Anthropological Review • Vol. 83(1), 31–41 (2020)

sciendo

ANTHROPOLOGICAL REVIEW
Available online at: https://doi.org/10.2478/anre-2020-0002

Paternal age and the risk of cerebral palsy

Marta Sternal, Barbara Kwiatkowska, Krzysztof Borysławski, Agnieszka Tomaszewska

Department of Anthropology, Wroclaw University of Environmental and Life Sciences, Poland

ABSTRACT: In the literature there are no unequivocal assessments of the effect of paternal age on the risk of cerebral palsy (CP). The objective of the studies was the analysis of the influence of paternal age on this risk, considering all the important risk factors and division into singleton and twin, as well as term- and preterm-born infants. The inquiry included a group of 278 children with cerebral palsy from selected education-therapeutic institutions in Poland. The control group consisted of the data from medical records of 435 neonates born in God's Mercy Hospital in Limanowa, Poland. The data were based on a questionnaire designed to obtain information which would make it possible to ascertain the probable etiological factors. Constructed models of logistic regression were used in statistical analysis. The results were presented as the odds ratio (OR) with 95% confidence interval (CI). Though the estimation with a complex model of logistic regression showed no significant effect of paternal age on the occurrence of cerebral palsy, it confirmed it as a stronger predictor compared to maternal age. Disregarding paternal of age while considering maternal age and other risk factors may lead to a bias in the estimations of the risk cerebral palsy.

KEY WORDS: cerebral palsy, singleton, twin, term- preterm-born infants, paternal age, maternal age

Introduction

Cerebral palsy includes various, non-progressive, kinds of motor impairment and posture disorders, combined with other symptoms of permanent brain damage at early development stages (Novotny 1993; Rosenbaum et al. 2007). It is not a definite syndrome, but a set of pathological symptoms which are etiologically and clinically diverse; hence it is difficult to determine its frequency. Literature data show that the rate of cerebral palsy ranges from 1 to even 5 per 1000 live born children (Kiely et al. 1981; Johnson and Catterson 1988; Platt and Pharoah 1995; Colver 2000; Jóźwiak 2001; Kwolek et al. 2001; Zgorzalewicz et al. 2001; Lin 2003; Jacobsson et al. 2004; Koman et al. 2004;; Sankar and Mundkur 2005; Blair and Watson 2006; Bober and Kobel-Buys 2006; Korzeniewski 2006; Thorngren-Jerneck and Herbst 2006; Robertson et al. 2007; Öztürk et al. 2007; Schaefer 2008; Baxter 2009; Selier et al. 2010; Wu et al. 2010; Milewska et al. 2011; Reid et al. 2011). This makes

Original Research Article Received: November 26, 2019; Revised: February 3, 2020; Accepted: February 7, 2020
DOI: 10.2478/anre-2020-0002

it the most frequent reason for motor disability and the second most frequent (next to intellectual disturbances) reason for neurodevelopmental disturbances in children (Michalska et al. 2012).

The underlying pathophysiological mechanisms are heterogenous and still incompletely known which is the main reason for continuous search for independent factors which favor the occurrence of cerebral palsy (Sternal et al. 2011). In our opinion the role of father in the etiopathogenesis of cerebral palsy should be considered as one of potential factors responsible for the condition.

According to Fletcher and Foley (Fletcher and Foley 1993) father's elderly age may be associated with the dyskinetic form of cerebral palsy and hemiplegia which, as pointed out by the authors, may be an effect of a dominant mutation. This is no doubt associated with paternal age at the moment of fertilization, and the effect of age on the formation and maturation of spermatozoa. Spermatogenesis is a short-lasting process and occurs successively; spermatozoa do not age, but with age the production of male gametes poses a risk of a higher number of errors and thus of mutations in the spermatozoa. With age, the DNA information in the spermatozoa becomes increasingly unstable, their morphology and total count change, the semen volume and the sperm viability decrease. This is also associated with the effect of long-lasting stress, inappropriate malnutrition or lifestyle of the male parent (Carothers et al. 1986; Fletcher and Foley 1993; Auger et al. 2001; Slama et al. 2005; Reichman and Teitler 2006; Zhu et al. 2008; Johnson et al. 2012; Schnekenberg et al. 2015). Clinical studies revealed that late fatherhood implied a higher risk of *de novo* mutations; their proportion increased by 4.28 % per year, and their number doubled every 16.5 years of the man's lifetime (Schnekenberg et al. 2015).

