AUTISM IN NORTHERN FINLAND

A prevalence, follow-up and descriptive study of children and adolescents with autistic disorder

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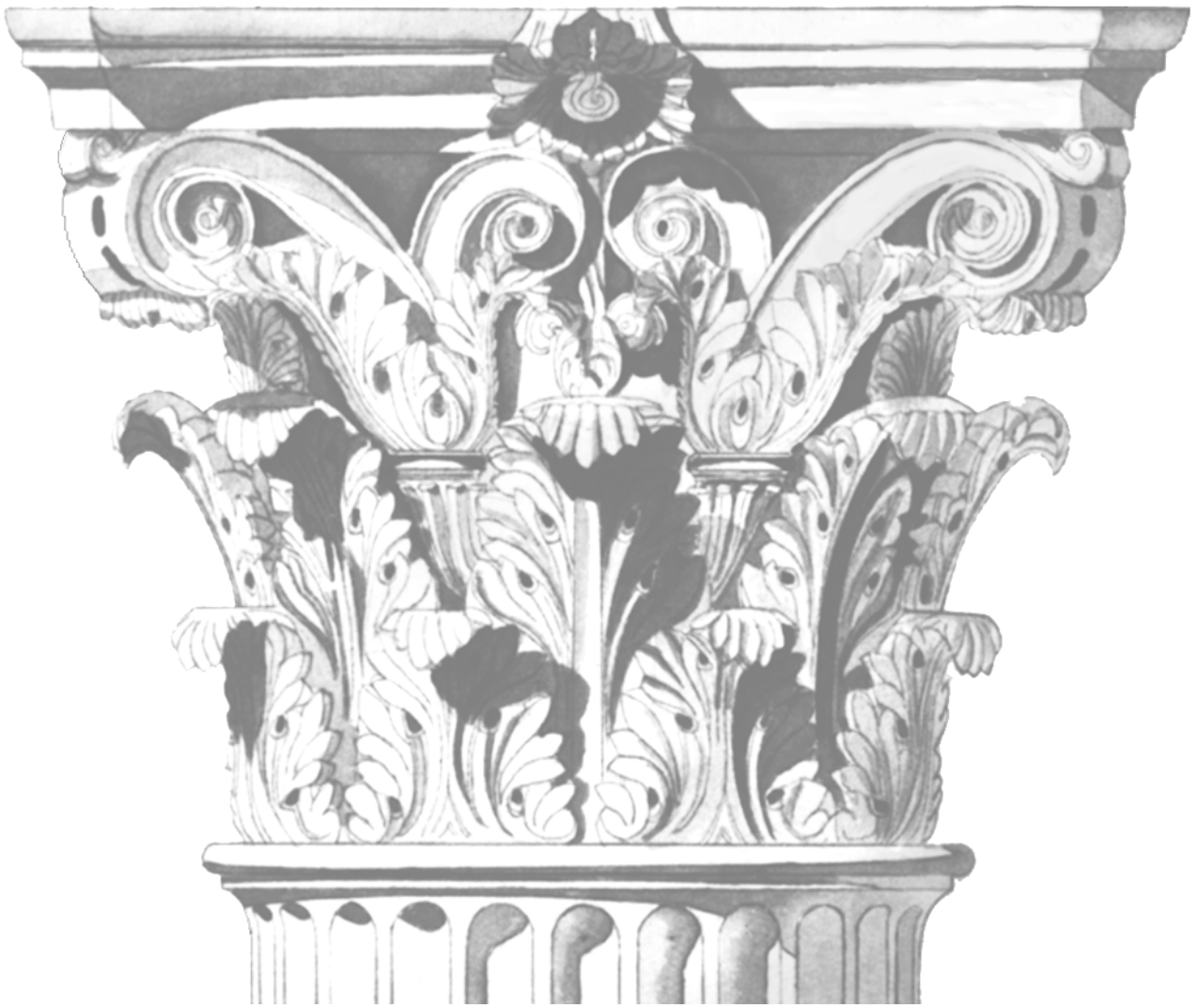

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Abstract

The aims of this study were to estimate the prevalence of autism in Northern Finland and to assess retrospectively the associations of autistic disorder with identified medical conditions and additional disabilities in this defined population of children and adolescents with autistic disorder. In order to find out the factors influencing the outcome, the methods of treatment/habilitation and the interventions used were studied in detail. The last aim was to elicit reliable information for decision-makers as well as ideas for giving support and, because of the presumed better outcome, saving resources in the long run.

The data were collected from hospital records and the records of the central institutions for the intellectually disabled in the Provinces of Oulu and Lapland in 1996–1997. The age-specific prevalences obtained in this study showed the prevalence to be lowest, i.e. 6.1 per 10 000, in the oldest age group of 15- to 18-year-old adolescents and highest, i.e. 20.7 per 10 000, in the age group of 5- to 7-year-old children, when the criteria of ICD-10 and DSM-IV were used. In this study, almost 50% of the autistic cases had a tested IQ above 70. Associated medical disorders or associated disorders of known or suspected genetic origin were diagnosed in 12.3%. Other associated medical disorders were epilepsy, hydrocephalus, fetal alcohol syndrome and cerebral palsy. Severe impairment of vision was evident in 3.7%. The most common therapies were physiotherapy and speech, occupational and music therapy. 43.9% of the children and adolescents with autism received specific training according to the TEACCH (Treatment and Education of Autistic and related Communication-Handicapped Children), 10.2% according to the Lovaas and 30.5% according to the Portage program. Antiepileptic medication had been prescribed to 23.9% and psychopharmacals to 14.9% of the individuals with autistic disorder (AD).

About three- to fourfold prevalence of AD in Northern Finland was found when compared to 16 years ago. Early, effective and regular interventions in autism have a good impact and should be provided as early as possible to children with autism. Based on the poorer prognosis of those without any early intervention, it can be anticipated that these methods will save resources in the long run.

Keywords: autism, autistic spectrum, comorbidity, outcome, prevalence, treatment

To my family

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Abbreviations

AD	Autistic Disorder
AS	Asperger Syndrome
ASD	Autism Spectrum Disorder
ADI-R	Autism Diagnostic Interview-Revised
ADOS	Autism Diagnostic Observation Schedule
CARS	Childhood Autism Rating Scale
CDD	Childhood Disintegrative Disorder
fMRI	functional Magnetic Resonance Imaging
HFA	High-Functioning Autism
IQ	Intelligence Quotient
DSM-III	Diagnostic and Statistical Manual of Mental Disorders. Third edition
DSM-III-R	Diagnostic and Statistical Manual of Mental Disorders. Third edition.Revised
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders. Fourth edition
ICD-8	International Classification of Diseases. Eighth edition
ICD-9	International Classification of Diseases. Ninth edition
ICD-10	International Classification of Diseases. Tenth edition
PDD	Pervasive Developmental Disorders
PET	Photon Emission Tomography
SPECT	Single Photon Emission Tomography
SSRI's	Selective Serotonin Reuptake Inhibitors
TEACCH	Treatment and Education of Autistic and related Communication-Handicapped Children
ToM	Theory of Mind
WHO	World Health Organization
WISC	Wechsler Intelligence Scale for Children

List of original papers

I Kielinen M, Linna S-L & Moilanen I (2000) Autism in Northern Finland. European Child & Adolescent Psychiatry 9:162–167.

II Kielinen M, Rantala H, Timonen E, Linna S-L & Moilanen I (2004) Associated medical disorders and disabilities in children with autistic disorder: a population-based study. Autism, The International Journal of Research and Practice 8:49–60.

III Kielinen M, Linna S-L & Moilanen I (2002) Some aspects of treatment of children and adolescents with autistic disorder in Northern-Finland. International Journal of Circumpolar Health 2:61–71.

IV Kielinen M, Hjelmquist E, Moilanen I & Syrjälä L. Intervention, treatment and care in autistic disorder. Case reports from Northern-Finland. International Journal of Circumpolar Health, In Press.

In addition, some unpublished data have been included in this thesis. The published original papers are reprinted by permission of Steinkopff Verlag, International Journal of Circumpolar Health and Sage Publications Ltd.

Contents

1 Introduction

Autism is a behaviourally defined syndrome manifested as impairment in social relatedness and communication and as repetitive routines and restricted interests (American Psychiatric Association, DSM-IV 1994; WHO, ICD-10 1993). Its prevalence is low, ranging from early estimates of 4–5/10 000 (Lotter 1966) to recent estimates of 16–17/10 000 (Chakrabarti & Fombonne 2001). The recognition of childhood autism and the availability of services have improved over time, which means that the higher prevalence rates also reflect the improved sensitivity of the case-finding methods. The differences in prevalence might be accounted for by methodological factors or regional and cultural differences rather than by true secular changes in the incidence of the disorder. The explanations for higher prevalence might include changes in diagnostic criteria, development of the concept of 'wide autistic spectrum', different research methods, growing awareness and knowledge among parents and professional workers and development of specialist services as well as the possibility of a true increase in numbers.

