■ CHILDREN'S ORTHOPAEDICS

The epidemiology of hip dysplasia in the nationwide Norwegian Mother, Father, and Child Cohort Study

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Aims

Developmental dysplasia of the hip (DDH) is a congenital disorder with several assumed risk factors, including breech presentation, female sex, and familial predisposition. Although several of these risk factors are included in national screening programmes, delayed diagnoses of DDH still occur. The aim of this study was to examine the prevalence of these and other risk factors in order to improve the current screening programmes.

Methods

This study used data from the Norwegian Mother, Father, and Child Cohort Study (MoBa) and the Medical Birth Registry of Norway (MBRN). We used descriptive statistics and logistic regression analyses. Children with cerebral palsy, syndromic disorders, or developmental delay were excluded from the study.

Results

A total of 107,194 children were included, with parents reporting using questionnaires if their child had a diagnosis of, or treatment for, DDH. A total of 3,460 children (3.2%) in MoBa had a diagnosis of DDH, with 1,453 (1.4%) being treated for DDH. Statistically significant risk factors included female sex, breech presentation, and pes equinovarus, whereas plural births and maternal diabetes were protective factors for DDH. Having a Caesarean section did not increase the prevalence of DDH.

Conclusion

We were able to confirm previously proposed risk factors such as breech presentation and female sex, whereas other variables such as plural births and Caesarean section were not found to be risk factors. However, regression analysis suggested that there are additional factors which affect the prevalence of DDH. These could be both environmental and genetic factors, highlighting the need for further research on DDH to improve the current screening programmes.

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Introduction

Developmental dysplasia of the hip (DDH) is a congenital disorder with a prevalence of 2% to 3% in neonates.[1,2] Several risk factors have been proposed, including both prenatal and perinatal factors. Some are well established, such as breech presentation, female sex, and familial predisposition.[3] Others, such as Caesarean section and associated deformities of the foot, have been more debated.[4] One of the primary theories about the risk factors for DDH is the so-called 'packaging theory', in which it is proposed that decreased intrauterine space increases the risk of DDH.[5] Parity, plurality, gestational age, oligohydramnios, and birth weight are among the factors included in this theory.[6] DDH can usually be treated during infancy with low-risk, noninvasive hip abduction braces such as a Frejka's pillow or a Pavlik harness. Delayed diagnosis can lead to the need for more invasive procedures, including surgery.

Although screening programmes for DDH have been established in many countries for decades, delayed diagnosis still occurs, even in countries with universal screening.[7,8] In Norway, the current

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screening programme for DDH is based on an ultrasound evaluation of the hips in neonates with abnormal clinical findings from Ortolani or Barlow tests and/or risk factors including breech delivery, foot deformities, or a familial disposition.[9,10] An increased knowledge of risk factors for DDH might lead to improvements in the current screening programmes, ensuring that fewer children experience diagnostic delay, and possibly allowing for a tailored length of treatment. Residual or recurrent DDH is associated with an increased risk of the development of early secondary osteoarthritis (OA) of the hip, a condition which severely affects quality of life in adulthood and often leads to the need for total hip arthroplasty (THA).[11–13]

The aim of this study, using a large, national study ('The Norwegian Mother, Father, and Child Cohort' (MoBa)), including > 300,000 individuals, of whom nearly 115,000 are children, was to investigate the risk factors for DDH.[14,15] All women in Norway who were pregnant between July 1999 and December 2008 were invited to join the study. The response rate was 41%. At the time of recruitment, there were about 55,000 births per year in Norway, according to Statistics Norway.[16] Parents answered questionnaires about themselves, the pregnancy, delivery, and (later) the child at regular intervals, including questions about DDH. We did a literature search and established the known and proposed risk factors which were also available from the MoBa cohort data. From this, we sought to examine risk factors for DDH and determine the prevalence of both DDH and any major risk factors in the MoBa cohort.

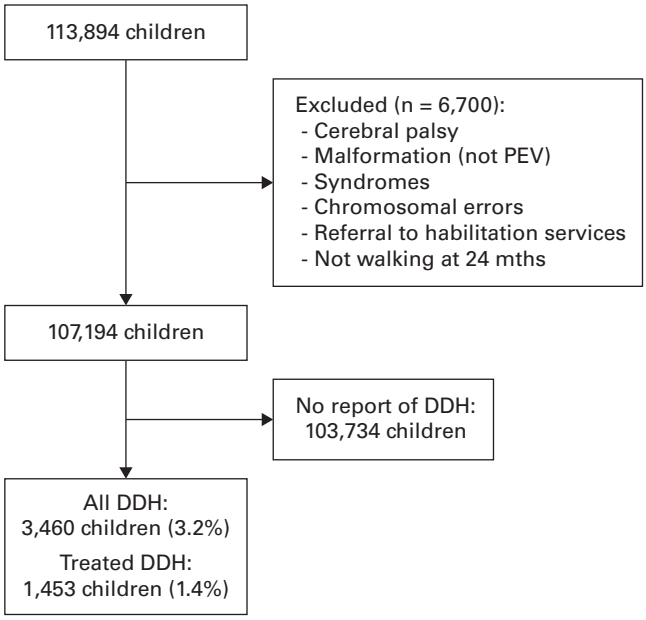

Fig. 1

Flowchart of the exclusion process in the Mother, Father, and Child Cohort Study cohort. The 1.4% of children reported to have had treatment for developmental dysplasia of the hip (DDH) (Treated DDH) are nested within the All DDH group reported to have DDH (3.2% of children in the cohort). PEV, pes equinovarus.