To date it was shown that father's elderly age was associated with low birth weight, preterm births, osteogenesis imperfecta (OI), achondroplasia, thanatophoric dysplasia, Apert, Marfan, Pfeiffer and Crouzon syndromes, autism, epilepsy, schizophrenia, multiple endocrine type 2A and type 2B, childhood cancer, congenital malformations, as well as spontaneous abortions (Carothers et al. 1986; Fletcher and Foley 1993; Auger et al. 2001; Slama et al. 2005; Reichman and Teitler 2006; Zhu et al. 2008; Johnson et al. 2012; Schnekenberg et al. 2015).

As far as we know, there is no information in the literature on the influence of the age of teenage fathers on the occurrence of cerebral palsy in their offspring.

Material and Methods

The study included a group of 278 children with diagnosed cerebral palsy, born in 1976–2010, and frequenting education-therapeutic centers in Poland. The questionnaire was filled by the mothers of the children with cerebral palsy or, in the absence of such possibility, by the persons most involved in the care of the disabled child. The data were collected in 40 Polish institutions dealing with children with cerebral palsy.

This paper presents only a fragment of extensive studies aimed at identifying independent risk factors for cerebral palsy among demographic, antenatal, perinatal and neonatal factors. For this purpose the questionnaire was divided in three parts. Since an important part in the etiology of cerebral palsy is played by the factors that are active during gestation and childbirth,

the first part included medical, family and obstetric record of the mother, it also included questions about childbirth, pregnancy, gynecological disturbances, as well as information on the child's father. The second part contained questions pertaining to medical and anthropological information, as well as to the occurrence of accompanying disturbances and child's diseases. The last part included environmental diagnose pertaining to the family structure, address, education level and profession of the parents and the mother's material situation during pregnancy.

Cases of acquired cerebral palsy, congenital hydrocephalia, genetic syndromes (Arnold-Chiari malformation, Dandy-Walker syndrome) and congenital microcephalia were excluded from the analysis.

The control group included data from the medical records of 435 neonates born without congenital defects, genetic syndromes, metabolic diseases or other hereditary diseases, in 1990–2010 in God's Mercy Hospital in Limanowa, Poland. The data on the mothers and neonates were based on the available medical record (hospital information card, nursing care card, obstetric care card, neonate card – information on parents and neonate's condition).

It follows from epidemiological studies that the etiology of cerebral palsy differs between preterm- and term-born neonates, and between neonates from singleton and twin pregnancies. Thus in order to obtain a reliable assessment of the risk of cerebral palsy the material was divided into categories according to singleton and twin, term (≥37 week) and preterm (<37 week) births (Table 1).

In the studies we used the assumption that the main predictors of cerebral palsy were gestational age, as well as singleton vs. twin pregnancy. Accordingly, all the research procedure was aimed at showing that the risk factors of cerebral palsy differed depending on the category of gestational age. Consequently, the proposed models estimating the risk should be different. The categories distinguished in the material considering singleton, twin, term and preterm births differ significantly in the etiopathogenesis of cerebral palsy. Thus the initial assumption was an existence of variables which differentiate between the above-mentioned categories. The planned univariate analyses, using such variables as birth weight, birth length, gestational age, birth order and sex, were aimed at comparative characteristics of children from the cerebral palsy group and the control group within the categories, and at identifying the variables which, once included into logistic regression models, served as accompanying variables and controlled the correctness of statistical conclusions.