Autism does not have a single etiology. This could mean that a series of totally different diseases may present clinically through a common final pathway, so that they resemble each other and can be confused with each other (Gillberg & Coleman 2000). Autism could also be defined as Autism Spectrum Disorders (ASD), which in terms covers clinical variations of the same disease (Wing 1996). The manifestations of autism change over time and depending on other developmental impairments, personality and concurrent medical or mental health problems. A study by Folstein & Rutter (1977) and recent genetic studies have shown genetic factors to play a key role in the etiology of autism (Szatmari 2003). Diagnosable neurological conditions or signs of central nervous system dysfunction have been found in 9 to 37 per cent of cases in population studies of autistic disorders (Wing & Could 1979, Ritvo *et al.* 1990, Gillberg *et al.* 1991, Steffenburg 1991, Rutter *et al.* 1994, Gillberg 1995, Chakrabarti & Fombonne 2001). The rate of other associated disorders is also high, although co-morbidity estimates vary according to the IQ level, the strictness of the diagnostic criteria applied and the rigour with which medical investigations are conducted.

Children with autism are in need of special help and adjustment in everyday life, so as to minimise the risk of educational and social failure. The TEACCH, Lovaas and Portage

projects have developed methods of special treatment, care and habilitation for children and adolescents with autism (Schopler *et al.* 1983, Lovaas 1981, Schearer & Schearer 1972). Treatment at an early age is known to influence the outcome. Thus, care, treatment and habilitation at the age of 2 to 4 years are most effective and give the best influence on outcome (Lovaas 1987, Mesibov *et al.* 1989, Howlin 1998, Volkmar *et al.* 1999).

Intensive research has also opened up new perspectives into the understanding, care, treatment and habilitation of ASD. Psychopharmacology is one of the most rapidly advancing fields of medicine. Psychopharmacals are prescribed for many symptoms in children with autism, most commonly hyperactivity, aggressive and destructive behaviours, self-injury, stereotypies, obsessions, anxiety, depression and sleeping problems. In adolescence and adulthood, particularly individuals with high-functioning autism (HFA) and Asperger's syndrome (AS) may develop depression or obsessive-compulsive phenomena that interfere with functioning and can be relieved with medication. On the other hand, some individuals may have such overwhelming side-effects that medication is contraindicated for them.

Autism may occur together with any level of cognitive ability, from profound general learning disability to average or even superior cognitive skills in areas not directly affected by the basic impairments (Wing 1997). Therefore, each person's development will be individualistic, depending on the interaction between their abilities and disabilities, the environments they are in and the education and training they receive. However, the choice of therapeutic methods is very often determined by availability. Speech therapy, physiotherapy, occupational therapy, music therapy, holding therapy and riding therapy are examples of the wide range of possible choices.

2 Review of the literature

2.1 Definition

The term ‘autism’ was coined by Bleuler (1911) to describe a category of thought disorder present in schizophrenic syndromes. He saw egocentric thinking as a core symptom of schizophrenia, and derived the word autism from the Greek word “autos” which means “self” (Gillberg & Coleman 2000). After Leo Kanner’s description of autism in 1943, the climate of opinion, especially in the U.S.A. (Kanner 1943), was heavily influenced by psychoanalytical theory. The Austrian paediatrician Hans Asperger described AS in Europe in 1944 in a remarkably similar account at much the same time (Asperger 1944). He described what he considered to be an unusual personality variant in young children, most of whom were boys. He alluded to ‘autistic psychopathy’, but after an influential paper by Wing (1981), the particular combination of problems that Asperger described is now generally referred to as Asperger syndrome.

As a consequence of Leo Kanner’s description of autism and the psychoanalytic attitude in 1943 autism was regarded as an emotional disorder without any neurological basis. The treatment of AD was also influenced by the psychoanalytic theory. The tide began to turn in the 1960s. Parents in the USA and UK disclaimed the assumption that they were to be blamed for their children’s problems. Also, some researchers proved that autism was a disorder of the developing brain, mainly genetic in origin and a part of a wider spectrum of disorders (Folstein & Rutter 1977, Wing & Gould 1979, Wahlström *et al.* 1989, Volkmar 1996, Wing & Potter 2002, Szatmari 2003). The research on the neurological causes of autism and common knowledge of autism have helped to temper the guilt so often experienced by parents when the disorder was considered to be of psychogenic origin. Today, autism is regarded as a complex developmental syndrome manifesting in a heterogeneous group of individuals with similar symptoms but multiple biological etiologies (Coleman & Gillberg 2000, Szatmari 2003).

The prevalence surveys conducted in the 1960s and 1970s only dealt with AD (as opposed to ASD) and used a rather narrow definition of autism based on Kanner’s descriptions, including only subjects who were not mentally retarded (Kanner 1943, Wing & Potter 2002). Autism was not officially included as a diagnostic category until in

the third edition of the American Psychiatric Association's Diagnostic Statistical Manual of Mental Disorders (DSM III; APA 1980), where it was included in a new class of disorders; the Pervasive Developmental Disorders (PDD). Changes in the definition of autism were made in DSM-III-R (APA 1987). These guidelines included a larger number of criteria and a more developmental orientation; the requirement for the onset of autism in the first 3 years of life was not an essential diagnostic feature. Numerous alternatives in DSM-IV were conceptually the same as in ICD-10. The diagnostic categories that fall within the ICD-10 systems and were named by Wing (1996) as ASD, include autism or autistic disorder, atypical autism, Rett's syndrome, other childhood disintegrative disorder (CDD), AS, other PDD, unspecified PDD, and added by Wing (1996) semantic pragmatic disorder. According to the ICD and the DSM criteria, autism is a PDD (American Psychiatric Association, APA 1980, 1987, 1994, 2000; WHO 1993) characterised by disturbances in three major areas:

a) qualitative impairment in reciprocal social interaction
b) qualitative impairment in verbal and non-verbal communication and imaginative activity
c) markedly restricted repertoire of activities and interests.

Additionally information on early history and development of the individual are essential for the diagnosis of autism. The developmental course of linguistic and communicative abilities in children without major disabilities has its roots in infant pre-verbal communication, and even newborn babies demonstrate social intent and active interest in communicating (Murray & Trevarthen 1986). Newborn children are interested in other people's faces and have an ability for facial imitation. Children with autism have different imitation ability than typically developing children and this might show itself as early as around birth (Heimann *et al.* 1992). In many cases the behavioural problems are associated with communication skills. The absence of communicative speech at 5–6 years of age is indicative of a poor long-term overall outcome (Gillberg & Steffenburg 1987, Rutter *et al.* 1992). Because of their difficulties in social contact, autistic children are often thought to be deaf or blind, although their hearing and vision are normal (Wing & Gould 1979). Impairment of speech and communicational difficulties are one of the core symptoms and deficits in autistic disorder. Almost half of the persons with AD do not develop any spoken language at all, whereas the other half display various deviances in language, such as pragmatic deficiencies, echolalia and lexical idiosyncrasies (Wing 1989). It is also common in autism to have problems in understanding prosodic features, such as intonation, pitch and word stress, which influence the understanding of the content of communication. The dysfunction in language understanding and speech is likely to lead to specific impairment in social relationships and imaginative play (Frith 1989). However, some non-speaking children with autism may show speech development after the age of five and still have a fair overall outcome (Nordin & Gillberg 1998).

2.2 Differential diagnosis

According to the DSM IV differential diagnosis of AD (American Psychiatric Association, APA 1994, 2000) periods of developmental regression may be observed in normal development, but these are neither as severe or as prolonged as in AD. AD must be differentiated from other Pervasive Developmental Disorders. Rett's Disorder differs from AD in its characteristic sex ratio and pattern of deficits. Rett's Disorder has been diagnosed mainly in females, whereas AD occurs much more frequently in males. In Rett's Disorder, there is a characteristic pattern of head growth deceleration, loss of previously acquired purposeful hand skills, and the appearance of poorly coordinated gait or trunk movements. Particularly during the preschool years, individuals with Rett's Disorder may exhibit difficulties in social interaction similar to those observed in AD, but these tend to be transient. Gillberg & Coleman (2000) argue that Rett's Disorder and autism can co-exist, and that in such cases both diagnoses should be made.

AD differs from Childhood Disintegrative Disorder, which has a distinctive pattern of developmental regression following at least 2 years of normal development. CDD, or Heller's syndrome, which was first described as early as the early 20th century by Heller (Heller 1930), is very rare condition. In CDD, development is normal or near normal for at least the first two years. After this period of normal or near normal development, children with CDD gradually lose their speech, become aggressive or hyperactive and begin to show autistic behaviour. This condition hence differs from autism in the pattern of onset, course and outcome (Volkmar 1992). The etiology is unknown, but recent evidence suggests that it also arises as a result of some type of central nervous system pathology (Malhotra & Gupta 1999). However, ICD-10 and DSM-IV have included CDD as one particular variant of ASD.