Methods

The MoBa is a population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health.[14,15] Participants were recruited from all over Norway during the study period, with an ongoing follow-up. The women consented to participation in 41% of all pregnancies during this period. The cohort included approximately 114,500 children, 95,200 mothers, and 75,200 fathers. The current study was based on v.12 of the quality-assured data files released for research in January 2019. The establishment of MoBa and the initial data collection were based on a licence from the Norwegian Data Protection Agency and approval from the Regional Committees for Medical and Health Research Ethics. The MoBa cohort is currently regulated by the Norwegian Health Registry Act. The Medical Birth Registry (MBRN) is a national health registry containing information about all births in Norway, where hospitals are obliged to report, and data from this registry was used in addition to the questionnaire data from the MoBa study. For this study, we used data concerning the birth such as parity, the foetal position, mode of delivery, congenital malformations, pes equinovarus (PEV) and anthropomorphic data. The study was approved on 15 August 2018 by the Regional Committee for Medical and Health Research Ethics in Western Norway (no. 24714).

The aim of the MoBa study is to gain information about the causes of diseases and disorders, including genetic causes, with questionnaires covering a wide spectrum of information. The first questionnaires are filled out by the parents during the pregnancy, and then successively as the child gets older. In this study, we looked at questions from questionnaires at 15, 21, and 30 weeks of pregnancy, and six, 18, and 36 months after birth. Although familial disposition is an important risk factor, this information was unfortunately not available in the data which were collected. Selective ultrasound screening was gradually introduced in Norway in the early 1990s, when children with risk factors such as familial disposition, breech presentation, and/or findings during the Ortolani or Barlow test started to be referred for an ultrasound examination of the hips.

Previously studied risk factors were selected based on the strongest evidence from the available literature, along with the availability in the MoBa cohort dataset. This included the maternal and paternal ages, sex, parity, gestational age, birth weight, mode of delivery, presentation, plurality, oligohydramnios, maternal diabetes, maternal folate and alcohol intake during pregnancy, PEV, and early postnatal changes in weight.[4–6,17–24] Descriptive statistics were used to determine the prevalence of DDH and various risk factors in the MoBa cohort and the MBRN. A difference in risk factors among males compared with females has previously been reported.[17] We therefore reported risk factors in the overall cohort and by sex, separately.[22]

In order to avoid losing statistical power, we grouped some questions: for example, alcohol use at various timepoints during the pregnancy was amended to 'any alcohol during pregnancy'. In terms of data related to treatment, the questionnaires at six and 18 months of age for the child included questions about the the treatment of DDH. The question about diagnosis in both questionnaires was "Does your child have, or has he/she had hip problems/hip dislocation?". With regard to treatment at six months, the question was: "Has your child been treated for a hip problem (hip dysplasia)?", while at 18 months it was: "Has your child been treated with a 'cushion' for a hip problem?",

Table I. Descriptive statistics of the risk factors for developmental dysplasia of the hip (DDH) in the Norwegian Mother, Father, and Child Cohort Study.