Table 1. Numbers of children with cerebral palsy and in control group in the categories adopted in the analysis

Category	N
All children	713
Singleton pregnancies	669
Twin pregnancies	44
Term births	548
Term singleton births	527
Term twin births	21
Preterm births	165
Preterm singleton births	142
Preterm twin births	23

Several logistic regression models were proposed to assess the probability of occurrence of cerebral palsy depending on the category and co-occurring accompanying variables:

– simple model determining the probability ratio of cerebral palsy depend-

ing on the studied factors in each category,
– adjusted model considering the effect of the accompanying variables determined in univariate analysis,
– complete model which, besides the accompanying variables and significant risk factors, considered also the effect of paternal and maternal age (in order to estimate independent influence of maternal and paternal age depending on the studied factors and category).

In order to reduce the probability of occurrence of augmented matrix the studies considered also logistic regression models constructed with and without data aggregation. Furthermore, considering the possibility of skewness of accompanying variables, we also used a regression model with transformed variables.

In each logistic regression model, in order to more precisely estimate the odds ratio, we used parameterization of the father's age variable with the separation of the reference group against which we assessed the risk of cerebral palsy. We categorized the variable in four variants: ≤20 years, 30–34 years, 35–39 years, ≥40 years, with the group of 21–29 years serving as a reference group.

For the ultimate assessment of independent factors of risk of cerebral palsy, we used logistic regression model considering all the demographic factors which were significant in multivariate statistics: demographic, antenatal, perinatal and neonatal, and also significant accompanying variables.

The analysis was performer using STATISTICA 12.5 program. The comparative characteristics of the group of children with cerebral palsy and the control group were made using the Mann-Whitney test, Student t-test (for quantitative date) and Chi-square test (for qualitative date). A *p*-value of less than 0.05 was selected as statistically significant.

The variables, which differentiated between the group with cerebral palsy and control group, and in univariate analyses proved to be statistically significant, were included in the principal component analysis. This made it possible to identify non-correlated accompanying variables which were included in further multivariate analyses.

The odds ratio was calculated to ascertain the dependence between the studied variables and the prevalence of cerebral palsy. The OR values were given with 95% confidence interval (CI), the significance level being *p* for the Wald Chi-square test. The data fit of the model was expressed using Cox-Snell and Nagelkerke coefficients, and Hosmer-Lemeshow test.

Results

The univariate analysis showed that in the category of all children and the category of children from singleton pregnancies, the variables birth weight and birth length, birth order and gestational age, differed significantly between the children with cerebral palsy and those from the control group. Within the category of children from twin pregnancies, sex, birth order and gestational age, differed significantly between the children with cerebral palsy and those from the control group (Table 2).

Within the category of children from term and singleton term births, the variables birth length, birth weight and birth order, differed significantly between the children with cerebral palsy and the control group. Among the children from term twin births, the significantly differ-

entiating factors were sex and birth order (Table 3).

In the categories of children from preterm births and from preterm singleton births, the variables birth weight and birth length significantly differentiated between the children with cerebral palsy and the control group. In the category of children from preterm twin births no variable was statistically significant (Table 4).

Following principal component analysis, birth weight and birth order were included in logistic regression models as

Table 2. Comparative characteristics of all children from experimental and control group and separately for singleton and twin pregnancies