In AD, developmental abnormalities are usually noted within the first year of life. When information on early development is unavailable or when it is not possible to document the required period of normal development, the diagnosis of Autistic Disorder should be made. Asperger's Disorder can be distinguished from AD by the lack of delay in language development. Asperger's disorder is not diagnosed if criteria are met for AD. A child with AS shows similar impairments from early childhood onwards as an autistic child, but exhibits clearly better verbal skills and social adaptation. AS refers to a disorder of early onset characterized by impairments in social interaction, difficulties in initiating and sustaining conversation, abnormalities of nonverbal communication and obsessive interest in obscure topics in the absence of a clinically significant delay in language and cognitive development (APA 1994, WHO 1993). The label 'semantic pragmatic disorder' was often used before the diagnosis of AS became common, and it is still used by some professionals, mainly speech and language therapists. Current research on AS suggests that there are few substantive differences, either in early history or in outcome, between HFA and AS (Wing 1981, Lord & Rutter 1994). However, there continues to be some debate as to whether the two conditions described, AD and AS, are quantitatively or qualitatively different (Schopler *et al.* 1998, Gillberg 1998). The disorders that include AS, atypical autism and CDD are often conceptualised as falling within the same spectrum as autism (Wing 1996, Szatmari 2003).

Schizophrenia with childhood onset usually develops after years of normal, or near normal, development. An additional diagnosis of Schizophrenia can be made if an individual with AD develops the characteristic features of Schizophrenia with active-phase symptoms of prominent delusions or hallucinations that last for at least 1 month. In Selective Mutism, the child usually exhibits appropriate communication skills in certain contexts and does not have the severe impairment in social interaction and the restricted patterns of behavior associated with Autistic Disorder, In Expressive Language Disorder and Mixed Receptive-Expressive Language Disorder, there is a language impairment, but it is not associated with the presence of a qualitative impairment in social interaction and restricted, repetitive, and stereotyped patterns of behaviour.

According to the DSM IV it is sometimes difficult to determine whether an additional diagnosis of AD is warranted in an individual with Mental Retardation especially if the Mental Retardation is Severe or Profound. An additional diagnosis of AD is reserved for those situations in which there are qualitative deficits in social and communicative skills and the specific behaviours characteristic of AD are present. Motor stereotypies are characteristic of AD; an additional diagnosis of Stereotypic Movement Disorder is not given when these are better accounted for as part of the presentation of AD (APA 1994, 2000).

2.3 Prevalence

The prevalence rate of ”classic” autism, also called infantile autism or Kanner’s autism, has been reported to range within 2–5 per 10 000, as determined based on the original narrow definition by Kanner (Fombonne *et al.* 1997, Lord & Rutter 1994, Lotter 1966, Wing & Gould 1979). When various definitions have been included, such as the less strict criteria of DSM-IV and ICD-10, the rates have ranged from 3.3 to 16.0 per 10,000 (Wing 1997).

There is still an ongoing debate regarding the diagnostic boundaries of autism and autism-like syndromes. The prevalence is much higher if the mildly autistic or Asperger type individuals are included, as AS has been reported to occur in 3–5 per 1000 children (Ehlers & Gillberg 1993, Gillberg *et al.* 1991, Wing 1997, Kadesjö *et al.* 1999, Magnusson & Sæmundsen 2001, Chakrabarti & Fombonne 2001). In more recent studies, AD has been reported to occur in 7–17 per 10 000 children (Bryson *et al.* 1988, Baron-Cohen *et al.* 1996, Kadesjö *et al.* 1999, Study I, Bertrand *et al.* 2001, Chakrabarti & Fombonne 2001), while the rate of ASD may approach 0.6 to 0.9 per cent of the population (Wing 1996, Yeargin-Allsopp *et al.* 2003). The population-based studies from 1996–2003 are presented in Table 1. The diagnostic procedures commonly used have become conceptually more uniform upon the adoption of the new criteria in the DSM-IV and ICD-10 diagnostic manuals (APA 1994, WHO 1993).

Table 1. Population-based studies of autism in 1966–2003

Author/country (year of publication)	Criteria	Prevalence	Population	Age range(yr.)
Prevalence studies from Europe from 1996–2001				
Lotter/UK (1966)	Kanner (1956)	4.5/10 000	780 000	8–10
Wing & Gould/UK (1979)	Kanner (1956)	4.9/10 000	46 500	3–17
Bohman *et al.*/Sweden (1983)	Rutter (1978)	6.1/10 000	69 600	3–20
McCarthy *et al.*/Ireland (1984)	Rutter (1978)	4.3/10 000	65 500	8–10
Gillberg/Sweden (1984)	Rutter-DSM III (APA1980)	4.0/10 000	128 600	4–18
Steffenburg & Gillberg/Swe (1986)	Rutter-DSM III -DSM III-R	7.5/10 000	780 400	4–18
Cialdella & Mamelle/France (1989)	Rutter-DSM III (APA 1980)	10.8/10 000	103 700	5–9
Gillberg *et al.*/Sweden (1991)	DSM-III-R(APA 1987)	11.6/10 000	78 100	4–13
Fitzgerald *et al.*/Ireland (1997)	DSM III and III-R, Wing (1987)	4.94/10 000	549 255	0–25
Baron-Cohen *et al.*/UK (1996)	ICD-10 (WHO 1990)	6.3/10 000	16 000	1.5
Fombonne *et al.*/France (1997)	ICD-10 (WHO 1990)	5.35/10 000	325 347	12–21
Kielinen *et al.*/Finland (2000)	ICD-10, DSM IV (APA 1994)	12.2/10 000	152 732	3–18
Kielinen *et al.*/Finland (2000)	Age group, 5–7 years (APA 1994)	20,7/10 000	16 000	5–7
Chakrabarti & Fombonne (2001)	ICD-10, DSM IV (WHO, APA 1994)	16,8/10 000	15 500	2,5–6,5
Prevalence studies from Asian from 1982–1996				
Hoshino *et al.*/Japan (1982)	Kanner (1956)	5.0/10 000	217 600	4–10
Tanoue/Japan (1989)	DSM III	13.8/10 000	95 400	6–12
Sagiyama & Abe/Japan (1989)	DSM III	13.0/10 000	12 300	1.5–3
Wignyosymarto *et al.*/Indonesia (1992)	CARS	11.7/10 000	5 100	4–7
Honda *et al.*/Japan (1996)	ICD-10(WHO 1990)	21.1/10 000	8 500	5
Prevalence studies from North America in 1982–2003				
Burd/Dakota (1987)	DSM III	3.3/10 000	181 000	2–18
Ritvo/U.S. (1989)	DSM III -DSM III-R (APA 1987)	3.6/10 000	184 800	8–12
Bryson *et al.*/Canada (1988)	DSM III-R	10.1/10 000	20 800	6–14
Yeargin-Allsopp *et al.* (2003)	DSM IV (APA 1994)	34.0/10 000	289 000	3–10

2.4 Genetic and environmental factors

An important advance in changing our understanding of the cause of autism was the discovery that genetic factors have a key role. In 1977, Folstein and Rutter published the first twin study in autism and showed that the concordance rate was very much higher in identical twins than in non-identical twins.

Molecular genetics has begun to contribute to our understanding of autism. Besides the apparently monogenetic disorders with a variety of mutations, molecular techniques have uncovered genetic patterns other than classical Mendelian genetics. Autism is a strongly genetic, yet strikingly complex disorder, and evidence from different cases supports the presence of chromosomal disorders, rare single gene mutations and multiplicative effects of common gene variants (Gillberg & Coleman 2000).

In particular, recent studies in families with autism spectrum disorder have identified uncommon occurrences of a novel genetic syndrome caused by disruptions of the NLGN4 gene on chromosome Xp22. Previous work has identified another uncommon syndrome caused by maternal duplications of the chromosome 15q11-13 region (Veenstra-VanderWeele & Cook 2004). Auranen *et al.* (2002) analysed in Finland the two chromosomal regions on Iq and 3q showing the highest lod scores in our genome-wide scan as well as the AUTS1 locus on chromosome 7q. For markers on 3q25-27, a more significant association was observed in families from a sub-isolate compared to families from the rest of Finland. In contrast, no clear evidence for association on the AUTS1 locus was obtained. The wide interval showing association particularly on chromosome 3q suggests a locus for autism spectrum of disorders in this chromosomal region in this previous study from Finland.

Rett's syndrome (RS) has been shown to be caused by mutations in MECP2, located at Xq28, which encodes the methyl CpG-binding protein 2 (Amir *et al.* 1999). However, this mutation has not been found in all RS cases. Male cases are extremely rare, and there is a hypothesis that RS is likely to be X-linked, and that mutations are lethal in hemizygous males (Schannen *et al.* 1998).

Genetic data do not exclude environmental risk factors, as long as it is understood that 'environmental' in this context can include any event after fertilisation (Szatmari 2003). The only environmental factors for which we have preliminary evidence of such causation are thalidomide, which causes embryopathy (Folstein & Rutter 1977, Stromland *et al.* 1994), and anticonvulsants taken during pregnancy (Bailey *et al.* 1995). An association between autistic spectrum problems and fetal alcohol syndrome was also reported earlier in a Swedish follow-up study, where two out of 24 fetal alcohol syndrome children had autistic spectrum disorders (Aronsson *et al.* 1997). Various environmental causes for a genuine rise in incidence have also been suggested, including the triple vaccine for measles, mumps and rubella (MMR) (Wing & Potter 2002). In spite of recent publicity, there is good epidemiological evidence to show that these vaccines are not a risk factor for autism (Taylor *et al.* 2002). Even deprivation seems to have some etiological role in autistic-like conditions, as shown by a recent study of infants raised in Romanian orphanages: autistic-like patterns of behaviour have been noted in a small percentage of these truly abandoned children (Rutter *et al.* 1999).