Characteristic	Study cohort		p-value†	Study cohort		p-value†
	No DDH	All DDH*		No treated DDH	Treated DDH‡	
Mean maternal age, yrs (SD)	30.13 (4.63)	30.33 (4.54)	0.012	30.13 (4.63)	30.65 (4.42)	< 0.001
Mean paternal age, yrs (SD)	32.72 (5.44)	32.93 (5.39)	0.023	32.72 (5.44)	33.13 (5.35)	0.005
Any type of diabetes, n (%)						
No	102,174 (98.51)	3,428 (99.08)	0.007	104,177 (98.52)	1,444 (99.38)	0.007
Yes	1,541 (1.49)	32 (0.92)		1,564 (1.48)	9 (0.62)	
Folate supplements during pregnancy, n (%)						
No	44,979 (43.37)	1,560 (45.09)	0.045	45,893 (43.40)	650 (44.74)	0.308
Yes	58,736 (56.63)	1,900 (54.91)		59,848 (56.60)	803 (55.26)	
Alcohol use during pregnancy, n (%)						
No	70,260 (86.82)	2,559 (84.88)	0.002	71,774 (86.79)	1,060 (83.93)	0.003
Yes	10,670 (13.18)	456 (15.12)		10,925 (13.21)	203 (16.07)	
Parity, n (%)						
None (primiparous)	45,181 (43.62)	1,572 (45.51)	0.027	46,106 (43.66)	655 (45.11)	0.267
One	37,457 (36.16)	1,166 (33.76)	0.004	38,123 (36.10)	508 (34.99)	0.381
Two	16,231 (15.67)	562 (16.27)	0.338	16,577 (15.70)	218 (15.01)	0.478
Three	3,594 (3.47)	115 (3.33)	0.658	3,655 (3.46)	55 (3.79)	0.499
Four or more	1,127 (1.09)	39 (1.13)	0.819	1,150 (1.109)	16 (1.10)	0.962
Plural births, n (%)						
Single birth	100,209 (96.62)	3,364 (97.23)	0.052	102,151 (96.60)	1,440 (99.11)	< 0.001
Multiple birth	3,506 (3.38)	96 (2.77)		3,590 (3.40)	13 (0.89)	
Presentation, n (%)						
Normal cephalic	93,513 (90.93)	2,802 (81.79)	< 0.001	95,219 (90.82)	1,111 (77.21)	< 0.001
Breech	4,162 (4.05)	477 (13.92)	< 0.001	4,377 (4.17)	268 (18.62)	< 0.001
Transverse	372 (0.36)	6 (0.18)	0.071	376 (0.36)	2 (0.14)	0.165
Anomaly cephalic	4,558 (4.43)	133 (3.88)	0.123	4,636 (4.42)	55 (3.82)	0.271
Other	235 (0.23)	8 (0.23)	0.952	240 (0.23)	3 (0.21)	0.872
Oligohydramnios, n (%)						
No	101,112 (97.49)	3,363 (97.20)	0.278	103,080 (97.48)	1,414 (97.32)	0.686
Yes	2,603 (2.51)	97 (2.80)		2,661 (2.52)	39 (2.68)	
Caesarean section, n (%)						
No Caesarean	88,682 (85.51)	2,811 (81.24)	< 0.001	90,367 (85.46)	1,140 (78.46)	< 0.001
Planned Caesarean section	5,557 (5.36)	293 (8.47)	< 0.001	5,719 (5.41)	132 (9.08)	< 0.001
Emergency Caesarean section	9,403 (9.07)	352 (10.17)	0.026	9,580 (9.06)	179 (12.32)	0.346
Unspecified Caesarean section	73 (0.07)	4 (0.12)	0.329	75 (0.07)	2 (0.14)	0.267
Pes equinovarus (clubfoot), n (%)						
No	103,570 (99.86)	3,452 (99.77)	0.161	105,595 (99.86)	1,446 (99.52)	0.001
Yes	145 (0.14)	8 (0.23)		146 (0.14)	7 (0.48)	
Child's mean weight, kg	3.57 (0.59)	3.61 (0.54)	< 0.001	3.57 (0.59)	3.66 (0.53)	< 0.001
Sex, n (%)						
Male	53,550 (51.72)	1,095 (31.70)	< 0.001	54,301 (51.44)	355 (24.45)	< 0.001
Female	49,992 (48.28)	2,359 (68.30)		51,262 (48.56)	1,097 (75.55)	
Mean gestational age, days (SD)	278.47 (14.69)	279.43 (11.41)	< 0.001	278.47 (14.64)	280.36 (10.80)	< 0.001
Mean slope for weight 0 to 6 mths of age, g/day (SD)	24.13 (5.68)	23.03 (5.63)	< 0.001	24.11 (5.68)	22.69 (5.42)	< 0.001

*Includes a parental report of a diagnosis of DDH.
†Tests assume equal variances. Tests are adjusted for all pairwise comparisons within a row of each sub-category using the Bonferroni correction.
‡Includes a parental report of the child receiving any type of treatment for DDH.

referring to the Norwegian practice of treatment with a Frejka's pillow. As versions of the question about treatment were slightly different with regard to treatment using a pillow, brace, or cast, we grouped the questions into 'No treatment' or 'Any treatment'. The individual growth slopes for weight from birth until approximately six months of age were calculated using the method described by Pfister et al,[25] with weight at birth and at about six weeks and three and six months being regressed against the age of the child at the time of the different measurements. We also chose to be inclusive when combining variables across several questionnaires, such as DDH treatment at six and 18 months, meaning that a 'yes' in either form was included. This choice was made based on the fact that the MoBa questionnaires are quite substantial and time-consuming to fill out, there being about 100 main questions with a risk that parents skip questions. We chose to include both outcomes ('All DDH'

Table II. Results of the logistic regression analysis for developmental dysplasia of the hip (DDH).

Variable	All DDH		Treated DDH	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Parity overall		0.274		0.709
Gestational age	1.00 (1.00 to 1.00)	0.792	1.01 (1.00 to 1.01)	0.069
Any type of diabetes (reference: no)	0.68 (0.46 to 1.01)	0.054	0.36 (0.16 to 0.81)	0.013
Folate supplements during pregnancy (reference: no)	0.86 (0.79 to 0.93)	< 0.001	0.88 (0.78 to 0.99)	0.034
Presentation overall		< 0.001		< 0.001
Breech presentation	4.54 (3.95 to 5.22)	< 0.001	6.95 (5.74 to 8.42)	< 0.001
Transverse presentation	0.73 (0.30 to 1.78)	0.484	0.46 (0.06 to 3.35)	0.447
Anomaly cephalic presentation	0.98 (0.81 to 1.20)	0.866	0.91 (0.67 to 1.24)	0.563
Other presentations	1.72 (0.84 to 3.54)	0.140	1.58 (0.50 to 5.03)	0.436
Caesarean section overall		0.812		0.248
Oligohydramnios (reference: no)	1.26 (1.00 to 1.59)	0.051	1.15 (0.80 to 1.66)	0.444
Plurality (reference single birth)	0.68 (0.53 to 0.87)	< 0.001	0.19 (0.10 to 0.36)	< 0.001
Sex (reference: male)	2.30 (2.12 to 2.50)	< 0.001	3.55 (3.08 to 4.10)	< 0.001
Child's weight (kg)	1.27 (1.16 to 1.39)	< 0.001	1.45 (1.26 to 1.66)	< 0.001
Slope for weight 0 to 6 months	0.98 (0.98 to 0.99)	< 0.001	0.98 (0.97 to 0.99)	< 0.001
Maternal age (years)	0.99 (0.98 to 1.00)	0.149	1.01 (0.99 to 1.03)	0.294
Paternal age (years)	1.01 (1.00 to 1.02)	0.057	1.01 (1.00 to 1.03)	0.210
Pes equinovarus (clubfoot; reference: no)	1.72 (0.77 to 3.82)	0.183	3.05 (1.18 to 7.89)	0.021
Alcohol consumption during pregnancy (reference: no)	1.14 (1.02 to 1.27)	0.018	1.21 (1.03 to 1.42)	0.022
Constant	0.01	< 0.001		< 0.001

