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## Trends in prevalence and characteristics of post-neonatal cerebral palsy cases: A European registry-based study



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### ABSTRACT

The present paper aims to analyze trends over time in prevalence of cerebral palsy of post-neonatal origin, to investigate whether changes are similar according to severity and to describe the disability profile by etiology. Post-neonatal cases, birth years 1976 to 1998, were identified from the Surveillance of Cerebral Palsy in Europe collaboration (19 population-based registries). A recognized causal event occurring between 28 days and 24 months of age was considered to define the cases. Trends in prevalence were explored using graphical methods (Lowess and Cusum control chart) and modeled with negative binomial regressions. Over the study period, 404 cases were identified as post-neonatal cases (5.5% of the total). Mean prevalence rate was 1.20 per 10,000 live births (95% CI [1.08–1.31]). A significant downward trend was observed ( $p = 0.001$ ), with an accentuated decrease in the 1990s. The prevalence of severe cases which account for around one third of the total also significantly decreased over time ( $p < 0.001$ ). In 46% of cases, an infectious aetiology was reported; the corresponding prevalence significantly decreased since 1989. No significant decrease was observed for the rate of cases due to a vascular episode or of traumatic origin. Our results emphasize the need of large population-based surveillance systems to reliably monitor trends in prevalence in rare subgroups of children like those with acquired cerebral palsy. The decrease of the overall prevalence as well as those of the most severe cases may be partly due to public health actions targeted to prevent such events.

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## 1. Introduction

Cerebral palsy (CP) is an umbrella term used to describe a wide spectrum of disabilities in children due to a non-progressive lesion of the brain (Bax et al., 2005). It is the most common motor disorder in early childhood with a prevalence rate around 2/1000 live births (S.C.P.E., 2000). Other deficits in cognitive performance, behavior, epilepsy, visual-spatial or perceptual problems, or a combination of these features may accompany motor disorders resulting in a variability of disability profiles (Himmelmann, Beckung, Hagberg, & Uvebrant, 2006; Shevell, Dagenais, & Hall, 2009). Most situations have heavy and lifelong impact on affected children, on their quality of life (Dickinson et al., 2007) and participation (Fauconnier et al., 2009) as well as on their families. As the probability of survival even among those with the most severe impairments has kept increasing (Aisen et al., 2011; Field, Dorling, Manktelow, & Draper, 2008), questions arise about the continuity of support and adequacy of services available for teens and also adults with CP.

According to previous studies (Himmelmann, Hagberg, Beckung, Hagberg, & Uvebrant, 2005; Parkes, Dolk, & Hill, 2005; Stanley, Blair, & Alberman, 2000), CP cases with a post-neonatal origin (PNN CP) are rare events which account for 5 to 18% of total CP cases. They are of particular interest as the main underlying causes of brain lesions (infections, head injuries or surgical complications) are potential targets for preventive actions. Indeed, educational programs were implemented to reduce the occurrence of brain lesions consecutive to abusive head trauma, drowning, household injuries or accidents in the community and environmental awareness improvements have also been made (safer homes and leisure areas, greater product safety). At the same time, progress in intensive care and dissemination of immunization programs for toddlers have had a major impact on mortality and morbidity (McCormick, 1993; Saigal & Doyle, 2008; Tudehope et al., 1995; Wallis, Paton, Beaton, & Jardine, 2011; Zeitlin, Ancel, Delmas, Breart, & Papiernik, 2010). However, continuing efforts are needed to prevent head trauma and enhance vaccine uptake (Durkin, Olsen, Barlow, Virella, & Connolly, 1998). The impact of such preventive health strategies on both incidence and severity cannot be evaluated without long-term surveillance systems. Information from population-based registries remains fragmented because of the limited number of children with PNN CP in geographical areas covered by each registry, which makes obtaining reliable estimates of the prevalence of PNN CP difficult. The evolution of profiles in the underlying causes remains also difficult to assess. In total, studies already published aimed to point out the documentary on evolution of the prevalence of CP PNN are scarce and their results partly contradictory.