2.5 Neurological and neuropsyhological findings

2.5.1 Neurological findings

Autism, in most cases, appears to be due to neurodevelopmental pathology of the central nervous system – a cascade of neurodevelopmental abnormalities that lead to neural misconnection, which leads, in turn, to impaired behaviours (Gillberg & Coleman, 2000). Neurobiological early warning signs for autism have not been discovered yet (Courchense *et al.* 2003). Knowledge of such signs could lead to objective, quantifiable and reliable clinical tests for autism, earlier identification and intervention and, eventually, insight into the original causes and/or mechanisms present at the earliest stages of the disorder. The disease process usually begins in utero, although occasional cases occur postpartum (Gillberg & Coleman 2000).

Postmortem examinations and magnetic resonance imaging have revealed larger volumes of white matter in general and subtle structural changes in cell density and alignment, particularly in the limbic system (Casanova *et al.* 2002).

The clinical onset of autism appears to be preceded by 2 phases of brain growth abnormality: a reduced head size at birth and a sudden and excessive increase in head size between 1 to 2 months and 6 to 14 months (Courchense *et al.* 2003). Existing brain imaging data suggest that the amygdala and its collaborating cortical systems, mainly the prefrontal and temporal cortices, are involved in the pathobiology of ASD (Baron-Cohen *et al.* 1999, Schultz *et al.* 2000, Critsley *et al.* 2000). Chritsley *et al.* (2000) found that individuals with autistic disorder differed significantly from controls in the activity of the cerebellar, mesolimbic and temporal lobe cortical regions when processing facial expressions, which may account for some of the abnormalities in social behaviour associated with autism. Recently, Nieminen-von Wendt *et al.* (2003) reported support for this notion when they found no indication of activation of the left amygdala during ToM tasks. There was no evidence for AS being linked to right hemisphere dysfunction. Instead, Nieminen-von Wendt *et al.* (2003) emphasised the role of the cerebellum in the pathogenesis of ASD.

2.5.2 Neuropsychology and theory of mind

The neuropsychological profile in autism is not consistent with that seen in mental retardation or in any other general deficit syndrome (Gillberg & Coleman 2000). Rather, it appears to involve selective impairment in complex information processing, which does not usually affect visuospatial processing (Baron-Cohen *et al.* 2000). There is now a considerable amount of literature on autism documenting the following issues: (1) mentalizing or theory of mind (ToM), 2) drive for central coherence and (3) executive functions (Frith 1989, Happé 1994, Ozonof & Strayer 1997, Baron-Cohen *et al.* 2000). Of these, ToM has been best able to explain the deficits in social communication (Baron-Cohen *et al.* 1985). The ToM construct covers the mental capacities that enable a person

to understand, explain and predict psychological states of others and self. There is a substantial body of literature showing that problems in ToM constitute a consistent part of ASD (Baron-Cohen *et al.* 2000). During the 1980s, the new hypothesis about the basic dysfunctions in autism soon began to dominate the way clinicians and researchers conceptualised the core autistic dysfunction (Baron-Cohen *et al.* 1985). Normal children acquire ToM skills during the first 3–4 years of life and process non-verbal social cues implicitly, unless their circumstances are exceptional. Many of the social impairments in people with autism are consistent with a deficit in ToM and/or differences in the processing of other people's emotions (Baron-Cohen *et al.* 1994). By the year 1990s, it was taken almost for granted that ToM deficit was at the root of autism and accounted for most of the variance in clinical presentation.

Happe *et al.* (1996) found in a PET scan study that AS volunteers scored worse than normal volunteers when performing ToM stories, and that they, contrary to the controls, did not show any significant task-related activity in the left medial prefrontal cortex (Brodmann's area 8/9) when processing ToM stories. Instead, significant activation was observed in the adjacent areas of the medial prefrontal cortex (Brodmann's area 9/10). However, it soon became evident that ToM deficits are not specific for all individuals with autism, nor can they explain all of the clinical and neuropsychological problems in ASD (Mayes *et al.* 1996, Charman & Campebell 1997, Klin 2000). Although not all people with autism have ToM deficits, the theory has helped both parents and professionals to understand the different way of thinking in the autism spectrum. ToM is deficient in many individuals with autism, and its relation to other neuropsychological impairments – including executive dysfunction and weak central coherence – remains to be established (Dahlgren 2002).

AD shows a fairly characteristic pattern on neuropsychological tests, such as the Wechsler scales (Frith 1989), the Tower of Hanoi or Tower of London tests and other assessments of executive functions (Ozonow & Miller 1995). Individuals with autism often fail on tasks measuring the drive for central coherence – such as elaborate versions of the intelligence tests, e.g. the block design subtest (Happé 1996).

Although high-functioning individuals with ASD are of normal intelligence, they have life-long abnormalities in social communication and emotional behaviour. However, the biological basis of social difficulties in autism is poorly understood. According to Critchley *et al.* (2000), individuals with AD participating in a fMRI study did not activate the cortical `face area' when explicitly appraising expressions or the left amygdala region and the left cerebellum when implicitly processing emotional facial expressions. High-functioning adults with AD, compared with controls, have abnormalities in regional brain activity during explicit and implicit processing of emotional facial expressions, indicating dysfunction of the pathways between the limbic and paralimbic regions, the cerebellum and the extrastriate visual cortices. These findings may partly explain the social impairments of people with AD (Critchley *et al.* (2000). This may account for some of the abnormalities in social behaviour associated with autism. Facial expressions of motion are important and culturally universal social signals (Ekman *et al.* 1998) and can be processed both explicitly (consciously) and implicitly (unconsciously). The rules and skills guiding normal social interactions are complex, but include the appreciation and understanding of other people's thoughts and intentions (ToM) and the explicit and implicit processing of emotional expression.

Prosopagnosia, or face blindness, has been reported in case reports of individuals with ASD (Duchaine *et al.* 2003, Pietz *et al.* 2003), indicating that the deficient perception of facial emotions may be an important pathogenic symptom of autistic behaviour (Kracke 1994, De Sonneville *et al.* 2002). In a study of Duchaine *et al.* (2003), the developmental processes that resemble the procedures used for face recognition in AS are separate from the procedures used for place recognition, for example. The overall prevalence of face blindness in AD is not known.

2.6 Unusual sensory responses

Idiosyncratic responses to sensory stimuli and unusual motor patterns have been reported clinically in young children with autism (Baranek 2002). Unusual sensory responses (such as hypo- and hyper-responses, preoccupations with the sensory features of objects, perceptual distortions, paradoxical responses to sensory stimuli, i.e. touch, pain, heat, cold, sound and light), have been reported in 42 to 88 percent of children with autism (Kientz & Dunn 1997, Baranek 2002). Of these symptoms, abnormal responses to sounds are the most characteristic. Many children with AD cover their ears to shut out even normal noise levels. Jansson-Verkasalo *et al.* (2003) demonstrated abnormalities, in a study of auditory event-related potentials (mismatch negativity), in transient sound-feature encoding and in sound discrimination. These results indicate that auditory sensory processing is deficient in children with ASD**.** Abnormal responses to visual stimuli are probably present in a large majority of young children with autism, who often give the impression of having difficulty to recognize the things they see. In a recent study, toddlers with autism were more abnormal in their responses to taste and smell than toddlers with other developmental disorders (Rogers *et al.* 2003). Sensory processing abilities also appear to be uneven and variable in autism, so that both hyper- and hypo-responses are evident in the same child (Gillberg & Billstedt 2000). Many authors would consider a fifth criterion important for a definitive diagnosis to be made, namely abnormal responses to sensory stimuli (Gillberg & Coleman 2000).

2.7 Associated medical disorders and comorbidities

Associated medical disorders are sometimes called associated medical conditions or double syndromes. Comorbidity with AD and another syndrome makes the diagnosis of each individual much more complicated. The findings highlight the need to rely on multiple sources of ascertainment in epidemiological studies of AD and caution against findings based on single databases. Diagnosable neurological conditions or signs of central nervous system dysfunction are found in 9 to 37 per cent in population studies of autistic disorder (Wing & Could 1979, Olsson *et al.* 1988, Ritvo *et al.* 1990, Gillberg *et al.*, 1991, Steffenburg 1991, Rutter *et al.* 1994, Gillberg 1995, Chakrabarti & Fombonne 2001). The rate of other associated disorders is also high, although co-morbidity estimates vary according to IQ level, depending on how strictly the diagnostic criteria for autism are applied and the rigour with which medical investigations are conducted.

According to Gillberg & Coleman (2000), one factor could be the mix of disease entities within any given population, which is known to vary from country to country, as determined by infant screening programmes to detect rare diseases.

Many other conditions have been reported in association with autistic disorder (eg. Wing 1989, Rutter *et al.* 1994, Gillberg & Coleman 2000, Chakrabarti & Fombonne 2001). These include neurofibromatosis, congenital rubella, hydrocephalus, impairments of ambulation, foetal alcohol syndrome and visual and hearing impairments. Developmental delay, epilepsy, dysmorphic features, obstetric complications, an unequal sex ratio and extremes of head size represent non-specific signs showing that autism is a neuropsychiatric disorder (Chakrabarti & Fombonne 2001, Szatmari 2003). Organic aetiologies include prenatal insults, such as phenylketonuria, anticonvulsants taken during pregnancy, localised lesions as in tuberous sclerosis and postnatal infections, such as encephalitis (Gillberg & Coleman 2000, Szatmari 2003). However, estimates of the frequency of such problems and conclusions about the nature of the association have differed from one research group to another.