OR, odds ratio.

and 'Treated DDH') in further analysis. The All DDH group probably contains children with immature hips who are monitored with further ultrasound appointments but are not treated for DDH.

Dysplasia of the hip may also be seen among various other disorders and syndromes. In order to avoid conflating these types of dysplasia with DDH, we excluded children with cerebral palsy, chromosomal disorders, congenital malformations apart from PEV, and 'other syndromes'. Children who were reported by their parents to have been referred to rehabilitation services, and those reported to have been not walking by the age of > 24 months, were also excluded. PEV was the only malformation among those previously investigated in relation to DDH specifically mentioned in either MoBa or MBRN.

Statistical analysis. Each risk factor was analyzed for significant differences between the DDH and non-DDH groups by column-wise proportion comparison, using Bonferroni correction with a p-value of 0.05 as the threshold for significance. Logistic regression analyses were performed to evaluate each factor and their contribution in a complete model of risk factors. We used binary logistic regression entering all variables at once and reporting a Hosmer-Lemeshow test of fit and Nagelkerkes pseudo-R^2 as an estimate of the proportion of variance explained by the model. As for the descriptive statistics, we also stratified regression analyses by sex.[22] Results are reported as odds ratios (ORs) with 95% CI and accompanying p-values. All analyses were performed using SPSS software v. 29 (IBM, USA). The threshold for significance in all analyses was $p < 0.05$.

Results

Questionnaire data were available for 113,894 children. The question regarding hip dysplasia at six months of age was used in relation to exclusion criteria, as it had minimal missingness and represented a broad phenotype including both immature hips and DDH. A total of 6,700 children were excluded, of whom 548 (8.1%) had reported hip dysplasia. A total of 107,194 children remained for further analysis (Figure 1).

From the question "Does your child have or has he/she had hip problems/hip dislocation?" at six months, a total of 3,460 children were identified, corresponding to a prevalence of 3.2% (All DDH). Treatment for hip dysplasia (Treated DDH) was reported for 1,453 children with a mean duration of treatment of 3.55 months (SD 1.78), representing 1.4% of the total cohort. Thus, the questionnaire provided information about how many children's parents reported their child as having DDH (All DDH) and how many were reported to have had treatment for DDH (Treated DDH). However, out of the 1,453 children reported as Treated DDH only 1,253 were reported as having DDH (All DDH), resulting in a rate of treatment (Treated DDH/All DDH) of 36%. When including the 200 children who were reported as having been treated for DDH without having been reported as having DDH, the rate of treatment increased to 39%.

Significant findings included a proportion of females in the All DDH group of 68.3%, increasing to 75.6% in the Treated DDH group, whereas the non-DDH groups had approximately 48% of females. Breech presentation was significantly more prevalent in the DDH groups (13.9% in All DDH and 18.6% in Treated DDH), compared with about 4% in non-DDH groups. The prevalence of PEV was 0.5% in the Treated DDH group compared with 0.1% in the non-DDH group. There was a significantly decreased proportion of maternal diabetes (0.9% vs 1.5%), decreased folate intake (54.9% in the DDH group vs 56.6% in non-DDH group), and increased alcohol intake (15.1% vs 13.2%) in mothers of children with All DDH. There was a significantly higher proportion of primiparous mothers in the All DDH group (45.5% vs 43.5%), and plural births

were significantly less common in the DDH group, at 2.8% of births compared with 3.4% in the non-DDH group. Some of the statistically significant risk factors did not have large absolute differences, including parental age (30.3 vs 30.1 years for mothers and 32.9 vs 32.7 years for fathers), gestational age (279 vs 278 days), and birth weight (3.61 kg vs 3.57 kg). The prevalence of risk factors stratified by sex can be found in Table I.