All children			
Variable	Children with cerebral palsy N = 278	Control group N = 435	*p*
Sex m/f	152/126	219/216	NS
Birth weight (g)	440.0–5600.0 2554.9±997.2	1290.0–5170.0 3367.1±552.0	<0.001
Birth length (cm)	20–65 50.0±7.2	41–63 54.8±3.1	<0.001
Birth order	1–9 2.0±1.3	1–15 2.6±1.8	<0.001
Gestational age (weeks) <37 Gestational age (weeks) ≥37	126 151	39 396	<0.001
Singleton pregnancies			
Variable	Children with cerebral palsy N = 250	Control group N = 419	*p*
Sex m/f	136/114	217/202	NS
Birth weight (g)	440.0–5600.0 2615.2±1005.6	1430.0–5170.0 3403.87±514.07	<0.001
Birth length (cm)	20–65 50.3±7.12	45–63 54.9±2.9	<0.001
Birth order	1–9 2.0±1.3	1–15 2.6±1.8	<0.001
Gestational age (weeks) <37 Gestational age (weeks) ≥37	106 141	35 381	<0.001
Twin pregnancies			
Variable	Children with cerebral palsy N = 28	Control group N = 16	*p*
Sex m/f	16/12	2/14	<0.01
Birth weight (g)	830.0–3750.0 2025.7±745.4	1290.0–3230.0 2403.1±650.4	NS
Birth length (cm)	33–63 47.4±7.0	41–56 49.9±4.4	NS
Birth order	1–4 1.8±1.0	1–5 2.6±1.5	<0.05
Gestational age (weeks) <37 Gestational age (weeks) ≥37	20 8	4 12	<0.01

Data expressed as number in category, range or mean, standard deviation, *p* from Student *t*-test, Mann-Whitney *U*-test or Chi-square test, NS-non-significant.

accompanying variables (controlling statistical conclusions) in the category of all children, children from singleton, term and singleton term births. In the category of children from preterm and preterm singleton births, only birth weight was considered. In the categories of children from twin, term twin and preterm twin births no accompanying variables were identified since the categories were too small for the estimate with the maximum likelihood method for logistic regression models.

The mean age of fathers in the group of children with cerebral palsy was 30.1 years and in the control group 31.2 years. In the group of children with cerebral palsy 2% of fathers were aged ≤20 years and 18.3% ≥ 35 years. In the control group only 0.5% of fathers were aged ≤20 years, and 24.8% ≥ 35 years. The limiting factor in the analysis was the lack of information on paternal age in 14.2% of cases in the group of children with cerebral palsy and in 5.8% of cases in the control group.

Table 3. Comparative characteristics of children from term, singleton term and twin term births from experimental and control group

Term births			
Variable	Children with cerebral palsy N = 152	Control group N = 396	*p*
Sex m/f	86/66	203/193	NS
Birth weight (g)	2180.0–5600.0 3307.4 ±540.3	1480.0–5170.0 3453.3±472.5	<0.01
Birth length (cm)	35–63 54.3±3.9	46–63 55.2±2.7	<0.05
Birth order	1–9 1.9±1.2	1–5 2.6±1.5	<0.001
Singleton term births			
Variable	Children with cerebral palsy N = 143	Control group N = 384	*p*
Sex m/f	80/63	201/183	NS
Birth weight (g)	2225.0–5600.0 3339.7±529.7	2050.0–5170.0 3479.3±446.3	<0.01
Birth length (cm)	35–63 54.4±3.9	46–63 55.3±2.6	<0.05
Birth order	1–9 1.9±1.2	1–15 2.6±1.8	<0.001
Twin term births			
Variable	Children with cerebral palsy N = 9	Control group N = 12	*p*
Sex m/f	6/3	2/10	<0.05
Birth weight (g)	2180.0–3750.0 2801.1±470.9	1480.0–3230.0 2620.8±548.9	NS
Birth length (cm)	49–63 52.9±4.9	47–56 51.8±2.8	NS
Birth order	1–4 1.8±0.9	1–5 3.2±1.4	<0.05

Data expressed as number in category, range or mean, standard deviation, *p* from Student *t*-test, Mann-Whitney *U*-test or Chi-square test, NS – non-significant.

Of all the logistic regression models estimating the risk of cerebral palsy for demographic factors (including paternal age), those adjusted to accompanying variables (both with and without data aggregation) reproduced the original data best.