Epilepsy occurs more commonly than usual in autism and was one of the early indications that autism is a neurobiological disorder rather than one caused by parental behaviour. The incidence figures of epilepsy range from 18 to 29 per cent (Olsson *et al.* 1988, Volkmar & Nelson 1990, Nordin & Gillberg 1998, Rapin & Katzman 1998). In follow-up studies of adolescents and adults with autism, one third developed epilepsy before the age of 30 years (Gillberg & Steffenburg 1987, Olsson *et al.* 1988, Gillberg *et al.* 1991, Tuchman *et al.* 1991). The risk of seizures is high in individuals who are severely mentally retarded, although epilepsy is also common among those with normal IQ (Gillberg *et al.* 1987) Epileptic seizures are most likely to occur in infancy, with a second peak in early adolescence (Sidenvall *et al.* 1993). Autism may follow infantile spasms or the manifestation of Lennox-Gastaut syndrome in early childhood, but all types of seizures may occur in autism and also later in adolescence (Rapin & Katzman 1998). Individuals with autism are more likely to have seizure disorder than most categories of individuals with mental retardation (Gillberg & Coleman 2000).

For example, although no children with cerebral palsy and autistic disorder were found in the 1980´s in the study by Gillberg *et al.* (1991). Fombonne *et al.* (1997) reported the rate of 2.9% for cerebral palsy among autistic children, even higher than the rate of 2.1/1000 founded by Pharoah *et al.* (1998).

Until recently the association with Down syndrome has been considered relatively rare with only few cases of Down syndrome and autistic disorder (Bregman & Volkmar 1988, Howlin *et al.* 1995, Fombonne *et al.* 1997, Ghaziuddin 1997), but in a recent UK study, the minimum prevalence rate of autistic disorder in Down syndrome was 7% (Kent *et al.* 1999).

There are a few single case studies in the literature of autism in connection with the XYY and Klinefelter syndromes (Gillberg *et al.* 1984). Such syndromes do not necessarily cause autism but might give rise to autistic-like behaviour.

The genetic condition of greatest potential causal significance for autism is tuberous sclerosis which occurs in 6% of subjects with autistic disorder (Fombonne *et al.* 1997), far higher than the occurrence of 1/10 000 in the general population (Shepherd *et al.* 1991, Ahlsen *et al.* 1994, Smalley 1998). Early screening studies of autistic individuals suggested that up to one quarter of the cases could be associated with the fragile X

syndrome. However, recent studies indicate that the predominant behavioural phenotype of the fragile X syndrome is distinct from autism as usually defined, and that a variety of methodological factors contribute to the variability of the prevalence estimates (Bailey *et al.* 1993, Gillberg & Coleman 1996).

Visual and hearing deficits are common in autism, while actual blindness and deafness are much rarer (Steffenburg 1991). Steffenburg (1991) reported 24% of children with ASD to have hearing deficits of more than 25dB. Co-occurrence of full deafness and infantile autism has been reported by Ritvo *et al.* (1990) and Fombonne *et al.* (1997). Ocular problems that affect vision are often found in children with AD. In a population-based survey by Steffenburg (1991), 50% of children with ASD had refraction error, mostly hypermetropia, but myopia and astigmatism were also found. A small percentage of totally blind children also have AD (Fombonne *et al.* 1997).

High-functioning individuals with ASD are at an enhanced risk for psychiatric disorders (such as depression and obsessive compulsive disorder) (Fombonne 1999, Ghaziuddin *et al.* 2002). In Wing's series of 34 adults with AS, the most common psychiatric diagnosis was depression, which occurred in at least 10 subjects (about 30%) (Wing 1981). Similar rates were reported by Ghaziuddin *et al.* (1998), who surveyed the occurrence of psychiatric disorders in a series of patients with AS. Twenty-three out of 35 individuals (65%) had an additional psychiatric disorder at the time of evaluation or during the 2-year follow-up. Children were most likely to suffer from attention deficit hyperactivity disorder, while depression was the most common diagnosis in adolescents and adults (Ghaziuddin *et al.* 1998). In a Canadian study, 17% of HFA children were more than two standard deviations above the population mean for attention deficit hyperactivity disorder and depression, and 13.5% were similarly rated for anxiety (Baird *et al.* 2003). Other problems of motor coordination, sleeping, eating and elimination are also common in autism (Gillberg & Billstedt 2000, Tani *et al.* 2003).

Learning difficulties occur in about 75% of children with autism (Howlin 1998, Jordan 1999, Baird *et al.* 2003). Associated developmental comorbidities include specific learning difficulties of attention, processing speed, working memory and other tasks often thought of as executive skills. They are often impaired in ASD and contribute to the common observation of a gap between structured test results, where no or few problems are shown, and daily functioning in real life, where marked impairment is evident. Learning disabilities in general are a risk factor for behavioural problems; 41% of children with mild to moderate or severe learning difficulties have severe emotional behavioural disturbance.

2.8 Care, treatment and habilitation

Although it is apparent that no specific treatment has proved superior to all others, research into psychological or educational interventions more generally has highlighted what seem to be the important components of any programme for children with autism (Howlin 1998, Jordan 1999, Lord 2000, Schreibman 2000, Bryson *et al.* 2003). The approaches that have tended to offer most are those that:

- account for the characteristic behavioural patterns of children with autism in developing interventional approaches.
- emphasise the development of skills instead of only focusing on deficits.
- employ a structured, behaviourally based approach to intervention.
- utilise functional analysis to understand behavioural problems.
- focus on the development of communication skills, both verbal and non-verbal.
- modify the environmental setting in order to enhance communication and understanding, reduce stress and facilitate learning.
- use naturally occurring opportunities for teaching and reinforcement.
- involve the parents in therapy or intervention and use their knowledge of the child.
- foster integration with typically developing peers.

Especially behaviour-based interventions have proven their efficacy in the treatment of this syndrome and similar disorders. The recognition that autism is not one disease but a syndrome can only enhance the understanding of etiologies and the prevention and treatment of this puzzling disorder. Only a small proportion of individuals with AD are able to live independent adult lives, while individuals with HFA or AS generally improve enough to live independently in adulthood (Nordin & Gillberg 1998). Behavioural thinking has guided our understanding of the antecedents and consequences of autistic behaviour. Behavioural treatment may produce long-lasting and significant gains for many young children with autism (Lovaas 1993). The TEACCH, Lovaas and Portage projects have developed special treatment and habilitation principles suitable for children and adolescents with autism (Lovaas 1987, Ozonoff & Miller 1998, Sturmey *et al.* 1992, Venter *et al.* 1992).

In Finland, the tide began to turn in the early 1980s, and the first set of behavioural training principles for individuals with AD was the Portage program. Other specialised interventions, such as TEACCH and Lovaas, were adopted into use in Northern Finland in the early 1990's. Personnel in day care centres, at schools, in hospitals and in institutions as well as parents were trained to use these interventions in everyday life. The parental organisation for autism in Finland also had an important role in launching intervention and habilitation methods for autistic children.

No standard pharmacological treatment is prescribed in autism, as the target symptoms determine the choice and length of psychopharmacological treatment. The medication given to individuals with AD is not always followed systematically, and the results do not indicate superior usefulness of any special medication in autism itself (Study III). Campbell *et al.* (1996) conclude that *neuroleptics*, such as haloperidol, *β-blockers*, such as naltrexone and clonidine, *antidepressants*, such as fluvoximine, nonselective tricyclic clomipramine, and selective serotonin reuptake inhibitors (SSRI's), such as fluoxetine, have been appropriately investigated (Tsai 1999). Recently, there has also been some interest in the research results on *atypical neuroleptics,* e.g. risperidone, which may be effective in the treatment of explosive, aggressive and disruptive behaviours in autism (Horrican & Barnhill 1997, Tsai 1999). *Mood stabilizers,* such as lithium, have sometimes effectively control the manic disorder or mood swings in adults with autism (Tsai 1999). Depression is common in children and adolescents with AD (Ghaziuddin & Greden 1998, Scifo *et al.* 1996). The minimal use of SSRI's in Finland might have been due to the late advent of these drugs only in 1989 and their restricted use for children in

prescriptions. The use of stimulants is increasing today in Finland, also for the combination of AD, hyperactivity and attention deficit (Social Insurance Institution of Finland and National agency for medicine 2003).

Speech therapy and the linguistic approach are common in the treatment and habilitation of ASD. Training studies by using computers have also shown promising effects on autistic children's communication, reading and language skills (Heimann *et al.* 1995).

In the follow-up studies from the 70's to the early 90's only 5–15% of cases with AD had a good outcome, with near normal or normal social life and acceptable functioning at work or school despite certain difficulties in social relationships and oddities in behaviour (Lotter 1974, Gillberg & Steffenburgh 1987, Venter *et al.* 1992, von Knorring & Hägglöf 1993).