The logistic regression model included the following risk factors: parental age, maternal diabetes, folate use, parity, plurality, presentation, oligohydramnios, Caesarean section, PEV, birth weight, sex, gestational age, and growth during the first six months of life. Table II shows the results of these analyses. Those stratified by sex are shown in Supplementary Table i. The model had a good fit for All DDH, with a Hosmer-Lemeshow p-value of 0.890, but low explanatory power (Nagelkerke R^2 5.1%). For Treated DDH, the model explained slightly more variance (8.4%). Significant risk factors for All DDH included folate use, presentation, plurality, sex, weight, growth, and alcohol use (Table II). For the Treated DDH group, maternal diabetes and PEV were also significant risk factors (Table II). The OR for females compared with males was 2.3 (95% CI 2.1 to 2.5) in All DDH and 3.5 (95% CI 3.1 to 4.1) in Treated DDH. Breech presentation had an OR of 6.9 (95% CI 5.7 to 8.4) in Treated DDH, increasing to 7.2 (95% CI 4.9 to 10.5) in treated males. For PEV, the OR in the treated group was 3.1 (95% CI 1.2 to 7.9; p = 0.021), increasing to 5.5 (95% CI 1.6 to 18.3) in treated males.

Sex-stratified analyses revealed that folate use, plurality, and early growth were not significant factors in males, whereas alcohol use was not a significant factor in females. Oligohydramnios and paternal age became significant in females; no risk factors became significant in males. In the Treated DDH group, maternal diabetes, folate use, birth weight, and growth were not significant factors in males, whereas folate use had borderline significance and alcohol use was not significant in females. The details of the logistic regression model stratified by sex are shown in Supplementary Table ii.

Discussion

We examined the prevalence of parent-reported DDH in the Norwegian MoBa Cohort in this study, and identifed several factors which were significantly associated with the prevalence of DDH in early life, including several known risk factors such as breech delivery, gestational age, sex, and Caesarean section.

We found a prevalence of DDH of 3.2% based on parent-reported diagnosis (All DDH). This decreased to 1.4% when based on parent-reported treatment for DDH (Treated DDH). A recent meta-analysis showed a large variation in the prevalence of DDH in different areas of Europe. Within Sweden, the prevalence ranged from 3.5 to 10.0 per 1,000 live births.[26] The prevalence of DDH for the All DDH group was at the higher end of previous reports, whereas the prevalence for the Treated DDH group was closer to that reported in other studies.[26] This probably reflects the fact that hips which warranted one or more ultrasound examinations without being treated were being reported as DDH by parents. This compares well with previous findings in a meta-analysis in which the prevalence of DDH at birth was 4.2%, decreasing to 2.9% after four to six weeks.[27]

The MoBa data support the well-established risk factor of female sex, consistent with previous large meta-analyses showing a relative risk (RR) for females of 2.54 (95% CI 2.11 to 3.05) and an OR of 2.50 (95% CI 1.74 to 3.59).[21,28] Previous studies have looked at the influence of oestrogren and relaxin without conclusive results.[27,29] However, the level of oestrogen receptors in the ligaments and capsule of the hip joint are increased in infants and children with DDH, and the children of women with pelvic instability have an increased risk of DDH, suggesting that the level of receptors or affinity play a role, rather than the levels of hormones.[6,27]

Another strongly established risk factor for DDH is breech presentation, with a RR of 3.75 and OR of 4.15 in two large meta-analyses.[21,28] We found a prevalence of breech presentation consistent with previous results in Norway, Denmark, and Finland.[27] Among the Treated DDH group, breech presentation had a high OR at 6.9, increasing to 7.2 in males. This is substantially lower than in a UK study, in which the OR was 24.3; however, the exclusion criteria in the two studies may have differed.[5] Breech presentation is associated with several complications of pregnancy, and the issue of cause and effect in relation to DDH and breech presentation under these circumstances can be questioned.[30–32] One theory is that hyperflexion of the hips, especially with straight knees (frank breech), puts pressure on the hips and leads to ligamentous and capsular laxity and acetabular dysplasia.[27,33] A reverse cause and effect theory is that abnormal hips may predispose to a breech presentation as opposed to healthy hips, as seen in children who are born breech with normal postnatal ultrasonography and clinical examination who subsequently develop a shallow acetabulum after three years of age.[3] These findings suggest that the mechanisms responsible for DDH and breech presentation continue to affect the hip after birth, supporting the need for long-term follow-up of infants with breech presentation.[3]

Both planned and unplanned delivery by Caesarean section has a higher prevalence in All DDH groups, with a statistically significant difference in most sub-groups of type of DDH and sex. Caesarean section is, however, likely to be a secondary outcome rather than a primary risk factor. In Norway, the rate of planned Caesarean section for breech presentation is high at 53% compared with 6% for cephalic presentation.[34] Caesarean section has previously been cited as being a risk factor for DDH, but was not a significant factor in the latest meta-analysis (RR 1.22, 95% CI 0.46 to 3.23), nor in our logistic regression models, suggesting that it is a proxy variable for breech presentation or the complications of pregnancy which lead to DDH.[21] Similarly, Woodacre et al[5] found a crude increased risk with Caesarean section, which did not remain in their logistic regression analysis. This agrees with the view that a vaginal birth even with a breech presentation is unlikely to cause DDH.[35,36]

Being the first-born child was significantly more common in the All DDH group, but not in Treated DDH, nor in any of the logistic regression analyses, indicating a higher risk for having immature hips rather than DDH requiring treatment. Our findings contrasts with previous findings of a RR of 1.44 for first-born children, yet other studies report varying prevalences of first-borns among children with DDH.[5,21,27,29] There was a statistically increased mean gestational age in DDH groups, but the difference

in the mean was only one to two days. Similar results were seen for birth weight, with a small but significant difference which remained in the regression models. This is consistent with the findings of Tirta et al,[28] in which a high birth weight had an OR of 2.00 for DDH. Gestational age was not a significant risk factor in any of the regression models, suggesting its role as a confounder.