The studies on the effect of demographic factors (including paternal age) on the occurrence of cerebral palsy for all the children from the experimental group and the control group showed that fathers aged 30–34 years were 45% less likely to have child with cerebral palsy than fathers aged 21 to 29 years (OR 0.55, 95% CI: 0.31–0.99). Similar results were obtained in the analysis of the effect of paternal age in the category of children from singleton pregnancies, term births and term singleton births. Also in this categories fathers aged 30–34 years were less likely to have child with cerebral palsy than fathers aged 21 to 29 years (OR 0.54, 95% CI: 0.29–0.98; OR 0.48, 95% CI: 0.24–0.93; OR 0.45, 95% CI: 0.22–0.90, respectively) (Table 5).The associ-

Table 4. Comparative characteristics of children from preterm, preterm singleton and preterm twin births from experimental and control group

Preterm births			
Variable	Children with cerebral palsy N = 126	Control group N = 39	*p*
Sex m/f	66/60	16/23	NS
Birth weight (g)	440.0–4600.0 1644.9±572.8	1290.0–3580.0 2491.3±499.4	<0.001
Birth length (cm)	20–65 44.5±6.6	41–57 50.2±3.6	<0.001
Birth order	1–9 2.1±1.4	1–7 2.2±1.3	NS
Singleton preterm births			
Variable	Children with cerebral palsy N = 107	Control group N = 35	*p*
Sex m/f	56/51	16/19	NS
Birth weight (g)	440.0–4600.0 1642.4±581.0	1430.0–3580.0 2576.0±485.7	<0.001
Birth length (cm)	20–65 44.5±6.7	45–57 50.8±3.1	<0.001
Birth order	1–9 2.2±1.5	1–7 2.3±1.3	NS
Twin preterm births			
Variable	Children with cerebral palsy N = 19	Control group N = 4	*p*
Sex m/f	10/9	0/4	NS
Birth weight (g)	830.0–2360.0 1658.4±540.2	1290.0–2300.0 1750.0±502.1	NS
Birth length (cm)	33–55 44.6±6.4	41–48 44.8±3.8	NS
Birth order	1–4 1.9±1.1	1 1±0	NS

Data expressed as number in category, range or mean, standard deviation, *p* from Student *t*-test, Mann-Whitney *U*-test or Chi- square test, NS – non-significant.

Table 5. Result of logit model estimations from prevalence of cerebral palsy considering paternal age for particular categories

Category	Paternal age (years)	OR (95% CI)	Wald test
All children	30–34	0.55 (0.31–0.97)	$p = 0.04$
Singleton pregnancies	30–34	0.53 (0.31–0.97)	$p = 0.03$
Term births	30–34	0.51 (0.27–0.98)	$p = 0.04$
Singleton term births	30–34	0.48 (0.24–0.93)	$p = 0.03$

ation between paternal age and cerebral palsy remained significant, even when we controlled for the other demographic characteristics (including maternal age).

Using the constructed logit models we selected potential independent variables which had a significant effect on the risk of cerebral palsy. Based on the set of obtained significant variables (among demographic, antenatal, perinatal and neonatal factors) an estimation was done using the complete model (all significant factors adjusted to accompanying variables), to obtain a model which would predict the effect of paternal age as an independent risk factor of cerebral palsy in each category.

Inclusion of paternal age in the ultimate model (combined analysis of significant demographic, antenatal, perinatal and neonatal factors) showed no effect of this factor on the prevalence of cerebral palsy.

Discussion

Though maternal age is often mentioned in the literature on the effect of social-demographic factors on the risk of cerebral palsy, there aren't unequivocal estimates of the effect of paternal age on the probability of the condition. With the technological development and the advent of new techniques of molecular cytogenetics and molecular biology, it has become possible to tie paternal age with the occurrence of *de novo* mutations which may have causal links to children's diseases or abortions (Carothers et al. 1986; Kadotani et al. 2001; Slama et al. 2005; Zhu et al. 2008).

It is thought at present that father's elderly age is the reason for a much greater number of children's diseases than previously believed, even though their probability is relatively small (Carothers et al. 1986; Zhu et al. 2008).