3 Aims of the study

The purpose of the present study was to investigate the prevalence of autistic disorder and other identified conditions associated with autism and to find out the present status of care, treatment and habilitation of autism in childhood and adolescence in Northern Finland.

The numerals I–IV hereafter refer to the original publications. The specific aims of this study were:

1. to estimate the prevalence of autism in the two northernmost provinces of Finland, the Provinces of Oulu and Lapland, and to test for a secular change in its incidence.
2. to assess retrospectively the association of autistic disorder with identified medical conditions and additional disabilities (other than mental retardation) in this defined population of children and adolescents with autistic disorder.
3. to describe and find out the present status of treatment/habilitation and especially the early interventions used in Northern Finland.
4. to find out the problems and challenges influencing the outcome in the intervention, treatment and care of AD and to elicit reliable information for decision-makers as well as ideas for giving support and, because of the presumed better outcome, saving resources in the long run.

There are no recently published studies where prevalence, care, treatment, habilitation and outcome would have been assessed and followed up in this geographical area.

4 Material and methods

4.1 Study group

The study was carried out in the Provinces of Oulu and Lapland with a total area of 149,925 km². The population on December 31, 1996 was 653,464, representing 12.7% of the total population of Finland (Statistics Finland, 1997). The population consists of Finnish-, Swedish- and Saami-speaking people. The proportion of males was 50.1% and that of females 49.9%. The number of children born in 1979–1994, representing the age group of 3- to 18-year-olds on the census day, December 31, 1996 was 152,732. Males accounted for 49.8% and females for 50.2% of this population. The selected individuals fulfilled the criteria of autism according to ICD-10 and DSM-IV, and they were resident in the Provinces of Oulu and Lapland.

The data were collected from hospital records and the records of the central institutions for the intellectually disabled in the Provinces of Oulu and Lapland in 1996–1997. The selected cases had, at some point in their lifetime, used communal health services and, according to the hospital records, had been diagnosed for AD or ASD. Each child with AD had been evaluated and assigned a diagnosis of autism based on the clinical judgement by a panel consisting of a child psychiatrist or a child neurologist, a psychologist, a speech therapist and a physiotherapist. The final diagnoses had been made by child psychiatrists or child neurologists.

The children included in this study had been diagnosed according to different classification systems.

I ICD-8 from 1969 to 1986 (WHO, 1980).
II ICD-9 (WHO, 1987) from 1987 to 1995, similar to DSM-III-R (APA, 1987).
III ICD-10 (WHO, 1993) from 1996 up to present, similar to DSM-IV (APA, 1994).

4.2 Procedure

It was necessary to re-review the children's and adolescents' health reports by using a single classification system, i.e. ICD-10 and DSM-IV (APA 1994). The raters (S-LL, MK) determined whether the criteria for the various diagnostic systems were met in each particular case. Children meeting the full criteria for autistic disorder (AD) according to DSM-IV and childhood autism according to ICD-10 were diagnosed as having autistic disorder. AD was defined in accordance with DSM-IV, i.e. all children had six or more of the 12 symptoms, at least two of which were in the social, one in the communication and one in the behavioural domain and had been diagnosed before the age of three years. The original sample thus consisted of the 187 children and adolescents with autistic disorder. The diagnostic criteria for autistic disorder according to DSM-IV (APA, 1994), similar to ICD-10 (WHO, 1993), are presented in Table 2.

Table 2. Diagnostic criteria for autistic disorder according to DSM-IV similar to ICD-10

Class	Description
A	A A total of six (or more items from 1,2 and 3 with at least two from 1 and one each from 2 and 3:
1.	Qualitative impairment of social interaction, as manifested by at least two of the following:
a)	marked impairment in the use of multiple non-verbal behaviours, such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
b)	failure to develop peer relationships appropriate to developmental level
c)	a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g. by a lack of showing , bringing, or pointing out objects of interest)
d)	lack of social or emotional reciprocity
2.	Qualitative impairment in communication as manifested by at least one of the following:
a)	delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mine)
b)	in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
c)	stereotyped and repetitive use of language or idiosyncratic language
d)	lack of varied, spontaneous make-believe play, or social imitative play, appropriate to developmental level
3.	Restricted, repetitive and stereotyped patterns of behaviour, interests and activities, as manifested by at least one of the following:
a)	encompassing preoccupation with one or more stereotyped and restricted patterns of interest, which is abnormal either in intensity or focus
b)	apparently inflexible adherence to specific, non-functional routines or rituals
c)	stereotyped and repetitive motor mannerisms (e.g. hand or finger-flapping or twisting, or complex whole-body movements)
d)	persistent occupation with parts of objects
B	Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years:
1.	Social interaction.
2.	Language used in social communication.
2.	Symbolic or imaginative play
C	The disturbance is not better accounted for by Rett's disorder or childhood disintegrative disorder.

In addition, nine children and adolescents with AD out of 187 (4.8%) were evaluated in more detail. These children's hospital records and all available information were re-evaluated through pregnancy to adolescence. Eight of these nine children and adolescents have been further followed by the author (MK) after the follow-up period, as in 1999 a new project was launched in Northern Finland to support families, professionals and persons with ASD (Study IV).

No children with AS were included in this study, because the ICD-10 classification only included the diagnosis of AS after 1.1.1996. Up till then, the children with these problems had been diagnosed for autistic-like conditions, minimal brain dysfunction, emotional disorder, affective disorder, borderline disorder, obsessive-compulsive disorder, mutism or anorexia nervosa. At the time of data collection at the beginning of 1997, only a few children with AS had been diagnosed. Additionally, neither Rett's syndrome nor CDD were included because they are specific entities of their own.

The level of intellectual functioning was defined based on the intelligence quotient (IQ or IQ-equivalent) obtained with one or more standardised, individually administered intelligence tests. Intellectual functioning had been assessed with the Griffiths Developmental Scale II (Griffiths, 1980) or the Wechsler Intelligence Scale for Children (WISC, Wechsler, 1974, 1992) by clinical psychologists. Childhood Autism Rating Scale (CARS) had been used to classify the severity of autism, and it was here also used as an outcome measure (Schopler *et al.* 1988). If not previously available, CARS analyses were made for the purposes of the present study based on all available information. The CARS classification was divided into three grades: the scores were less than 30 points for no or some autistic features, 30–36 for mild or moderate autism and > 37 for severe autism. Furthermore, the authors estimated in general, based on all the available data, the overall usefulness of the treatment of each autistic person. As the criteria of autism diagnosis in CARS presume at least mild/moderate autism (≥ 30), no cases with autistic features (< 30) were included in the initial CARS assessment. The outcome of the subjects was analysed in relation to intelligence and the methods of treatment/habilitation, such as TEACCH, Lovaas and Portage.

The pharmacotherapy affecting the central nervous system was classified into five subgroups: neuroleptics, antidepressants, sedative-hypnotics, mood stabilisers and anticonvulsants (AACAP 1999, Bazire 2000).

4.3 Statistical methods

Poisson's distribution for approximating the 5% confidence limits was used in the prevalence calculations. Statistical significances were calculated by using the Chi-Square test. Hypothesis testing with p-values does not necessarily give information about the actual differences observed, and it depends to a large extent on the size of the study (Gardner & Altman 1989, Campbell & Machin 1999). Thus, a small-scale study like this may fail to reveal important differences. All statistical analyses were performed with SPSS for Windows, release 7.5.1.

4.4 Ethical considerations

The research design was approved by the ethical committee of the Faculty of Medicine of the University of Oulu. The project was also approved by the Ministry of Health and Social Affairs. Confidentiality of information was ensured by giving each participant a code under which his/her data was entered, which made it impossible to recognise single persons.

5 Results

5.1 Prevalence of autism in Northern Finland

The prevalence (± 5% CI) of "classic" autism or Kanner's autism in the area was 5.6 (4.4–6.8)/10 000, being 8.2 (6.6–10.8)/10 000 in boys and 2.5 (1.5–3.7)/10 000 in girls. When the criteria of ICD-10 and DSM-IV were used, the age-specific prevalence was lowest, 6.1 (3.7–8.6) per 10 000, in the oldest age group of 15- to 18-year-old children and highest, 20.7 (15.3–26.0) per 10 000, in the age group of 5–7 years, The degree of autism, as assessed by CARS, was mild in 8.5% of the cases, moderate in 58.5 and severe in 33.0%. Half of all autistic persons (50.2%) had intelligence scores indicating intellectual disability (IQ < 70), while more than one fourth (26.6%) had normal or even superior cognitive abilities (IQ > 80).

5.2 Associated medical disorders

Associated disorders of known or suspected genetic origin were demonstrated in 23 (12.3%) children and adolescents (Table 3). All nine children with autosomal disorder and the child with 47, XYY had IQ under 70. Of single-gene disorders, all the four children with fragile X syndrome had IQ under 70, while the child with tuberous sclerosis or mitochondriopathy had IQ over 70. Two of the six children with suspected genetic abnormality had IQ over 70. Epilepsy was present in 34 (18.2%), cerebral palsy in 8 (4.3%) and other associated neurological disorders in 14 (7.5%) individuals (out of 187) (Table 4). Nineteen individuals had at least two associated medical conditions.