In contrast to the 'packaging theory', we did not find that oligohydramnios or plural birth were risk factors. Children from plural births were in fact less likely to have DDH. The latter finding might be explained by the fact that most plural pregnancies in Norway do not last to term, but are ended by induction or Caesarean section in weeks 36 to 39, depending on the choriocity.[37] In the MoBa cohort, the mean gestational age for plural births is 255 days, as opposed to 279 for single births. Oligohydramnios did become a significant risk factor among females in the All DDH group, consistent with the study by Onay et al,[22] who found a significant increase in female children in Turkey. However, this is inconsistent with the results from a large meta-analysis in which oligohydramnios had an OR of 3.76.[28] These disparities may be due to differences in the prevalence of oligohydramnios and the underlying relationship between oligohydramnios and DDH.

Foot deformities, including PEV, have been much debated as a risk factor for DDH.[38] Håberg et al[4] found that children with a foot deformity had an increased risk of DDH, thus recommending ultrasonography for children born with foot deformities. In our study, there were only 153 children with PEV leading to a low power to detect significant effects. This low prevalence might be due to difficulties separating PEV from other foot deformities at birth, leading to errors in the medical records. Despite this, there was a significantly increased prevalence of PEV in the Treated DDH group, which remained significant in the logistic regression model. Interestingly, this was found only in the male sub-group, an opposite effect when compared with the findings of Onay et al[22] in Turkish children, in whom PEV was a risk factor among females. The prevalence of DDH in children with PEV was 5.2% for All DDH and 4.6% for Treated DDH, in line with previous studies reporting the prevalence of DDH among children with foot deformities.[4,38]

A slower gain in weight postnatally was significantly associated with DDH, and remained significant in the logistic regression models. This risk factor has mainly been postulated for canine DDH, in which the relative age of walking is much earlier than in humans.[39] However, one might speculate that underweight infants might have a more adducted hip. This is in contrast to our previous findings in a different cohort, in which increased growth was found to be a risk factor for acetabular dysplasia in young adulthood.[20]

Parental age was increased in both DDH groups, and for both parents, mainly for female children. Increased paternal age has previously been established as a risk factor, while the maternal age is seen in conjunction with primiparity and has been lower in several previous studies.[23,24] The increasing age of women at the time of their first child possibly influences the interaction between these two risk factors, with studies finding opposite effects of maternal age in crude and regression analysis.[5]

Maternal diabetes has been postulated as a risk factor due to an increased birth weight in the child. In our study, the prevalence of maternal diabetes was significantly lower in both DDH groups. For the logistic regression, female children had a significantly lower risk of DDH with maternal diabetes. This aligns with a previous study of congenital malformations in relation to maternal pregestational diabetes, in which the prevalence of hip dislocation in the child was lower.[19] The authors related this to a shorter gestational age in the diabetic group, which we also found in our dataset.[19]

The use of folate was significantly lower and the use of alcohol was significantly higher among mothers of children with DDH, and this remained in the logistic regression analyses. Although the use of folate is not among the most studied risk factors for DDH, a South American study found that fortifying flour with folate significantly decreased the prevalence of subluxed hips;[18] neither has maternal alcohol consumption in relation to hip dysplasia been widely studied. Reece and Hulse[17] did not find a significant association between the rates of alcohol consumption and the prevalence of hip dysplasia in 14 European countries. However, chronic alcohol consumption can affect the foetal absorption of folate, and both factors could be associated with other lifestyle factors.[40]

The study had limitations. First, the definition of DDH is challenging. In the MoBa dataset, this diagnosis is based on parental reports, unsupported by a clinical diagnosis from medical records, radiological findings, or a clinical examination. The questions about DDH are also not sufficiently detailed to allow the optimal collection of data. However, the questionnaires are answered according to the child's age, reducing the effect of recall bias compared with purely retrospective studies with a longer time delay. Second, the most common protocol for undertaking an ultrasound examination in Norway is a modified Graf technique (Rosendahl's method).[41] This classification includes immature neonatal hips, which mostly resolve spontaneously within 12 weeks of birth and are not classified as DDH. However, many parents might report these findings in the questionnaire as representing DDH, explaining the discrepancy with Treated DDH. Third, the other variables which we used are also questionnaire-based, with questions answered either by midwives through the MBRN or by parents before and/or during pregnancy and at six months of age for the child. Limitations of the study thus included recruitment bias, reporting errors, plotting errors, recall bias, and missingness due to questionnaire fatigue or misunderstandings. However, our dataset was thoroughly inspected for outliers to remove errors and, apart from the low prevalence of PEV, the variables have a distribution which is close to what would be expected. A final limitation was the lack of information about familial disposition to DDH. This is a known risk factor, but the information was not available at the time of this study. We hope to gather more information about this by linking the data with patients' medical records in the future.

Although we successfully confirmed several risk factors in this dataset, including breech presentation, sex, and birthweight, the logistic regression models were unable to fully explain the variation in the prevalence of DDH. This highlights the need for more studies on DDH to confirm risk factors in order to improve screening and diagnosis, including genetic risk factors.