The present paper aims to analyze the trends over time in prevalence of cerebral palsy of post-neonatal origin, to investigate whether changes are similar according to severity and to describe the disability profile by aetiology.

## 2. Material and methods

### 2.1. Design and case definition

The collaborative network of CP population-based registries, Surveillance of Cerebral Palsy in Europe (SCPE), established a common database that pooled annual anonymised data for children with CP born since 1976. For full information on geographic areas concerned, inclusion and exclusion criteria, go to <http://www.scpenetwork.eu/>. Following international recommendations, CP cases registered in SCPE database fulfilled inclusion criteria after their 4th birthday. To guarantee a high level of completeness they usually are recorded between 5 and 8 years. Children with PNN CP born between 1976 and 1998 and whose mothers lived at birth in an area covered by a contributing registry were collected from the SCPE database. A case of PNN CP was considered when the motor deficit was the result of a recognized causal event independent of antenatal and perinatal periods occurring between the 28th day after birth and 24 months. To get accurate estimations of yearly prevalence rates and to precisely assess trends over time, only registries with at least 8 years of data collection of PNN CP cases and at least 16,000 live births over the follow-up period were eligible.

### 2.2. Characteristics of children

Following SCPE recommendations (S.C.P.E., 2000), the CP subtypes were classified as unilateral spastic, bilateral spastic, dyskinetic or ataxic. Walking ability was used as a measure of severity of motor impairment. Severe intellectual impairment was defined as Intelligence Quotient (IQ) below 50 (based on IQ measure using a validated tool) or equivalent clinical assessment. The condition was considered as severe when the child was unable to walk even with assistive devices and had an  $IQ < 50$ , of moderate severity in case of ability to walk, even with limitations/assistive devices and  $IQ < 50$ , or inability to walk without assistive devices and  $IQ \geq 50$ , and of mild severity when the child was able to walk without assistive devices and had an  $IQ \geq 50$ . Additional impairments were also recorded: severe visual impairment (defined by visual acuity  $< 6/60$  (Snellen scale) or 0.1 (decimal scale) in the better eye following correction), severe hearing impairment (loss  $> 70$  dB on the better ear before correction) and active epilepsy (two unprovoked seizures – excluding febrile or neonatal seizures – still on medication at the time of assessment).

Age at brain insult, expressed in months, was reported. Aetiology as reported by each registry was coded by ICD-10 taxonomy (or ICD-9 depending on year of registration). According to previous published classification (Stanley et al., 2000), the following main groups of the presumed cause of the brain damage were considered: (1) infection (e.g. central nervous system infection, Reye's syndrome or severe dehydration following infection); (2) head injury, including accidental traumatic head injury (such as road traffic accident, fall) and non-accidental head injury (abuse, shaken baby syndrome);

(3) vascular episode (cerebral-vascular accident, heart surgery or other surgery complications, vascular aetiologies associated with congenital heart defects); (4) miscellaneous factors which include all other known causes (e.g. drowning sequelae, near-miss sudden infant death syndrome, brain tumor sequelae).

### 2.3. Analysis strategy

Yearly prevalence rates were calculated per registry and per 10,000 live births (presented with exact 95% confidence intervals (CI)) and considered as time series to analyze trends over time. Except in both French registries where the prevalence estimates were based upon the number of children with PNN CP living in the area at the time of registration to take into account the specific migration patterns, the denominator of prevalence rates was the total number of live births provided by each registry.

The plots of raw and LOWESS (LOcally WEighted Smoothing Scatter, parameter 0.8) (Cleveland, 1979) smoothed data were first generated. The CUSUM (Cumulative Sum) control chart was applied to graphically identify trend and potential break points (Leprêtre & Carpentier, 1997), using the average prevalence over the whole period as reference value. The significance of time trend was assessed with an extension of the Poisson regression model to take account over-dispersion (Jourdan & Célestin, 2002) in the distribution of the dependant variable. Thus, negative binomial regression models were applied, considering the number of PNN CP cases by registry and year as the dependant variable, time and registry as covariables and the logarithm of number of live births per year in each area as offset term. The assumption of linearity between time and the outcome and the interaction between year and registry were tested. When a break point was highlighted by the CUSUM chart, a piecewise binomial negative regression model was applied. The same strategy was used when analyzing trends by severity and aetiology.

