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Ulla Heikura

INTELLECTUAL DISABILITY IN THE NORTHERN FINLAND BIRTH COHORT 1986

FACULTY OF MEDICINE,
DEPARTMENT OF PUBLIC HEALTH SCIENCE AND GENERAL PRACTICE,
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Abstract

The objective of this study was to investigate intellectual disability (ID) in children, with focus on occurrence, associated biomedical and sociodemographic factors, probable psychiatric problems and temporal variations in the occurrence of ID and the associated factors in an interval of 20 years.

The study population consisted of two birth cohorts of children born in northern Finland, the Northern Finland Birth Cohort 1986 (NFBC 1986, N = 9,432 live-born children) and the Northern Finland Birth Cohort 1966 (NFBC 1966, N = 12,058 live-born children). Temporal changes in ID were studied by comparing NFBC 1986 with NFBC 1966. The same definition of intellectual disability (intelligence quotient ≤70), time of follow-up (up to 11.5 years), case ascertainment methods and data sources were used. Data were collected from questionnaires, registers and records.

In NFBC 1986 the incidence of ID was 12.62/1,000 by age 11.5 years and prevalence 11.2/1,000 live-born at age 11.5 years. Associated biomedical aetiology could be found in two thirds of the cases. Genetic disorders were the largest aetiological category (36.1%) associated with ID. Maternal disadvantage (unskilled worker, basic education only) had the largest impact on the incidence of ID, while among single independent factors, maternal prepregnancy obesity (body mass index ≥30) showed the highest risk for ID (OR 2.8, 95% CI 1.5, 5.3) in the offspring. According to the assessments by the teachers at school children with ID had 4.9 times more likely probable behavioural problems than their peers not having ID.

In an interval of 20 years, there was no change in the incidence or in the prevalence of ID between NFBC 1986 and NFBC 1966. However, a shift occurred from more severe levels of ID towards mild ID, so that both the incidence and prevalence of mild ID increased by 50% whereas more severe ID decreased by 50%. Temporal changes appeared in the proportions of aetiological categories (NFBC 1986 vs. NFBC 1966) with a statistically significant decrease of Down syndrome and paranatally originating causes (traumas/asphyxia). The proportion of chromosomal disorders other than Down syndrome increased, as did malformations of the central nervous system. Among sociodemographic factors associated with ID, indicators of socio-economic disadvantage retained their status as having the largest impact on the incidence of ID. Over the 20 years, the mother being single, living in a remote area and mother's older age at time of delivery had lost their association with ID. Only one new maternal sociodemographic factor, prepregnancy obesity, had emerged as having an association with ID with statistical significant difference between NFBC 1986 and NFBC 1966.

In conclusion, these results indicate that although the occurrence of ID remained the same in northern Finland over a period of 20 years, temporal changes have taken place in the biomedical and sociodemographic factors contributing to the incidence and prevalence of ID. There are also factors that have retained their status as associated disadvantageous factors. Studies like this with repeatedly collected data in the same geographical area, describing the occurrence of ID, and analysing associated biomedical and sociodemographic factors, are valuable for evaluating developments in the health care and service system. They are also of value for future planning of services for individuals with ID.

Keywords: aetiology, children, cohort study, epidemiology, incidence, intellectual disability, prevalence, sociodemographic factors

Heikura, Ulla, Kehitysvammaisuus Pohjois-Suomen syntymäkohortti 1986:ssa
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Tiivistelmä

Tämän tutkimuksen tavoitteena oli selvittää kehitysvammaisuuden esiintyvyyttä lapsilla, siihen liittyviä lääketieteellisiä etiologisia ja sosiodemografisia tekijöitä, mahdollisia psykiatrisia ongelmia sekä kehitysvammaisuuden esiintyvyydessä ja siihen liittyvissä tekijöissä tapahtuneita muutoksia 20 vuoden aikana.

Tutkimusjoukko muodostui kahden syntymäkohortin lapsista, jotka olivat syntyneet Pohjois-Suomessa, Pohjois-Suomen syntymäkohortti 1986 (NFBC 1986, N = 9432 elävänä syntynyttä lasta) ja Pohjois-Suomen syntymäkohortti 1966 (NFBC 1966, N = 12058 elävänä syntynyttä lasta). Kehitysvammaisuudessa tapahtuneita ajallisia muutoksia tutkittiin vertaamalla Pohjois-Suomen syntymäkohortti 1986:ta Pohjois-Suomen syntymäkohortti 1966:een. Tutkimuksessa käytettiin samaa kehitysvammaisuuden määritelmää (älykkyysosamäärä ≤70, seuranta-aika 11.5 vuoteen saakka), tiedonkeruun menetelmiä ja tietolähteitä. Tiedot kerättiin kyselylomakkeista, rekistereistä ja asiakirjoista.

Pohjois-Suomen syntymäkohortti 1986:ssa kehitysvammaisuuden ilmaantuvuus oli 12.62/1000 11.5 vuoden ikään mennessä ja vallitsevuus 11.23/1000 11.5 vuoden iässä. Kehitysvammaisuuteen liittyvä lääketieteellinen etiologia pystyttiin selvittämään kahdessa kolmasosassa tapauksia. Geneettiset häiriöt muodostivat suurimman etiologisen luokan (36.1 %). äitiin liittyvillä epäedullisilla sosiaalisilla tekijöillä (kouluttamaton työntekijä, vain peruskoulutus) oli suurin vaikutus kehitysvammaisuuden ilmaantuvuuteen, kun taas yksittäisistä sosiodemografisista tekijöistä korkein riski (vaarasuhde 2.8, luottamusväli 1.5, 5.3) oli äidin lihavuudella (painoindeksi ≥30) raskauden alussa. Koulussa opettajien arvioiden mukaan kehitysvammaisilla lapsilla esiintyi mahdollisia käytöshäiriöitä 4.9 kertaa useammin kuin ei-kehitysvammaisilla lapsilla.

20 vuoden aikana Pohjois-Suomen syntymäkohorttien 1986 ja 1966 välillä ei ollut tapahtunut muutoksia kehitysvammaisuuden kokonaisilmaantuvuudessa eikä -vallitsevuudessa. Kuitenkin tuli esiin siirtymä vaikeammasta lievempään asteeseen siten, etta lievän kehitysvammaisuuden ilmaantuvuus ja vallitsevuus lisääntyivät noin 50%, kun taas vaikeamman väheni 50%. Lääketieteellisten etiologisten luokkien osuuksissa tuli esiin ajallisia muutoksia (Pohjois-Suomen syntymäkohortti 1986 vs. Pohjois-Suomen syntymäkohortti 1966) siten, että Downin syndrooman sekä syntymän aikaan ajoittuvan vamman ja hapenpuutteen osuudet vähenivät tilastollisesti merkitsevästi. Keskushermoston epämuodostumien sekä muiden kromosomihäiriöiden kuin Downin syndrooman osuudet kasvoivat. Kehitysvammaisuuteen liittyvistä sosiodemografisista tekijöistä sosioekonomisen huono-osaisuuden osoittimet säilyttivät asemansa suurimpana ryhmänä. 20 vuoden aikana äidin naimattomuus, asuminen syrjäseudulla sekä korkeampi ikä lapsen syntymän aikaan olivat menettäneet yhteytensä kehitysvammaisuuteen. Pohjois-Suomen syntymäkohortti 1986:n ja Pohjois-Suomen syntymäkohortti 1966:n välillä tuli esiin vain yksi uusi kehitysvammaisuuteen tilastollisesti merkitsevästi liittyvä sosiodemografinen tekijä, äidin lihavuus raskauden alussa.

Yhteevetona voidaan todeta, etta vaikka kehitysvammaisuuden kokonaisesiintyvyys oli pysynyt samana Pohjois-Suomessa 20 vuoden aikana niin esiintyvyyteen liittyvät etiologiset ja sosiodemografiset tekijät olivat osittain muuttuneet. Tämänkaltaiset tutkimukset, joissa peräkkäisinä ajanjaksoina kerätään tietoja samalla maantieteellisellä alueella ja jotka kuvaavat kehitysvammaisuuden esiintyvyyttä sekä analysoivat siihen liittyviä lääketieteellisiä ja sosiodemografisia tekijoitä, ovat hyödyllisiä arvioitaessa terveydenhoidossa ja palvelujärjestelmässä tapahtunutta kehitystä. Niitä voidaan hyödyntää myös suunniteltaessa tulevaisuudessa palveluja kehitysvammaisille henkilöille.

Asiasanat: epidemiologia, etiologia, insidenssi, kehitysvammaisuus, kohorttitutkimus, lapset, prevalenssi, sosiodemografiset tekijät

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Abbreviations

AAMR	American Association on Mental Retardation
AGU	Aspartylglycosamiuria
BMI	Body mass index
CDI	Child Depression inventory
CI	Confidence interval
CNS	Central nervous system
DSM	Diagnostic and Statistical Manual of Mental Disorders
FAS	Fetal alcohol syndrome
ICD	International classification of diseases
ID	Intellectual disability
INCL	Infantile neuronal ceroid lipofuscinosis
IQ	Intelligence quotient
NFBC 1966	Northern Finland Birth Cohort with expected date of birth in 1966
NFBC 1986	Northern Finland Birth Cohort with expected date of birth in 1985/ 1986
NKH	Nonketotic hyperglycinaemia
OR	Odds ratio
PAR	Population attribution risk
PDD	Pervasive development disorder
RB2	Children's Behaviour Questionnaire for Completion by Teachers by Rutter
RR	Risk ratio
SES	Socioeconomic status
WHO	World Health Organization
WISC	Wechsler Infant Scale for Children
WISC-R	Wechsler Infant Scale for Children – Revised
WPPSI	Wechsler Preschool and Primary Scale of Intelligence

List of original publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals:

I Heikura U, Taanila A, Olsen P, von Wendt L, Hartikainen A-L & Järvelin, M-R (2003) Temporal changes in incidence and prevalence of intellectual disability between the birth cohorts 1966 and 1985–86 in Northern Finland. Am J Ment Retard 108 (1): 19–31.

II Taanila A, Ebeling H, Heikura U & Järvelin M-R (2003) Behavioral problems of 8-year-old children with and without intellectual disability. J Pediatr Neurol 1 (1): 15–24.

III Heikura U, Linna S-L, Olsen P, Hartikainen A-L, Taanila A & Järvelin M-R (2005) An etiological survey of intellectual disability in the Northern Finland Birth Cohort 1986. Am J Ment Retard 110(3): 171–180.

IV Heikura U, Taanila A, Hartikainen A-L, Olsen P, Linna S-L, von Wendt L & Järvelin M-R (2007) Variations in prenatal sociodemographic factors associated with intellectual disability: A study of 20 year interval between two birth cohorts in Northern Finland. Am J Epidemiol in press. doi:10.1093/ajc/kwm291

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1 Introduction

Intellectual disability (ID) has always been a socioculturally determined phenomenon. The definition of ID is based on the classification practices stemming from the prevailing cultural beliefs and norms. (Scheerenberger 1986, Vehmas 2004). Traditionally individuals, who were considered mentally or physically different, have been treated with ambivalence in the Western societies. On one hand there was a "humanitarian" tradition aiming to help those perceived to be in need of help, while on the other hand there was a "social control" or exclusion tradition tending to hide the individuals perceived as not fitting the norms of society. (Woodill & Velche 1995). It is probable that there have always been individuals with ID, but the modern concept of ID in Western civilization emerged well after the recognition of other disabilities such as blindness, epilepsy and malformations (Berkson 2004). In ancient Greece, being born with a physical difference was seen as a sign of anger of the gods. During the Middle Ages, the Renaissance and the Reformation, a period extending from approximately 476 to 1600 A.D., attitudes towards and treatment of disabled persons varied considerably in different parts of Europe. The religious influence of Christianity spread throughout Europe and was also manifested as a number of hospitals and foundling homes intended to provide care for children who were abandoned. These facilities did not provide education or training, but were primarily dedicated to physical care. ID and mental illness were considered synonymous, and persons so afflicted were not believed to suffer from hunger, cold or pain. (Scheerenberger 1986). In the agrarian, labour-intensive and largely illiterate medieval society, individuals who would in our times be labelled as intellectually disabled were not considered as being particularly disabled (Stainton 2001).

Changes occurred during the late 17th and 18th centuries as philosophers and scientists initiated ideas aimed at educating individuals (Scheerenberger 1986). John Locke (1632–1704) introduced the idea that individuals are not born with the principles and ideas of absolute doctrinal truth, only with the machinery by which they will eventually grasp them. The infant mind is a blank slate, which receives external data empirically; individuals become intelligent if they possess the normal psychological equipment to process the external data. Locke stated that the disability of some individuals is their failure to form "abstract ideas". (Goodey 2001). Jean Etienne Esquirol (1772–1840), a French psychiatrist, was the first to differentiate between mental illness and ID as well as to establish levels of ID. He believed that rather than being a single phenomenon, ID existed in a continuum (i.e., there

were degrees of ID) and so he differentiated between idiots, whose ID was severe, and imbeciles, whose ID was not as significant. (Taylor *et al*. 2005).

After the differential diagnosis of ID and mental illness there emerged a need in the educational system for a practical, empirically based identification and classification system for ID. The emergence of public school education for children required an accurate, yet efficient assessment tool so that those who could not benefit from normal schooling could be identified. (Lewis & Sullivan 1985). The French psychologist Alfred Binet (1857–1911) discovered an appropriate way to measure intelligence or mental ability (Matarazzo 1972). In 1904, Binet and his co-worker Simon were asked by the Minister of Public Instruction to develop a method to identify the children attending an ordinary school who cannot profit from the instruction given because of the state of their intelligence and who should be admitted into special class. The Minister of Public Instruction had decided that no child suspected of ID should be eliminated from ordinary school and admitted into special class without being subjected to a pedagogical and medical examination. (Binet & Simon 1905). Binet developed a psychological method to distinguish mentally retarded children from children of normal intelligence. The scales developed by Binet and his co-worker Theodore Simon became the basis for individual intelligence tests in a number of countries and languages. (Matarazzo 1972)

The definition of ID in the USA and England had different emphases from the beginning of the 19th century until the second half of the 20th century, so that in USA the criterion for ID was solely an IQ score of 70 or below based on a standardized psychometric test (Cronbach 1975), while in England social behaviour along with IQ formed the criteria for ID. In England's Mental Deficiency Act in 1913 the definition of ID comprised emphasis on the incapability of adaptation to the normal environment of one's peers as well incapability to maintain existence independently of supervision, control or external support. In 1959 the American Association on Mental Deficiency published the Manual on Terminology and Classification in Mental Retardation by Heber, which established objective criteria for diagnosis, classification, and reporting of ID. ID was defined as sub-average intellectual functioning, which originates during the developmental period and is associated with impairment in adaptive behaviour. (Matarazzo 1972).

The attitudes towards individuals with disabilities have changed from institutionalization and exclusion and they have been developed, so that from the 1960s until recently a tendency for inclusion and promoting independence of individuals with disabilities has dominated (Woodill & Velche 1995). This attitude has been called the normalization principle which can be understood as making available to

all individuals with ID patterns of life and conditions of everyday living which are as close as possible to the regular circumstances and ways of life of society. The principle concerns all individuals with ID and it should serve as a guide for e.g. medical, educational and social work with individuals with ID. (Nirje 1980). These same trends have also been in evidence in Finland (Kaski *et al*. 2001).

The incidence and prevalence as well as aetiological risk factors for intellectual disability in a birth cohort of children born in 1966 in northern Finland (Northern Finland Birth Cohort 1966, NFBC 1966) were studied by Paula Rantakallio and Lennart von Wendt as part of a large prospective study on long-term morbidity of children born in 1966 in northern Finland (Rantakallio 1969). The prevalence of severe level of intellectual disability reported by Rantakallio and von Wendt (1986) in their study was higher than in the corresponding studies carried out in other developed countries, which has been assumed to be due to a thorough method of case ascertainment in the review-articles by Kiely (1987), McLaren & Bryson (1987) and Roeleveld *et al*. (1997). Rantakallio and von Wendt (1985) presented that there was no evidence that the higher prevalence of severe level of ID would be attributable to any specific aetiological cause.

Comparison of two epidemiological studies on the occurrence of ID in the same geographic area would give an opportunity to estimate e.g. changes in associated factors such as aetiology. The comparison would also be valuable in assessing the development carried out in the study region aimed to prevent ID. However, many researchers have stated that it is difficult to find two single studies on the occurrence of ID that are comparable in methodology (Roeleveld *et al*. 1997). The aim of present study is firstly to examine intellectual disability (ID) in a birth cohort of children born in northern Finland in 1985/1986 (Northern Finland Birth Cohort 1986, NFBC 1986). The occurrence of ID, associated biomedical and sociodemographic factors as well as the prevalence of behavioural problems in children with ID were studied. Secondly, the aim is to examine temporal changes in the occurrence of ID as well as in the associated biomedical and sociodemographic factors in an interval of 20 years in northern Finland by comparing the results obtained from the NFBC 1986 with the corresponding results from the NFBC 1966. The same data collection methods and the same definition of ID were used with both cohorts.

2 Review of the literature

2.1 Definition and classification of intellectual disability

When defining a term such as ID it must be explained as precisely as possible, and the definition should establish the boundaries of the term. When classifying what has been included in the term by its definition different subgroups can be formed according to certain principles. Classification may be a component determining eligibility for services and benefits. It is important to update the classification system periodically, to incorporate new findings and accommodate changing concepts and philosophies. (Luckasson *et al*. 2002). Various terms referring to ID have been used and they reflect both the times and the context of their use. Examples of the terms that have been used in recent and past years are feebleminded, moron, imbecile, idiot, mental retardation, mental handicap, mental deficiency, learning disabilities, intellectual impairment, intellectual disability, mental disability. (Louhiala 2004). In this study, the term ID, intellectual disability, is used.

There are three widely used approaches in classifying ID. They are the International Classification of Diseases (ICD) by the World Health Organization (WHO), the definition and classification by the American Association on Mental Retardation (AAMR) and the Diagnostic and Statistical Manual of Mental Disorders (DSM) by the American Psychiatric Association.

In the ninth edition of the International Classification of Diseases (ICD-9, WHO 1977, pp. 212–213) ID is defined as a condition of arrested or incomplete development of mind, which is especially characterized by sub-normality of intelligence. The coding should be made on the individual's current level of functioning without regard to its nature or causation – such as psychosis, cultural deprivation, Down syndrome etc. The assessment of intellectual level should be based on all information available, including clinical evidence, adaptive behaviour and psychometric findings. The intelligence quotient (IQ) levels given are based on a test with a mean of 100 and a standard deviation of 15 – such as the Wechsler scales. They are provided only as a guide and should not be applied rigidly. ID frequently involves psychiatric disturbances and may often develop as a result of some physical disease or injury. In ICD-9, ID is classified into levels of mild ID (IQ 50–70), moderate ID (IQ 35–49), severe ID (IQ 20–34), profound ID (IQ under 20) and unspecified ID (ID not otherwise specified, NOS, and mental subnormality NOS). The classification presented in ICD-9 has been used in this study, except that the term mental retardation has been replaced with the term intellectual disability.

In the definition and classification of ID by AAMR ID is defined as ”a disability characterized by significant limitations both in intellectual functioning skills and in adaptive behaviour as expressed in conceptual, social, and practical adaptive behaviour skills. This disability originates before age 18.” In application of the definition of ID five assumptions have been specified: 1) Limitations in present functioning must be considered within the context of community environments typical of the individual’s age peers and culture, 2) Valid assessment considers cultural, and linguistic diversity as well as differences in communication, sensory, motor, and behavioural factors, 3) Within an individual, limitations often coexist with strengths, 4) An important purpose of describing limitations is to develop a profile of needed supports and 5) With appropriate personalized supports over a sustained period, the life functioning of the person with mental retardation will generally improve. (Luckasson *et al*. 2002, p.8). Diagnosis of ID is based on standardized IQ tests and tests of adaptive behaviour skills, coupled with team member observations. Intellectual ability is best represented by IQ scores and the criterion for diagnosis of ID is approximately two standard deviations below the mean, considering the standard error of measurement of the specific measurement. The classification of ID by severity can be based on different criteria, such as IQ range, support intensity and aetiology, depending on the purpose. (Luckasson *et al*. 2002)

According to the definition in Diagnostic and Statistical Manual of Mental Disorders (DSM) IV by the American Psychiatric Association (1994), ID is a developmental condition characterized by a significantly lower than average level of general intellectual functioning. Diagnostic criteria for ID exist when a) intellectual functioning is significantly below average, meaning an IQ of about 70 or lower in a person who can take an IQ test. b) Clinical judgment must be used on those individuals who cannot take an IQ test. Impairments or deficits in functioning should appear in at last two of the following areas: communication, health, leisure time, safety, school, self-care, social, taking care of a home and work. c) The onset must be before the age of 18. An individual with ID does not yield to an intelligence level of his or her peers. Levels of ID by severity are mild ID (IQ 50–55 up to about 70), moderate ID (IQ 35–40 to 50–55), severe ID (IQ 20–25 to 35–40) and profound ID (IQ below 20 or 25). ID with severity unspecified is defined when there is a strong presumption of ID but standard tests cannot be used to determine level of impairment.

Psychometric testing of intelligence

In all the classification systems mentioned earlier there is a notion that ID should be classified and the diagnosis of ID made when the IQ of an individual falls two standard deviations below the mean through an IQ obtained in a standardized psychometric test. The Stanford-Binet and the Weschler Scales are the most widely used standardized tests for evaluating IQ in individuals with ID (Nihira 1985). In the Weschler Scales IQ is defined as a method of defining relative intelligence at a given time among the age peers of the individual. General intelligence is, however, considered a noninterrupted continuum in an ordinal scale, but the classification of intelligence is based essentially on a statistical concept of tested intelligence. Each intelligence level has a class interval embracing IQs falling at measured distance from the mean, these distances being expressed as multiples of standard deviations. The scheme of classification follows the Gaussian symmetrical classification, comprising as many classes above the mean as there are below it. So, the choice of limiting individuals with ID from intellectually normal individuals is in part arbitrary. For example, the Weschler Scales defined as intellectually disabled those individuals who comprise about 2 to 3% of the total area of the normal curve, 0.13% falling in the severe (IQ below 55–50) and 2.14% in the mild ID (IQ 70 to 55–50) groups. (Anastasi 1988)

The advantage of classifying the severity of ID by the IQ score obtained in a standardized individually administered psychometric test is that it is an approach that is widely used internationally, which facilitates comparisons of the epidemiological studies on ID carried out in different countries. The major limitation is that classification based solely on IQ level emphasizes individuals' deficits and masks profiles of cognitive strengths and weaknesses, it does not convey information about adaptive behaviour and needed supports, and it also relies heavily on IQ testing, and may thus invite the possibility of cultural bias. (Durkin & Stein 2000). In addition, IQ test results in one child can vary according to mood, motivation and fatigue, which is why in clinical practice test results are always taken in the context of the respective test situation and the wider child-specific/environmental factors (Anastasi 1988, O'Brien 2001).