Table 3. Associated disorders of known or suspected genetic origin in 187 children and adolescents with AD.

Genetic background	N	%
Chromosomal disorders		
Autosomal disorders		
– Down syndrome	7	3.7
– Chromosome 46,XX,dup(8)(p)	1	0.5
– Chromosome 17-deletion	1	0.5
Sex chromosome disorders		
– 47, XXY (Klinefelter syndrome)	1	0.5
– 47, XYY	1	0.5
Single gene disorders		
Autosomal dominant		
– Tuberous sclerosis	1	0.5
– Mitochondriopathia	1	0.5
X-linked recessive		
– Fragile X syndrome	4	2.1
Suspected genetic abnormality NUD	6	3.2
Total	23	12.3

Table 4. Associated neurological disorders in 187 children and adolescents with AD.

Genetic background	N	%	Proportion of IQ > 70
Epilepsy	34	18,2	11 (34.4)
partial	10	5.4	4 (40.0)
primarily	12	6.4	1 (8.3)
infantile spasms	7	3.7	4 (57.1)
Lennox-Gastaut syndrome	2	1.1	0 (0.0)
not classifiable	3	1.6	2 (66.7)
Cerebral palsy	8	4.3	4 (50.0)
diplegia	4	2.1	2 (50.0)
triplegia	2	1.1	0 (0.0)
tetraplegia	2	1.1	0 (0.0)
Other	1	7.5	3 (21.4)
Hydrocephalus	1	3.2	1 (16.7)
Fetal alcoholic syndrome	2	1.1	1 (50.0)
Sotos syndrome	1	0.5	1 (100.0)
Neonatal meningitis/encephalitis	5	2.7	0 (0.0)

Thirty-four (18.2%) of the children had seizures. Forty-three (23%) children had impairment of vision in the clinical examination. Seven of the children (3.7%) were blind, and two of them were known to have been blind since birth. Impairment of hearing was detected in sixteen (8.6%) individuals. Fifty-eight (31%) children had had three to

five episodes of prolonged otitis media before the age of three years. Mild impairment of hearing was diagnosed in 13 (7.0%) children and adolescents and severe impairment in 3 (1.6%) individuals. Impairment of ambulation was present in 25 (13.4%) individuals. Eight individuals (4.3%) had severely or moderately impaired mobility. Because of the impairment of speech and language, one hundred and fifty-seven (84.0%) individuals received speech therapy (Table 5 and Figure 1). The severity of additional disabilities is presented in Table 5.

Table 5. Additional disabilities

Disabilities		N (%)
Seizures:		
mild	seizures < 1/month	24 (12.8)
moderate	seizures < 1/week	4 (2.1)
severe	seizures > 1/week despite medication	6 (3.2)
Impairment of vision:		
mild	correctable to > 0.1/< 1	36 (19.3)
severe	not correctable to > 0.1, blind;	7 (3.7)
Impairment of hearing:		
mild	audiometrically > 30 dB/< 60 dB; evoked potentials 70–80 dB (all frequences); hears normal speech 2m;	13 (7.0)
severe	audiometrically > 60 dB; evoked potentials > 90dB; some reaction to sound;	3 (1.6)
Impairment of speech:		
mild	speech comprehensible;	70 (37.4)
moderate	some words and communication;	36 (19.3)
severe	single words, cannot communicate by speech;	43 (23.0)
Impairment of ambulation:		
mild	walks with support from one hand;	17 (9.1)
moderate	crawls, uses two canes or wheelchair;	6 (3.2)
severe	bedridden;	2 (1.1)

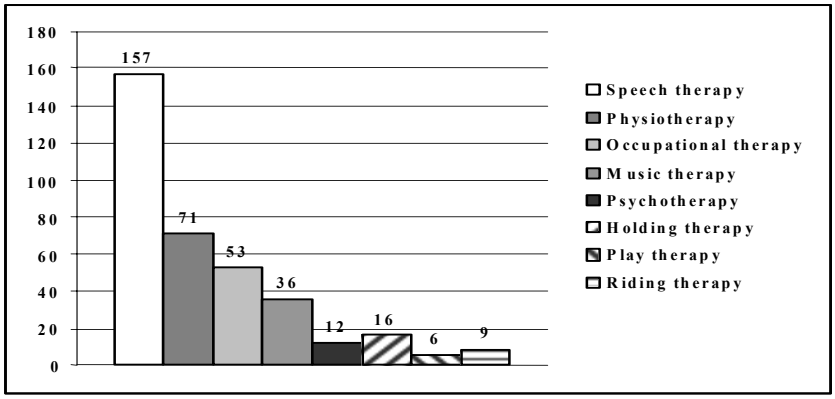

Fig. 1. Therapeutic interventions

5.3 Care, treatment and habilitation of autistic disorder

One hundred and fifty-two (82.9%) children and adolescents with autism had attended at least two therapeutic interventions and/or specific training programs. The most common therapies were speech, physio-, occupational and music therapy. The therapeutic interventions other than specific training programs used in the treatment are shown in Figure I. Eighty-two of the children and adolescents with autism (43.9%) attended training programs according to TEACCH, 19 (10.2%) according to Lovaas and 57 (30.5%) according to the Portage scenario. During the follow-up, the severity of autism changed in all programs (Fig. II,III,IV). Seventy-three (39.0%) individuals had no specific training program, such as TEACCH, Lovaas or Portage, but received additional therapies, such as speech therapy, physiotherapy or occupational or music therapy. In addition, parental counselling and instruction constituted a substantial part of all children's treatment.

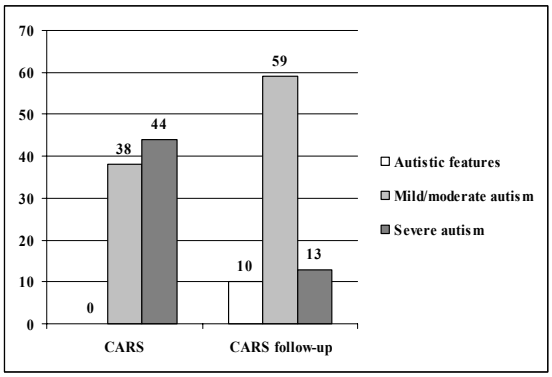

Fig. 2. TEACCH -program and the severity of autism in follow-up (N=82)

Statistically significant improvement, as assessed by CARS, took place in the TEACCH, Lovaas and Portage interventions ($p < 0.001$, Chi-Square) (Table 4). The individuals who had only one intervention, 18 out of 40 (45.0%) in the TEACCH program, 3 out of 4 (75.0%) in the Lovaas program and 12 out of 22 (54.5%) in the Portage program, had profited by at least one grade in CARS during the follow-up. Though the children in the Lovaas program seemed to profit most, the differences in efficacy between these three interventions were not statistically significant, probably because of the small study groups. Of the individuals who did not undergo a TEACCH, Lovaas or Portage intervention, but had some other therapy or treatment, 45 out of 73 (61,6%) seemed to profit by at least one grade in CARS during the follow-up.

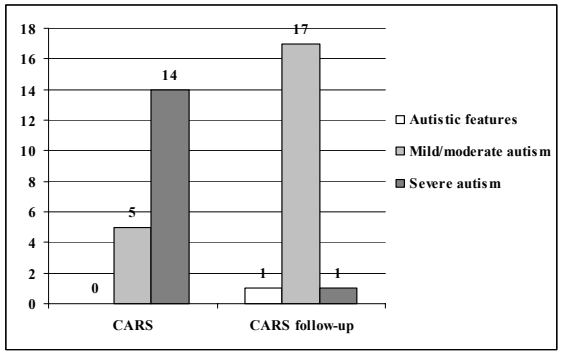

Fig. 3. Lovaas -program and the severity of autism in follow-up (N=19)

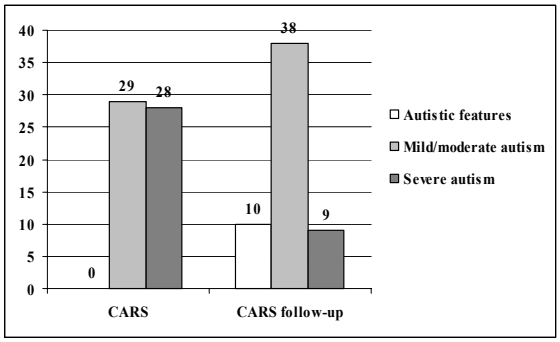

Fig. 4. Portage -program and the severity of autism in follow-up (N=57)

When recovery was assessed on the basis of the change in CARS raw scores, only nine children with AD out of 183 (4.9%) failed to derive any benefit from the treatment, irrespective of the treatment modality, and they were classified as "challenging". In addition, after the follow-up period, five of these nine (55.6%) individuals showed a more positive outcome when the level of autism had been taken into account in the planning of the intervention, treatment and care of AD. The average distance to the nearest central hospital was 80.9 km (min. 5 km and max. 390 km) in the whole group of 187 individuals with AD and 96.7 km (min. 10 km and max. 280 km) in the group of nine challenging children with AD.