All analyses were performed using STATA software (version 11.0 Stata Corp., College Station TX, USA).

## 3. Results

### 3.1. Study population: selection procedures and characteristics

The study population included 404 cases of PNN CP from ten registries (Fig. 1). Male accounted for 55.0% of cases. The sex ratio did not significantly differ between registries ( $p = 0.743$ ). The median age of the child at the onset of injury was 8.0 months, and for 79.0% of PNN CP cases, the injury occurred during the first year after birth. Compared with perinatal CP cases (Appendix SI, supplementary data published online), children with PNN CP were more often singletons, born of younger mothers, and more frequently with normal birth weight ( $BW \geq 2500$  g). Accompanying disabilities were more common in PNN CP. A higher proportion of severe cases was observed while the distribution of clinical subtypes didn't significantly differ.

### 3.2. Prevalence and trend over time

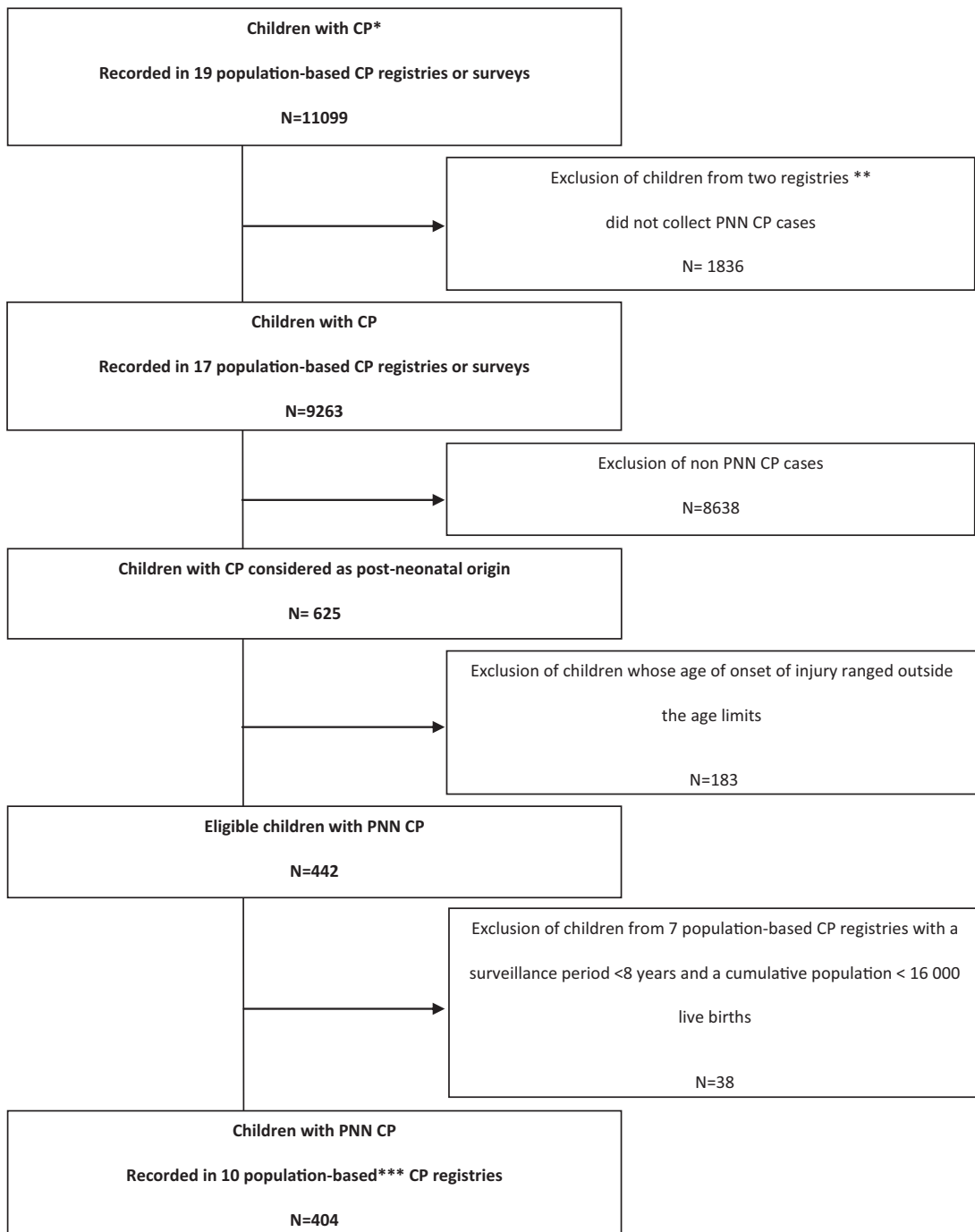
PNN CP cases represented 5.5% of the total CP cases (Table 1). Prevalence rates ranged from 2.05 (95% CI [1.07–3.02]) in 1976 to 0.41 (95% CI [0.14–0.67]) per 10,000 live births in 1998, the latter being the lowest. The plot of raw and smoothed data (Fig. 2a) suggested a downward trend over the period. No significant interaction between time and registries was found. An overall significant downward trend in the evolution of the prevalence of PNN CP over the period 1976–1998 was observed with a change in the decreasing trend in 1989. More precisely, the piecewise binomial negative regression showed a non significant downward trend from 1976 to 1989 ( $\beta = -0.032$ , 95% CI [-0.093; 0.030];  $p = 0.309$ ), associated with a significant downward trend from 1990 to 1998 ( $\beta = -0.115$ , 95% CI [-0.199; -0.031];  $p = 0.007$ ) (Fig. 2b).