2.2 Incidence and prevalence of intellectual disability

Studies on the prevalence of ID indicate that rates vary according to temporal, demographic and social factors as well biomedical risk factors. This variability

resembles the variability of any other chronic condition. The methods of case acquisition are analogous to the method of detection of most disabilities and chronic conditions. Prevalence rates of ID rely on a numerator that includes only those cases who were referred for further clinical study, because there is rarely an opportunity to screen an entire population for a disabling condition like ID. Although ID is usually considered a life-long disability, the analysis of prevalence rates indicates that ID is most often identified during the mid-school years, when the individual is approximately 10 to 16 years of age, and more rarely in early life or in later adulthood. One important reason for the variability in the assignment of this label is because physicians, educators and the public consider the label ID in different ways. Physicians rarely diagnose ID unless it is severe or associated with a genetic or medical syndrome. They are mainly concerned with aetiology as well as the possibility of medical or surgical intervention, and prediction about reoccurrence in subsequent pregnancies. Educators are interested in academic achievement, and intellectual level is considered an important determinant of basic potential affecting success at school. Finally, the public may use the label to describe poor adaptive skills. Many individuals characterized in childhood or adolescence as having mild ID may gradually become indistinguishable from general population in adulthood. Adults with ID who have adapted to the mainstream of society by holding jobs and living independently are not always described as having ID. (McDermott 1994, Luckasson *et al*. 2002)

Strategies for estimating the prevalence of ID are registers, birth cohort studies, administrative agencies and population-based surveys: 1) Registers are a common method of estimating prevalence in Western countries other than the United States. They tend to be complete in their identification of persons with ID through mandated reporting. A major limitation with registers is that they may overestimate the prevalence of ID if individuals are not removed from registers due to death, changes in diagnosis status or changes in geographical location. 2) Birth cohort: the benefit of studies like this is that they eliminate the likelihood of duplication found in registers. They are useful in identifying factors that may be associated with differing prevalence rates among different population groups (e.g. people living in poverty, low birth-weight children). They are also useful in attending to changes in diagnosis and status over time. The main limitation of the cohort method is attrition. The number and characteristics of persons lost to the study scope can affect prevalence estimates. 3) Administrative prevalence: in this method, researchers survey service agencies (or schools within a particular geographic area) to obtain an unduplicated count of all persons receiving or qualifying

for the agency's services. However, this method tends to identify only persons who are receiving services and overlooks persons who lack services. Case finding or case census is an extension of administrative prevalence. 4) Population-based surveys: Researchers conducting population-based or cross-sectional prevalence surveys select a random sample of persons from a population in a specific geographical region. This sample is screened either through interviews or diagnostic examination to identify those with the condition of interest. (Larson *et al*. 2001)

Table 1. Epidemiological studies on the incidence and prevalence of intellectual disability (ID) from 1970.

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Wing (1971) UK	N=38,460. Children who had home addresses in the area of Camberwell, UK, on 31 Dec 1967.	0–14	ICD 8 Severe ID=IQ<50	The Camberwell register (Children, who have a contact to a psychiatric or mental retardation services, children excluded from education in school)	Severe ID in children aged 5–14 years: 3.99
Brask (1972) Denmark	N=52,880 All children registered at Aarhus county, Denmark, in 1960 and 1965.	0–14	Severe ID=IQ<50, Mild and borderline ID=IQ 50–80.	Administrative prevalence: registers of the MRS (Mental Retardation Service) (administrative prevalence = the number of mentally retarded persons for whom services would be required in a community having provisions).	Severe ID 2.55, mild and borderline 2.36. Total ID 4.92
McDonald (1973) Canada	N=141,500 Children born in 1958 and living in the Province of Quebec, Canada, between 1966–1969.	8–12	ID=IQ<50.	Hospitals, institutions, schools, and homes for retarded children, Individual assessments by author	Severe ID 3.8. Incidence of severe ID 5.4.
Bernsen, (1976) Denmark	N=50,667 Children born between 1 July 1955 and 30 June 1970, and living in Aarhus, Denmark, on 1 July 1970	0–14	Severe ID=IQ<50 (=traditional definition in use in Denmark	Registers, hospital records, child guidance clinics.	Severe ID 3.38
Laxova, Ridler & Bowen-Bravery (1977) UK	N=46,960 Children born between 1 Jan 1965, and 31 Dec 1967 and living in Hertfordshire, UK, in 1972.	7–9	Severe ID=IQ≤50.	Registers, medical officers, general practitioners, schools for severely retarded children, individual investigations by the authors.	Severe ID 3.1
Gustavson, Holmgren, Jonsell & Blomqvist (1977) Sweden	N=40,871 Children born between 1959–1970 and alive at the age of 1 year in the county of Västerbotten, Sweden.	1–16	ID=IQ<50 judged by conventional tests and present before the age of 18 years.	Register for mentally retarded children, (Board for the Provision and Services to the Mentally Retarded), death registers, hospital records, school records, and intelligence assessments	The mean yearly incidence of severe ID 3.9. Prevalence at 11–16 years 3.5.
Gustavson Hagberg, Hagberg & Sars (1977) Sweden	N=37,210 Children born between 1959–1970 and alive at age of 1 year in Uppsala, Sweden. Follow-up until year 1975.	5–16	Severe ID=IQ<50	Register for children with ID for receiving services, case records in hospitals, death register.	Cumulative incidence of severe ID at 1–16 years of age 3.3. Prevalence 2.8 (11–16 y.)

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Hagberg, Hagberg, Lewerth & Lindberg (1981) Sweden	N=24,498 children born 1966–1970 and living in Gotherburg, Sweden on 31 Dec 1978.	8–12	WHO (1968) Mild ID=IQ 50–70	The Registers of the Board for Provisions and Services to the Mentally Retarded, case records in special schools and institutions, hospital registers.	Mild ID 3.7
Blomquist Gustavson & Holmgren (1981) Sweden	N=40,871, Children born between 1959–1970 and alive at age of 1 year, a yearly incidence of mild ID, end of follow-up on 1 Jan 1979 in the county of Västerbotten, Sweden	8–19	ID=IQ 50–69	Registered for mentally handicapped children for receiving services**,** (Board for the Provision and Services to the Mentally Retarded), hospital case-records, and other relevant records.	Mild ID: Mean yearly incidence of period 1959–1970 divided into three four-year periods 4.2, prevalence 3.8
Koller, Richardson & Katz (1984) UK	N=8,274 Children born between 1952 and 1954 and who were residents of a British city in 1962.	8–10.	Mild ID=IQ 55–69.	An administrative prevalence: Individuals were defined as having ID if they were placed in any mental retardation faculty.	Mild ID 4.5
Baird & Sadovnick (1985) Canada	N=73,9785 Children born 1952–1966 and alive at the end of 1981 in British Columbia, Canada.	15– 29	ICD 9 ID=IQ<70 Profound ID IQ<20, severe ID IQ 20–34, moderate ID IQ 35–49, mild ID IQ 50–69	The British Columbia Health Surveillance Registry = A registry based on more than 80 sources of ascertainment,	Profound ID 0.5, severe ID 0.6, moderate ID 1.1. Severe total, 2.2, mild ID 1.7 Unspecified ID 1.7 Total ID 7.7
Rantakallio & von Wendt (1986) Finland	12,058 (incidence) Children born in 1966 in northern Finland, a birth cohort study with a follow-up until the age of 14 yrs. (N of children alive at the age of 14 11,766, prevalence)	0–14	ICD 8 ID=IQ 70, mild ID=IQ 50–70, severe ID=IQ<50.	Hospital and institutional records, national registers, child subsidies for chronically sick children, questionnaires.	Cumulative incidence: Severe ID 7.4, mild ID 5.5, Total 13.4 Prevalence at age 14: Severe ID 6.3, mild ID 5.6 Total 13.7
McQueen, Spence, Garner, Pereira & Winsor, (1987) Canada	N is not given. All children living in the Maritimes, Canada, on the census date of 1 Jan 1980 and who were born between 1 July 1969 and 30 June 1972.	7–10	Grossman 1977 (AAMD) ID=IQ<55, Profound ID IQ<25, Severe ID IQ 25–39, Moderate ID IQ 40–54	200 school boards, health and social service agencies and institutions, local parent associations, regional clinics, special purpose handicap registers)	IQ<55: 3.65. Profound ID 0.34 severe ID 1.19, moderate ID 2.12

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Diaz-Fernandez (1988) Spain	N=2,750,000 Population registered living in Galicia, Spain, on 1 Jan 1983.	All ages	ID=IQ<50	Administrative prevalence: Registers for handicapped persons of the Spanish Health Ministry's Diagnosis and Therapeutic Guidance Centres and the Department of Social Security's lists of registered handicapped persons from 1965 to 1983.	Severe ID 3.40
Benassi, Guorino, Cammarata, Cristoni, Fantini, Ancona, Manfredini & D'Alessandro (1990) Italy	N=26,494 Children living in Bologna Italy on 31 Dec 1986.	6–13	ID=IQ≤50	A diagnosis of ID in the record of the school service of local health unit (type and degree of impairment, and support needs	Severe ID 3.4
Mallon, MacKay, McDonald & Wilson (1991) Northern Ireland	N=about 1,500,000 All individuals living in Northern Ireland.	All ages	Severe ID=IQ<50	Files of all registered, mentally handicapped individuals.	Severe ID between 2.76–3.85
Wellesley, Hockey, Montgomery & Stanley (1992) Australia	N=210,789. Birth cohorts of children born in Western Australia between 1967 and 1976.	6–16	American Association of Mental Deficiency (Grossman 1983), ID=IQ<70, Profound ID IQ<20, severe ID IQ 20–34, moderate ID IQ 35–54, mild ID IQ 55–69.	Governmental agencies providing services for intellectually handicapped and schools and hospitals.	Profound ID 0.6, severe ID1.0, moderate ID 2.4, mild ID 3.0. Total ID 7.6. (0.8 unknown IQ)

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Sonnander, Emanuelsson & Kebbon (1993) Sweden	N=7,989 29 municipalities representative of Swedish districts were selected; a systematic selection of school classes in these municipalities.	12–13	Psychometric criterion two SD below the mean. ID=IQ≤70,	Information from the school offices collected yearly and the information collected from the individuals, parents, and teachers. All the pupils were tested once at 13 years of age. Tests developed at the institute for educational research, administered by teachers. The questionnaires to the pupils and parents were given once. Standard achievement tests were completed on several occasions as part of the regular school routine.	Mild ID 14.5
McDermott (1994) USA	N=616,713 Children at school during the school year 1980–1981 in South Carolina, USA	School-age children	South Carolina State Department of Education handbook. ID=IQ≤70 profound mental handicap IQ<25, trainable IQ 25–50, educable IQ 50–70.	The data were collected from two South Carolina State Department of Education annual publications, mental retardation placements of children in the state	ID IQ≤70 41.66. Profound 0.67, trainable 3.57, educable 37.41
Katusic, Colligan, Beard, O'Fallon, Bergstralh, Jacobsen & Kurland (1995) USA	N=5,919, A birth cohort of children born 1976–1980 in Rochester, Minnesota, USA	From birth until 8 years	ID=IQ<71.	Community medical records and files from public school system.	Cumulative Incidence of total ID 9.1. Severe ID 4.9, mild ID 4.3,
Murphy, Yeargin-Allsop, Decouflé, & Drews (1995) USA	N=89,534 Children who were born between 1 Jan 1975 through 31 Dec 1977 and who resided in Metropolitan Atlanta area, USA, at 10 years of age.	10	DSM III ID=IQ≤70. Profound ID=IQ<20, severe ID=IQ 20–34, moderate ID=IQ 35–49. Severe ID=IQ<50. Mild ID=IQ 50–70.	Public schools, hospitals, health departments, service agencies etc.	Profound ID 0.9, severe ID 0.6, moderate ID 2.1. Severe (IQ<50) 3.6, mild 8.4. Total ID 12.0

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Matilainen Airaksinen, Mononen, Launiala & Kääriäinen (1995) Finland	N=12,882 Four birth cohorts of children born in 1969–1972 and living in the province of Kuopio Finland were screened at the age of 8–9 years of age.	8–9.	American Association of Mental Deficiency (Grossman 1973), ID=more than 2 sd below the mean of the tests. Severe ID=IQ 55, mild ID=IQ 56–70	First: School achievement test in reading and arithmetic, second: psychological tests for the weakest children in the school achievement tests and for children of the same births cohorts registered by the provincial social services as mentally retarded.	Severe ID=6.3, mild ID=7.5. Total ID 13.8.
Yaqoob, Bashir, Tareen, Gustavson Nazir, Jalil, von Dobeln & Ferngren (1995) Pakistan	N=1,476 live born children A cohort of children born in and around Lahore, Pakistan during 1984–1987.	2–24 months old	Severe ID=IQ<50. (WHO 1980)	A house-to-house survey by the field team. A prospective cohort study. The Griffith's Mental Developmental Scales.	Incidence of severe ID=11.
Beange & Taplin (1996) Australia	N=104,584 Individuals born between 30 June 1938 and 30 June 1968 and who were living in a designated area in Australia for 6 months between June 1988 and December 1988.	20–50	AAMR, (Grossman 1983), severe ID=IQ<56, mild ID=IQ 56–70.	Administrative prevalence: Subjects who were receiving services for ID and/or who had a diagnosis of ID	Severe ID 2.19, mild ID 1.12. Total ID 3.31 (Down syndrome 0.96)
McDonald & MacKay (1996) Northern Ireland	N=about 290,000 Individuals living in three administrative areas in Northern Ireland.	All ages	WHO 1980 (ICD), Profound ID=IQ<20, severe ID=IQ 20–34, moderate ID IQ 35–49, Severe ID=IQ<50, mild ID (IQ 50–70).	Individuals registered in the files of registers in the Special Care Service, paediatric clinics, child guidance clinics, residential homes.	Profound ID 0.40–0.82, severe ID 1.42–2.19, moderate ID 2.05–2.99). Severe ID (IQ<50) between 3.87–6.00, mild ID between 1,98–3.04
Fernell (1996) Sweden	N=6,397 Children residing in Botkyrka, Sweden on December 31, 1994	9–15	DSM IV and stanine 1 in the WISC test Mild ID=IQ 50–72.	The records of the Paediatric Clinic at Huddinge hospital, the records of the BPSMR and the records of the administrative unit for special schools of children with ID.	Prevalence of mild ID 12.8.

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Rumeau-Rouguette, Grandjean, Cans, du Mazaubrun & Verrier (1997) France	N=325,347. Children born between 1976–1985 whose parents lived in one of the three French "departements" (Haute-Garonne, Isere, Saone-et-Loire) at the time of the study in 1992–1993.	8–17	ID=IQ≤50.	Authority (CDES) that is authorized under French law to refer children with ID for special education, and care in public or private institutions and to provide financial assistance to their families. Also institutions without CDES authorization.	Severe ID 3.56. (Down syndrome 0.98)
Hou, Wang & Chuang (1998) Taiwan	N=423,000 Children, living in Taipei City, Kaoshiung City and 21 counties/cities of Taiwan province, Republic of China. Study was made between 1991 and 1996.	6–18	ID=IQ<71 Profound ID=IQ<20, severe ID=20–34, moderate ID=35–50, mild ID=50–70.	All children with ID from outpatient clinic, all special schools and institutions.	The incidence of ID 28
Strømme & Valvatne (1998) Norway	N=30,037 Children born in 1980–1985 and living in Akershus county Norway, on 1 Jan 1993.	8–13	DSM-IV, IQ<71, Profound (IQ<20), severe (IQ 20–34), moderate (IQ 35–49), mild (IQ 50–70)	Multiple sources: Ascertainment in all institutions with responsibility to diagnose or treat children with ID.	Profound ID 0.8, severe ID 0.4, moderate ID 1.5, mild ID 3.5. Total ID 6.2
Fernell (1998) Sweden	N=14,138 Children born between 1979 and 1992 who resided in one of the 24 suburban municipalities in Stockholm, Sweden on the census day 31 Dec 1995.	3–16	IQ of a level that the individual would need special education. Severe ID=IQ of less than 50 to 55	Files at the Paediatric Clinic, University Hospital, and the local Board for the Provision and Services to the Mentally Retarded (BPSMR) centre.	4.5/1,000. (3.7 European population, 5.9 non-European population).
Larson, Lakin, Anderson, Kwak, Lee & Anderson (2001) USA	N=3,887,158 Noninstitutionalized population in the United States. A random sample of approximately 116,000 members of 46,000 households from 21 primary sampling units nationally in USA.	All ages	Mental retardation = the household answered "yes", MR if the cause of an age-specific general activity limitation or if it was the primary cause of limitations of communication, getting along with others etc. (ICD code was listed)	Data gathering in two stages: An initial visit to each sampled household, the core NHIS interview and Phase I Disability Supplement was completed for all members in the selected households. In the 2nd phase interviewers returned to households that included members with disabilities.	7.8

Table 1. Continued

Author, year, country	Population Base	Age, years	Definition of intellectual disability	Study design, data ascertainment	Prevalence of ID (per 1,000)
Bashir, Yaqoob, Ferngren, Gustavson, Rydelius, Ansari, & Zaman (2002) Pakistan	N=1,476 live born children, A cohort of children born in and around Lahore, Pakistan during 1984 – 1986	6–10	Mild ID=IQ 50–69 (WHO 1980) and adaptive malfunctiong	A prospective cohort study. Two phases: 1) Ten Questions Screening (TQS) difficulties. 2) All the children who were screened positive on TQS (n=132) were assessed by WISC, the Griffith's Mental Developmental scales for children, Goodenough Drawing Test and a detailed interview with the parents or the caretakers of the child in order to assess the adaptive functioning of the child.	Mild ID=62.
Leonard, Petterson, Bower, & Sanders (2003) Australia	N=240,358 Children born in Western Australia 1983–1992 and alive at the end of 1999.	6–15	DSM IV. Severe ID (incl. profound) IQ<35 or 40, moderate ID IQ 35–40 to 40–54, mild ID IQ 50–55 to 69	The Disability Services Commission, a multidisciplinary body to co-ordinate the medical and therapeutic support and accommodation needs of people with ID, cases registered with agencies providing educational support. A record linkage with the Maternal and Child Health research Database.	Severe/profound ID 1.4, moderate/mild ID 10.6. Total ID 14.3.
Westerinen, Kaski, Virta, Almqvist & Iivanainen (2007) Finland	N=5 184 980 Finnish residents. Data was collected from the registers in year 2000.	All ages	The diagnosis of ID in the register, based on ICD-9 or ICD-10.	A register-based study, eight national registers.	Mean prevalence of ID 7.0

Studies on the prevalence of ID from the 1970s until recently are shown Table 1. They have all been carried out in the developed countries except for two studies from a developing country, Pakistan. The rates obtained in the two Pakistani studies based on the same cohort of 1,476 live-born children exceed the respective rates obtained in the studies from developed countries for both severe ID and mild ID. The authors of the Pakistani studies explain that there were more children having severe ID in their study compared with studies from the developed countries due to less adequate pre- and perinatal services, lack of genetic counselling, a high prevalence of intermarriages and a high proportion of older mothers giving birth to children with Down syndrome in Pakistan (Yaqoob *et al*. 1995). The higher prevalence of mild ID, 62/1,000, compared to corresponding studies in developed countries was due to poor socio-economic conditions in Pakistan (Bashir *et al*. 2002).

In developed countries, the variability of the prevalence of mild ID (IQ 50–70) is greater compared to severe ID (IQ<50), the range within severe ID varying between 2.19/1,000 and 6.30/1,000 (Table 1). The lowest rate in the prevalence of severe ID has been obtained in the study by Beange and Taplin (1996) in Australia based on administrative prevalence. The highest prevalences of severe ID were obtained in two Finnish studies, the study by Rantakallio and von Wendt (1986) in northern Finland in which the data ascertainment was based on multiple sources (re-reviewed for the present study), and by Matilainen *et al*. (1985) in eastern Finland data with a data ascertainment method based on psychometric testing of the entire study population. In mild ID the prevalence in the developed countries varied between 2.19/1,000 and 37.41/1,000; the former rate is from the study by Beange and Taplin in Australia (1996) based on the administrative prevalence and the latter from the US study by McDermott (1994) based on the publications of school placements. In general, higher prevalence rates for mild ID are obtained in studies that comprise referrals for IQ testing from school authorities than in studies based on administrative prevalence of mild ID. Studies monitoring the incidence of ID are few (Katusic *et al*. 1995) and they indicate that the incidence rates for total ID and for severe and mild ID separately fall within the ranges of the prevalence rates (Gustavson *et al*. 1977, Rantakallio & von Wendt 1986). In general, the variation found between the studies focusing on the prevalence of ID can be explained as being due to inconsistent criteria of case definition, varying age of the study populations, ascertainment methods and definition of ID (Bernsen 1976, Zigler *et al*. 1984, Kiely 1987, Strømme & Hagberg 2000) as well true variations over populations (Roeleveld *et al*. 1997).

Roeleveld *et al*. (1997) have stated that although many studies focusing on the occurrence of ID have been conducted in a number of regions in developed countries, only few studies count as repeated surveys in the same geographical area. In their studies, Dupont (1989) and Fryers (1984) have discussed changes in the prevalence of ID over time in a large population. Dupont (1989) examined the age-specific prevalence of ID in Denmark in the years 1888, 1965 and 1979 based on the administrative register of the individuals who have ID and who are in need of and entitled to special services. The total prevalence of ID was 0.2/1,000 in 1888, while in 1965 and 1979 it remained unchanged, 0.43/1,000. The general pattern of age-specific prevalence of ID was much the same throughout the time period of about 100 years. The peak period in the age-specific prevalence of ID was between the age of 10–14 years in 1888, and between the age-range of 7–22 years in 1965 and 1979. According to Dupont, regional differences remained the same during the study period, so that the prevalence was higher in rural than in urban areas. The age-specific prevalence has been influenced by a law according to which individuals with ID had the right to education from the age of 7 years until the age of 21 years in since 1959, while the total prevalence was influenced by an excess mortality of individuals with ID.

In his study Fryers (1984) explored the age-specific prevalence of severe ID and the aetiologies associated with ID in children in Salford, United Kingdom, between 1961 and 1980 based on the Salford Register. The annual age-specific prevalence ratio of ID in children aged 5 to 14 years varied from the low figure 2.37/1,000 in 1961 to a high 5.42/1,000 in 1976, and then gradually fell to 4.89/1,000 in 1980. Fryers discussed that there were factors that at the same time increased and decreased the prevalence of ID. According to Fryers, the factors that contributed to the increase of severe ID were improved maternal health and perinatal care leading to a reduced mortality of the babies with preexisting neurological impairments and a better survival of children at a high risk of subsequent brain damage, improved neonatal care leading to increased survival of neurologically disordered babies of any aetiology, as well all the general factors that have improved life expectancy for all children once they have survived the hazardous perinatal period. The factors leading to the decrease of severe ID were the reduced incidence of specific disorders such as phenylketonuria and congenital hypothyroidism as well as the reduced amount of neurological impairment in survivors of very low birth weight due to advances in perinatal care.