Take home message

- In the Norwegian Mother, Father, and Child Cohort Study, significant risk factors included female sex, breech position, and pes equinovarus, while delivery by Caesarean section was not a risk factor when controlling for confounders.
- The data suggest that more risk factors remain undetected.

Supplementary material

Tables of descriptive statistics of risk factors by sex and by All developmental dysplasia of the hip (DDH) and Treated DDH, and logistic regression of risk factors by sex and by All DDH and Treated DDH.

References

1. **Kuitunen I**, **Uimonen MM**, **Haapanen M**, **Sund R**, **Helenius I**, **Ponkilainen VT**. Incidence of neonatal developmental dysplasia of the hip and late detection rates based on screening strategy: a systematic review and meta-analysis. *JAMA Netw Open*. 2022;5(8):e2227638.
2. **Dezateux C**, **Rosendahl K**. Developmental dysplasia of the hip. *Lancet*. 2007;369(9572):1541–1552.
3. **Humphry S**, **Hall T**, **Hall-Craggs MA**, **Roposch A**. Predictors of hip dysplasia at 4 years in children with perinatal risk factors. *JB JS Open Access*. 2021;6(1):e20.00108.
4. **Håberg Ø**, **Foss OA**, **Lian ØB**, **Holen KJ**. Is foot deformity associated with developmental dysplasia of the hip?. *Bone Joint J*. 2020;102-B(11):1582–1586.
5. **Woodacre T**, **Ball T**, **Cox P**. Epidemiology of developmental dysplasia of the hip within the UK: refining the risk factors. *J Child Orthop*. 2016;10(6):633–642.
6. **Swarup I**, **Penny CL**, **Dodwell ER**. Developmental dysplasia of the hip. *Curr Opin Pediatr*. 2018;30(1):84–92.
7. **Broadhurst C**, **Rhodes AML**, **Harper P**, **Perry DC**, **Clarke NMP**, **Aarvold A**. What is the incidence of late detection of developmental dysplasia of the hip in England: a 26-year national study of children diagnosed after the age of one. *Bone Joint J*. 2019;101-B(3):281–287.
8. **Poacher AT**, **Hathaway I**, **Crook DL**, **et al**. The impact of the introduction of selective screening in the UK on the epidemiology, presentation, and treatment outcomes of developmental dysplasia of the hip. *Bone Jt Open*. 2023;4(8):635–642.
9. **Barlow TG**. Early diagnosis and treatment of congenital dislocation of the hip. *Proc R Soc Med*. 1963;56(9):804–806.
10. **Ortolani M**. Congenital hip dysplasia in the light of early and very early diagnosis. *Clin Orthop Relat Res*. 1976;119:6–10.
11. **Seo LJ**, **Gabor J**, **Novikov D**, **Feng JE**, **Schwarzkopf R**, **Vigdorchik JM**. Outcomes in 385 developmental dysplastic hips requiring total hip arthroplasty. *Arch Orthop Trauma Surg*. 2019;139(5):723–728.
12. **Rahm S**, **Hoch A**, **Tondelli T**, **Fuchs J**, **Zingg PO**. Revision rate of THA in patients younger than 40 years depends on primary diagnosis - a retrospective analysis with a minimum follow-up of 10 years. *Eur J Orthop Surg Traumatol*. 2021;31(7):1335–1344.
13. **Halvorsen V**, **Fenstad AM**, **Engesæter LB**, **et al**. Outcome of 881 total hip arthroplasties in 747 patients 21 years or younger: data from the Nordic Arthroplasty Register Association (NARA) 1995-2016. *Acta Orthop*. 2019;90(4):331–337.
14. **Magnus P**, **Birke C**, **Vejrup K**, **et al**. Cohort profile update: the Norwegian mother and child cohort study (MoBa). *Int J Epidemiol*. 2016;45(2):382–388.
15. **Magnus P**, **Irgens LM**, **Haug K**, **et al**. Cohort profile: the Norwegian mother and child cohort study (MoBa). *Int J Epidemiol*. 2006;35(5):1146–1150.
16. **No authors listed**. Statistics on Births. Statistics Norway. https://www.ssb.no/en/befolkning/fodte-og-dode/statistikk/fodte (date last accessed 28 May 2025).
17. **Reece AS**, **Hulse GK**. Effects of cannabis on congenital limb anomalies in 14 European nations: a geospatiotemporal and causal inferential study. *Environ Epigenet*. 2022;8(1):dvac016.
18. **López-Camelo JS**, **Castilla EE**, **Orioli IM**, **INAGEMP (Instituto Nacional de Genética Médica Populacional)**, **ECLAMC (Estudio Colaborativo Latino Americano de Malformaciones Congénitas)**. Folic acid flour fortification: impact on the frequencies of 52 congenital anomaly types in three South American countries. *Am J Med Genet A*. 2010;152A(10):2444–2458.
19. **Garne E**, **Loane M**, **Dolk H**, **et al**. Spectrum of congenital anomalies in pregnancies with pregestational diabetes. *Birth Defects Res A Clin Mol Teratol*. 2012;94(3):134–140.
