespecified covariates were included: maternal age at birth (<20, 20–24, 25–29, 30–34, 35–39, and ≥40 years), parity (1, 2, and ≥3), maternal education (low, middle, and high), cohabiting status (yes, no), parental income (quartiles), and the child's birth year (2000, 2001, 2002, 2003, 2004, 2005, 2006, 2007, and 2008). In addition, we included gestational age at birth in weeks (<33, 34, 35, 36, 37, 38, 39, 40, 41, and ≥42), and the child's birth weight was grouped into categories with equal numbers of children (≤2879, 2880–3119, 3120–3289, 3290–3419, 3420–3549, 3550–3679, 3680–3809, 3810–3989, 3990–4199, and 4200–7253 g). By the use of log–log plots, we verified that the assumption for proportional hazards was not seriously violated. All analyses were performed by using Stata version 11 (StataCorp, College Station, TX).

RESULTS

Table 1 shows maternal and child characteristics of all singleton deliveries after spontaneous onset of labor between 2000 and 2008 in Denmark. Among the 546 146 deliveries, medical augmentation of labor was used in 139 473 (25.5%), and 4617 (0.9%) of the children were subsequently diagnosed with or treated for ADHD. Of all the deliveries, when medical augmentation of labor was used, 68.0% were nulliparous women, and 29.4% were born at term (40 weeks of gestation). The statistical differences regarding maternal and child characteristics between the groups with and without medical augmentation of labor were tested. We found a statistically significant difference for all covariates (data not shown).

Table 2 presents the hazard ratios for ADHD when adjusted for medical augmentation, maternal age, parity, maternal education, cohabiting status, parental income, birth year, and different combinations of gestational age and birth weight. As shown in detail, the estimates did not change substantially even with adjustment for different combinations of covariates. An elevated risk of

TABLE 1 Medical Augmentation of Labor According to Maternal and Child Characteristics: Live-Born Singletons Born After Spontaneous Onset of Labor, Denmark, 2000–2008 (*N* = 546 146)

Characteristic	Without Medical Augmentation of Labor (*N* = 406 673)	With Medical Augmentation of Labor (*N* = 139 473)
Maternal age at delivery, y		
<20	5497 (1.3)	2462 (1.8)
20–24	42 209 (10.4)	18 015 (12.9)
25–29	134 571 (33.1)	51 882 (37.2)
30–34	150 971 (37.1)	46 117 (33.1)
35–39	63 131 (15.5)	17 884 (12.8)
≥40	10 294 (2.5)	3113 (2.2)
Parity		
1	140 133 (34.5)	94 837 (68.0)
2	175 007 (43.0)	29 840 (21.4)
≥3	91 533 (21.5)	14 796 (10.6)
Maternal education		
Low	109 407 (26.9)	39 041 (28.0)
Middle	127 532 (31.4)	42 405 (30.4)
High	169 734 (41.7)	58 027 (41.6)
Cohabiting status		
Not married[a]	165 507 (40.7)	68 848 (49.4)
Married[b]	241 166 (59.3)	70 625 (50.6)
Parental income (quartiles)		
1st (lowest)	27 366 (6.7)	11 020 (7.9)
2nd	135 550 (33.3)	45 237 (32.4)
3rd	179 821 (44.2)	60 584 (43.4)
4th	63 936 (15.7)	22 632 (16.2)
Child's birth year		
2000	48 032 (11.8)	15 032 (10.8)
2001	46 732 (11.5)	14 575 (10.5)
2002	45 287 (11.1)	15 080 (10.8)
2003	45 160 (11.1)	15 736 (11.3)
2004	45 042 (11.0)	15 833 (11.4)
2005	44 025 (10.8)	16 219 (11.6)
2006	44 714 (11.0)	15 903 (11.4)
2007	43 986 (10.8)	15 530 (11.1)
2008	43 695 (10.7)	15 565 (11.2)
Gestational age, wk		
<33	4801 (1.2)	492 (0.4)
33–36	17 841 (4.4)	4301 (3.1)
37	21 586 (5.3)	5716 (4.1)
38	60 307 (14.8)	13 150 (9.4)
39	96 261 (23.7)	27 103 (19.4)
40	112 310 (27.6)	41 016 (29.4)
41	69 494 (17.1)	32 244 (23.1)
≥42	22 208 (5.5)	15 207 (10.9)
Gender of child		
Boy	205 474 (50.5)	74 784 (53.6)
Girl	201 199 (49.5)	64 689 (46.4)

Data are presented as number (%).

[a] Single parenthood or widow.

[b] Married or living with new partner.