In studying the epidemiology of ID one should not focus only on the overall changes in the prevalence in general, but also take into account specific groups,

like the most severely disabled who require many services, preventive measures as well as associated social factors (Leonard & Wen 2002, Louhiala 2004). O'Brien (2003) has stated that at present there are no indicators that the incidence of severe ID is declining, nor is there an overall reduction in the incidence and prevalence of ID. On the contrary, an annual increase of 1% in the population of people with ID is anticipated for the foreseeable future, and there is a trend that a greater proportion of individuals with very severe and multiple disabilities survive as a result of improved health care. This is due to a range of factors: increased life expectancy in some conditions such as Down syndrome, improved survival rates in children with multiple and complex disabilities, increased prevalence of genetic conditions among some populations, and increased diagnostic awareness.

2.3 Biomedical aetiology associated with intellectual disability

Intellectual functions depend to a great degree on the integrity of the central nervous system (CNS). A variety of biomedical causes can disrupt this integrity and start the process leading to ID. A biomedical cause, whether genetic or acquired, may be the primary cause that will start the process of developmental delay, but it will not necessarily be the only factor responsible for the functional outcome, which will depend on the synergistic or cumulative effects of all factors involved. (Szymansky & Wilska 1996, Wilska & Kaski 2001). ID is not a medical term per se, but it is a symptom of brain origin and has an administrative function by defining a group of persons who are in need of support and educational services. Thus, ID must be differentiated from the biomedical diagnosis of the underlying medical condition (Szymanski & Wilska 1996).

Aetiology is an important aspect in diagnosing and classifying ID. Among other things, the aetiology may be associated with other health-related problems that may influence physical and psychological functioning. The aetiology may be treatable, which could permit appropriate intervention to minimize or prevent ID; accurate information on aetiology is needed for the design and evaluation of programmes aimed to prevent specific aetiologies of ID; the aetiology may be associated with specific phenotypes that allows anticipation of actual, potential, or future functional support needs, and individuals and families can be referred to other people and families with the same aetiological diagnosis for desired information and support. (Luckasson *et al*. 2002)

In the aetiological classification of ID, the chronological point of the causative insult to the CNS involves categories of genetic and other prenatal and perinatal,

postnatal and unknown factors (Yannet 1956). Most studies aim to elucidate the earliest developmental biomedical cause leading to ID, even though it is known that a large proportion of children with ID have more than one possible causal factor appearing in early life (McLaren & Bryson 1987). Variation between the studies in the prevalence of cases with a known aetiology is likely to be influenced by how a "known aetiology" is defined, by the extent of the clinical investigations undertaken and possibly by the speciality of the diagnostician. It could be that differences in the prevalence of genetic conditions are real and related both to the genetic characteristics and the rates of consanguinity of the population. On the other hand, it may be that unknown aetiology of ID is more common in some communities because of underlying psychosocial or unidentified environmental determinants. Alternatively, because cases with unknown aetiology are more likely to be identified through the educational than the medical system, they may have been better ascertained in studies using multiple sources. (Leonard & Wen 2002)

Genetic factors account for about 35% of the separate aetiological factors leading to ID (Szymanski & King 1999). The two most common causes of ID, Down syndrome and Fragile X syndrome, both have genetic aetiology (Skellern *et al*. 2000). Down syndrome is the most common known cause for ID, appearing in 14–15% of the individuals with ID (Leonard & Wen 2002). About one third of the aetiological factors involve an external prenatal, perinatal or postnatal factor including infections, injuries, toxins, and delivery complications and premature birth; fewer than 10% involve a malformation syndrome of unknown origin. The aetiology remains unknown in a quarter or a third of the ID cases. (Szymanski & King 1999)

The proportion of known aetiology varies along with the severity of ID so that in severe ID (IQ<50) biomedical aetiology can be found in 70–80% of the cases, while in mild ID the aetiology leading to ID is only found in about 40% of the cases. Aetiology leading to severe ID is more often diagnosed in the child's early years, e.g. such chromosomal disorder as Angelman syndrome, but in some conditions with late onset of symptoms or with deteriorating course, the diagnosis may be possible to ascertain later. On the other hand, diagnosing mild ID is often connected to referrals from the educational system, especially when the child has been suspected to have learning difficulties, which become apparent later, up into the teenage period. (Gillberg 1997)

Repeatedly collected epidemiological data on the incidence and prevalence of biomedical causes leading to ID are valuable, for they inform of the success of prenatal care and indicate the efficacy of preventive activities such as prenatal diag-

nostic tests (Fryers 1986). ID associated with infectious disease, trauma, and maternal phenylketonuria as well as a variety of infectious diseases during the prenatal period (e.g. congenital syphilis, rubella) and the postnatal period (e.g. measles, tuberculosis meningitis) has decreased due to vaccination of women prior to pregnancy as well as newborn screening followed by targeted treatment and other specific medical interventions. ID now appears to be an unlikely complication of perinatal trauma such as asphyxia, unless cerebral palsy is an accompanying disability. (Kiely 1987). In the US, based on a study exploring the existing literature between 1950 and 2000, ID due to congenital syphilis, Rh haemolytic disease of the newborn, measles, Haemophilic influenza type b (Hib) meningitis, congenital hypothyroidism, phenylketonuria and congenital rubella syndrome has decreased so that these condition-specific causes of ID accounted for approximately 16.5% of the total number of the cases with ID in 1950 and for 0.005% of the total number of cases with ID in 2000 (the prevalence of ID was estimated at 1.2%). (Brosco *et al*. 2006)

In studying and predicting trends in the aetiological factors leading to ID it is necessary to take into account the sociodemographic factors connected to them, possible prenatal interventions, current prevalence of ID, future estimated prevalence of ID, survival, associated health problems and facilities. Regarding time trends for certain syndromes leading to ID such as Down syndrome, it is reasonable to assume that the incidence of Down syndrome is decreasing, because a downward shift in the maternal age has emerged as a result of the decrease in the proportion of pregnancies among women aged 35 years or older. However, due to improvements in postnatal medical care, survival of infants with chromosome anomalies is increasing, which might result in an increase in the prevalence of these conditions including Down syndrome. An increase in survival because of improved medical care has also occurred for other conditions that are associated with ID. Data on temporal variations and future prediction of the trends on the incidence and prevalence of separate aetiological factors leading to ID are valuable for the authorities for the purposes of planning health, education and social services. (Leonard & Wen 2002)

2.4 Sociodemographic factors associated with intellectual disability

In 1933 E.O. Lewis discussed social aspects of ID. Based on the recognition that ID varies by degrees he classified ID into primary and secondary amentia and

introduced the terms pathological type and subcultural type Lewis presented that the pathological group comprises all those individuals with ID whose condition is due to some definite organic lesion or abnormality such as Down syndrome, trauma, inflammatory conditions or hydrocephalus. Most of the individuals affected by these diseases have ID that is more severe, while the individuals in the subcultural group usually have mild ID. The subcultural group includes, on the other hand, the individuals with ID in whom no organic lesion or abnormality is found. Lewis presented that there is a close biological kinship between the subcultural defective and the population with normal intelligence who have an IQ in the low extreme in the variation of general intelligence. He stated that the families having a child with a pathological type of ID are evenly distributed in the continuum of the various socio-economic status (SES) levels of the community, whereas the families whose child has ID originating from subcultural type are to a large extent concentrated to lower SES levels. According to Lewis, subcultural type of ID arises in disadvantaged conditions in children whose mental capacities are below the average, but who are not intellectually disabled; however, adverse conditions affecting their mothers during pregnancy and/or adverse conditions during their earlier years give rise to reduced mental capacity and social inefficiency. (Lewis 1933)

Since the study by Lewis in the 1930s a large part of the studies on the association between ID and social/environmental factors have focused on the association between the distribution of SES of the families having a child with ID and the severity of the child's ID (Penrose 1938, Zigler 1995, Strømme & Magnus 2000). In their review Leonard and Wen (2002) presented that many studies have consistently found that mild ID is strongly associated with low familial SES. Some studies suggest that mild ID is rarely found in the highest familial SES groups unless accompanied by evidence of organic damage, whereas severe ID is not associated with low SES of the families. Also such sociodemographic factors as low educational level of the mother, high birth order of the child and older age of the mother at the time of delivery have turned out to associate with ID. (McQueen *et al*. 1987, Drews *et al*. 1995, Camp *et al*. 1998, Chapman *et al*. 2002). However, during the last decade, with increasing awareness of the role of social determinants in population health, the social factors associated with ID have proved to create a more complex entity than was thought in earlier studies (Leonard *et al*. 2005). For instance in Australia, Leonard *et al*. (2006) found that there was an increased risk for mild to moderate ID with unknown aetiology in children of mothers with asthma, diabetes, a renal or urinary condition and epilepsy. The increased risk for

ID in the offspring of mothers with such conditions as asthma and diabetes is particularly important for disadvantaged population, in whom these conditions are more prevalent and may be less well managed. Results of this kind bring up the concept of intergenerational effects, which recognizes that reversible adverse environmental factors in the lives of some families may be related to the aetiology of ID. When this is noticed, enhanced individual and family supports should follow, for example targeted maternal/parental care and social support during pregnancy. (Luckasson 2002)

In the panorama of sociodemographic associations with ID the entity is mixed not only by uncertainties of the nature of these associations but also by the accumulation of detrimental social and psychological factors that are found individually in all SES groupings. In his study Sameroff (1987) found that different combinations of equal numbers of risk factors produced similar effects on IQ, providing evidence that no single factor identified here uniquely enhances or limits early intellectual achievement and that cumulative effects from multiple risk factors increase the probability that development will be compromised. The multiple risk indexes predicted substantially more variance in the outcome measure than did any single risk factor alone, including socio-economic status. High-risk children were more than 24 times more likely to have IQs below 85 than low-risk children. It is possible that in passing of time shifts occur in these risk factors as is the case in some biomedical factors associated with ID reflecting the development in the society and that's why it is important to collect up-to-date data widely on these factors. For instance, a recent small-scale study by Neggers *et al.* (2003) showed that maternal prepregnancy obesity was associated lower cognitive ability in the offspring compared with normal weight mothers (general intellectual score 80.0 vs. 84.5).

Defining that a child is at risk for poor or detrimental outcome means that a child has not yet manifested a developmental delay, but has a high probability of doing so because of the risk condition or factors. A proposed list of risk factors influencing human development and functioning includes factors such as mother's younger or older age than normal childbearing years, low parental education attainment, inadequate income, low occupational status of head of household, low SES, repeated job changes or unemployment, unplanned pregnancy, more than four children, repeated relocations, absence of spouse or partner, high exposure to toxic substances, frequent accidents, no alternative caregivers, presence of no or few extended family, poor/unsupportive extra family support, inadequate nutri-

tional intake, poor parental social skills, poor parental physical health. (Dunst 1993)

2.5 Behavioural problems among individuals with intellectual disability

Studies on the prevalence of behavioural problems in children with ID have two main functions, administrative and scientific. The administrative function is to provide service planners with accurate information on which to base decisions regarding the resources required to assist families and other carers of children who have ID and behavioural disturbance. Service planners need to know not only the overall requirements for health and special educational and other services in their area of responsibility, but also which particular groups are most in need. The scientific purpose of an epidemiological study is to examine associations between incidence, prevalence of behavioural problems and other variables in order to provide hypothesis about potential aetiological links of behavioural problems in children with ID. (Einfeld & Tonge 1996a)

Since the study by Rutter *et al*. (1970) on the Isle of Wight in the 1960s it is a well-documented result that individuals with ID have more often psychiatric and/or behavioural problems than individuals in general population (Gillberg *et al*. 1986, Maes *et al*. 2003). The risk of significant psychopathology in the population of children with ID increases at least threefold in comparison to children without ID (Dekker *et al*. 2002). The factors contributing to the increased prevalence of psychopathology among children with ID include limited communication skills, additional stressors due to ID (e.g. personal limitations in adaptive behaviour during the developmental years), higher prevalence of neurological deficits and genetic syndromes as well as limited independence (de Ruiter *et al*. 2007). Estimates of the prevalence of psychopathology in children with ID, collected from studies with representative community samples including school-aged children, range from 30% to 60%. This wide range may be accounted for by variability in the definition of both ID and psychopathology, the use of different instruments to assess psychopathology, the use of different samples (e.g. children with ID referred to services versus general population studies), and including populations with different levels of ID. (Dekker *et al*. 2002)

Mental disorders in persons with ID are basically the same as in general population (Bregman 1991, Szymanski & Wilska 1996, Luckasson *et al*. 2002). In DSM-IV the diagnosis of specific mental disorders with onset in childhood com-

prise pervasive development disorders (PDD, a major impairment in interpersonal reciprocal interaction and in interpersonal communication), attention-deficit/ hyperactivity disorder (ADHD, can be situational, e.g. related to the task being too difficult or too boring), stereotypic movement disorder, schizophrenia and other psychiatric disorders, mood disorders, anxiety disorders, personality disorders, aggression and adjustment disorders. Adjustment disorders comprise emotional and behavioural symptoms that are a response to a stressor (Szymanski & Wilska 1996). In his review on epidemiological findings regarding the prevalence of various neuropsychiatric disorders, Bregman (1991) presented that there is variation in the prevalence of types of psychiatric symptoms by level of ID as well as by aetiology for ID. Among persons with mild ID, an elevated prevalence has been reported for disorders of conduct, activity level and attention, mood and affect and thought processes. In severe ID level, an increased prevalence of autism and PDD, stereotypic behaviours and self-injurious behaviours has been found. Specific patterns of psychopathology have been reported to be associated with such genetic syndromes as fragile X (attention deficit disorders, autism) and Prader-Willi (eating disturbance, oppositional-defiant behaviour).

It is important to study the association between ID and emotional and behavioural problems among individuals with ID, because behavioural and emotional problems are a major source of additional handicap, especially in adaptation. According to the definition of ID by AAMR, merely analysing someone's limitations is not enough, but the specification of the limitations should lead to the development of the support the individual needs in order to improve his or her functioning. (Luckasson *et al*. 2002). It has been found that behavioural and emotional problems in children with ID add to the suffering of the affected individual, cause distress to parents and reduce social integration and later employment of the individual with ID (Einfeld & Tonge 1996a, Maes *et al*. 2003, Emerson & Hatton 2007, Koskentausta *et al*. 2007). Although it is generally accepted that the rate of behavioural and emotional problems in children with ID exceeds that of general child population many such problems remain undiagnosed and untreated. (Einfeld & Tonge 1996a, 1996b, Moss *et al*. 1997, Linna *et al*. 1999, Molteno *et al*. 2001, van Schrojenstein Lantman-deValk 2005). The services provided by the surrounding society do not often meet the individual needs of a family and its child having both ID and psychiatric problems (Gillberg *et al*. 1986, Einfeld & Tonge 1996b, Maes *et al*. 2003, Emerson & Hatton 2007).

In treatment of behavioural and other disturbances in individuals with ID, the interventions that are typically used in psychiatric treatment can be considered for

individuals with ID, too (Bregman 1991). However, one of the dilemmas faced by the service planners has been whether to try and cater to the needs of the individuals with ID within generic provision, or whether it is appropriate to provide specialist services. Advocates of normalization support the generic approach, arguing that specialist services lead to stigmatization, labelling and negative professional attitudes. The benefit of using specialist psychiatric services for treating psychiatric problems of the individuals with ID is that special expertise is needed for accurate diagnoses and therapy, for behavioural and emotional problems, and that other psychiatric problems often manifest in individuals with ID in ways different from the general population. (Moss *et al*. 1997). However, more often than non-ID children, individuals with ID suffer besides mental health problems also of disadvantages in social and physical well-being. Their own self-reports of health problems are often lacking and so far there is hardly any idea of their own perceptions of their needs. (van Schrojenstein Lantman-de Valk 2005)

2.6 Summary of the literature review and further research needed

The three widely used systems for classification of ID are the ICD by the World Health Organization, the American Association of Mental Retardation and the DSM from the American Psychiatric Association. The diagnosis of ID and the classification into levels of severity should be based on IQ obtained through an individually administered standardized psychometric test. The criterion of ID is fulfilled when an individual's tested IQ is two standard deviations below the mean.

The prevalences of ID in different studies vary according to temporal, demographic, and social factors as well by medical risk factors. Variation found between the studies focusing on the prevalence of ID can be explained by inconsistent criteria of case definition, varying age of the study populations and case ascertainment methods, by definition of ID and by true variations over populations. The peak period of prevalence of ID is approximately from 10 to 16 years of age. In industrialized western countries the prevalence of severe ID is estimated between 2.6 and 7.3 per 1,000 and of mild ID from 3.5 to 7.5/1,000.

In the aetiological classification of ID, the chronological period of the causative insult to the CNS involves categories of genetic and other prenatal, perinatal, postnatal and unknown factors. The classification by Yannet (1956) and Wilska and Kaski (1999) presents the so-called paranatal period as its own category. Variation between the studies in the prevalence of a known aetiology is likely to be influenced by how a "known aetiology" is defined and by the extent of the clinical

investigations undertaken. Aetiology associated with severe ID is found in 70–80% of the cases and is more often diagnosed in the child's early years, while in mild ID the respective proportion is only about 40% and it is often diagnosed later as a result of a referral from the educational system. Time trends in the incidence and prevalence of biomedical causes leading to ID are valuable for providing information of the success of prenatal care and indicating the efficacy of preventive activities such as prenatal tests. Future prediction of these trends is valuable for the authorities in planning health, education and social services.

Many studies have consistently found that the prevalence of mild ID is strongly associated with low SES, some suggesting that mild ID is rarely found in the highest SES groups unless accompanied by evidence of organic damage, whereas severe ID is not associated with low SES of the families. However, during the last decade, with increasing awareness of the role of social determinants in population health, the social factors associated with ID have proved to create a more complex entity than was thought in earlier studies. Deeper understanding of mediating factors between the sociodemographic factors and ID is needed in order to develop more precisely targeted intervention programmes for promoting the living conditions of individuals with ID and their families.

Previous studies have shown that the risk of significant psychopathology in the population of children with ID increases at least threefold in comparison to children without ID. Mental disorders in persons with ID are basically the same as in general population. Behavioural problems are a major source for additional handicaps of ID and they make adaptation more difficult. The types of psychiatric symptoms vary by level of ID as well as by aetiology for ID. Among persons with mild ID elevated prevalence for disorders of conduct, activity level and attention, mood and affect and thought processes have been reported. In severe ID level, increased prevalence of autism and PDD, stereotypic behaviours and self-injurious behaviours has been found.

Epidemiological studies on the occurrence of ID as well as on the associated aetiological and sociodemographic factors suffer from methodological and definitional discrepancies that make comparisons difficult. That is why data on temporal trends on these associations are difficult to achieve. Such changes in the field of biomedical aetiology as the emergence or discovery of new syndromes, changes or variations in sociodemographic environments, changes in maternal age at time of delivery and improvements in the availability of high-standard medical care most likely contribute to the incidence and prevalence of ID. It is important to follow populations and to collect repeatedly data on the factors related to ID to be able to

offer targeted services for groups and individuals with ID most in need. In examining sociodemographic factors associated with ID the factors selected for the study have in many cases been limited to socio-economic factors only, based on the repeatedly found result on the association between the family SES and the level of ID in the offspring. Not until recently has the scope of the sociodemographic factors been expanded and extended to cover the indicators of maternal health during pregnancy as well.

3 The aims and objectives of the study

The overall aim was to examine the incidence and the prevalence of ID, the associated aetiological and sociodemographic factors and the possible changes in them over a twenty-year period in the same geographical area in Northern Finland. The prevalence of behavioural problems in children with ID was also investigated. The specific objectives of the study were:

1. To investigate the incidence and prevalence of ID in children born in 1985–86
2. To investigate biomedical aetiology associated with ID in children born in 1985–86
3. To investigate maternal and familial sociodemographic factors associated with ID in children born in 1985–86
4. To investigate the prevalence of behavioural problems among children with ID born in 1985–86.
5. To investigate temporal changes over 20 years' time in the incidence/ prevalence of ID and associated biomedical and maternal/familial sociodemographic factors (from 1 to 3 separately) between NFBC 1986 and NFBC 1966.

4 Subjects and methods

4.1 Study design and study populations

This is an epidemiological, mainly prospective cohort study with some follow-back features, which applied both descriptive and analytical approaches. In this thesis the incidence and prevalence of ID in NFBC 1986 and NFBC 1966 as well as the distributions of the potentially associated aetiological and sociodemographic factors and the probable behavioural problems describe the general characteristics of these study populations. An analytic approach is used to study the associations of the biomedical, potentially causative, factors as well as sociodemographic risk factors for ID both in and between NFBC 1986 and NFBC 1966 (Cummings *et al*. 1988).

The study is based on data collected prospectively from two one-year birth cohorts of children with a follow-up of 11.5 years of age in each cohort. The study populations comprise all children of the mothers whose expected dates of delivery fell within the time period of the cohort and whose pregnancy continued after the 24^{th} gestational week in the study area, the provinces of Oulu and Lapland, in NFBC 1986 between July 1, 1985, and 30 June, 1986 (Rantakallio & Oja 1990). The number of mothers and deliveries was 9,362 (99% of all eligible) and there were 9,432 children born alive. (Järvelin *et al*. 1997). In NFBC 1966 the expected dates of delivery fell between January 1 and 30 December in 1966 and the number of mothers and deliveries was 12,068, representing 96% of the total number of deliveries occurring in the area. 12,058 of the children were born alive (Rantakallio 1969). Data for NFBC 1966 used in this study was originally collected by Rantakallio and von Wendt (1985, 1986), but re-reviewed in the present study for comparison purposes. All maternal health care centres in the area participated in the study, and the deliveries took place in the hospitals located in the area in both of the NFBC cohorts.

4.2 Data collection and methods

4.2.1 Identification of children with intellectual disability

Identification of children with intellectual disability in NFBC 1986

The follow-up data were collected until the children belonging to the cohort reached the age of 11.5 years. The children who potentially had ID were traced by a) birth and neonatal data (e.g. Apgar scores, convulsions); b) register data including Hospital Discharge Register and National Insurance and Medication Reimbursement Register (screened for more detailed scrutiny if the child had a diagnosis/disorder potentially connected with ID) as well as Cause-of-Death Register (if the child had obtained a passing diagnosis potentially connected with ID); c) hospital records (all including outpatient records systematically reviewed until the age of 7 years), family counselling centre and institutional records, d) questionnaires filled in by the parents of the children on the growth, development and health and school type at the age of 7 and 8 years (if there were symptoms of developmental disorders, handicaps, or diseases potentially related to ID), and the questionnaire filled by the child's teacher with the parents' permission (if there was evidence of difficulties in school achievement or behavioural disturbances); and e) by results of psychometric tests. All the relevant records were requested from all the health care units in the study area where the children had potentially been examined. The study had high coverage, for all but six children were traced at age 7–8 years. (Table 2, Figure 1)

Screening procedures were applied for the 722 children who had moved away from the study area within the period of the follow-up to other parts of Finland by using similar methods as for those who resided in the target area. For the 43 children who had moved abroad, the information described earlier was available until the date of emigration. Using these screening procedures, 154 children with possible ID were found and their data underwent a more comprehensive review. A paediatrician (MRJ) and the author scrutinized the child's hospital/institutional, child welfare, and/or family counselling centre records. Six of these children turned out to have ID.