20. **Laborie LB**, **Lie SA**, **Rosendahl K**. Radiographic markers of hip dysplasia in young adults: predictive effect of factors in early life. *BMC Musculoskelet Disord*. 2023;24(1):119.
21. **Ortiz-Neira CL**, **Paolucci EO**, **Donnon T**. A meta-analysis of common risk factors associated with the diagnosis of developmental dysplasia of the hip in newborns. *Eur J Radiol*. 2012;81(3):e344–51.
22. **Onay T**, **Gumustas SA**, **Cagirmaz T**, **Aydemir AN**, **Orak MM**. Do the risk factors for developmental dysplasia of the hip differ according to gender? A look from another perspective. *J Paediatrics Child Health*. 2019;55(2):168–174.
23. **Wynne-Davies R**, **Littlejohn A**, **Gormley J**. Aetiology and interrelationship of some common skeletal deformities. (Talipes equinovarus and calcaneovalgus, metatarsus varus, congenital dislocation of the hip, and infantile idiopathic scoliosis). *J Med Genet*. 1982;19(5):321–328.
24. **Mendez-Dominguez N**, **Alvarez-Baeza A**, **Estrella-Castillo D**, **Lugo R**, **Villasuso-Alcocer V**, **Azcorra H**. Ethnic and sociodemographic correlates of developmental dysplasia of the hip in newborns from Yucatan, Mexico. *Am J Hum Biol*. 2022;34(6):e23724.
25. **Pfister R**, **Schwarz K**, **Carson R**, **Jancyzk M**. Easy methods for extracting individual regression slopes: comparing SPSS, R, and Excel. *TQMP*. 2013;9(2):72–78.
26. **Laskaratou ED**, **Eleftheriades A**, **Sperelakis I**, **et al**. Epidemiology and screening of developmental dysplasia of the hip in Europe: a scoping review. *Reports*. 2024;7(1):10.
27. **Loder RT**, **Skopelja EN**. The epidemiology and demographics of hip dysplasia. *ISRN Orthop*. 2011;2011:238607.
28. **Tirta M**, **Rahbek O**, **Kold S**, **Husum H-C**. Risk factors for developmental dysplasia of the hip before 3 months of age: a meta-analysis. *JAMA Netw Open*. 2025;8(1):e2456153.
29. **Hinderaker T**, **Daltveit AK**, **Irgens LM**, **Udén A**, **Reikeräs O**. The impact of intrauterine factors on neonatal hip instability. *Acta Orthop Scand*. 1994;65(3):239–242.
30. **Toijonen AE**, **Heinonen ST**, **Gissler MVM**, **Macharey G**. A comparison of risk factors for breech presentation in preterm and term labor: a nationwide, population-based case-control study. *Arch Gynecol Obstet*. 2020;301(2):393–403.
31. **Shaw BA**, **Segal LS**, **Orthopaedics SO**, **Otsuka NY**, **Schwend RM**, **Ganley TJ**. Evaluation and referral for developmental dysplasia of the hip in infants. *Pediatrics*. 2016;138(6):e20163107.
32. **Rayl J**, **Gibson PJ**, **Hickok DE**. A population-based case-control study of risk factors for breech presentation. *Am J Obstet Gynecol*. 1996;174(1 Pt 1):28–32.
33. **Holen KJ**, **Tegnander A**, **Terjesen T**, **Johansen OJ**, **Eik-Nes SH**. Ultrasonographic evaluation of breech presentation as a risk factor for hip dysplasia. *Acta Paediatr*. 1996;85(2):225–229.
34. **Bjellmo S**, **Andersen GL**, **Martinussen MP**, **et al**. Is vaginal breech delivery associated with higher risk for perinatal death and cerebral palsy compared with vaginal cephalic birth? Registry-based cohort study in Norway. *BMJ Open*. 2017;7(4):e014979.
35. **Koureas G**, **Wicart P**, **Seringe R**. Etiology of developmental hip dysplasia or dislocation: review article. *Hip Int*. 2007;17 Suppl 5(5_suppl):S1–S7.
36. **Kraus T**, **Pellegrin MD**, **Dubs B**. DDH: definition, epidemiology, pathogenesis, and risk factors. In: *Developmental Dysplasia of the Hip*. 2022: 11–15.
37. **Johnsen SL**, **Aarseth T**, **Helbig A**, **et al**. Norsk Veileder i Fødselshjelp-Tvillinger. 2025. https://metodebok.no/index.php?action=topic&item=2CcR7D8M (date last accessed 28 May 2025).
38. **Perry DC**, **Tawfiq SM**, **Roche A**, **et al**. The association between clubfoot and developmental dysplasia of the hip. *J Bone Joint Surg Br*. 2010;92-B(11):1586–1588.
39. **Rhodes AML**, **Clarke NMP**. A review of environmental factors implicated in human developmental dysplasia of the hip. *J Child Orthop*. 2014;8(5):375–379.
40. **Hutson JR**, **Stade B**, **Lehotay DC**, **Collier CP**, **Kapur BM**. Folic acid transport to the human fetus is decreased in pregnancies with chronic alcohol exposure. *PLoS One*. 2012;7(5):e38057.
41. **Rosendahl K**, **Markestad T**, **Lie RT**. Developmental dysplasia of the hip: prevalence based on ultrasound diagnosis. *Pediatr Radiol*. 1996;26(9):635–639.

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Data sharing:
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