The available epidemiological studies provide little information on the effect of paternal age on the probability of cerebral palsy (Fletcher and Foley 1993; Kadotani et al. 2001). A few previous studies (Fletcher and Foley 1993; Kadotani et al. 2001) found detrimental effects of advanced paternal age on cerebral palsy in their children, but they didn't investigate association between teenaged fathers and cerebral palsy. Whether such association occurs is an empirical question that has not been explored. Likewise, the pertinent literature lacks information on the combined effect of paternal and maternal age on the risk of cerebral palsy.

In our own studies the inclusion of paternal age into the ultimate model (combined analysis of significant demographic, antenatal, perinatal and neonatal factors) failed to demonstrate its effect on the occurrence of cerebral palsy in any of the gestational age categories. However, testing the effect of paternal age with different models, separately for demographic, antenatal, perinatal and neonatal factors, revealed that paternal

age was a stronger risk factor than maternal age (even after correction for maternal age, parents' material status and accompanying variables). The observations suggest that disregarding paternal age while analyzing maternal age and other risk factors of cerebral palsy may bias the real picture. At the same time it seems justified to suppose that the previous studies, which indicated a significant effect of maternal age, may have involved a bias of estimators evaluating the risk of CP because of disregarding paternal age. Further analyses should be aimed at estimating the effect of maternal age combined with monitoring paternal age in order to reduce the error resulting from incorrect estimation.

Our findings that paternal age is independently and negatively associated with the probability of cerebral palsy is hard to explain. A number of potential mechanisms could have had affect this association between paternal age and cerebral palsy. Some involve the sample size. The sample size underrepresented fathers aged younger than 20 years, and overrepresented fathers aged 30 to 34 years, as well as overrepresented children with spastic type of cerebral palsy (the previous studies indicated a paternal age effect in athetoid/dystonic CP, but not in spastic type of cerebral palsy (Fletcher and Foley 1993)). Moreover, as a limiting factor in this study, it should be also mentioned in the method used. The authors are aware of the limitations of the retrospective method and further studies, if possible, should be conducted by the prospective method as well. Because our study was subject to these limitations. It should be replicated and the association further explored.

There is also another explanation. Previous studies (Giblin et al. 1988; Auger et al. 2001; Bigelow et al. 1998) revealed that some factors related to the sociological and lifestyle backgrounds (eg alcohol habits) of the men were found to possibly modulate sperm morphogenesis. The men who had moderate alcohol habits had less sperm defects than those drinking a lot. Maybe men aged 21–29 years are at extremely high risk of sperm defect owing to greater exposure to harsh living condition (higher alcohol consumption). This is the only one biologically meaningful explanation for this intriguing result, which warrants further studies.

The statistical analyses identified birth weight and birth order as significant accompanying variables which significantly changed the values of estimated parameters and hence they should be used as controlling variables when attempting to assess the risk of cerebral palsy.

Furthermore, the multivariate analyses showed that depending on the kind of pregnancy (twin, singleton, term, singleton term, preterm, singleton preterm) the risk factors of cerebral palsy vary, so models estimating the probability of cerebral palsy should be constructed considering this fact.

The presented study is the first one in Poland to consider the effect of paternal age as potential risk factor of cerebral palsy. It demonstrated the need to include, besides the basic clinical information also paternal age in neuroepidemiological studies. The future studies should consider forms of cerebral palsy. We also suggest extending the studies to include a greater number of twin pregnancies and the health of the male (including obesity, drug and alcohol intake) as well. The need to focus on this aspect of research results from the fact that still many pathogenic factors of cerebral palsy remain unexplained.

Authors' contributions

MS conceived the paper, collected the data, performed statistical computations and drafted the manuscript; BK was project supervisor, co-edited the final version of the manuscript; KB performed statistical computations and co-edited the final version of the manuscript; AT performed statistical computations, drafted the manuscript, co-edited the final version of the manuscript. All authors carefully read and accepted the final version of the manuscript.