Data on psychometric test results were collected from hospitals, institutions for children with intellectual disability, family counselling centres, and school psychologists. No separate evaluations or examinations were made for the purposes of the study. The information collected was based on the routine clinical practice to

refer the child for further examinations due to developmental or learning disorders, for example. If the psychometric test had been administered to the child more than once during the follow-up time, the result of the most recent and complete test was used. The identification of children with ID was carried out by a stepwise procedure starting with children who scored a full-scale IQ of 70 or under according to their most recent standardized psychometric test. In cases where no IQ estimation was available, hospital records were searched for an assessment by a doctor or psychologist of the child's intellectual level. If neither of the above assessments was found, but it was evident that the child had ID based on a diagnosis of a disorder or a disease (e.g. chromosomal disorders, specific syndromes, brain anomalies), then the classification was made by the author, a paediatric neurologist (PO) and a paediatrician (MRJ). This procedure concerned mainly neonatal and infant deaths. The results of the psychometric tests were collected between 1995–2000. The author collected the data on psychometric tests.

Psychometric tests used in the classification of the severity of ID were Wechsler Intelligence Scale for Children – Revised (WISC-R, Wechsler, 1984), Terman-Merrill (Hellström, Terman & Merrill 1967, Lehtovaara 1950), Merrill-Palmer Performance Preschool Tests (Stutsman, 1948), the Wechsler Infant Scale for Children (WISC, Wechsler 1974), Vineland Social Maturity Scale (Doll 1950), Bayley Scales for Infant Development (Bayley 1969), the Wechsler Preschool and Primary Scale of Intelligence (WPPSI, Wechsler 1977), Leiter International Performance Scale (Leiter 1961), Cattell Infant Intelligence Scale (Cattell 1970). The method was unknown in eight cases that were reported to have been given a psychometric test. Twenty-two children were not given a psychometric test; instead, a psychologist or a clinician made the assessment of the level of ID on a clinical basis. (Table 2)

Table 2. Identification of children with intellectual disability in the Northern Finland Birth Cohort 1986 (NFBC 1986) and the Northern Finland Birth Cohort 1966 (NFBC 1966).

	NFBC 1986	NFBC 1966
Expected date of birth	1 July 1985 – 30 June 1986	1 Jan 1966 – 30 Dec 1966
Number of live-born	9,432	12,058[3]
Number of stillborn	47	173
Population base in the provinces of Oulu and Lapland	633,084[1]	640,025[2]
End of follow-up	30 Dec .1996	30 June 1977
Number of deceased children during the follow-up	81	268
Number of children alive at end of follow-up	9,351	11,790[3]
Data ascertainment and data reviewed	Hospital discharge register, cause-of-death register, hospital and institutional records, psychometric tests, questionnaires, National Insurance and Medication Reimbursement Register.	Hospital discharge register, cause-of-death register, hospital and institutional records, psychometric tests, questionnaires, National Insurance and Medication Reimbursement Register.
Psychometric tests used for the assessment of the level of ID	Terman-Merrill (n=16) WISC (n=5), WISC-R (n=52), WPPSI (n=2), Bayley (n=2), Merrill-Palmer (n=7), Leiter (n=1) Vineland (n=3), Cattell (n=1), Unknown (n=8)	Terman-Merrill (n=71) WISC (n=11) Merrill-Palmer (n=2) Leiter (n=3) Vineland (n=3) Cattell (n=15) Unknown (n=3)
Assessment of the child's level of ID on a clinical basis	n=22	n=43

[1] Central Statistical Office of Finland 1985/1986
[2] Central Statistical Office 1966
[3] 93 subjects of NFBC 1966 refused the use of their data at the 31-year-old-study leaving 11,965 subjects for the denominator in the incidence of ID and respectively for the prevalence 11,697 subjects, which figures have been utilized in Publication I

Identification of children with intellectual disability in NFBC 1966

Rantakallio and von Wendt (1986) originally traced the children with ID. The follow-up data were prospectively collected during the period 1966 to 1983 in the following ways: a) questionnaires on the pregnancy and on morbidity and mortality among the infants during the perinatal period were filled in by the midwives at the antenatal and postnatal clinic (Rantakallio 1969); b) questionnaires on the children's health and development at the age of one year were filled in by the public

health nurses at the children's welfare centres (Rantakallio & Mäkinen 1983); c) hospital records and special forms were filled in for those who had visited neurological out-patient clinics in the area; d) data on admissions to the four children's hospitals in the target area from 1966 to 1972 (Rantakallio & von Wendt 1986); e) death certificates; f) Hospital Discharge Register (since 1972), g) Medicine Reimbursable in full (since 1967); h) child subsidies for chronically sick children (since 1967), and children with ID (since 1979); i) questionnaire on the child's health and performance at the age of 14 years filled in by the child and family. The response rate of the questionnaire was 97%. In the remaining cases, school health nurses were interviewed concerning the health of the child and school social workers were queried concerning school performance. The latter method was also used if the data on school performance received from the child and family were inadequate or doubtful. The study had high coverage; all but the 14 children who had moved abroad could be traced. (Rantakallio & von Wendt 1986). (Table 2)

The author reviewed the data on psychometric tests and clinical data on ID collected by Rantakallio and von Wendt (1986) and reclassified the data based on the tests until the age of 11.5 years (until 30 June 1977). The result of the most recent psychometric test with respect to the end of the follow-up was selected as the basis of classification. The procedure in cases where no IQ estimation based on psychometric tests was available followed the one utilized in NFBC 1986. In these cases hospital and other relevant records were searched for a doctor's or psychologist's assessment of the child's intellectual level and in cases where it was not found, but it was evident that the child had ID based on a diagnosis of a disorder or a disease, the author, a paediatric neurologist (PO) and a paediatrician (MRJ) classified the level of ID. The medical and psychological data on children who had died during the follow-up period were also re-reviewed by a paediatric neurologist (PO). The re-revision was based on medical data, mainly hospital discharge registers, and the assessment of the level of ID was made based on the current medical knowledge on the association between the diagnosed disease, syndrome etc. and level of ID if the child had lived.

In the re-review, the psychometric tests used as the basis for the assessment of the level of ID were: The Terman-Merrill (Hellström *et al*. 1967, Lehtovaara 1950), Cattell Infant Intelligence Scale (Cattell 1970), the Wechsler Infant Scale for Children (WISC, Wechsler 1974), Leiter International Performance Scale (Leiter 1961), Vineland Social Maturity Scale (Doll 1947), Merrill-Palmer Performance Preschool Tests (Stutsman 1948). The psychometric test remained

unknown in three cases. Assessment of the child's level of ID was made on clinical basis in 43 cases. These were mainly children who had died in infancy. (Table 2)

4.2.2 Data collection on biomedical aetiology associated with intellectual disability

NFBC 1986

Data on aetiological factors associated with ID were collected from multiple sources: 1) Hospital and institutional records 2) National Hospital Discharge Register 3) National Cause-of-Death Register 4) Maternal and perinatal data, (Järvelin *et al*. 1997) and 5) Parental questionnaire on the child's health and development at the child's age of seven years. The data from the hospital and institutional records were collected from birth until the age of 11.5 years (for all until the age of 7 years – both from hospitals and institutions – but until 11.5 years for those whose diagnoses and aetiology needed further assessment). The diagnoses in the child's case records or institutional registers were regarded as aetiological when the causal factor related to the diagnosis was considered to be responsible for ID. An experienced clinician with special competence in the medical aspects of intellectual disability (SLL) scrutinized all the data on the aetiological diagnoses. (Table 2, Figure 1)

NFBC 1966

In NFBC 1966 data on the aetiological factors associated with ID was originally collected by Rantakallio and von Wendt (1985). Data sources were 1) questionnaires on the pregnancy and morbidity and mortality among the infants during the perinatal period filled in by the midwives at the antenatal and postnatal clinics for the mothers; 2) in 1967, a form with 72 items was filled in for children admitted to children's hospitals during the first 28 days of life. Data were available for 81.2% of the total of 773 children who were admitted at that age and for whom diagnoses were available. The details that were used were blood sugar and bilirubin concentrations, body temperature on hospital admission, and the diagnoses for the mother and child; 3) diagnoses on admission to the children's hospitals in the area between 1966 and 1972; 4) a questionnaire on the child's health and development at the age of one year was filled in by the public health nurses at the child welfare centres; 5) hospital records and special forms were filled in for those children who visited a

neurological outpatient clinic in the area either because of their symptoms or because requested to do so for the purposes of the study; 6) national registers of death certificates (from 1965), hospital discharge register (from 1972), child subsidies for chronically sick children (from 1967), children with ID (from 1979).

For the purposes of this study, an experienced clinician with special competence in the medical aspects of intellectual disability (SLL) re-reviewed data of the original file folders on aetiology presented in the earlier chapter. The data of original death certificates were not available, but the corresponding information concerning the deceased children was collected from hospital discharge registers/original files.

The data on biomedical aetiology associated with ID in NFBC 1966 presented in this study is unpublished data.

4.2.3 Data collection on sociodemographic factors associated with intellectual disability

In both NFBC 1986 and NFBC 1966, the data on sociodemographic factors were based on the structured self-completed questionnaire filled by the mothers. The core questions were similar and comparable. The questionnaire was given to the mothers and collected from them at the maternal health care centre usually in the 24^{th} to 28^{th} gestational week. If this failed, the questionnaire was completed later in the pregnancy or after the delivery. (Rantakallio 1969; Järvelin *et al*. 1997) (Figure 1). In NFBC 1986 the questionnaire was filled in by the mothers later than the 28^{th} gestational week or after birth in about 12% of the sample (Järvelin *et al*., 1997).

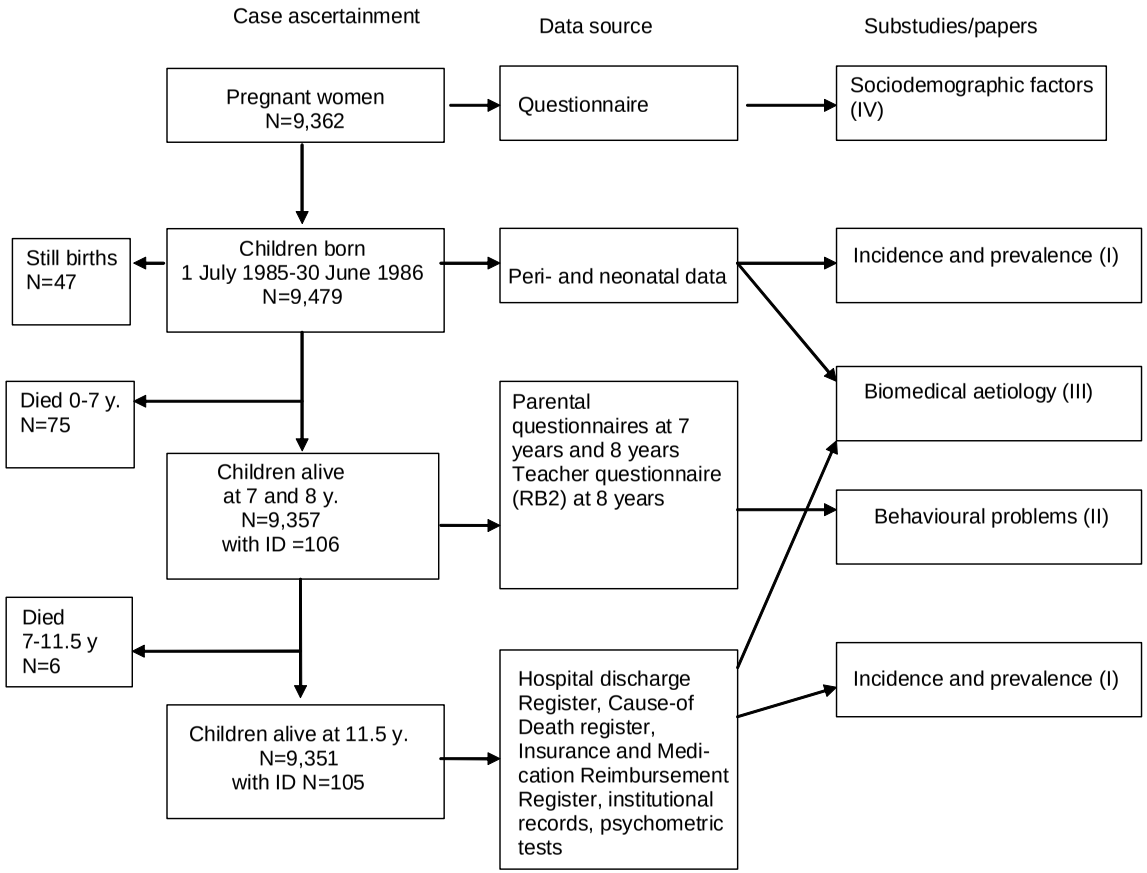

Fig. 1. Study population and data collection in the Northern Finland Birth Cohort 1986 (NFBC 1986).

4.2.4 Data collection on behavioural problems in children with intellectual disability in NFBC 1986

In NFBC 1986, data were collected by a two-stage procedure. In the first stage, in the autumn of the children's first school year, information about the child's growth, development and health, school and family type and social situation were gathered from the parents using a postal questionnaire. In the second stage, seven months later, in the spring of the children's first school year, teachers assessed the children's behaviour using the Children's Behaviour Questionnaire for Completion by Teachers (RB2, Rutter 1967) and the parents filled in the questionnaire on children's psychomotor development and behaviour. The parents returned the postal questionnaire on the child's health and development for 8,416 children (90%) and the questionnaire on psychomotor development and behaviour for 8,370 children (90%) The Children's Behaviour Questionnaire for Completion by Teachers (RB2) was returned for 8,525 children (92%). For the children with ID the response rate was 70% (n=74). (Figure 1).

4.3 Definition of the study variables

Outcome variable (the number of publications in which they were used in parentheses):

1. Intellectual disability (I–IV): ID is defined as an intelligence quotient (IQ) of 70 or under, based on either the most recent individually administered standardized psychometric test or developmental assessment on a clinical basis. The severity of ID is classified according to the International Classification of Diseases, 9th edition (ICD-9), Finnish version (National Board of Health in Finland 1987) into four subcategories: profound ID (IQ<20), severe ID ((IQ=20–34), moderate ID (IQ=35–49), and mild ID (IQ=50–70).

2. ID by severity is classified into two groups as follows (I, III, IV): mild ID (IQ 50–70) and severe ID (IQ<50).

3. Incidence of ID (I, III, IV) is defined as the number of new cases arising from birth until the end of the follow-up time per 1,000 live-born. In this study, per 1,000, arising from birth until the end of the follow-up time, 11.5 years, from 1 July 1985 to 30 December 1996 for the NFBC 1986 and from 1 January 1966 to 30 June 1977 for the NFBC 1966.

4. Prevalence (I–III) is defined as the number of cases present at the end of the follow-up per 1,000 population alive, on the 30 of December 1996 for the NFBC 1986 and 30th of June 1977 for the NFBC 1966.

5. Biomedical aetiology (III): A hierarchical method based on timing and type of damage to the CNS was used in classification to organize the most probable cause of ID. This was done according to Wilska and Kaski (1999) with the exception of recent knowledge about multifactorial prenatal disorders concerning autism (Rutter 2000). We also used a traditional main categorization: a) prenatal, from fertilization until birth: genetic disorders with a proven chromosomal aberration, single-gene disorders, multifactorial disorders (including CNS malformations, multiple syndromes of unknown origin) and external prenatal disorders (maternal infections, medications, toxins, nutrition, intrauterine growth retardation, prematurity, other external prenatal disorders such as radiation); b) paranatal: considered to have occurred in the period between 1 week before and 4 weeks after delivery, including infections, delivery, and other neonatal complications; c) postnatal: including CNS damage occurring after the neonatal period up to the age of 18 years (e.g. infections and other factors damaging the CNS, such as toxic agents, vascular accidents, hypoxia); and d) unknown, cases not classified into any of the previously mentioned categories, pure non-familial intellectual disabilities, and the CNS symptom group of individuals with CNS symptoms or signs, without malformations or dysmorphic features.

6. Behavioural and emotional problems (II) were based on the Children's Behaviour Questionnaire for the Teachers developed by Michael Rutter (1967). The questionnaire includes 26 items, producing a total score from 0 to 52. Children with a total score of 9 or more were designated as indicating probable psychiatric disturbance (the term "probable psychiatric disturbance " is used, because the children were not clinically diagnosed but screened by a validated questionnaire). Of these children, those with the emotional subscore (4 items, max score 8 points) higher than the behavioural subscore (6 items, max score 12 points) were designated as "children with emotional problems" and those with the behavioural subscore higher than the emotional subscore as "children with behavioural problems". The children with equal emotional and behavioural subscores were called a mixed group. A child with emotional, behavioural, or mixed problems can also be classified as hyperactive if he or she gets a score of 9 or more on the total scale and 3 or more on the

hyperactivity items (3 items, max 6 points). In the analysis of the 26 items, if there were responses to 20 or more items, the missing items were replaced by the mean of the items responded by each subject. For the emotional, behavioural and hyperactivity subscores, the inclusion criterion was 3 out of 4, 5 out of 6 and 2 out of 3 items, respectively.

7. Socioeconomic status (IV): The same classification system of the socioeconomic status was utilized in both NFBC studies. Classification by socioeconomic status consisted of the social standing and prestige of the mother's occupation. Group I usually required academic education. Group II had occupations with somewhat lower vocational prestige than those of social class I. Class III consists of skilled workers, while those in social class IV were unskilled workers. Farmers formed social class V. (Finnish Bureau of Statistics 1954, Rantakallio 1979).

8. Socioeconomic status of the family (IV): Classification by socio-economic status of the family was assessed by the same categorization as in the socioeconomic status of the mother, but as determined by the father's occupation and its prestige; in case this was missing, the mother's social class determined the socio-economic status of the family.

9. Body mass index (IV): Prepregnancy weight was (kg) was asked of the mothers by their first visit at maternal health care centre in both cohorts (Olsen *et al*. 1995). In NFBC 1966, when prepregnancy weight was not known, the weight at the first antenatal clinic visit was used if obtained before the 4^{th} month of gestation (Rantakallio 1969). Estimates of height (cm) were based on the measurement carried out at the maternal health care centre or on self-report of the mother. In NFBC 1986, height was measured at the maternal health care centre in 52% of the subjects. For the remaining women, these data were based on self-report. These proportions were unknown in NFBC 1966. (Olsen *et al*. 1995). The prepregnancy body mass index (prepregnancy weight (kg)/height (m^2) was divided into four categories: 1) thin (<20.0 kg/m^2), 2) normal (20.0–24.9 kg/m^2), 3) slightly overweight (25.0–29.9 kg/m^2), and 4) obese (≥30.0 kg/m^2).

10. Parity (IV): a nulliparous woman is defined as one with no earlier deliveries and a multiparous woman as one with four or more earlier deliveries.

11. Education (IV): Education was divided dichotomously as 1) compulsory or less and 2) more than compulsory. In NFBC 1966, mothers belonging to the

category of compulsory education had at most eight years of basic education, while in NFBC 1986 the education of the mothers in this category usually comprised nine years of basic education.

12. Smoking (IV): In the questionnaire filled in by the mothers they were asked if they smoked regularly after the second month of pregnancy. The answering categories were “yes” or “no”.
13. Maternal age (IV): maternal age at time of delivery was categorized into age-groups below 20 years, 20 – 34 years and 35 years or over.

4.4 Statistical analysis

Incidence and prevalence of ID

The results are described by numbers, proportions, incidences and prevalences with 95% confidence intervals. Comparisons between NFBC 1986 and NFBC 1966 are presented as risk ratios and their 95% confidence intervals. The results obtained in NFBC 1986 are compared with the results obtained in NFBC 1966.

Biomedical aetiology associated with ID

The results are described by numbers, proportions, incidences and prevalences with 95% confidence intervals. In order to study the magnitude of the difference in the proportions of the distribution of aetiological categories and single disorders/ syndromes by level of ID between NFBC 1986 and NFBC 1966 the Fischer exact probability test was used.

Maternal and familial sociodemographic factors associated with ID

The sociodemographic conditions to which mothers/families in each cohort were exposed were considered as exposure factors or independent variables. Outcome or dependent variable was intellectual disability in live-born children in NFBC 1986 and NFBC 1966. Firstly, the unadjusted incidences and odds ratios in all the categories of each factor were estimated in both cohorts separately. Secondly, a Breslow-Day test for homogeneity of the odds ratios (Breslow & Day 1994) was computed within each factor between NFBC 1986 and NFBC 1966. Thirdly, the logistic regression analysis was conducted separately in both cohorts. The factors

representing the statistically (and clinically) most significant unadjusted odds ratios associated with ID were forwarded into adjusted analyses to explore their independent association with ID. A population attribution risk (PAR) for ID was also calculated separately for NFBC 1986 and NFBC 1966 (Griffith *et al*. 1993). PAR takes into account both the individual-level risk and the prevalence of a given risk factors in the population. The population-attributable fraction percentage estimates the effect of a risk factor on the population as a whole and is the proportion by which the rate of a given outcome (e.g. intellectual disability) would be reduced in the population if the rate associated with a given risk factor were reduced to that of the referent group. Even if a causal relationship cannot be necessarily established, the population-attributable fraction percentage will still serve to identify the group that is having the largest impact on the overall rate or number of cases in the population. (Fleiss 1979, Chapman *et al*. 2002)

Behavioural problems among children with ID

Numbers, proportions, odds ratios and their 95% confidence intervals were used to present data and to show associations. The comparison group was composed of the children in NFBC 1986 who had not been identified as having intellectual disability.

4.5 Ethical considerations

This study has been approved by the ethics committee of the Faculty of Medicine at the University of Oulu. The permission for the case record review was approved by the Ministry of Health and Social Affairs. Informed written consent was requested from members of both cohorts (in NFBC 1986 at 15/16 years; in NFBC 1966 at 31 years), and at that time participants and/or their parents had an option to refuse the use of their data. In NFBC 1966 at 31 years there were 93 subjects who refused the use of their data (and 268 deceased subjects during the follow-up), leaving 11,965 subjects for the denominator in the incidence of ID and 11,697 subjects in the prevalence of ID (in Paper I).

5 Results, specific discussion and comments

5.1 Incidence and prevalence of intellectual disability

5.1.1 Incidence and prevalence of intellectual disability in NFBC 1986

Incidence and prevalence by severity of ID is presented in Table 3. In NFBC 1986, the total incidence of ID was 12.62/1,000 live-born by age of 11.5 years and prevalence 11.23/1,000 alive at age of 11.5 years. 40% of the incident cases had severe ID (IQ<50) and the rest mild ID; the corresponding percentages for prevalent cases were 33 and 67, in accordance.

Comments

Studies on the incidence of ID comparable with the present study are few. One of them has been carried out in Rochester, Minnesota, US (Katusic *et al*. 1995). The total incidence of ID in our study was 1.4 times higher than that reported by Katusic *et al*. (12.6/1,000 vs. 9.1/1,000), the incidence of severe ID (IQ<50) being about the same (5.1/1,000 vs. 4.9/1,000), but the incidence of mild ID was 1.8 times higher (7.5/1,000 vs. 4.3/1,000). The differences between the NFBC 1986 study and the US study by Katusic *et al*. (1995) probably reflect the well-known factors that have an effect on comparisons of this kind, especially when comparing the incidence of mild ID. Although there can be true differences in the occurrence of ID between populations, data collection practices (Kebbon 1987) and follow-up periods differ in epidemiological studies on ID. In NFBC 1986 the follow-up lasted until the age of 11.5 years, while in the study by Katusic *et al*. (1995) it was up to the age of eight years. It is possible that more children with mild ID would have been diagnosed after the age of eight years (Baird & Sadovnick 1985, Katusic *et al*. 1995), while the incidence of severe ID might be more reliably diagnosed by the age of eight years.