### 3.3. Severity of impairments

Level of severity was known for 90.8% of the cases. Approximately one third of the cases were defined as severe, one-third as moderate and one third as mild. The mean age at onset tended to be lower in severe cases ( $7.0 \pm 0.5$  months compared with moderate or mild ones ( $9.0 \pm 0.6$  months;  $p = 0.078$ ). The mean prevalence rates [95% CI] per 10,000 live births were 0.41 [0.31; 0.52], 0.36 [0.26; 0.46] and 0.37 [0.29; 0.45] for severe, moderate and mild cases respectively. Over the period, there was a significant monotonic downward trend in the overall prevalence of severe PNN CP ( $\beta = -0.096$ , 95% CI [-0.147; -0.046];  $p < 0.001$  for time effect) whereas the prevalence rates of moderate and mild cases remained stable (Fig. 2c).

### 3.4. Aetiology

Aetiology was known in 87.4% of the cases and detailed in Table 2. Infections represented the most prevalent group of postneonally acquired CP (46.2%) with meningitis and encephalitis as the most numerous causes. Active epilepsy was significantly more frequent in this group (55.0%) compared to the others (37.3%;  $p < 0.007$ ). Non accidental injuries represented a large group of children with severe disability profiles (mainly bilateral lesions resulting in higher frequency of severe cases, of presence of active epilepsy, of associated severe visual impairment compared to accidental head traumas).



**Fig. 1.** Selection of children with post-neonatal cerebral palsy, born between 1976 and 1998. \*Whose mother lived inside the area at birth, except French registries. \*\*These registries were C10: Tübingen, Germany and C12: Copenhagen, Denmark. \*\*\*All CP cases from the 10 population-based CP registries selected accounted for 7285 cases.

We can notice that mothers of children with head injuries (accidental or not) were significantly younger at delivery ( $23.9 \pm 0.9$  years) compared to mothers of children with other causes of brain damage ( $27.0 \pm 0.2$  years,  $p = 0.023$ ). The “vascular events” group accounted for 19.5% of the cases and referred to various causal pathways. Near-miss-cot-death cases were characterized by a young age at onset ( $4.0 \pm 0.5$  months), bilateral lesions of the brain (90.9%) and a very severe disability profile.