Conflict of interest

With the submission of this manuscript I would like to undertake that the above mentioned manuscript is without any conflict of interest and has not been published elsewhere.

Corresponding author

Agnieszka Tomaszewska Department of Anthropology, Wroclaw University of Environmental and Life Sciences, Kożuchowska 5, 51-631 Wroclaw, Poland e-mail: agnieszka.tomaszewska@upwr.edu.pl

References

Auger J, Eustache F, Andersen AG, Irvine DS, Jørgensen N, Skakkebæk NF, Suominen J, Toppari J, Vierula M, Jouannet P. 2001. Sperm morphological defects related to environment, lifestyle and medical history of 1001 male partners of pregnant women from four European cities. Hum Reprod 16(12):2710–17.

Baxter P. 2009. Preventing cerebral palsy: hidden improvements. Dev Med Child Neurol 51(3):335.

Bigelow PL, Jarrell J, Young MR. 1998. Association of semen quality and occupational factors: comparison of case-control analysis and analysis of continuous variables. Fertil Steril 69:11–8.

Blair E, Watson L. 2006. Epidemiology of cerebral palsy. Semin Fetal Neonat M 11:117–25.

Bober T, Kobel-Buys K. 2006. Mózgowe porażenie dziecięce z doświadczeń trzyletniego programu rehabilitacyjnego. Wydawnictwo AWF Wrocław.

Carothers AD, McAllion SJ, Paterson CR. 1986. Risk of dominant mutation in older fathers: evidence from osteogenesis imperfect. J Med Genet 23:227–30.

Colver AF, Gibson M, Hey EN, Jarvis SN, Mackie PG, Richmond S. 2000. Increasing rates of cerebral palsy across the severity spectrum in north-east England 1964–1993. Arch Dis Child- Fetal 83:7–12.

Fletcher NA, Foley J. 1993. Parental age, mutation, and cerebral palsy. J Med Genet 30(1):44–6.

Giblin PT, Poland ML, Moghissi KS. 1988. Effects of stress and characteristics adaptability on semen quality in healthy men. Fertil Steril 49:127–32.

Jacobsson B, Ladfors L, Milson I. 2004. Advanced Maternal Age and Adverse Perinatal Outcome. Am Coll Obstet Gynecol 104(4):727–32.

Johnson A, Catterson J. 1988. Trends in birth prevalence of cerebral palsy. Arch Dis Child 63(3):340.

Johnson JA, Tough S, Wilson RD, Audibert F, Blight C, Brock JA, Cartier L, Désilets V, Gagnon A, Langlois S., Murphy-Kaulbeck L, Okun N. 2012. Delayed Child-Bearing. J Obstet Gynaecol 34(1):80–93.

Jóźwiak M. 2001. Mózgowe porażenie dziecięce – postęp w diagnostyce i terapii. Ortop Traumatol Rehab 3(4):445–49.

Kadotani T, Watanabe Y, Saito T, Sawano K, Minstozaki K, Kadotani TA. 2001. Chromosomal Study on 100 Cases of Cerebral Palsy. Ind J Hum Gens 2:109–12.

Kiely J, Paneth N, Stein Z, Susser M. 1981. Cerebral palsy and newborn care. I: Secu-