The overall prevalence of ID at age 11.5 years in NFBC 1986 is similar to that seen in another Finnish cohort study (Matilainen *et al*. 1995) and a US (Atlanta) cohort study (Murphy *et al*. 1995). However, it is 1.8 times higher than that seen in a corresponding study carried out in Norway in the 1990s (Strømme & Valvatne, 1998), but lower than obtained in an Australian study (Leonard *et al*. 2003; 11.0/1,000 vs. 14.3/1,000). The rate of severe ID (IQ<50) is the same as the “average” value (3.7/1,000) reported in the review by Roeleveld *et al*. (1997).

Table 3. The incidence of intellectual disability (ID) by age 11.5 years (per 1,000 born alive) and prevalence of intellectual disability (ID) at age 11.5 years (per 1,000 alive) in the Northern Finland Birth Cohort 1986 (NFBC 1986) and the Northern Finland Birth Cohort 1966 (NFBC 1966).

	NFBC 1986				NFBC 1966				Incidence		Prevalence	
Level of ID	Incidence (N=9,432)	N (%)	Prevalence (N=9,351)	N	Incidence (N=11,965)	N (%)	Prevalence (N=11,697)	N (%)	RR †	CI (95%)*	RR †	CI (95%)*
Profound (IQ<20)	1.48	14 (12)	1.28	12 (11)	1.42	17 (11)	1.37	16 (12)	1.04	(0.52, 2.12)	0.94	(0.44,1.98)
Severe (IQ 20–34)	1.06	10 (8)	0.75	7 (7)	2.34	28 (19)	2.05	24 (19)	0.45	(0.22, 0.93)	0.36	(0.16,0.85)
Moderate (IQ35–49)	2.54	24 (20)	1.71	16 (15)	3.84	46 (30)	2.56	30 (23)	0.66	(0.40, 1.08)	0.67	(0.36,1.22)
Subtotal (IQ<50)	*5.08*	*48 (40)*	*3.75*	*35 (33)*	*7.61*	*91 (60)*	*5.98*	*70 (54)*	0.67	(0.47, 0.95)	0.63	(0.42,0.94)
Mild (IQ 50–70)	7.53	71 (60)	7.49	70 (67)	5.01	60 (40)	5.04	59 (46)	1.50	(1.07, 2.11)	1.48	(1.05,2.10)
Total (IQ≤70)	12.62	119 (100)	11.23	105 (100)	12.62	151 (100)	11.03	129 (100)	1.00	(0,79, 1.27)	1.02	(0.79,1.32)

*95% confidence interval (CI). †Risk Ratio (RR) between the cohorts is calculated by dividing the rates of the NFBC 1986 with the rates of the NFBC 1966.

The prevalence of mild ID is similar to that seen in another Finnish study (Matilainen et al., 1995), but clearly below the rate (12.8/1,000) obtained in a Swedish study by Fernell (1996).

5.1.2 Incidence and prevalence of intellectual disability between NFBC 1986 and NFBC 1966

There was no change in the total incidence (12.62/1,000 in both) by age 11.5 yeas or in the total prevalence (11.23/1,000 vs. 11.03/1,000) of ID at age of 11.5 years between NFBC 1986 and NFBC 1966. However, changes appeared by the levels of severity with a statistical significance between NFBC 1986 and NFBC 1966, so that the incidence/prevalence of severe ID (IQ<50) decreased and the incidence/prevalence of mild ID increased. The increase by 50% in the incidence/prevalence of mild ID was due to a same-size decrease in the incidence/prevalence of severe and moderate levels combined. The incidence/prevalence of profound ID remained the same between the cohorts (Table 3, Figure 2, Figure 3).

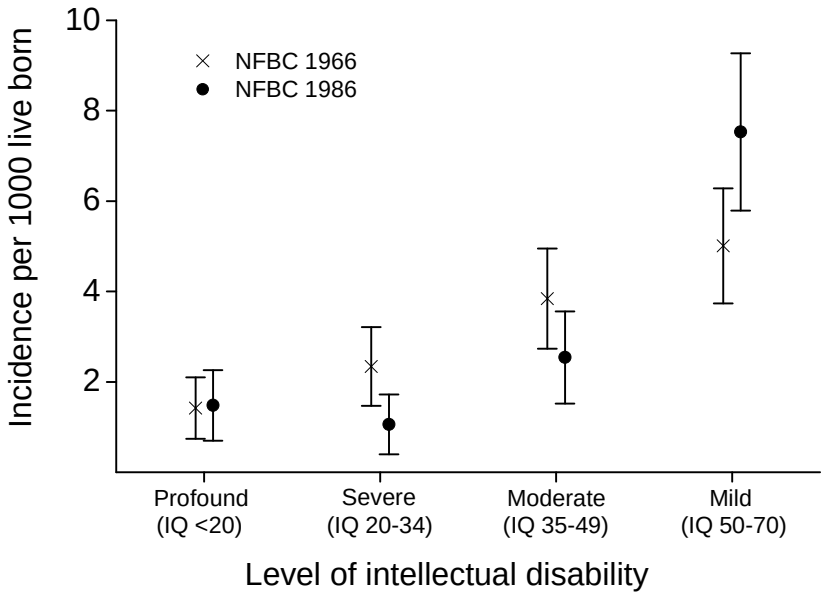

Fig. 2. Incidence of intellectual disability by age of 11.5 years by levels of severity with 95% confidence intervals in the Northern Finland Birth Cohort 1986 (NFBC 1986) and the Northern Finland Birth Cohort 1966 (NFBC 1966).

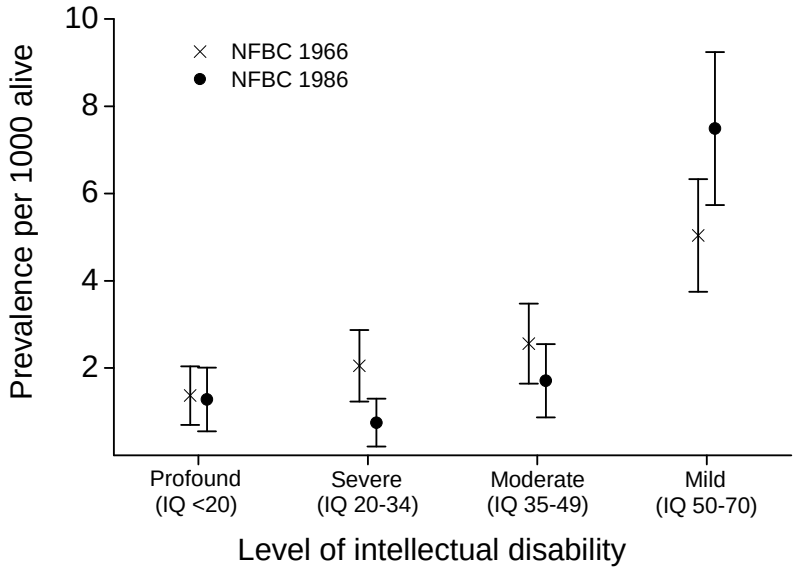

Fig. 3. Prevalence of intellectual disability at age of 11.5 years by levels of severity with 95% confidence intervals in the Northern Finland Birth Cohort 1986 (NFBC 1986) and the Northern Finland Birth Cohort 1966 (NFBC 1966).

In NFBC 1986, the male to female ratio among children with ID was 1.2:1 both in the incident and prevalent cases, whereas in NFBC 1966 the respective ratio was 1.1:1, which indicates that there were no significant differences between the genders in the prevalent or incident cases having ID in either cohort. There was no statistically significant difference between the genders in the separate levels of severity in each cohort, either. (Publication I, Table 4 and Table 5). There emerged no statistically significant shifts in the ratios between the genders in incident or prevalent cases on any level of severity or in the total ID between NFBC 1986 and NFBC 1966 (Publication I, Table 6).

Comments

In general, the variation found in the occurrence of ID can be explained due to inconsistent criteria for case definition, varying age of cohorts, case ascertainment methods and definition on ID (Bernsen 1976, Zigler *et al*. 1984, Kiely 1987, Strømme & Hagberg 2000, Leonard *et al*. 2003), true variation over populations, or other discrepancies between studies (Murphy *et al*. 1995, Roeleveld *et al*.

1997). Our study showed that using similar definition, period of follow-up, geographical study area and case ascertainment methods the incidence and prevalence between the NFBC cohorts were very similar indicating that the differences observed in the severity can be taken as reflecting in large part true differences.

To conclude, in an interval of 20 years between NFBC 1986 and NFBC 1966, the incidence and the prevalence of ID remained similar. However, a clear shift from severe and moderate ID to mild ID has occurred, even though the most severe form of ID, profound ID, remained unchanged. There were no differences in the gender ratio within and between the cohorts by levels of severity. As ID is not a single syndrome or disease, but depends on underlying associated aetiology (Gillberg 1992), in order to be able to analyse temporal changes in the incidence and prevalence one has to explore the determinants contributing to the temporal changes. In this kind of exploration, in addition to aetiology, social and cultural factors as well as advances in medical practice and service system in general between the 1960s and 1990s should be considered (Grossman 1983, Fryers 1984). In general, when exploring the causes or associated factors of trends and distributions of ID, studies based on incident cases are the most appropriate (Stein & Susser 1971).

5.2 Aetiological factors associated with intellectual disability

5.2.1 Aetiological factors associated with intellectual disability in NFBC 1986

Table 4 shows the distribution of main aetiological categories and single disorders/diseases associated with ID both in the incident and prevalent (in brackets) cases categorized into two levels of ID. In NFBC 1986, a biomedical factor associated with ID could be found in 66.4% of the cases with ID, in 93.7% with severe (IQ<50) ID and in 47.9% with mild ID. Genetic causes accounted for slightly over one third of the aetiological factors of ID and they dominated both in the severe (IQ<50) and the mild levels of ID. Of the genetic factors, trisomy-21 (Down syndrome) was the most common, accounting for 13.4% of all incident cases (15.2% of prevalent cases) with ID, giving an incidence of 1.70/1,000 live-born.

Malformations and malformation syndromes of unknown cause accounted for 16.8% of the associated aetiology with ID, while external prenatal and postnatal disorders were more rare. Unknown causes comprised one third of all incident cases with ID.

The major associated biomedical factors among the children with ID who had died during the follow-up of 11.5 years were malformations and malformation syndromes of unknown cause (9 out 14 cases). The rest of the associated biomedical aetiology comprised one autosomal chromosomal disorder, two autosomal recessive single gene mutations, one unknown paranatal intracranial lesion and one case of unclassified CNS symptoms with epilepsy and ataxia. All but one had severe (IQ<50) ID.

Table 4. The distribution (%) of children with severe and mild intellectual disability by aetiological main categories and by single causes of ID among incident and prevalent cases in the Northern Finland Birth Cohort 1986. Prevalent cases at age 11.5 years are shown in brackets. N=9,432 at birth, N=9,351 alive at 11.5 years. #=deaths

Aetiological category/single cause	Severe ID IQ<50 N=48 (35)		Mild ID IQ 50–70 N=71 (70)		Total IQ≤70 N=119 (105)	
	n	%	n	%	n	%
Prenatal causes, total	41 (30)	85.4 (85.7)	29 (28)	40.8 (40.0)	70 (58)	58.8 (55.2)
I Genetic disorders	24 (21)	50.0 (60.0)	19 (19)	26.8 (27.1)	43 (40)	36.1 (38.1)
1. Chromosomal aberrations	12 (11)	25.0 (31.4)	11 (11)	15.5 (15.7)	23 (22)	19.3 (21.0)
Autosomal						
Trisomy 21 (Down syndrome)	10		6		16	
46,XY,-5, +der(5)t(5;9)(q35; p13)pat	1#				1	
46, XY/47, XY, +8			1		1	
46, XY, (4)dup(q28;q31.3)			1		1	
Sex chromosomal						
47, XYY			1		1	
47, XXX			1		1	
Microdeletions						
Angelman syndrome	1				1	
Prader-Willi syndrome			1		1	
2. Single gene mutations	7 (5)	14.6 (14.3)	2 (2)	2.8 (2.9)	9 (7)	7.6 (6.7)
Autosomal dominant						
Tuberous sclerosis	1				1	
Cornelia de Lange syndrome	1				1	
Mitochondriopathy[1)]			1		1	
Autosomal recessive						
Mitochondriopathy	1				1	
Mucopolysaccharidosis	1#				1	
Lipoprotein-metabolic disease	1#				1	

Table 4. Continued

Aetiological category/single cause	Severe ID IQ<50 N=48 (35)		Mild ID IQ 50–70 N=71 (70)		Total IQ≤70 N=119 (105)	
	n	%	n	%	n	%
Sex-linked						
Menkes syndrome	1				1	
Rett syndrome	1				1	
Fragile-X			1		1	
3. Multifactorial	5 (5)	10.4 (14.3)	6 (6)	8.5 (8.6)	11 (11)	9.2 (10.5)
Pure familial	3		4		7	
Autism	2		2		4	
II Malformations and malformation syndromes of unknown cause	14 (6)	29.2 (17.1)	5 (4)	7.0 (5.7)	19 (10)	16.0 (9.5)
CNS malformations	12		3		15	
Anencephaly	1#				1	
Holoprosencephaly	1				1	
Congenital hydrocephaly	2				2	
Craniosynostosis	2		1		3	
Cerebral anomaly, not otherwise specified			1		1	
Multiple congenital malformations	6#		1#		7	
Multiple malformation syndromes	2		2		4	
Syndrome, not otherwise specified	1+1#		2		4	
III External prenatal disorders	3 (3)	6.3 (8.6)	5 (5)	7.0 (7.1)	8 (8)	6.7 (7.6)
Toxins						
Foetal alcohol syndrome	1				1	
Nutrition / intrauterine growth retardation / prematurity						
Embryopathy	1				1	
Foetopathy	1		1		2	
Prematurity			4		4	
IV Paranatal disorders (between 1 week before and 4 weeks after birth)	1 (0)	2.1	3 (3)	4.2 (4.3)	4 (3)	3.4 (2.9)
Delivery complications						
Intracranial lesion, not otherwise specified	1#				1	
Asphyxia			3		3	
V Postnatal disorders	3 (3)	6.3 (8.6)	2 (2)	2.8 (2.9)	5 (5)	4.2 (4.8)
Infections						
Encephalitis	1		1		2	
Meningitis			1		1	
Traumas						
Cerebral contusion	1				1	
Anoxia	1				1	

Table 4. Continued

Aetiological category/single cause	Severe ID IQ<50 N=48 (35)		Mild ID IQ 50–70 N=71 (70)		Total IQ≤70 N=119 (105)	
	n	%	n	%	n	%
VI Unknown causes	3 (2)	6.3 (5.7)	37 (37)	52.1 (52.9)	40 (39)	33.6 (37.1)
Pure non-familial	2		29 (29)		31 (31)	
With central nervous system symptoms			8 (8)		9 (8)	
Lennox epilepsy and ataxia					1	
Cerebral palsy			2		2	
Epilepsy			1		1	
Minimal brain dysfunction			2		2	
Attention deficit hyperactivity disorder			1		1	
Muscular hypotony			1		1	
Epilepsy, cerebral palsy, amaurosis			1		1	
Total	48 (35)	100 (100)	71 (70)	100 (100)	119 (105)	100 (100)

[1] Progressive encephalopathy, sister has mitochondriopathy

Comments

In NFBC 1986, genetic factors were responsible for 38.1% of the prevalent ID cases. The major single factor was Down syndrome, which was the causative factor for ID in 15.2% of the cases. The prevalence as well as the proportion of Down syndrome as the causative factor for ID was higher than the corresponding rates from other population-based studies (Matilainen *et al*. 1995, Yeargin-Allsop *et al*. 1997, Hou *et al*. 1998, Cans *et al*. 1999, Strømme & Hagberg 2000) and has been so since at least from the 1960s, when the first population-based studies were conducted (Leisti *et al*. 1985, Rantakallio & von Wendt 1985). The relatively high portion of older mothers (35 years or over) has had an effect on the high prevalence rates in Down syndrome (Hartikainen 1973), but while the proportion of older mothers decreased, a trend of an increase in the chromosomal nondisjunction rate in the younger age groups occurred between 1975 and 1979 in northern Finland. (Leisti *et al*. 1985).

The prevalence of genetic disorders (4.23/1,000) associated with ID was higher in NFBC 1986 than in the contemporary Norwegian study by Strømme and Hagberg (2000) and the eastern Finnish Study by Matilainen *et al*. (1995). The difference is partly due to a higher prevalence in the chromosomal aberrations in NFBC 1986 (Publication III, Table 2).

Fragile X has been considered the most common inherited cause of ID, with an occurrence of approximately one in 1,300 (Kähkönen *et al*. 1987, Szymanski & Wilska 1996). However, in NFBC 1986 there was only child with fragile X, possibly due to the targeted screening for the families at risk and the availability of abortion (Leisti 2002).

Some rare autosomal recessive disorders are found in Finland due to the enrichment of recessive genes (Norio 1981, Linna 1989); these disorders are called Finnish disease heritage conditions. Disorders belonging this group such as aspartylglycosaminuria (AGU), a sialic acid storage disorder (Salla disease), nonketotic hyperglycinaemia (NKH) and infantile neuronal ceroid lipofuscinosis (INCL) have been found to be aetiological causes for severe ID in 4% to 5% in the northern (Linna 1989) and eastern part of Finland (Matilainen *et al*. 1995). In NFBC 1986 there were no children with ID suffering from these conditions.

Among external prenatal disorders associated with ID, the prevalence of foetal alcohol syndrome (FAS) was low – only one child, a result that is consistent with corresponding studies (Matilainen *et al*. 1996, Yeargin-Allsop *et al*. 1997, Strømme & Hagberg 2000). The low prevalence of FAS is presumably a result of low alcohol consumption among pregnant women in NFBC 1986 (Kotimaa *et al*. 2003).

In NFBC 1986, 64% of the children with ID who had died during the follow-up had the diagnosis of CNS malformation or malformation syndromes of unknown cause. This tendency has been found in autopsy studies of severe ID; for example cerebral developmental abnormalities have been found in 25% of autopsy series of severe ID (Hagberg & Hagberg 1985), ID per se is not necessarily associated with an increased death rate, but the underlying aetiology or associated complicating handicaps may predispose the child with ID to an early death (Rantakallio & von Wendt 1986).

Paranatal causes (asphyxia, anoxia and maldisposition) appeared only in mild ID with a proportion of 4%. Strømme and Hagberg (2000) found the same kind of result in their contemporary corresponding study in Norway. In northern Finland the rate of paranatal causes resulting in ID has decreased remarkably between 1960 and 1980s. In the study by Linna (1989), ID was due to paranatal factors in 9.1% cases born between 1960 and 1974, a rate similar to that stated in the review by McLaren & Bryson (1987) and by Matilainen *et al*. (1995). The spectrum of paranatal disorders in our study was also different from that of Linna (1989) for in our study group there were for example no cases with such neonatal disorders as hyperbilirubinaemia.

5.2.2 Aetiological factors associated with intellectual disability between NFBC 1986 and NFBC 1966

Table 5 has been made for descriptive purposes to present the distribution of the proportions of aetiological factors associated with ID by single disorders/diseases and by main aetiological categories in all levels of severity of ID between NFBC 1986 and NFBC 1966. When comparing total proportions of aetiological categories between NFBC 1986 and NFBC 1966, statistically significant differences appear in the proportions of Down syndrome (a decrease from 25.2% of all in NFBC 1966 to 13.4% in NFBC 1986) as well as in other chromosomal disorders (an increase from 0.7% to 5.9%) and CNS-malformations (an increase from 2.6% to 12.6%). However, the total proportion of prenatal disorders was about the same in both NFBC cohorts. Traumas and asphyxia of paranatal origin decreased from 11.9% in NFBC 1966 to 3.4% in NFBC 1986 with a high statistical significance. (Table 5, Figure 4). Due to low numbers of cases in different aetiological categories any statistical testing needs to interpreted with caution.

The distributions of the proportions of aetiological categories by level of ID between NFBC 1986 and NFBC 1966 are very similar in profound ID, while there were greater differences in all the other levels of severity. Down syndrome was more often associated with mild ID in NFBC 1986 than in NFBC 1966. CNS malformations were more seldom associated with severe ID, and external prenatal disorders were more rarely associated with moderate ID in NFBC 1986 than in NFBC 1966. Altogether, prenatal disorders as well as its subcategory genetic disorders were more rarely associated with severe/moderate ID and more often associated with mild ID in NFBC 1986 than in NFBC 1966. This trend was apparent in the subcategory of unknown aetiology, too. In the single gene defects the distributions of the proportions in all levels of severity were alike between the cohorts. During the follow-up to the age of 11.5 years, 14 children with Down syndrome had died in NFBC 1966, while none of the children with Down syndrome had died during the follow-up in NFBC 1986.

Table 5. Aetiological main categories and single syndromes/disorders by the severity of intellectual disability (ID) until age of 11.5 years in the Northern Finland Birth Cohort 1986 (NFBC 1986, N=9,432 live-born) and the Northern Finland Birth Cohort 1966 (NFBC 1966, N=12,058 live-born). Percentages are given of the total ID population in each cohort and of the aetiology they denote.