The prevalence rates of PNN CP due to infections remained fairly stable from 1976 to 1988 ( $\beta = 0.0016$ , 95% CI  $[-0.085; 0.089]$ ;  $p = 0.971$ ), and since then tended to decrease ( $\beta = -0.130$ , 95% CI  $[-0.229; -0.032]$ ;  $p = 0.010$ ). The regression models also showed a slight decline in the prevalence rate of the heterogeneous group of PNN CP cases induced by miscellaneous

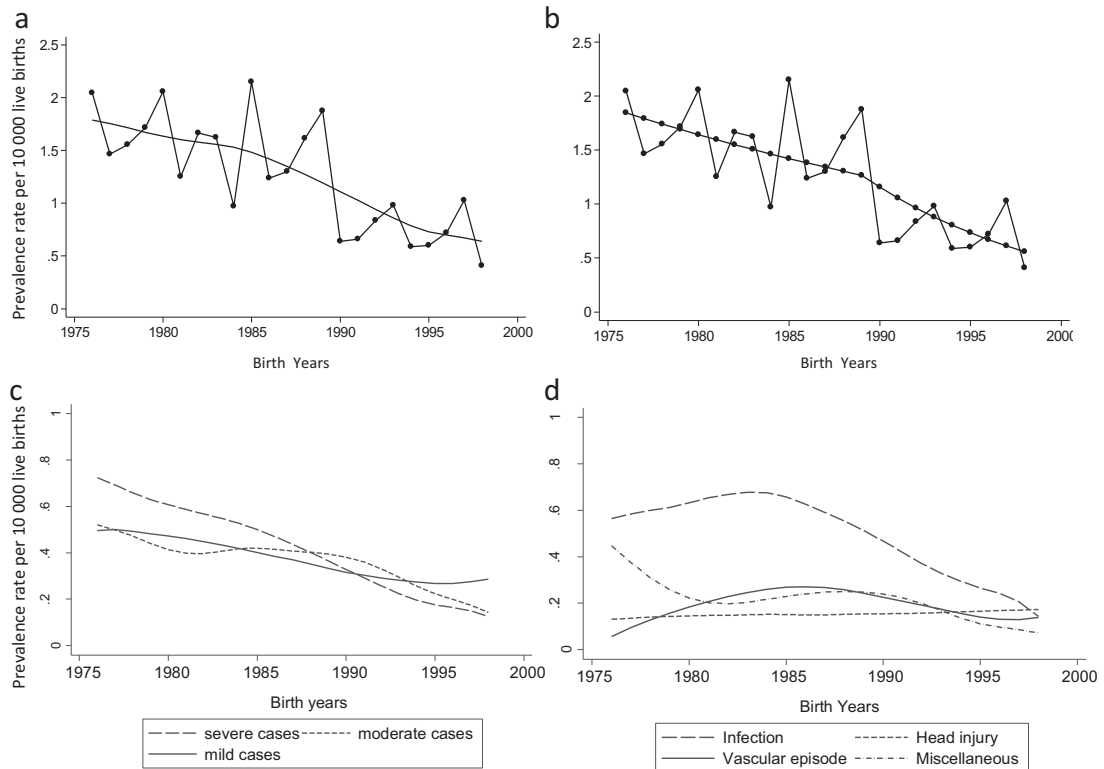
**Table 1**  
Proportions and rates of post-neonatal cerebral palsy in Europe (10 population-based registries), 1976–1998.