ADHD was found in Danish children when the mother was aged <25 years, when the mother had already given birth to >1 sibling, and when the parents were not married. Children born between 2001 and 2003 and children born before 32 weeks of gestation had an increased risk of ADHD. Furthermore, the risk of ADHD was associated with a birth weight <3000 g or >4100 g.

Table 3 presents the hazard ratios for ADHD in the offspring according to maternal medical augmentation of labor. With various multivariate adjusted Cox regression models, no association was found between medical augmentation during labor and subsequent ADHD in the offspring. Analyzing the data using medication exclusively or diagnosis codes exclusively revealed no association.

DISCUSSION

Using a nationwide cohort design with complete follow-up, no association was found between maternal medical augmentation of labor and ADHD in the offspring. Our findings are not in line with either of the 2 previous studies that reported a positive and a negative association, respectively, between augmentation of labor with oxytocin and ADHD in the offspring. The study by Kurth and Haussmann[7] found an association, which may be due to random error by multiple testing or to the rather small and highly selected population of 172 children. It may also be due to the fact that oxytocin used for both labor induction and labor augmentation counted as exposure. Our study had considerably higher statistical power, and we only included deliveries with a spontaneous onset of labor, because the indications for labor induction may confound the results; thus, only augmentation counted as exposure. Silva et al[8] found reduced odds of ADHD in girls (but not in boys) after medical augmentation of labor. The authors used a case-control

TABLE 2 Adjusted Cox Proportional HRs and 95% CIs for ADHD in Danish Children Prenatally Exposed to Mothers' Medical Augmentation of Labor Relative to Nonaugmented Deliveries: Live-Born Singletons Born After Spontaneous Onset of Labor, Denmark, 2000–2008 (N = 546 146)

Characteristic	Adjusted for Background Covariates (Maternal Age, Parity, Education, Cohabiting Status, Parental Income, and Birth Year)		Adjusted for Background Covariates + Gestational Age		Adjusted for Background Covariates + Birth Weight		Adjusted for Background Covariates + Gestational Age + Birth Weight	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Maternal age at delivery, y								
<20	2.18	1.86–2.56	2.17	1.85–2.56	2.16	1.84–2.54	2.17	1.84–2.55
20–24	1.39	1.28–1.52	1.40	1.28–1.52	1.40	1.28–1.53	1.40	1.29–1.53
25–29	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
30–34	0.89	0.82–0.96	0.88	0.82–0.95	0.89	0.82–0.96	0.88	0.82–0.95
35–39	0.89	0.80–0.99	0.88	0.79–0.97	0.88	0.80–0.98	0.88	0.79–0.97
≥40	0.90	0.72–1.13	0.88	0.70–1.10	0.87	0.70–1.10	0.86	0.69–1.09
Parity								
1	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
2	1.08	1.01–1.16	1.10	1.03–1.18	1.11	1.03–1.19	1.10	1.02–1.18
≥3	1.17	1.06–1.28	1.18	1.07–1.29	1.19	1.08 to1.30	1.17	1.06–1.28
Maternal education								
Low	0.99	0.92–1.07	0.99	0.92–1.06	0.99	0.92–1.06	0.99	0.92–1.06
Middle	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
High	0.72	0.67–0.77	0.72	0.67–0.78	0.72	0.67–0.78	0.72	0.67–0.78
Cohabiting status								
Not married[a]	1.38	1.30–1.47	1.38	1.30–1.47	1.38	1.29–1.46	1.37	1.29–1.46
Married[b]	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Parental income (quartiles)								
1st (lowest)	0.68	0.56–0.83	0.69	0.56–0.84	0.70	0.57–0.85	0.69	0.57–0.84
2nd	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
3rd	0.88	0.82–0.94	0.88	0.82–0.94	0.88	0.82–0.95	0.88	0.82–0.95
4th	0.73	0.65–0.82	0.74	0.66–0.82	0.74	0.66–0.83	0.74	0.66–0.82
Child's birth year								
2000	0.91	0.80–1.05	0.92	0.80–1.06	0.92	0.80–1.06	0.93	0.81–1.07
2001	1.05	0.92–1.21	1.07	0.93–1.23	1.06	0.92–1.22	1.08	0.94–1.24
2002	1.08	0.94–1.24	1.08	0.94–1.24	1.08	0.94–1.24	1.08	0.94–1.25
2003	1.13	0.98–1.31	1.14	0.98–1.31	1.13	0.98–1.30	1.14	0.98–1.31
2004	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
2005	0.81	0.65–1.01	0.82	0.66–1.01	0.82	0.66–1.02	0.82	0.66–1.02
2006	0.46	0.32–0.68	0.47	0.32–0.68	0.47	0.32–0.69	0.47	0.32–0.69
2007	0.52	0.29–0.92	0.52	0.29–0.92	0.53	0.30–0.93	0.53	0.30–0.93
2008	0.29	0.09–0.93	0.29	0.09–0.93	0.30	0.09–0.94	0.30	0.09–0.95
Gestational age, wk								
<32	NA	NA	1.67	1.33–2.10	NA	NA	1.42	1.12–1.80
33–36	NA	NA	1.05	0.90–1.24	NA	NA	0.96	0.81–1.13
37	NA	NA	Ref	Ref	NA	NA	Ref	Ref
38	NA	NA	0.80	0.70–0.91	NA	NA	0.83	0.72–0.95
39	NA	NA	0.70	0.61–0.79	NA	NA	0.73	0.64–0.83
40	NA	NA	0.65	0.58–0.74	NA	NA	0.67	0.59–0.77
41	NA	NA	0.66	0.58–0.76	NA	NA	0.58	0.59–0.78
≥42	NA	NA	0.63	0.54–0.73	NA	NA	0.63	0.54–0.75
Birth weight, g								
≤2879	NA	NA	NA	NA	1.59	1.41–1.80	1.27	1.11–1.46
2880–3119	NA	NA	NA	NA	1.06	0.93–1.21	0.98	0.86–1.12
3120–3289	NA	NA	NA	NA	0.96	0.83–1.10	0.91	0.79–1.04
3290–3419	NA	NA	NA	NA	0.95	0.83–1.09	0.92	0.80–1.05