Aetiological category		Level of ID																		
	NFBC	Profound (IQ<20)				Severe (IQ 20–34)				Moderate (IQ 35–49)				Mild (IQ 50–70)				Total (IQ≤70)		
		N	%all*	%†	p†	N	%all*	%†	p†	N	%all*	%†	p†	N	%all*	%†	p‡	all	%	p‡
1. Prenatal causes	1986	12	10.1	17.1		9	7.6	12.9		20	16.8	28.6		29	24.4	41.4		70	58.8	
	1966	11	7.3	12.9	0.50	23	15.2	27.1	0.05	36	23.8	42.4	0.09	15	10.0	17.6	0.00	85	56.3	0.71
1.1 Genetic disorders	1986	6	5.0	14.0		5	4.2	11.6		13	5.9	30.2		19	16.0	44.2		43	36.1	
	1966	7	4.6	12.3	1.00	14	9.3	24.6	0.13	26	17.2	45.6	0.15	10	6.6	17.5	0.01	57	37.7	0.80
1.1.1 Down syndrome	1986	2	1.7	12.5		1	0.8	6.3		7	5.9	43.8		6	8.5	37.5		16	13.4	
	1966	4	2.6	10.5	1.00	10	6.6	26.3	0.14	21	13.9	55.3	0.55	3	2.0	7.9	0.01	38	25.2	0.02
1.1.2 Other chromosomal	1986		–	–		1	0.8	14.3		1	0.8	14.3		5	4.2	71.4		7	5.9	
aberrations	1966		–	–			–	–	1.00	1	0.7	100.0	0.25		–	–	0.38	1	0.7	0.02
1.1.3 Single gene mutations	1986	2	1.7	22.2		3	2.5	33.3		2	1.7	22.2		2	1.7	22.2		9	7.6	
	1966	3	2.0	25.0	1.00	3	2.0	25.0	1.00	3	2.0	25.0	1.00	3	2.0	25.0	1.00	12	7.9	1.00
1.1.4 Multifactorial	1986	2	1.7	18.2			–	–		3	2.5	27.3		6	8.5	54.5		11	9.2	
	1966		–	–	0.52	1	0.7	16.7	0.35	1	0.7	16.7	1.00	4	2.6	66.7	1.00	6	4.0	0.08
2. Malformations of the CNS	1986	5	4.2	26.3		2	1.7	10.5		7	5.9	36.8		5	4.2	26.3		19	16.0	
	1966	3	2.0	21.4	1.00	7	4.6	50.0	0.02	2	1.3	14.3	0.24	2	1.3	14.3	0.67	14	9.3	0.13
2.1 CNS malformations	1986	5	4.2	33.3		2	1.7	13.3		5	4.2	33.3		3	2.5	20.0		15	12.6	
	1966		–	–	0.53	2	1.3	50.0	0.18	1	0.7	25.0	1.00	1	0.7	25.0	1.00	4	2.6	0.00
2.2 Malformation syndromes	1986		–	–			–	–		2	1.7	50.0		2	1.7	50.0		4	3.4	
	1966	3	2.0	30.0	0.51	5	3.3	50.0	0.22	1	0.8	10.0	0.18	1	0.8	10.0	0.18	10	6.6	0.28
3. External prenatal disorders	1986	1	0.8	12.5		2	1.7	25.0			–	–		5	7.0	62.5		8	6.7	
	1966	1	0.7	7.1	1.00	2	1.3	14.3	0.60	8	5.3	57.1	0.02	3	2.0	21.4	0.08	14	9.3	0.51
3.1 Infections	1986		–	–			–	–			–	–			–	–		–		
	1966		–	–			–	–		2	1.3	100.0			–	–		2	1.3	
3.2 Toxins	1986		–	–			–	–		1	0.7	–			–	–		1	0.8	
	1966		–				–	–			–				–			–		
3.3 Prematurity	1986	1	0.8			1	0.8				–	–		5	7.0	71.4		7	5.9	
	1966	1	0.7	9.1		2	1.3	18.2		5	3.3	45.5	0.10	3	2.0	27.3	0.15	11	7.3	0.81
3.4 Other	1986		–				–	–.			–	–			–	–		–	0.7	
	1966		–	–			–	–		1	0.7	100.0			–	–		1		

Table 5. Continued

Aetiological category	NFBC	Level of ID																	
		Profound (IQ<20)				Severe (IQ 20–34)				Moderate (IQ 35–49)				Mild (IQ 50–70)				Total (IQ≤70)		
		N	%all*	%†	p†	N	%all*	%†	p†	N	%all*	%†	p†	N	%all*	%†	p‡	all	%	p‡
4. Paranatal disorders	1986		–	–			–	–		1	0.8	25.0		3	2.5	75.0		4	3.4	
	1966	4	2.6	17.4	1.00	4	2.6	17.4	1.00	4	2.6	17.4	1.00	12	7.9	50.0	0.60	24	15.9	0.00
4.1 Infections	1986		–				–	–			–	–			–	–		–		
	1966	1	0.7	25.0		1	0.7	25.0			–	–		2	1.3	50.0		4	2.6	
4.2 Traumas, asphyxia	1986		–				–	–		1	0.7	25.0		3	2.5	75.0		4	3.4	
	1966	2	1.3	11.1		3	2.0	16.7		4	2.6	22.2	1.00	9	6.0	50.0	0.59	18	11.9	0.01
4.3 Toxic	1986		–				–				–				–			–		
	1966	1	0.7	50.0			–	–			–	–		1	0.7	50.0		2	1.3	
5. Postnatal disorders	1986	1	0.8	20.0		1	0.8	20.0		1	0.8	20.0	1.00	2	1.7	40.0		5	4.2	
	1966		–	–	1.00		–	–	1.00	2	2.0	40.0		3	2.0	60.0	1.00	5	3.3	0.75
5.1 Infections	1986		–	–		1	0.8	33.3			–	–		2	1.7	66.7		3	2.5	
	1966		–	–			–	–	1.00	1	0.7	100.0	0.25		–	–	1.00	1	0.7	0.32
5.2 Traumas	1986	1	0.8	50.0			–	–		1	0.8	50.0			0	0.0		2	1.7	
	1966		–	–	1.00		–	–			–	–	1.00	1	0.7	100.0	0.33	1	0.7	0.59
5.3 Psychosocial deprivation	1986		–				–				–				–			–		
	1966		–	–			–	–			–	–		1	0.7	100.0		1	0.7	
5.4 Psychosis	1986		–				–				–				–			–		
	1966		–	–			–	–		1	0.7	50.0		1	0.7	50.0		2	1.3	
6. Unknown causes	1986	1	0.8	2.5			–	–		2	2.5	5.0		37	52.1	92.5		40	33.6	
	1966	2	1.3	5.4	0.61	1	0.7	2.7	0.48	4	2.6	10.8	0.42	30	50.0	81.1	0.18	37	24.5	0.11
TOTAL	1986	14	11.8	11.8		10	8.4	8.4		24	20.2	20.2		71	59.7	59.7		119	100	
	1966	17	11.3	11.3	1.00	28	18.5	18.5	0.02	46	30.5	30.5	0.07	60	39.7	39.7	0.00	151	100	

* percentage of all cases with ID in the NFBC cohort

† percentage of the cases with ID by the level of ID

‡ p from Fisher's exact probability test, NFBC 1986 vs. 1966

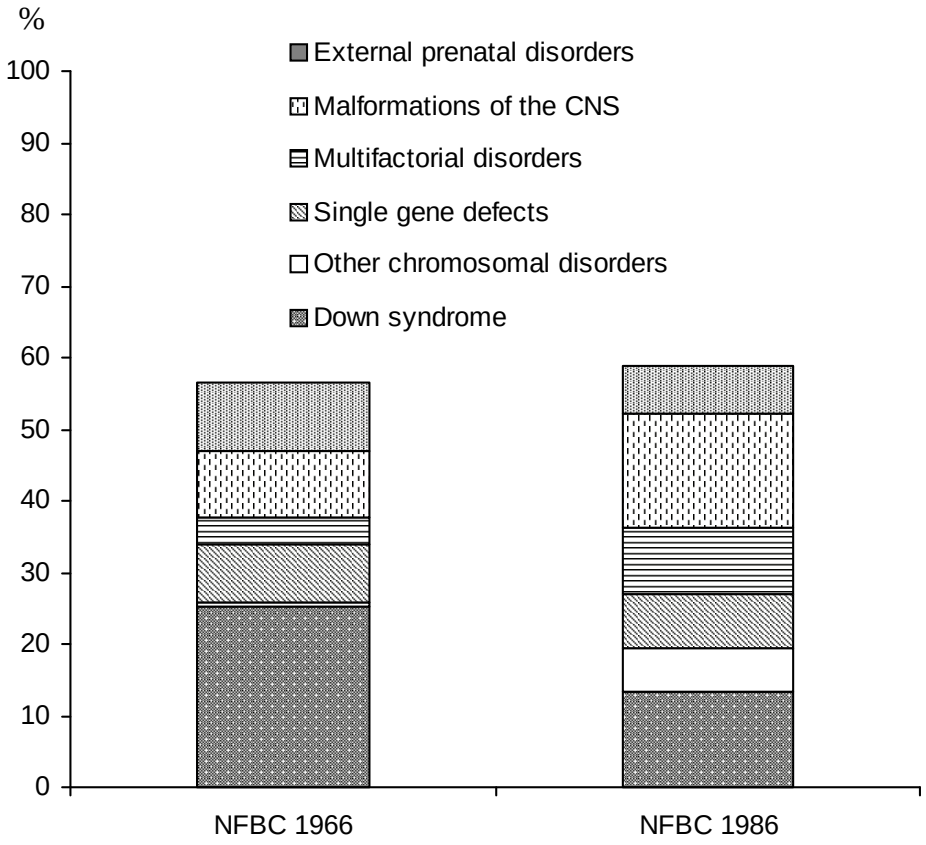

Fig. 4. a. The proportion (%) of prenatal aetiological main categories and single disorders associated with intellectual disability (ID) in the Northern Finland Birth Cohort 1986 (NFBC 1986) and in the Northern Finland Birth Cohort 1966 (NFBC 1966).

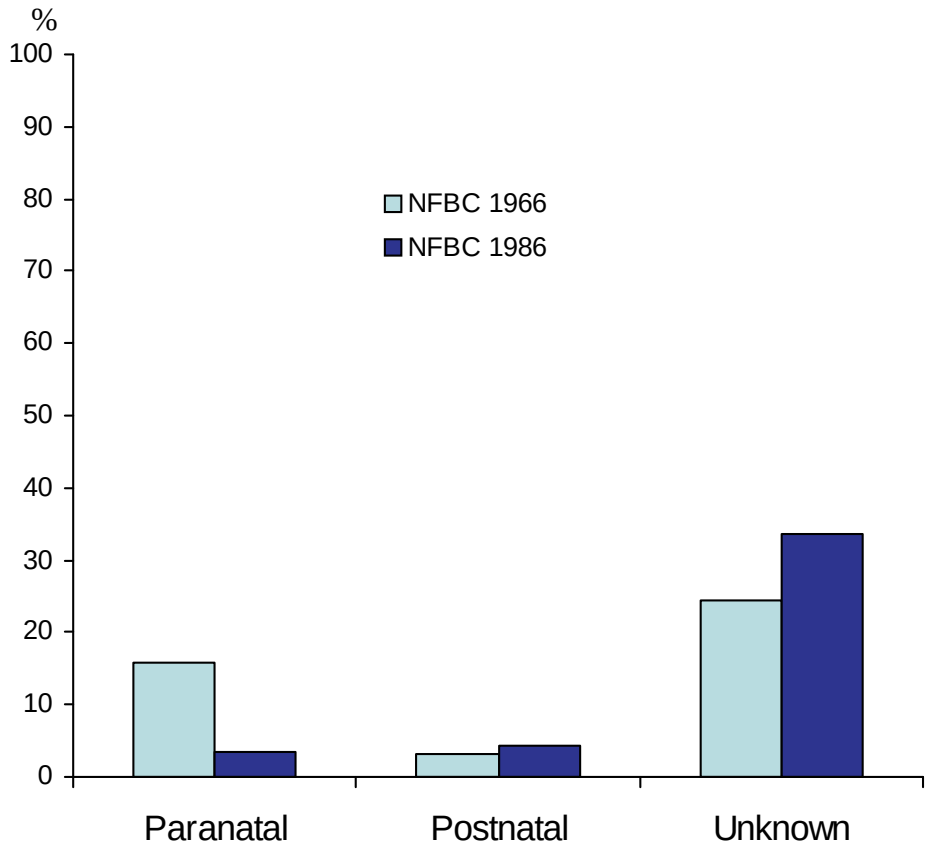

Fig. 4. b. The proportion (%) of paranatal, postnatal and unknown aetiological factors associated with intellectual disability (ID) in the Northern Finland Birth Cohort 1986 (NFBC 1986) and in the Northern Finland Birth Cohort 1966 (NFBC 1966).

Comments

Although there was no change in the total incidence (12.62/1,000 in each) between the NFBC cohorts, it could be expected that in an interval 20 years between the 1960s and the 1980s some shifts and changes had occurred in the distribution of the proportions of aetiological main categories and single disorder/diseases associated with ID. Fryers (1986) has stated that prevalence of severe ID varies in time and space, for it is dependent on the incidence of associated specific aetiologies, mortality of the individuals with ID and migration. Sources of this variation are

different genetic, environmental, economic, political, religious, attitudinal and health care factors. However, in most communities the most important factors that have an effect on the prevalence of severe ID is maternal age distribution through its effect on the incidence of Down syndrome, nutritional and health care factors affecting pre- and perinatal causes and mortality as well as health and social care factors affecting survival. Also intervention programs may influence the prevalence of ID, e.g. amniocentresis and abortion, immunization and combined screening for inherited disorders of metabolism and congenital hypothyroidism. These factors causing variation to the prevalence of ID may reflect general social and economical level in the given society.

Comparison between NFBC 1986 and NFBC 1966 shows that the incidence of profound ID remained the same, for the proportions of associated separate aetiological factors did not change. The incidence of severe ID decreased, mainly because there were fewer cases with Down syndrome, but also proportionally fewer cases with CNS malformations and paranatally originated traumas/asphyxia in NFBC 1986. In moderate-level ID, the decrease in the incidence was largely due to fewer cases with Down syndrome, but the decrease in the proportion of prematurity and traumas/asphyxia also had an effect. There was an increase of about 50% in the incidence of mild ID in NFBC 1986 compared with NFBC 1966, which was due to an increase in the proportion of all the other prenatal disorders/syndromes except single-gene defects and multifactorial disorders.

Interestingly, as expected based on existing literature on predicting trends in the proportions of aetiologies associated with ID (Fryers 1986, Kiely 1987, O'Brien 2003), in an interval of 20 years in northern Finland a decrease occurred in the proportion of Down syndrome, which might at least partly be due to a reduction of older maternal age at time of delivery. In addition, an increase in the proportion of CNS malformations was seen, a result that can be assumed to be connected with improved pre- and perinatal care enabling survival of children with malformations; an increase in the proportion of other chromosomal disorders than Down syndrome may be linked to increasing diagnostic awareness and improvements in the diagnostics of disorders of this kind. The decrease in the proportion of paranatally originated asphyxias and traumas can be considered to result from improvements in the pre- and perinatal medical care of pregnant mothers and foetuses.

5.3 Sociodemographic factors associated with intellectual disability

5.3.1 Sociodemographic factors associated with intellectual disability in NFBC 1986

In NFBC 1986, the unadjusted maternal sociodemographic factors that associated statistically significantly with total ID in the offspring were prepregnancy obesity (BMI ≥30), being a farmer or unskilled worker, multiparity and compulsory level of education. By level of ID, in severe ID (IQ<50) being an unskilled worker was the only statistically significant associated factor, while in mild ID the corresponding factors were obesity (BMI ≥30), multiparity, only compulsory education and having the SES of an unskilled worker or a farmer. (Table 6)

Table 6. Distribution of children and incidence of intellectual disability (ID) in the Northern Finland Birth Cohort 1986 (N=9,432), according to sociodemographic factors (incidence per hundred are given as well as odds ratios with a 95% confidence interval).

	Children with ID* (n=119)											
	All (n=9, 432)		Severe ID (IQ<50)			Mild ID (IQ 50–70)			Total (IQ≤70)			P Hom OR†
Maternal characteristics	N	%	N	Incidence (%)	Odds ratio (95% CI‡)	N	Incidence (%)	Odds ratio (95% CI‡)	N	Incidence (%)	Odds ratio (95% CI‡)	
Age (years)												
<20	399	4.2	1	0.25	0.4 (0.0, 3.6)	4	1.00	1.4 (0.5, 4.1)	5	1.25	1.0 (0.4, 2.6)	0.9554
20–34	7795	82.6	39	0.50	Referent	53	0.68	Referent	92	1.18	Referent	
≥35	1238	13.1	8	0.65	1.2 (0.6, 2.7)	14	1.13	1.6 (0.9, 3.0)	22	1.78	1.5 (0.9, 2.4)	0.1516
Parity												
0	3194	34.0	20	0.63	1.6 (0.8, 3.0)	19	0.59	0.9 (0.5, 1.6)	39	1.22	1.1 (0.7, 1.7)	0.6387
1–3	5438	57.9	21	0.39	Referent	35	0.64	Referent	56	1.03	Referent	
≥4	755	8.0	6	0.79	2.0 (0.8, 5.1)	16	2.12	3.3 (1.8, 6.7)	22	2.91	2.8 (1.7, 4.7)	0.5666
Miscarriages												
No	7545	80.4	33	0.44	Referent	55	0.73	Referent	88	1.17	Referent	
Yes	1841	19.6	14	0.76	1.7 (0.9, 3.2)	15	0.81	1.1 (0.6, 1.9)	29	1.58	1.3 (0.8, 2.0)	0.3881
Body mass index (kg/m^2)												
<20.0	2235	24.3	8	0.36	0.7 (0.3, 1.7)	14	0.63	0.9 (0.5, 1.7)	22	0.98	0.8 (0.5, 1.4)	0.2936
20.0–24.9	5400	58.7	25	0.46	Referent	35	0.65	Referent	60	1.11	Referent	
25.0–29.9	1209	13.1	8	0.66	1.4 (0.6, 3.1)	7	0.58	0.8 (0.3, 2.0)	15	1.24	1.1 (0.6, 1.9)	0.8697
≥30.0	353	3.8	4	1.13	2.4 (0.8, 7.1)	10	2.83	4.4 (2.1, 9.1)	14	3.97	3.6 (2.0, 6.6)	0.0525
Marital status												
Married, cohabiting	8917	94.9	45	0.50	Referent	65	0.73	Referent	110	1.23	Referent	
Unmarried	398	4.2	1	0.25	0.4 (0.0, 3.6)	5	1.26	1.7 (0.6, 4.3)	6	1.51	1.2 (0.5, 2.8)	0.1523
Widow, divorced	85	0.9	1	1.18	2.3(0.3, 17.2)	–	–	–	1	1.18	0.9 (0.1, 6.9)	0.3210
Education												
Compulsory	2167	23.0	10	0.46	0.9 (0.4, 1.8)	29	1.34	2.6 (1.5, 4.3)	39	1.80	1.7 (1.1, 2.6)	0.6520
More than compulsory	5996	63.6	30	0.50	Referent	31	0.52	Referent	61	1.02	Referent	
Unknown	1269	13.5	8	0.63	1.2 (0.5, 2.7)	11	0.87	1.6 (0.8, 3.3)	19	1.50	1.4 (0.8, 2.4)	0.8917

Table 6. Continued

	Children with ID* (n=119)											
	All (n=9, 432)		Severe ID (IQ<50)			Mild ID (IQ 50–70)			Total (IQ≤70)			P Hom OR†
Maternal characteristics	N	%	N	Incidence (%)	Odds ratio (95% CI‡)	N	Incidence (%)	Odds ratio (95% CI‡)	N	Incidence (%)	Odds ratio (95% CI‡)	
Socioeconomic status												
I–II (professional)	2385	25.3	13	0.55	1.9 (0.8, 4.5)	7	0.29	0.5 (0.2, 1.4)	20	0.84	1.0 (0.6, 1.9)	0.6228
III (skilled worker)	3657	38.8	10	0.27	Referent	18	0.49	Referent	28	0.77	Referent	
IV (unskilled worker)	2653	28.1	22	0.83	3.0 (1.4, 6.4)	35	1.32	2.7 (1.5, 4.7)	57	2.15	2.8 (1.8, 4.4)	0.5321
Farmer	432	4.6	1	0.23	0.8 (0.1, 6.6)	9	2.08	4.3 (1.9, 9.6)	10	2.31	3.0 (1.4, 6.3)	0.5705
Unknown	305	3.2	2	0.66	2.4 (0.5, 11.0)	2	0.66	1.3 (0.3, 5.7)	4	1.31	1.7 (0.6, 4.9)	0.7476
Socioeconomic status of the family												
I–II (professional)	3004	31.8	13	0.43	0.6 (0.3, 1.1)	13	0.43	0.6 (0.3, 1.1)	29	0.97	0.8 (0.5, 1.3)	0.8772
III (skilled worker)	4479	47.5	31	0.69	Referent	31	0.69	Referent	51	1.14	Referent	
IV (unskilled worker)	1366	14.5	18	1.32	1.9 (1.0, 3.4)	18	1.32	1.9 (1.0, 3.4)	28	2.05	1.8 (1.1, 2.8)	0.4042
Farmer	583	6.2	9	1.54	2.2 (1.0, 4.7)	9	1.54	2.2 (1.0, 4.7)	11	1.89	1.6 (0.8, 3.2)	0.9846
Place of residence												
Urban	4061	43.4	21	0.52	Referent	31	0.76	Referent	52	1.28	Referent	
Rural	3359	35.9	16	0.48	0.9 (0.4, 1.7)	23	0.68	0.8 (0.5, 1.5)	39	1.16	0.9 (0.5, 1.3)	0.9313
Remote area	1942	20.7	10	0.51	0.9 (0.6, 2.1)	16	0.82	1.0 (0.6, 1.9)	26	1.34	1.0 (0.6, 1.6)	0.2595
Number of visits to antenatal clinic/maternal healthcentre												
≤6	220	2.6	2	0.91	1.7 (0.4,7.5)	4	1.82	2.5 (0.9,7.2)	6	2.73	2.2 (0.9, 5.2)	0.8692
7–12	5901	70.3	30	0.51	Referent	42	0.71	Referent	72	1.22	Referent	
>12	2274	27.1	11	0.48	0.9 (0.5, 1.9)	17	0.75	1.0 (0.5, 1.8)	28	1.23	1.0 (0.6, 1.5)	0.2775
Smoking after 2 months of pregnancy												
No	7311	79.6	35	0.48	Referent	55	0.75	Referent	90	1.23	Referent	
Yes	1871	20.4	12	0.64	1.3 (0.6, 2.5)	15	0.80	1.0 (0.6, 1.8)	27	1.44	1.1 (0.7, 1.8)	0.1527

* ID, intellectual disability
† P Hom OR, P-value for a test for homogeneity of unadjusted odds ratios between the Northern Finland Birth Cohort 1986 and the Northern Finland birth Cohort 1966
‡ CI, confidence interval.

The factors that had a strong univariate association with ID in both of the NFBC cohorts were chosen for multivariate logistic regression model in order to find out their independent effect. The factors chosen for adjustment were maternal age at time of delivery, parity, prepregnancy BMI, marital status, socio-economic status and place of residence were the (Table 6). Being an unskilled worker turned out to have an independent elevated risk for ID in both severe and mild ID, being a farmer had the highest risk for ID in mild ID; in total ID maternal prepregnancy obesity appeared as having the highest risk with ID. The SES of unskilled worker, having only compulsory education, multiparity and prepregnancy obesity had the largest impacts on the incidence of ID calculated as population attribution risk (PAR) in NFBC 1986 (Table 7).

Table 7. Mutually adjusted sociodemographic factors for the risk of severe, mild and total intellectual disability (ID) in the Northern Finland Birth Cohort 1986 (NFBC 1986) and Northern Finland Birth Cohort 1966 (NFBC 1966) with 95% confidence interval.