Centre	Birth years	All CP <sup>a</sup> cases (N)	Post-neonatal cases (n)	Proportion %	Live births (over the period considered)	Rate per 10,000 live births [95% CI] <sup>b</sup>
C01: Iserre (France)	1980–1998	538	47	8.7	263,894	1.78 [1.27–2.29]
C02: Haute Garonne (France)	1986–1998	246	8	3.3	153,249	0.52 [0.18–0.88]
C04: Cork (Ireland)	1976–1998	370	22	5.9	201,183	1.09 [0.64–1.55]
C05: Belfast (UK)	1981–1998	1091	41	3.8	439,807	0.93 [0.65–1.22]
C06: Göteborg (Sweden)	1976–1998	993	48	4.8	472,208	1.02 [0.73–1.31]
C07: Dublin (Ireland)	1976–1998	1037	81	7.8	495,728	1.63 [1.28–1.99]
C08: Newcastle (UK)	1991–1998	737	30	4.1	280,780	1.07 [0.69–1.45]
C09: Oxford (UK)	1984–1998	1079	53	4.9	520,254	1.02 [0.74–1.29]
C11: Mersey (UK)	1976–1989	983	64	6.5	412,318	1.55 [1.17–1.93]
C15: Tønsberg (Norway)	1991–1998	211	10	4.7	132,486	0.75 [0.29–1.22]
Total		7285	404	5.5 [5.0–6.0] <sup>c</sup>	3,371,907	1.20 [1.08–1.31]

<sup>a</sup> CP cerebral palsy.

<sup>b</sup> 95% CI: confidence interval (exact) of the prevalence.

<sup>c</sup> 95% Confidence interval of the proportion.



**Fig. 2.** Trend over time of post-neonatal cerebral palsy cases from 10 registries. (a) Raw and smoothed LOWESS data. (b) Raw data and predicted data (piecewise binomial negative regression). (c) Prevalence of post-neonatal CP according to aetiology. (d) Prevalence of post-neonatal CP according to severity.

Table 2

Distribution of causes of post-neonatal CP by characteristics, causes was unknown in 51 cases.

	N (%)	Bilateral CP type (%)	Unable to walk (%) <sup>e</sup>	Known to have IQ < 50 (%)	Known to have active epilepsy (%)	Known to have visual impairment (%)
Infection	<b>163 (46.2)</b>	98 (60.5)	63 (41.2)	104 (63.8)	89 (54.6)	29 (17.8)
Meningitis/encephalitis <sup>a</sup>	85	63.5	39.0	65.9	49.4	15.3
Other infection <sup>b</sup>	19	73.7	50.0	47.4	73.7	21.1
Reye's syndrome post infection	28	60.7	46.4	67.9	42.9	32.1
Severe dehydration related to severe infection	31	43.3	36.7	64.5	67.7	9.7
Head injury	<b>54 (15.3)</b>	24 (44.4)	15 (30.6)	29 (53.7)	16 (29.6)	12 (22.2)
Road traffic accident	11	45.5	20.0	36.4	9.1	27.3
Other accidental injury	13	15.4	16.7	38.5	30.8	7.7
Non accidental injury	30	56.7	40.7	66.7	36.7	26.7
Vascular episode	<b>69 (19.5)</b>	30 (44.1)	23 (37.1)	30 (43.5)	29 (42.0)	11 (15.9)
Cerebrovascular accident <sup>c</sup>	20	50.0	29.4	45.0	40.0	10.0
Post-heart surgery	21	47.6	45.0	52.4	33.3	23.8
Post-other surgery	20	42.1	36.8	45.0	55.0	15.0
Association with congenital heart disease	8	25.0	33.3	12.5	37.5	12.5
Miscellaneous	<b>67 (19.0)</b>	47 (71.2)	31 (48.4)	43 (64.2)	27 (40.3)	14 (20.9)
Drowning	7	100.0	85.7	100.0	57.1	28.6
Near-miss cot death	22	90.9	63.6	72.7	50.0	45.5
Others <sup>d</sup>	38	54.1	28.6	52.6	31.6	5.3
Total	<b>353 (100)</b>	<b>199 (56.9)</b>	<b>132 (40.2)</b>	<b>206 (58.4)</b>	<b>161 (45.6)</b>	<b>66 (18.7)</b>
		$p = 0.003^f$	$p = 0.263^f$	$p = 0.021^f$	$p = 0.007^f$	$p = 0.780^f$

<sup>a</sup> Among "Meningitis/Encephalitis" there were cases with an identified responsible infectious agent responsible such as: Herpes virus, *Haemophilus influenzae*, *Pneumococcus*, *Meningococcus*, *E. coli*, *Streptococcus*, *Bordella pertussis*, *Klebsiella*, other virus or bacteria (agent was not identified).