TABLE 2 Continued

Characteristic	Adjusted for Background Covariates (Maternal Age, Parity, Education, Cohabiting Status, Parental Income, and Birth Year)		Adjusted for Background Covariates + Gestational Age		Adjusted for Background Covariates + Birth Weight		Adjusted for Background Covariates + Gestational Age + Birth Weight	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
3420–3549	NA	NA	NA	NA	1.00	0.87–1.14	0.99	0.86–1.13
3550–3679	NA	NA	NA	NA	Ref	Ref	Ref	Ref
3680–3809	NA	NA	NA	NA	0.93	0.82–1.07	0.94	0.82–1.08
3810–3989	NA	NA	NA	NA	1.04	0.91–1.19	1.06	0.93–1.21
3990–4199	NA	NA	NA	NA	1.04	0.91–1.19	1.06	0.93–1.22
4100–7253	NA	NA	NA	NA	1.14	1.00–1.29	1.18	1.04–1.34

CI, confidence interval; HR, hazard ratio; NA, not available.
[a] Single parenthood or widow.
[b] Married or living with new partner.

TABLE 3 Cox HRs and 95% CIs for ADHD in Danish Children Prenatally Exposed to Mothers' Medical Augmentation of Labor Relative to Nonaugmented Deliveries: Live-Born Singletons Born After Spontaneous Onset of Labor, Denmark, 2000–2008 (N = 546 146)

Variable	HR	95% CI
Crude estimate	1.02	0.96–1.09
Adjusted for background covariates (maternal age, parity, education, cohabiting status, parental income, and birth year)	1.01	0.94–1.08
Adjusted for background covariates + gestational age	1.06	0.98–1.13
Adjusted for background covariates + birth weight	1.03	0.96–1.10
Adjusted for background covariates + gestational age + birth weight	1.05	0.98–1.13

CI, confidence interval; HR, hazard ratio.

design, and the findings lacked statistical power.

The studies from Kurth and Haussmann[7] and from Silva et al[8] had conflicting results. We found no association between maternal medical augmentation of labor and ADHD in Danish children, and given the statistical power in the present study, we would expect to see an effect in 1 direction or another, if any strong association did exist. Data from Danish national registers are generated from the child's unique identification number assigned at birth, they are collected routinely, and any contact with the health care system is registered. We thus consider selection bias to be minimal. Furthermore, the fact that we used both diagnostic codes and information on ADHD-specific medications enhances the likelihood that we captured data on children with ADHD, thus securing the robustness of this study. Data on medical labor augmentation were based on a dichotomized variable in the DMBR; the register does not include information on dosage and administration of oxytocin throughout delivery, and we were therefore unable to analyze the total amount or the total duration of perinatal oxytocin exposure. Such analyses are required to rule out an association between labor augmentation and ADHD, and we recommend that future studies include a higher degree of detail in oxytocin exposure measure. Despite these limitations, our results provide an argument against an association between augmentation of labor with oxytocin and offspring ADHD. Our findings therefore do not support the hypothesis of a causal role of perinatal exposure to oxytocin during delivery on the development of ADHD.

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