Maternal characteristics	NFBC 1986			NFBC 1966		
	Severe ID (IQ<50) OR* (95% CI*)	Mild ID (IQ 50–70) OR* (95% CI*)	Total ID (IQ≤70) OR* (95% CI*)	Severe ID (IQ<50) OR* (95% CI*)	Mild ID (IQ 50–70) OR* (95% CI*)	Total ID (IQ≤70) OR* (95% CI*)
Age (years)						
<20	0.4 (0.0, 2.7)	0.9 (0.2, 3.1)	0.6 (0.2, 1.8)	1.5 (0.6, 3.9)	0.4 (0.1, 1.6)	0.9 (0.4, 2.0)
20–34	Referent	Referent	Referent	Referent	Referent	Referent
≥35	1.2 (0.5, 3.0)	1.0 (0.5, 2.1)	1.1 (0.6, 1.9)	2.8 (1.5, 5.1)	0.6 (0.2, 1.8)	1.8 (1.1, 2.9)
Socioeconomic status						
I–II (professional)	1.8 (0.8, 4.3)	0.6 (0.2, 1.4)	1.0 (0.6, 1.8)	1.1 (0.4, 2.9)	0.5 (0.1, 2.4)	0.9 (0.4, 2.0)
III, skilled worker	Referent	Referent	Referent	Referent	Referent	Referent
IV (unskilled worker)	3.1 (1.4, 6.7)	2.2 (1.2, 3.9)	2.5 (1.6, 4.0)	2.0 (0.8, 4.9)	1.7 (0.5, 5.8)	1.9 (0.9, 3.9)
Farmer	0.7 (0.1, 6.2)	3.7 (1.4, 9.8)	2.6 (1.1, 6.1)	0.9 (0.4, 2.1)	3.5 (1.1, 11.4)	1.4 (0.7, 2.9)
Unknown	1.4 (0.2, 11.1)	0.7 (0.1, 5.2)	0.9 (0.2, 4.0)	1.3 (0.6, 2.7)	2.8 (1.2, 6.9)	1.8 (1.0, 3.2)
Body mass index (kg/m^2)						
<20.0	0.7 (0.3, 1.7)	1.0 (0.5, 1.8)	0.9 (0.5, 1.4)	1.0 (0.5, 2.1)	2.1 (1.0, 4.1)	1.5 (0.9, 2.4)
20.0–24.9	Referent	Referent	Referent	Referent	Referent	Referent
25.0–29.9	1.4 (0.6, 3.1)	0.7 (0.3, 1.7)	1.0 (0.6, 1.8)	0.9 (0.5, 1.6)	0.6 (0.2, 1.6)	0.8 (0.5, 1.3)
≥30.0	2.6 (0.9, 7.7)	2.9 (1.3, 6.1)	2.8 (1.5, 5.3)	1.0 (0.4, 2.5)	0.5 (0.1, 3.8)	0.8 (0.3, 2.0)
Parity						
0	2.1 (1.1, 4.0)	0.8 (0.4, 1.5)	1.2 (0.8, 3.6)	1.0 (0.5, 1.9)	1.1 (0.6, 2.3)	1.0 (0.6, 1.7)
1–3	Referent	Referent	Referent	Referent	Referent	Referent
≥4	1.5 (0.5, 4.0)	2.4 (1.2, 4.9)	2.0 (1.2, 3.6)	1.8 (1.0, 3.4)	1.5 (0.6, 3.6)	1.7 (1.0, 2.8)
Place of residence						
Urban	Referent	Referent	Referent	Referent	Referent	Referent
Rural	1.0 (0.5, 2.0)	0.8 (0.5, 1.5)	0.9 (0.6, 1.4)	1.4 (0.7, 2.7)	0.3 (0.1, 0.7)	0.7 (0.4, 1.2)
Remote area	1.0 (0.4, 2.4)	0.6 (0.3, 1.3)	0.8 (0.4, 1.3)	1.9 (1.0, 3.6)	0.4 (0.2, 0.8)	1.0 (0.6, 1.6)
Marital status						
Married	Referent	Referent	Referent	Referent	Referent	Referent
Unmarried	0.4 (0.1, 3.4)	2.4 (0.9, 6.6)	1.4 (0.6, 3.3)	2.8 (1.1, 7.4)	1.4 (0.3, 6.4)	2.3 (1.0, 5.0)
Widow	2.3 (0.3, 17.4)	–	0.9 (0.1, 6.4)	–	–	–

* OR, odds ratio. CI, confidence interval.
† IQ, intelligence quotient.

Table 8. Maternal sociodemographic factors associated with 5% population attributable risk of intellectual disability in the Northern Finland Birth Cohort, 1986 and 1966.

Northern Finland Birth Cohort 1986	%	Northern Finland Birth Cohort 1966	%
Being an unskilled worker	27.5	Compulsory education only	25.2
Compulsory education only	12.7	Multiparity (≥4 earlier deliveries)	19.7
Multiparity (≥4 earlier deliveries)	11.7	Age ≥35 years	18.9
Family socioeconomic status unskilled worker	10.6	Residing in a remote area	18.4
Body mass index ≥30	9.1	<6 visits at antenatal/maternal health centre	13.6
Previous miscarriages	6.4	Being a farmer	9.5
Age ≥35 years	6.2	Being an unskilled worker	9.5
		Family socioeconomic status of a farmer	9.4
		Family socioeconomic status of an unskilled worker	6.1
		Being unmarried	5.2

Comments

Socioeconomic disadvantage was associated with ID in both severe and mild ID in NFBC 1986, a result that differs from previous studies showing that low SES is associated with mild ID only (Penrose 1938, Zigler 1995, Strømme & Magnus 2000). The mediating factors between low parental SES and poorer cognitive function in the offspring may be factors such as poor living conditions in general, impairments of children's physical health status at birth and inadequate provision of learning stimulation in the home environment (Roeleveld *et al*. 1997, McLoyd 1998). However, it has been a common general observation that children from lower SES groups have more health problems compared to the children from higher SES groups. (Gissler *et al*. 1998).

The distribution of the educational status of the mothers with a child who has severe ID was equal to the corresponding distribution in the background population, i.e. all mothers in NFBC 1986, while a low level of maternal education was statistically significantly associated with mild ID only. This result is consistent with corresponding studies (Drews *et al*. 1995, Camp *et al*. 1998, Leonard *et al*. 2005). Chapman *et al*. (2002) stated that low maternal education might operate during pregnancy so that mothers with a low educational level may not be aware of the risk factors that might cause poor developmental outcomes in the child. Also, during the child's early developmental years maternal education may be related, among many other child-rearing issues, to knowledge of and access to early intervention services for children born at risk. (McLoyd 1998).

Sameroff *et al*. (1987) have stated that no single sociodemographic factor uniquely limits early intellectual achievement, but it is cumulative effects from multiple risk factors that increase the probability that intellectual development will be compromised. Social disadvantage often implies the existence of other risk factors that serve to compromise a child's development. However, the term social disadvantage should not be used interchangeably with poorer development of child, which is the case in environmental view of disadvantage, but individual biological characteristics and supportive environmental factors should be considered, too. Disadvantage viewed from a child-focused orientation means that attention should be paid to individual characteristics and biological vulnerabilities of the child. Those children at both biological and environmental risk are referred to as being "doubly vulnerable", and they are especially susceptible to significant developmental problems. (Guralnick 1998). A genetic study of mild mental impairment from a sample of 3,88 twins presented that the heritability of low intellectual level is 50% (Spinath *et al*. 2004), but geneticists have, however, emphasized that there is no fixed level of heritability invariant over time and over differing physical and social conditions (Rutter *et al.* 2006). Based on the view presented by geneticists that a powerful adverse environmental factor can lower the impact of genetic factors accounting for the liability to show a particular trait in a particular population Rutter *et al.* (2006) have stated there is a gene-environment interplay modifying the phenotypic behaviour and it is possible that the contribution of heritability of intelligence is decreased in the presence of social disadvantage.

Maternal prepregnancy obesity (BMI≥30) associated with an increased risk for ID in the offspring in NFBC 1986, the association being stronger with mild ID than with severe ID. To author's knowledge this is the first study in which the association between mother's prepregnany weight and ID in the offspring has been studied. In a small-scale study carried out in the US the association between maternal prepregnancy BMI and general IQ was explored at the average age of 5.3 years of 355 low-income African-American children. The result was that after adjusting for other covariates (age, receptive language ability, zinc supplementation status, smoking, alcohol use, the child's birth weight, childcare status and home environment), the children with obese mothers (BMI>29) had significantly lower general cognitive ability and non-verbal scores as compared with the children with normal-weight mothers (general intellectual ability score (80.0±12.7 vs. 84.5±12.7) (Neggers *et al*. 2003). While the prevalence of obesity has increased dramatically, the association between low SES and obesity has weakened in industrialized countries (Zhang & Wang 2004). It is well known that overweight and obese women

are at increased risk for pre-eclampsia, eclampsia, dystocia, caesarean delivery, macrosomic offspring and late foetal death, and that the risks increase along with increasing BMI (LaCoursiere *et al*. 2005). Maternal prepregnancy obesity has also been found to associate with spina bifida, omphalocele, heart defects and multiple anomalies in the offspring. It is possible that obese women have metabolic alterations, such as hyperglycaemia, elevated insulin or oestrogen levels that increase the risk for birth defects, but they might also have nutritional deficits resulting from dieting behaviours or poor-quality diets that increase the risk for congenital anomalies. (Watkins *et al*. 2003). However, clear demonstration of the causal effects of early nutrition on long-term neurodevelopment requires an experimental approach, which is not easily accomplished with pregnant women (Neggers *et al*. 2003).

5.3.2 Sociodemographic factors associated with intellectual disability between NFBC 1986 and NFBC 1966

Temporal changes in the sociodemographic factors between all mothers of the children in NFBC 1986 and all mothers of the children in NFBC 1966 appeared in parity, number of visits at antenatal clinic/maternal health centre during pregnancy, BMI, education, SES and place of residence. In NFBC 1986 the mothers were less often multiparous and had more rarely only a few (six times or less) visits at antenatal clinic/maternity health centre. The proportion of lean mothers (BMI<20) increased 1.8 fold, while the amount of obese (BM≥I30) mothers remained the same between the cohorts (3.8%). Educational level improved and the proportion of higher SES females (professional and skilled workers) had increased from one third to two thirds. In NFBC 1986, 20% of the mothers lived in the remote areas compared to 41% in NFBC 1966. (Table 6, Table 9)

Comparison of the univariate maternal sociodemographic factors associated with total ID in the offspring between NFBC 1986 and NFBC 1966 indicated, that only one factor, maternal prepregnancy obesity (BMI≥30), appeared as having a marginally statistical significance (P value, a test for homogeneity, p=0.053) presenting its' strengthened association with ID (Table 7). By severity of ID in an interval of twenty years, new associated risk factors did not emerge, but mother's older age at time of delivery (p=0.01) as well as living in a remote area (p=0.056) had lost their effect as major unfavourable associated factors for severe ID in NFBC 1986. High BMI (P=0.03) and multiparity (p=0.05) were new unfavourable factors associated with an elevated risk of mild ID in NFBC 1986. (Table 6)

Table 9. Distribution of children and incidence of intellectual disability (ID) in the Northern Finland Birth Cohort 1966 (N=12,058), according to sociodemographic factors (incidence per hundred are given as well as odds ratios with a 95% confidence interval).

Maternal characteristics	All (n=12,058)		Children with ID* (n=151)								
			Severe ID (IQ<50)			Mild ID (IQ 50–70)			Total (IQ≤70)		
	N	%	N	Incidence of ID* (%)	Odds ratio (95% CI†)	N	Incidence of ID* (%)	Odds ratio (95% CI†)	N	Incidence of ID* (%)	Odds ratio (95% CI†)
Age (years)											
<20	906	7.5	7	0.77	1.6 (0.7, 3.5)	3	0.33	0.6 (0.1, 2.0)	10	1.10	1.0 (0.5, 2.1)
20–34	8936	74.1	43	0.48	Referent	47	0.53	Referent	90	1.01	Referent
≥35	2213	18.4	41	1.85	3.9 (2.5, 6.0)	10	0.45	0.8 (0.4, 1.7)	51	2.30	2.3 (1.6, 3.2)
Parity											
0	3892	32.3	21	0.54	1.0 (0.5, 1.7)	19	0.49	1.0 (0.5, 1.9)	40	1.03	1.0 (0.6, 1.5)
1–3	5939	49.3	32	0.54	Referent	27	0.45	Referent	59	0.99	Referent
≥4	2207	18.3	38	1.72	3.2 (2.0, 5.1)	14	0.63	1.3 (0.7,2.6)	52	2.36	2.4 (1.6,3.5)
Miscarriages											
No	9824	82.9	70	0.71	Referent	51	0.52	Referent	121	1.23	Referent
Yes	2028	17.1	20	0.99	1.3 (0.8, 2.2)	6	0.30	0.5 (0.2, 1.3)	26	1.28	1.0 (0.6, 1.5)
Body mass index (kg/m^2)											
<20.0	1464	13.4	8	0.55	0.8 (0.3, 1.7)	12	0.82	2.0 (1.0, 3.9)	20	1.37	1.2 (0.7, 2.1)
20.0–24.9	7125	65.0	47	0.66	Referent	29	0.41	Referent	76	1.07	Referent
25.0–29.9	1964	17.9	17	0.87	1.3 (0.7, 2.2)	5	0.25	0.6 (0.2, 1.6)	22	1.12	1.0 (0.6, 1.6)
≥30.0	413	3.8	5	1.21	1.8 (0.7, 4.6)	1	0.24	0.5 (0.0, 4.3)	6	1.45	1.3 (0.5, 3.1)
Marital status											
Married, cohabiting	11519	95.7	84	0.73	Referent	54	0.47	Referent	138	1.20	Referent
Unmarried	437	3.6	7	1.60	2.2 (1.2, 6.9)	6	1.37	2.9 (1.2, 6.9)	13	2.97	2.5 (1.4, 4.5)
Widow, divorced	86	0.7	–	–	–	–	–	–	–	–	–
Education											
Compulsory	7788	65.4	64	0.82	1.3 (0,8 2.2)	45	0.58	1.8 (0,9 3.5)	109	1.40	1.5 (1.0, 2.3)
More than compulsory	3905	32.8	23	0.59	Referent	12	0.31	Referent	35	0.90	Referent
Unknown	208	1.7	2	0.96	1.6 (0.3, 6.9)	1	0.48	1.5 (0.2, 12.1)	3	1.44	1.6 (0.4, 5.3)

Table 9. Continued

Maternal characteristics	All (n=12, 058)		Children with ID* (n=151)								
			Severe ID (IQ<50)			Mild ID (IQ 50–70)			Total (IQ≤70)		
	N	%	N	Incidence of ID* (%)	Odds ratio (95% CI†)	N	Incidence of ID* (%)	Odds ratio (95% CI†)	N	Incidence of ID* (%)	Odds ratio (95% CI†)
Socioeconomic status											
I–II (professional)	1483	12.3	7	0.47	1.1 (0.4, 3.1)	2	0.13	0.4 (0.0, 2.0)	9	0.61	0.8 (0.3, 1.9)
III (skilled worker)	2540	21.1	10	0.39	Referent	8	0.31	Referent	18	0.71	Referent
IV (unskilled worker)	1037	8.6	10	0.96	2.4 (1.0, 5.9)	6	0.58	1.8 (0.6, 5.3)	16	1.54	2.1 (1.1, 4.3)
Farmer	2704	22.4	29	1.07	2.7 (1.3, 5.6)	16	0.59	1.8 (0.8, 4.4)	45	1.66	2.3 (1.3, 4.1)
Unknown	4294	35.6	35	0.82	2.0 (1.0, 4.2)	28	0.65	2.0 (0.9, 4.5)	63	1.47	2.0 (1.2, 3.5)
Socioeconomic status of the family											
I–II (professional)	2798	23.2	4	0.14	0.2 (0.0, 0.6)	4	0.14	0.2 (0.0, 0.6)	24	0.86	0.8 (0.4, 1.3)
III (skilled worker)	4030	33.4	25	0.62	Referent	25	0.62	Referent	43	1.07	Referent
IV (unskilled worker)	3042	25.2	19	0.62	1.0 (0.5, 1.8)	19	0.62	1.0 (0.5, 1.8)	45	1.48	1.3 (0.9, 2.1)
Farmer	2188	18.1	12	0.55	0.8 (0.4, 1.7)	12	0.55	0.8 (0.4, 1.7)	39	1.78	1.6 (1.0, 2.6)
Place of residence											
Urban	3989	33.1	17	0.43	Referent	26	0.65	Referent	43	1.08	Referent
Rural	3155	26.2	23	0.73	1.7 (0.9, 3.2)	7	0.22	0.3 (0.1, 0.7)	30	0.95	0.8 (0.5, 1.4)
Remote area	4914	40.8	51	1.04	2.4 (1.4, 4.2)	27	0.55	0.8 (0.4, 1.4)	78	1.59	1.4 (1.0, 2.1)
Number of visits to antenatal clinic/maternal health centre											
≤6	1395	12.3	23	1.65	2.2 (1.3, 3.7)	11	0.79	1.7 (0.8, 3.5)	34	2.44	2.0 (1.3, 3.1)
7–12	6715	59.4	49	0.73	Referent	30	0.45	Referent	79	1.18	Referent
>12 times	3195	28.3	15	0.47	0.6 (0.3, 1.1)	12	0.38	0.8 (0.4, 1.6)	27	0.85	0.7 (0.4, 1.1)
Smoking after 2 months of pregnancy											
No	9957	84.5	79	0.79	Referent	49	0.49	Referent	128	1.29	Referent
Yes	1821	15.5	10	0.55	0.6 (0.3, 1.3)	7	0.38	0.7 (0.3, 1.7)	17	0.93	0.7 (0.4, 1.2)

* ID, intellectual disability
† CI, confidence interval.

Multivariate logistic regression models (Table 7) presented that mother's older age at time of delivery, multiparity and being unmarried associated independently with total ID and separately with severe ID in NFBC 1966. Also living in remote place of residence associated with severe ID in NFBC 1966. In mild ID, mother's low BMI and being a farmer appeared as having a statistically significant independent effect. Comparison with NFBC 1986 indicated that temporal changes had occurred in adjusted associated sociodemographic factors in passing of time, for older age at time of delivery and being unmarried were no more associated with an elevated risk of total ID, in severe ID all the associated unfavourable factors (older age, multiparity, living remote area) and prepregnancy leanness (BMI<20) in mild ID had lost their independent effect. On the other hand new unfavourable factors had emerged in NFBC 1986, namely nulliparity in severe ID, prepregnancy obesity (BMI≥30) with mild and total ID and unskilled worker in both levels of severity. However, a farmer in mild ID and multiparity in total ID had retained their status as unfavourable associated factors.

The interaction of maternal BMI and parity, i.e. the possible additive effects, were studied in further analyses (Figure 5 and Figure 6) in both NFBC cohorts. An increased trend was seen in the opposite categories of maternal prepregnancy BMI. In NFBC 1986 the incidence of ID was elevated for obese women in all parity categories with a linear increase from 1.4 fold in nulliparas to 3.4-fold in multiparas, when compared with total incidence of ID (36/1,000 vs. 12.6/1,000) (Figure 5). In contrast to that, an increased trend in the incidence of ID was apparent in lean mothers (BMI<20) in NFBC 1966, though the trend was weaker than in NFBC 1986. The highest incidence of ID among lean mothers appeared in multiparity category (3 earlier deliveries) with 2.9-fold risk compared with the total incidence of ID (36/1,000 vs. 12.6/1,000) (Figure 6).

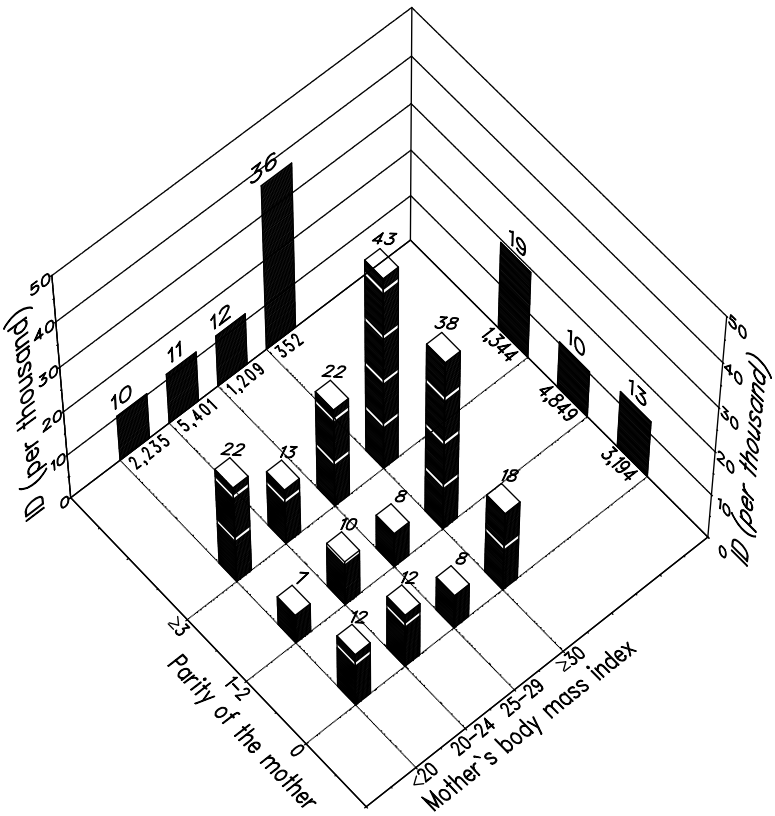

Fig. 5. Effect of the interaction between maternal parity and body mass index on risk of intellectual disability (ID) in the offspring in the Northern Finland Birth Cohort 1986. Numbers above bars indicate incidence of ID per 1000 live born. Total numer by parity and body mass index categories are beneath the bars.

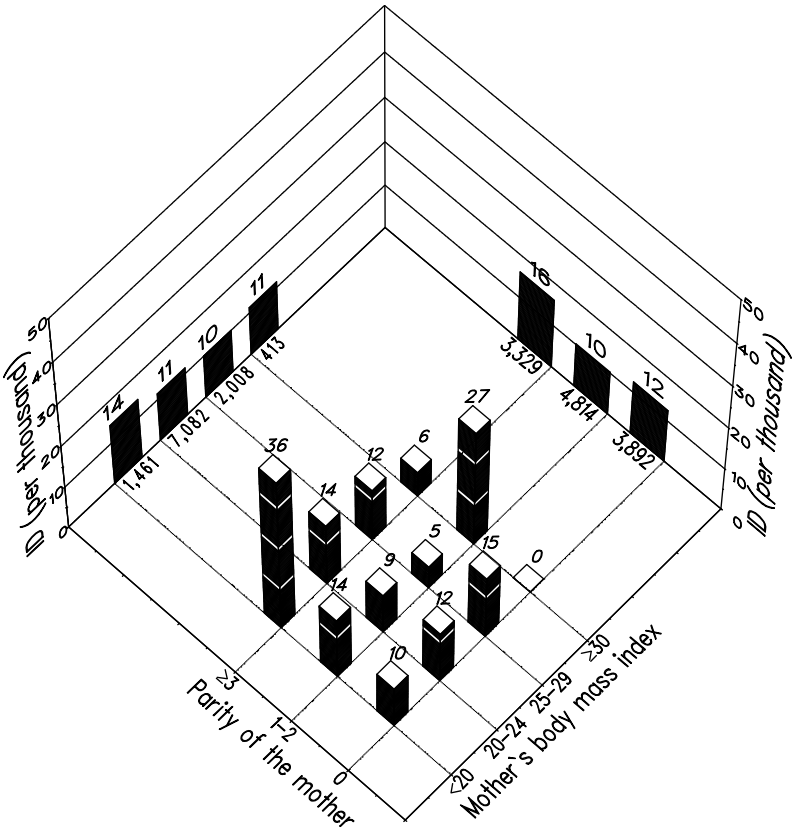

Fig. 6. Effect of the interaction between maternal parity and body mass index on risk of intellectual disability (ID) in the offspring in the Northern Finland Birth Cohort 1966. Numbers above bars indicate incidence of ID per 1000 live born. Total numer by parity and body mass index categories are beneath the bars.