<sup>b</sup> Among the other infections there were cases of bronchiolitis, endocarditis, septicemia, and other not well defined infections (viral, convulsion, acute epiglottitis...).

<sup>c</sup> Among "Cerebrovascular accident" there were cases of AVC, cerebral hemorrhage, ischemic episode if no further details were provided.

<sup>d</sup> Among the other "miscellaneous" aetiologies, the most frequently observed were brain tumor, epilepsy, unknown encephalopathy.

<sup>e</sup> Estimated percentage without missing data.

<sup>f</sup>  $p$ -Value Chi-square test.

causes ( $\beta = -0.060$ , 95% CI [-0.117; -0.003];  $p = 0.040$ ) while no significant trend was found in the prevalence rates of cases induced by head injuries ( $\beta = -0.038$ , 95% CI [-0.077; 0.070];  $p = 0.919$ ) or vascular events ( $\beta = -0.028$ , 95%CI [-0.052; 0.058];  $p = 0.922$ ) (Fig. 2d).

#### 4. Discussion

CP cases with a post-neonatal origin still account for a significant proportion of CP cases and although our study showed a significant downward trend in the prevalence and severity of PNN CP, this trend did not include all causes.

At the time of the study, various practices were reported within the SCPE network in recording and defining PNN CP cases. Some registries did not record any PNN CP cases and when PNN CP cases were recorded on a routine basis, a significant variation in the upper age limit was observed. However, all registries agreed on the lower age limit and most of them defined the post-neonatal period between 28 days and 2 years old. We know from previous studies that post-neonatal cases are more frequent in the first year of life and more severe during 2 years after birth (Cans et al., 2004; Reid, Lanigan, & Reddihough, 2006). Moreover, beyond this age, aetiological profiles seem different with cases more likely related to traumatic head injuries (Reid et al., 2006).

We chose to consider only data from registries with at least 8 years of data collection of PNN CP cases and at least 16,000 live births over the follow-up period, leading to exclusion of 7 population-based CP registries. These centers recorded very few cases of CP with post-neonatal origin and seemed that they had not yet a policy of systematic recording of these cases. Despite an arbitrary choice, relevant registries for monitoring these conditions were not excluded.

All registries participating in the SCPE collaboration agreed on case definition, inclusion and exclusion criteria, classification and standardized minimum dataset (S.C.P.E., 2000), and produced much work to maintain reproducibility of classifying and coding cases (Gainsborough, Surman, Maestri, Colver, & Cans, 2008; Sellier, Horber, Krageloh-Mann, De La Cruz, & Cans, 2012) so ensuring reliable estimates of trends over time in prevalence rates for CP subgroups. In addition, all registries were able to ascertain children from a geographically defined population in their own area and all together included large numbers of cases to have sufficient statistical power to study subgroups nevertheless a lack of completeness of registration especially for the least severe cases, and variation of case ascertainment over time cannot be excluded despite our results supported a consistent decline in PNN CP prevalence in most registries. However, the main objective of such databases is to monitor trends by detailed aetiological subgroups to suggest preventive strategies or evaluate their impact.

Although the SCPE collaboration consistently used the same method of classification of aetiological profiles over time, the choice of a broad classification as usually done in the literature did not allow monitoring such detailed trends. In fact, each of those broad groups (with sometimes very rare cases) includes a variety of causes and underlying pathological mechanisms. Unfortunately there is no universally accepted detailed classification (Stanley et al., 2000). Progress could be made by further specifying aetiologies and developing a consensus in their categorization to improve evaluation of public health actions and comparisons between studies.