lar trends in cerebral palsy. Dev Med Child Neurol 23:533–38.
Koman AL, Smith PB, Shilt JS. 2004. Cerebral palsy. Lancet 363:1619–31.
Korzeniewski S. 2006. The Descriptive Epidemiology of Cerebral Palsy. Clin Perinatol 33:251–67.
Kwolek A, Majka M, Pabis M. 2001. Rehabilitacja dzieci z porażeniem mózgowym – problemy, aktualne kierunki. Ortop Traumatol Rehab 3(4):499–07.
Lin JP. 2003. The cerebral palsies: a physiological approach. J Neurol Neurosurg Psychiatry 74:23–9.
Michalska A, Boksa E, Wendorff J, Wiktor PJ. 2012. Jakość życia dzieci i młodzieży z mózgowym porażeniem dziecięcym i niepełnosprawnością intelektualną. Wybrane uwarunkowania społeczno-demograficzne. Neurol Dziec 21(42):35–44.
Milewska A, Mileańczuk-Lubecka BA, Kochanowski J, Werner B. 2011. Analiza czynników ryzyka mózgowego porażenia dziecięcego. Nowa Pediatr 4:79–84.
Nowotny J. 1993. O racjonalne podejście do usprawniania dzieci z mózgowym porażeniem dziecięcym. Neurol Dziec 2(3):29–36.
Öztürk A, Demirici F, Yavuz T, Yildiz S, Degirmenci Y, Döşoglu M, Avşar Y. 2007. Antenatal and delivery risk factors and prevalence of cerebral palsy in Duzce (Turkey). Brain Dev 29:39–42.
Platt MJ, Pharoah POD. 1995. Epidemiology of cerebral palsy. Curr Paediatr 5:151–55
Reichman NE, Teitler JO. 2006. Paternal Age as a Risk Factor for Low Birthweight. Am J Public Health 96(5):862–66.
Reid S, Carlin J, Reddihough D. 2011. Rates of cerebral palsy in Victoria, Australia, 1970 to 2004: has there been a change? Dev Med Child Neurol 53:907–12.
Robertson ChMT, Watt M, Yasui Y. 2007. Changes in the Prevalence of cerebral Palsy for Children Born Very Prematurely Within a Population-Based Program Over 30 Years. JAMA-J Am Med Assoc 297(24):2733–40.
Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M. 2007. A report: the definition and classification of cerebral palsy. Developmental Medicine and Child Neurology 109:8–14.
Sankar C, Mundkur N. 2005. Cerebral palsy-definition, classification, etiology and early diagnosis. Indian J Pediatr 72(10):865–8.
Schaefer GB. 2008. Genetics Considerations in Cerebral Palsy. Semin Pediatr Neurol 15:21–6.
Schnekenberg RP, Perkins EM, Miller JW, Davies WIL, D'Adamo MC, Pessia M, Fawcett KA, Sims D, Gillard E, Hudspith K, Skehel P, Williams J, O'Regan M, Jayawant S, Jefferson R, Hughes S, Lustenberger A, Ragoussis J, Jackson M, Tucker SJ, Németh AH. 2015. De novo point mutations in patients diagnosed with ataxic cerebral palsy. BRAIN 138:1817–32.
Sellier E, Anderson G, Surman G, Cans Ch. 2010. Trends in prevalence of cerebral palsy in children born with a birthweight of 2500 g or over in Europe from 1980–1998. Eur J Epidemiol 25 (9):635–42.
Slama R, Bouyer J, Windham G, Fenster L, Werwatz A, Swan SH. 2005. Influence of Paternal Age on the Risk of Spontaneous Abortion. Am J Epidemiol 161(9):816–22.
Sternal M, Kwiatkowska B, Borysławski K. 2011. Czynniki zwiększające ryzyko mózgowego porażenia dziecięcego. Pediatr Pol 86(2):163–8.
Thorngren-Jerneck K, Herbst A. 2006. Perinatal Factors Associated with Cerebral Palsy in Children Born in Sweden. Obstet Gynecol 108(6):1499–05.
Wu YW, Croen L, Shah S, Newman T, Najjar D. 2010. Cerebral palsy in a Term Population: Risk Factors and Neuroimaging Finding. Pediatrics 118(2):69–97.
Zgorzalewicz B, Mieszczanek T. Zgorzalewicz M. 2001. Epidemiologia opisowa mózgowego porażenia dziecięcego. Ortop Traumatol Rehab 3(4):467–71.
Zhu JL, Vestergaard M, Madsen KM, Olsen J. 2008. Paternal age and mortality in children. Eur J Epidemiol 23:443–7.