The largest population attributable risks (PAR) for ID in both cohorts are presented in Table 8. Comparison of PAR fractions of ID between NFBC 1986 and NFBC 1966 shows that low level of education, low SES and multiparity had the largest impact on the incidence of ID in both cohorts. In the course of time living in remote area and rare visits at antenatal clinic had lost their impact on ID. The impact of older age for ID has decreased from 18.9% in NFBC 1966 to 6,2% in NFBC 1986.

Comments

Comparison of sociodemographic factors associated with ID between NFBC 1986 and NFBC 1966 showed that the indicators of socioeconomic disadvantage and multiparity of the mother had the largest impact on the incidence of ID in both cohorts. No other significant changes in single sociodemographic factors appeared in an interval of 20 years, but prepregnancy obesity of the mother emerged as an unfavourable factor associated with ID. NFBC cohorts differed in the interaction of BMI and parity so that in NFBC 1986 the highest incidence of ID appeared in obese mothers (BMI≤30) with three or more earlier deliveries, while in NFBC 1966 the highest incidence of ID was among mothers who were lean and had three or more earlier deliveries. Being single, living in a remote area and mother's older age at delivery had lost their association with ID over time.

The findings on temporal variations in sociodemographic factors associated with ID between NFBC 1986 and NFBC 1966 indicate firstly that the associations between intellectual disability and maternal/familial sociodemographic factors are wide-ranging (Leonard *et al*. 2006). Secondly they show that sociodemographic factors change in the course of time within the same region. Interestingly in NFBC 1986 and NFBC 1966 the opposite ends of prepregnancy BMI appeared as associated risk factors for ID especially in interaction with three or more earlier deliveries; obesity in NFBC 1986 and leanness in NFBC 1966, though not as strongly as in NFBC 1986. These kinds of results show that temporal variations occur in associated sociodemographic risk factors for ID. Variations like these reflect changes in the living circumstances of people in society. (Fryers 1984, 1986). It is important to collect repeatedly data on social indicators connected with ID and other disabilities in the same area for planning up-to-date targeted services and prevention. Based on the result that older age of the mother, being single and living in remote area had lost their effect on ID, it can be concluded that a rise in the living standard and educational level of the mothers as well as improvements in the availability of high-standard antenatal and obstetric care (Hartikainen 1973), health care and service system (Kaski *et al*. 2001) have at least partly contributed to these changes. (Fryers 1984, 1986)

5.4 Behavioural problems in children with intellectual disability in NFBC 1986

The risk for probable psychiatric disturbances was 4.9 times higher among children with ID than children without ID according to the Children's Behaviour Questionnaire (RB2) filled in by the teachers in the spring of the first school year of children in NFBC 1986. All problems under study were more common among children with ID than among children not having ID. The proportions of behavioural, emotional and hyperactivity problems in these children with ID and non-ID children in NFBC 1986 were 20.8% vs. 9.1%, 18.1% vs. 4.9% and 36.1% vs. 9.3%, accordingly. Behaviour indicating hyperactivity differentiated most distinctly these two groups of children; for example, 61.6% of the children with ID were assessed as having poor concentration or short attention span while the corresponding assessment was made for 20.9% of the children not having ID. (Table 10). Children with mild ID were assessed as having all the problems, probable psychiatric, behavioural, emotional and hyperactivity problems, more often than the children with more severe levels of ID (Publication II, Figure 1). The risk for behavioural, emotional or hyperactivity problems did not differ significantly between boys and girls in the group of children having ID in NFBC 1986, whereas in the comparison group, non-ID children in NFBC 1986, boys had an elevated risk for behavioural problems (OR 3.9, 95% CI 3.2, 4.6) and to be hyperactive (OR 3.9, 95% CI 2.9, 4.2) (Publication II, Table 3).

Table 10. Hyperactivity, emotional and behavioural problems indicated by the items in the Rutter scale in the Northern Finland Birth Cohort 1986 among children with intellectual disability (ID) and children without intellectual disability (ID). Numbers and proportions as well as odds ratios with 95% confidence interval (CI) for the different items on the Rutter scale indicating externalizing and internalizing problems and hyperactivity (the statements "certainly applies" and "applies somewhat" combined).

Parametres	Children with ID N=72		Children without ID N=8410		OR	95% CI
	n	%	n	%		
Hyperactivity problems						
Has poor concentration or short attention span.	45	61.6	1,761	20.9	6.09	3.70–10.05
Squirmy, fidgety child.	43	58.1	2,112	25.0	4.15	2.55–6.78
Very restless.	44	61.1	2,473	29.4	3.80	2.30–6.27
Behavioural problems						
Often destroys or damages own or others' property.	16	22.2	475	5.6	4.79	2.62–8.66
Has stolen things on one or more occasions.	6	8.2	197	2.4	3.73	1.44–9 08
Is often disobedient.	31	42.4	1,522	18.1	3.35	2.05–5.47
Bullies other children.	20	27.4	1,403	16.7	1.89	1.09–3.25
Often tells lies.	9	12.5	701	8.3	1.57	0.72–3.29
Frequently fights or is extremely quarrelsome with other children.	19	26.0	1,596	18.9	1.51	0.86–2.62
Emotional problems						
Tends to be fearful or afraid of new things or new situations.	41	57.7	1,649	19.6	5.62	3.41–9.26
Has had tears on arrival at school or has refused to come into the building this year.	8	10.9	296	3.5	3.38	1.49–7.36
Often appears miserable, unhappy, tearful or Distressed.	25	34.9	1,190	14.1	3.23	1.92–5.39
Often worried, worries about many things.	19	26.4	1,936	23.0	1.20	0.69–2.09

(Modified from Table 4 in Paper II)

Comments

Individuals with ID suffer from the same types of psychiatric problems as nondisabled individuals, but some disorders are more frequent among them than among nondisabled individuals (Bregman 1991, Szymanski & Wilska 1996, Linna *et al*. 1999). The result that 44.4% of the children with ID had probable psychiatric disturbances in NFBC 1986 is within the range of 30% to 60% reported in previous corresponding studies (Gillberg *et al*. 1986, Einfeld & Tonge 1996b, Linna *et al*. 1999, Molteno *et al*. 2001, Dekker *et al*. 2002). The proportion of cases assessed as having probable psychiatric disturbances was 4.9 times greater in the group of children with ID than among children without ID. The same kind of finding has

been made in corresponding studies, too (Gillberg *et al*. 1986; Emerson 2003). Variability between the studies on the prevalence of psychiatric disturbances among individuals with ID results from differences in the assessment procedures, diagnostic criteria and the level of ID of the subjects in the study population. An instrument such as Children's Behaviour Questionnaire for Teacher by Rutter (RB2) used in some of these studies has not been standardized among large samples of individuals with ID, and it should therefore be viewed as preliminary. (Bregman 1991) The questionnaire by Rutter has been developed for teachers to evaluate children's emotional and behavioural problems (Rutter 1967). According to Dekker *et al*. (2002), the use of instruments not specifically designed to assess the behavioural problems of children with ID may be too superficial, lacking preciseness in catching the prevalent disturbances.

The types of problems differed by the level of ID so that probable psychiatric disturbances, behavioural and emotional problems as well as hyperactivity were more common among children with mild ID than among children with more severe levels of ID in NFBC 1986. In the study by Einfeld and Tonge (1996a) another instrument was utilized in assessing psychopathology in children with ID, the prevalence of psychiatric disorder was considerably lower than among children with profound level of ID than among the children having mild, moderate or severe ID, whereas self-absorbed and autistic behaviours were more prominent in those with severe ID and disruptive and antisocial behaviours were more prominent in children with mild ID.

Although the results of behaviour problems among children with ID in NFBC 1986 can be viewed as preliminary because of the weaknesses in the assessment instrument used, it can be concluded that children with ID have more often than their peers not having ID difficulties in managing at school according to their teachers. The most prominent difficulties were hyperactivity problems and especially poor concentration or short attention span. The reason for the high prevalence of psychiatric disorders in children with ID may at least in some cases be that there is a common aetiology behind CNS dysfunction and behaviour problems in children with ID (Bregman 1991, Linna *et al*. 1999). Children with ID and psychiatric disturbances are in need of targeted support and psychiatric services, but however there is lack of these services. Also the specialized needs of the parents supporting a child with ID and with psychiatric services should be fulfilled by the social service system. (Einfeld & Tonge 1996a, Emerson 2003)

The behavioural disturbances of individuals with ID lead to limitations in adaptive behaviour and they decrease the capability to fulfil the societal expecta-

tions. Behavioural problems are a result of a complex interaction between multiple risk factors consisting of biomedical, social, behavioural and educational factors (Coulter 1996). The children with ID having behavioural problems and limitations in adaptation are in need of supports that enables them to access resources and relationship within an integrated environment, resulting in increased integration and enhanced personal growth and development. (Luckasson *et al*. 2002).

6 Discussion about the methods

The study is a descriptive and analytic epidemiological study. Two birth cohorts of children, NFBC 1986 and NFBC 1966, were followed up until the age of 11.5 years. The data on ID in NFBC 1986 are new data, and the data on NFBC 1966 was originally collected by Rantakallio and von Wendt and re-reviewed and scrutinized for the purposes of this study. The focus of this study was on NFBC 1986, while NFBC 1966 formed a comparison group for describing and analysing temporal changes in the occurrence of ID and in associated aetiological and sociodemographic factors. Behavioural problems among children having ID were studied in NFBC 1986 only.

In a cohort study, the quality of the results of the study will depend on the quality of the measurements of the main predictor and outcome variable, and the ability to draw inferences about cause and effect will also depend on how completely and accurately the investigator has measured potential important confounders. Outcomes should be assessed using standardized criteria. (Cummings *et al*. 1988). The validity of the results is influenced by the process of identifying the individuals with ID in a population (Louhiala 2004). In a cohort study design such as in this work, the reliability of case identification is increased since the likelihood of duplication found in registers can be eliminated.

Both NFBC cohorts comprise population-based study groups with a high coverage. In NFBC 1986 the proportion of mothers and deliveries was 99% of all eligible and in NFBC 1966 the corresponding proportion was 96%. The children belonging to the cohort who emigrated the study area during the follow-up were also traced; in NFBC 1986 there were six children who could not be traced whereas in NFBC 1966 the number of children who could not be traced was 14, from among over 9,000 in the younger and over 11,000 in the older cohort. The data for estimating the incidence and prevalence of ID in NFBC 1986 and NFBC 1966 were collected from all the health units having this kind of information in the study area. The data on ID regarding the children who had immigrated to other parts of Finland or abroad were traced in both cohorts. Data collection on biomedical aetiology and maternal/familial sociodemographic factors was also carried out with similar methods. The criterion for ID was the same, based on ICD-9. The author collected the data on the psychometric tests administered to the children in NFBC 1986 and she re-assessed the data on psychometric assessments in NFBC 1966. The same paediatric neurologist evaluated the data on ID of the children who had died during the follow-up. One clinician with special competence in the

medical aspects of intellectual disability evaluated the data on the aetiology associated with ID of children who were alive at the end of the follow-up. The results obtained in this study consisting of two births cohorts of children can be assessed as being reliable and not biased, for study design and data collection methods were mainly the same and the definitions of the study variables were similar.

In the process of identification of children with ID the most recent IQ test result until the end of the follow-up was used in each cohort. The psychometric tests used as the basis of the assessment of intellectual level of the child were mainly WISC-R in NFBC 1986 in NFBC 1986 and Stanford-Binet (Terman-Merrill) in NFBC 1966. The Stanford-Binet and Wechsler Scales have been by far the most widely used tests for the evaluation of intellectually disabled individuals. In the studies carried out in the US the correlations between Stanford-Binet IQs and both WISC and WISC-R full scale IQs varies between 0.81 and 0.83. The difference between Stanford-Binet and the Wechsler tests is that the Weschler Scales were not designated for the assessment of children with severe ID, as they do not have as low a floor as the Stanford-Binet. (Nihira 1985). The Finnish norms for the WISC-R were too strict, especially for 6- to 7-year-old children (Konttila 1998), and some borderline cases whose assessment fell in that age may have been included in the group of children with mild ID instead of being in the borderline (IQ 71–85). However, these issues should not cause a major bias between the two because the mean age in NFBC 1986 of the group of children whose level of ID was assessed through the WISC-R was over 8 years and our follow-up was up to the age of 11.5 years.

The data utilized in this study were collected during the follow-up until the age of 11.5 years from multiple sources and were collected prospectively except for the data on psychometric tests (although they were also based on contemporary records of the standardized tests which data were later transferred on the study forms). The comprehensive data enabled to carry out an expansive study on ID in both NFBC 1986 and NFBC 1966. Besides the incidence and prevalence of ID, the focus in these studies was also on the associated aetiological and sociodemographic factors. Studies utilizing such an extensive data set on ID as this study are rare. An extensive material was also utilized in a Norwegian study based on a cohort of 30,037 children (Strømme & Valvatne 1998, Strømme & Hagberg 2000, Strømme & Magnus 2000, Strømme & Diseth 2000).

In both of the NFBC cohorts all data were collected from multiple sources, such as registers and records. There were neither individual medical nor other individual examinations for the purposes of this study. This kind of method in assess-

ing the incidence and prevalence of ID using data ascertainment based on multiple sources has been utilized in such corresponding studies as Katusic *et al*. (1995), McDonald & McKay (1996) and Strømme & Valvatne (1998), but there are also studies in which the subjects of the study population have been given an individual or group IQ test or a corresponding psychometric test, e.g. Sonnander *et al*. 1993 (mild ID only) and Matilainen *et al*. 1995. In general, population-based studies collecting data on the prevalence of ID based on psychometric testing administered individually or in groups attain a higher prevalence for ID than studies using epidemiological methods.

While the study populations consisted of two birth cohorts of children born in the same geographical area, Northern Finland, with a follow-up and tracing of those who had emigrated the area in both cohorts, it was possible to examine temporal changes and variations in the occurrence of ID and factors affecting the variations. Studies focusing on temporal changes in the prevalence of ID in the same geographical area are rare. One of those has been carried out in Denmark by Dupont (1989), who examined the age-specific prevalence of ID in Denmark in the years 1888, 1965 and 1979 based on the administrative register of the individuals who have ID and are in need of and/or entitled to special services, and another study was conducted by Fryers (1984) in Salford, England. Fryers explored the age-specific prevalence of severe ID and the aetiologies associated with ID in children in Salford between 1961 and 1980 based on the Salford Register.

Data on aetiology associated with ID were mainly based on registers and hospital records in both of the NFBC cohorts. However, there are also studies in which individual medical examinations for detecting aetiology associated with ID have been carried out. Examples of the latter studies are Strømme & Hagberg (2000) in Norway, Hou *et al*. 1998 in Taiwan and Matilainen *et al*. 1995 in eastern Finland. The results on the aetiology for ID obtained in these studies are not consistent, but it is well known that there are differences e.g. in the classification systems of the aetiology of ID, real differences between the populations etc., which makes direct comparisons between studies of this kind difficult.

Both in NFBC 1986 and in NFBC 1966, data on sociodemographic factors were collected from the mothers during pregnancy, and the data described the situation at that time. For example, maternal education refers to the status at prepregnancy time and does not reflect further or final educational attainment of younger mothers in the cohorts in particular. In the corresponding studies the data on familial and maternal sociodemographic characteristics usually reflect the situation dur-

ing the time of delivery, too (Drews *et al*. 1995, Camp *et al*. 1998, Chapman *et al*. 2002, Leonard *et al*. 2005).

In examining behavioural problems among children with ID in NFBC 1986 Children's Behaviour Questionnaire for Teacher developed by Rutter (RB2) was used. It is meant for teachers to evaluate children's emotional and behavioural problems (Rutter 1967). However, it has not been standardized among large samples of individuals with ID, and therefore the results should be viewed as preliminary (Bregman 1991). Another widely used instrument used in assessing the prevalence of psychiatric/behavioural problems in children with ID is the Child Behaviour Checklist (Einfeld & Tonge 1996b). In the studies by Linna *et al*. (1999) and Dekker *et al*. (2002) several instruments for assessing psychiatric problems in children with ID have been utilized.

7 Summary and general discussion

Main results of the study:

1. Total incidence of ID was 12.62/1,000 live born up the age 11.5 years and total prevalence was 11.23/1,000 alive at age 11.5 years in NFBC 1986. The incidence for profound, severe, moderate and mild ID was 1.48/1,000, 1.06/ 1,000, 2.54/1,000 and 7.53/1,000 live born, accordingly. The respective prevalences of ID were 1.28, 0.75, 1.71 and 7.49 per 1,000 alive at age 11.5 years.

2. Associated biomedical aetiology for ID could be found in 66.4% of the incident cases in NFBC 1986. 92.5% of the cases with unknown aetiology associated with ID had mild ID. Genetic disorders accounted for a little over one third of the aetiological factors dominating in both severe and mild ID, Down syndrome being the most common genetic factor. Malformation and malformation syndromes of unknown cause accounted for 16% of cases. Paranatally and postnatally originated aetiological factors associated with ID were rarer.

3. In NFBC 1986, maternal prepregnancy obesity (BMI≥30) appeared as having the highest risk for total ID estimated both by unadjusted and adjusted odds ratio. It dominated also in mild ID. The indicators of socio-economic disadvantage (low level of education and low SES) and multiparity of the mother had the largest impact on the incidence of ID.

4. The risk for probable psychiatric disturbances was 4.9 times higher in children with ID than among their non-ID peers. The difference between children with ID and children not having ID was the greatest in hyperactivity (poor concentration, short attention span) in NFBC 1986.

5. In an interval of 20 years between NFBC 1986 and NFBC 1966 there was no change in total incidence or prevalence between the cohorts. However, there was a shift from severe and moderate levels towards mild ID both on the incidence and the prevalence. Both the incidence and prevalence of mild ID increased about 50% in NFBC 1986. The incidence of profound ID between remained the same. Comparison of the proportions of associated aetiological categories and single syndromes/disorders by level of ID between NFBC 1986 and NFBC 1966 presented that the incidence of profound ID remained the same, because there was no change in the incidence of aetiological factors

associated with this level of ID. The incidence of severe ID decreased, because there were fewer cases with Down syndrome, multiple malformation syndromes and paranatally originated traumas/asphyxia in NFBC 1986 compared with NFBC 1966. The decrease in the proportion of Down syndrome, prematurity and traumas/asphyxia had an effect on decrease in the incidence of moderate ID. The incidence of mild ID increased about 50% in NFBC 1986 compared with NFBC 1966 due to an increase of all the other prenatal disorders/syndromes except single gene defects. Regarding sociodemographic factors associated with ID in both of the NFBC cohorts the indicators of socio-economic disadvantage (low level of education and low SES) and multiparity of the mother had the largest impact on the incidence of ID. No other significant changes in single sociodemographic factors appeared in the interval of 20 years, but prepregnancy obesity of the mother turned to an unfavourable factor associated with ID in NFBC 1986. In the course of time being single, living in a remote area and older maternal age at delivery had lost their association with ID.

The important observation in this study was that although the total incidence and prevalence of ID have remained the same, shifts and changes in associated aetiological and sociodemographic factors contributing to the incidence of ID occurred between the cohorts. Data ascertainment in the same geographical area 20 years apart enabled to study these temporal changes in the occurrence of ID as well in associated factors. Repeated surveys performed within a stable and homogenous population provide a picture of the interaction between, and the relative importance of, the multiple factors influencing the incidence and the prevalence of ID (Dupont 1989). We assume that these shifts and changes are results of the improvements that have taken place in northern Finland between 1960s and 1990s. Just to mention a few: development in medical and antenatal care and the availability of health care services in all geographical areas, a social support system for families having a child with ID, provision of basic education to all children with ID, increase in the living standard, increase in the educational level of the mothers. Children with Down syndrome had more often mild ID instead of severe/moderate ID in NFBC 1986 compared with NFBC 1966. The difference was statistically significant. It can be concluded that the development and improvements in the society mentioned earlier have contributed to this positive change. Whether it is a matter of a longer-lasting increase in the approximate cognitive capacity among children with Down syndrome, the trajectory of IQ of these individuals should be followed

in the future. The proportion of children having ID associated with CNS malformations had increased, which should be noted in the planning of medical, social and educational services. The results on associated sociodemographic factors showed that although risk factors like as being unmarried or living in a remote area have lost their significance, new risks emerge, such as maternal prepregnancy obesity. The finding that low education and low SES of the mother as well as multiparity have remained their status as disadvantageous factors associated with ID should be considered in planning of targeted services for families and mothers since pregnancy.

8 Implications for further study

The association between maternal prepregnancy obesity and ID in the offspring is a new finding, but replications in other studies are needed in order to be able make further judgements on the nature of this association. The mechanism between maternal disadvantage and ID in the offspring is unclear and needs further study, in which factors related to maternal health characteristics, health behaviour and overall well-being during pregnancy should also be included.

There are two general questions which only longitudinal studies can examine: 1) what happens to children who are classified as having ID during the course of time, and 2) why do some adjust and manage better than others as adults. (Richardson & Koller 1985). As this is a longitudinal study these questions can be examined. Important aims for further study when gathering data in future in this longitudinal study concern adaptation into the basic education and later into vocational education system, possibilities for acquiring work as an adult and also characteristics of physical health and mental health. Information on the factors that contribute to the improvement in the functional level and well-being of individuals with ID would be valuable for the planning of services and other procedures that are carried out in the field of the promotion of public health.

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Original publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals:

I Heikura U, Taanila A, Olsen P, von Wendt L, Hartikainen A-L & Järvelin, M-R (2003) Temporal changes in incidence and prevalence of intellectual disability between the birth cohorts 1966 and 1985–86 in Northern Finland. Am J Ment Retard 108 (1): 19–31.

II Taanila A, Ebeling H, Heikura U & Järvelin M-R (2003) Behavioral problems of 8-year-old children with and without intellectual disability. J Pediatr Neurol 1 (1): 15–24.

III Heikura U, Linna S-L, Olsen P, Hartikainen A-L, Taanila A & Järvelin M-R (2005) An etiological survey of intellectual disability in the Northern Finland Birth Cohort 1986. Am J Ment Retard 110(3): 171–180.

IV Heikura U, Taanila A, Hartikainen A-L, Olsen P, Linna S-L, von Wendt L & Järvelin M-R (2007) Variations in prenatal sociodemographic factors associated with intellectual disability: A study of 20 year interval between two birth cohorts in Northern Finland. Am J Epidemiol in press doi:10.1093/ajc/kwm291

Original publications are not included in the electronic version of the dissertation.

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