Over the whole studied period, we showed a monotonic downward trend in prevalence of PNN CP with an acceleration of decrease in the 1990s. Few data on trends in prevalence of PNN CP are available in literature and results seem inconsistent. Over the period 1960–1999, the Western Australia CP registry reported a significant increase from 1990 in the prevalence of PNN CP cases defined as age at onset below 5 years (Report of the Western Australian Cerebral Palsy Register to birth year 1999., 2006). At the same time and considering the same upper age limit, the Victoria group (Australia) showed a significant decrease in the occurrence of PNN CP (birth cohorts from 1977 to 1999) (Reid et al., 2006). Previous analysis of the SCPE database using the same case definition as we did, also revealed a declining trend until 1990 (Cans et al., 2004). The detailed analysis of the particular profile of trend found in our study in the late 1980s would require contextual data for all the regions concerned but some explanations may be related to the evolution of the reported causes of brain damage. Infections still dominated the aetiological profile in our database (nearly half of the cases). A significant decline in the prevalence rate was observed since 1989, consistent with previous published results (Cans et al., 2004; Reid et al., 2006). We may hypothesize that the introduction of Haemophilus Influenza vaccine (Eskola, 1995; McVernon, Mitchison, & Moxon, 2004; Morris, Moss, & Halsey, 2008; O'Loughlin et al., 2010) and the implementation of programs of universal immunization of infants with pneumococcal conjugate vaccine (Clarke et al., 2006; Fitzwater, Chandran, Santosham, & Johnson, 2012; Ivady, 2010) may have contributed to this decrease. Unfortunately, the viral or bacterial agent responsible for central infections was rarely reported in the registries databases (less than one in two). It is therefore essential to improve the coding of causes in general and of pathogen agents particularly.

Even if substantial achievements have been made in prevention of head injuries, our study failed to report a significant decrease in traumatic head injury group that was mainly composed by non accidental head injury causes of CP in this study. This result is somewhat different from those of the Victorian group in Australia which reported a significant fall in PNN CP due to accidental head injury (which represented 64.3% of cases in the “head injury” group) and for a similar surveillance period (1977–1999). Indeed head injury is the most common aetiology reported after the age of 2 years (Cans et al., 2004) and decline may be related to extended definition of age at brain insult. The number of PNN CP cases between the age of 2 and 5 years old is insufficient in the SCPE database to provide trends in a larger age group even if we can reasonably assume that such an evolution also exists in Europe. Non-accidental injuries still represented 8.5% of the cases and did not decrease over time despite widely publicized prevention messages. However, data from various sources (Gilbert et al., 2012; Louwers et al., 2012) indicate that reporting of child abuse cases has improved and consequently could explain the fairly stable prevalence rate over the period.

Cases with a post-surgery aetiology also remained important in terms of proportion (10%). Recommendations for the prevention of neurological complications of cardiac surgery operative procedures in children were recently proposed and we may expect a diminution of such cases in the future (Fallon, Aparicio, Elliott, & Kirkham, 1995; Kirkham, 1998). Other causes, quantitatively less numerous, should also have their impact reduced. Drowning remains the second leading cause of death in children and a major cause of disability in Europe (Rogman, 2000) with often severe conditions. The figure reported in this study (7 cases over the entire period) seems to reflect the difficulty to measure the true incidence of near drowning, even if non-fatal cases are very few compared to fatal ones. A retrospective analysis conducted in the US from 2001 through 2009 showed that the proportion of non-fatal cases was less than 15% of the submersion events (Shields, Pollack-Nelson, & Smith, 2011). Near-miss cot death syndromes still accounted for 22 cases in our database. Since the supine or lateral sleeping position has been recommended as well as educational programs against active and passive smoking in child's vicinity implemented, there have been significant decreases in incidence of cot deaths in many countries (over 75% reduction) (Institut de veille sanitaire, 2011).

## 5. Conclusions

Our study emphasises the need of large population-based surveillance systems to reliably monitor trends in prevalence in rare but severe subgroups of children with CP like those with a post-neonatal origin. Our results should encourage systematic recording of PNN CP, which is not currently the case in all registries to evaluate the effectiveness of public health actions targeted to prevent such events. While the reported decline in post-neonatal CP cases in Europe is encouraging, children who suffer the most severe conditions may experience dramatic personal changes and require life-long medical care. It is then compulsory to strengthen existing public health measures and develop primary prevention which must take advantage of many innovations.

## Conflict of interest

The authors declare that they have no competing interests.

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## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.ridd.2013.02.016>.

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