The pharmacotherapy affecting the central nervous system included neuroleptics, antidepressants, sedative-hypnotics, mood stabilisers and anticonvulsants. Pharmacological interventions other than anticonvulsants were prescribed to 27 individuals (14.9%). The mean age of starting medication was 8.4 years (range from 3 to 15 years). Neuroleptics, such as haloperidol, chlorprotixen, levomepromazine, perphenazine and thioridazine, were administered to 23 persons (85.2%) and sedative-hypnotics, such as buspirone, nitrazepam and diazepam, to eight individuals (29.6%). One person received lithium as a mood stabiliser, to improve severely aggressive behaviour. Selective serotonin reuptake inhibitors (SSRI), such as fluoxetine and citalopram, were prescribed to two persons. Five persons had a combination of neuroleptics and sedative-hypnotics. A beneficial effect on the target symptoms was seen in 20 individuals out of 27 (74.1%).

Anticonvulsant medication, such as barbiturates, carbamazepine, valproate and ACTH, was given to 44 of the persons with AD (23.9%). In three of these cases, carbamazepine or valproate was also used to improve psychological functioning and to reduce behavioural problems. The mean age of starting epileptic medication was 2.9 years (range from 0.1 to 10 years). In 28 individuals out of 44 (63.6%), this medication successfully prevented epileptic seizures.

5.4 Outcome in the study group

The severity of autism and the outcome in relation to intelligence are shown in Table 6. A non-statistical trend suggested that the most intelligent individuals seemed to benefit most from the habilitation methods. After the therapeutic interventions during the follow-up, the CARS grade of autism became remarkably milder according to most recent hospital records. The difference between the initial and follow-up CARS scores was statistically significant in all age groups ($p < 0.001$ Chi-Square test).

Table 6. Intellectual functioning and outcome.

	IQ ≥ 86 N (%)	IQ 69–85 N (%)	IQ 50–69 N (%)	IQ 35–49 N (%)	IQ < 35 N (%)	All N (%)
Initial CARS						
Mild/Moderate	36 (76.6)	37 (84.1)	24 (49.0)	11 (36.7)	10 (58.8)	118 (63.1)
Severe	11(23.4)	7 (16.0)	25 (51.0)	19 (63.3)	7 (41.2)	69 (36.9)
Follow-up CARS						
Features	17 (36.2)	17 (38.6)	9 (18.4)	6 (20.0)	2 (11.8)	51 (27.3)
Mild/Moderate	27 (57.4)	25 (56.8)	36 (73.5)	18 (60.0)	12 (70.6)	118 (63.1)
Severe	3 (6.4)	2 (4.6)	4 (8.1)	6 (20.0)	3 (17.6)	18 (9.6)
Total	47	44	49	30	17	187

When the severity of autism was assessed by CARS for the first time at the age of three to five years, 24 of the children in the age group of 3–5 years (48.0%) had severe autism, whereas five adolescents in the older age group of 15–18 years (20.8%) had severe autism. In the follow-up CARS seven of the children in the age group of 3–5 years (14.0%) had severe autism, whereas only one adolescent in the older age group of 15–18 years (4.2%) had severe autism. However, the difference in outcome between the age groups was not statistically significant. The total numbers of individuals with severe autism were not high enough to show the difference in outcome or to reach statistical significance (Table 7).

Table 7. Overall outcome in different age groups

Age groups	Initial CARS		Follow-up CARS			Total
(yrs)	Mild/Moderate	Severe	Features	Mild/Moderate	Severe	
3–5	26(52.0)	24(48.0)	6(12.0)	37(74.0)	7(14.0)	50
Boys	21	18	5	28	6	39
Girls	5	6	1	9	1	11
6–14	73(64.6)	40(35.4)	36(31.9)	67(59.3)	10(8.9)	113
Boys	58	32	29	7	90	90
Girls	15	8	52	15	23	23
15–18	19(79.2)	5(20.8)	9(37.5)	14(58.3)	1(4.2)	24
Boys	16	3	8	10	1	19
Girls	3	2	1	4	-	5
3–18	118(63.1)	69(36.9)	51(27.3)	118(63.1)	18(9.6)	187
Boys	95	53	42	90	16	148
Girls	23	16	9	28	2	39

6 Discussion

6.1 Prevalence

Autism turned out to be more common in this geographical area than previously believed. In line with the recent prevalence studies from other countries, the prevalence of AD was close to 2/1000 (Baron-Cohen *et al.* 1996, Kadesjö *et al.* 1999, Study I, Bertrand *et al.* 2001, Chakrabarti & Fombonne 2001), more specifically 20.7 (15.3–26.0) per 10 000, in the lowest age group of 5–7 years, when the criteria of ICD-10 and DSM-IV were used. The evidence suggests that the majority, if not all, of the reported rise in incidence and prevalence is due to changes in diagnostic criteria and it is generally agreed that the definition of autism has been broadened over the last decades, particularly at the less severe end of the spectrum. These major changes occurred in nosology from the Diagnostic and Statistical Manual of Mental Disorders, Third Edition *(*DSM-III*) in 1980 to the* DSM-Revised Third Edition *in 1987 and the* DSM, Fourth Edition *in 1994*. Also increasing awareness and recognition of autistic spectrum disorders has increased the prevalence (Wing & Potter 2002, Fombonne 2003). Whether there is also a genuine rise in prevalence remains an open question. Also according to this study, the prevalence of AD in Northern Finland is threefold compared to 16 years ago (Vinni & Timonen 1988, Study I). There are methodological differences between these two studies (use of diagnostic criteria, data collection methods, knowledge of autism), and the detection and understanding of AD in Northern Finland has also been improved by the availability of specialised education and international collaboration with researchers from other Nordic countries. However, the prevalence is similar in many other countries according to recent studies, and the rates for AD seem to be 3 to 4 times higher than 30 years ago (Wing & Potter 2002, Fombonne 2003).

It has been argued that the Nordic nature and geographical specialities could influence the prevalence of AD. The Swedish studies from the 1980s showed autism to be more frequent in the northern part of the country (Bohman *et al.* 1983, Gillberg 1984). The Finnish study by Vinni and Timonen carried out in the 1980s (Vinni & Timonen 1988) showed a similar phenomenon. There seems to be a remarkable number of cases of autism and autistic-like syndromes in Northern Finland. These results were congruent

with the results from Sweden. On the basis of the present study, it is difficult to say if we have more autism in Northern Finland, because there is no recent comparative study available from the southern parts of Finland. However, we seem to have more children and adolescents who need care, treatment and intervention to AD than previously.

About 20% of the total autistic population have been considered to be well-functioning (IQ > 70) (Bryson *et al.* 1988, Vinni & Timonen 1988, Gillberg *et al.* 1991, Lotter 1966, Rapin & Katzman 1998). More recent studies have reported lower proportions of mental retardation in AD. In this study, almost 50% had a tested IQ above 70. Honda *et al.* also reported half of the children with autism to have an IQ over 70 (Honda *et al.* 1996). Chakrabarti & Fombonne (2001) found that only 25.8% of the 97 children with ASD had mental retardation. This may indicate better detection of well-functioning autistic individuals both in Japan and in England as well as in Finland in this study. The previous studies of gender and autism have shown autism to be more common in boys, but girls with autism and mental retardation to be more severely affected than boys, with regard to both the level of intellectual functioning and the overall measures of brain dysfunction (Nydén *et al.* 2000, Yeargin-Allsopp *et al.* 2003). The male:female ratio in this study was about three boys to one girl, but the girls with autism and mental retardation were not more severely affected than boys. The connection between autism and low intelligence represents a diagnostic challenge, for the autistic triad threatens to be swamped by the overall picture of mental retardation.

6.2 Outcome

In this retrospective study, CARS was the only measure of which the initial evaluations were available. In addition, CARS as a rating instrument might involve considerable variation and errors across ratings and raters. The time intervals between the assessments also influence the outcome measure, and the CARS instrument might not adequately take into account the different developmental ages. Other standardized tests, such as psychoeducational profile-revised (PEP-R) (Schopler *et al.* 1990), might have been more appropriate in evaluating the outcome.

Only a small proportion of individuals with AD are able to live independent adult lives, while the individuals with HFA and AS generally improve enough to live independently in adulthood (Nordin & Gillberg 1998). In the follow-up studies from the 70's to the early 90's, only 5–15% of individuals with AD had a good outcome with nearly normal or normal social life and acceptable functioning at work or school despite certain difficulties in social relationships and oddities in behaviour (Lotter 1974, Gillberg & Steffenburgh 1987, von Knorring & Hägglöf 1993, Howlin 1998). However, it was not possible to follow up the children and adolescents into adulthood in this research. Follow-up studies of these individuals with AD would be an important research issue in the future. Cognitive ability, level of mental retardation and other comorbidities, including medical syndromes, neuropsychiatric disorders and epilepsy, were important prognostic factors in this study (Study II) as well in other outcome studies (Nordin & Gillberg 1998, Volkmar *et al.* 1999, Lord 2000).

Although behavioural approaches to intervention were the most desirable form of treatment for children and adolescents with AD in this study, there were times when, because of severe behavioural disturbance (especially self-injury or aggression), sleeping problems, overactivity, anxiety or depression or marked obsessional and compulsive behaviours, medication was prescribed (Howlin 1998, Baird *et al.* 2003, Tani *et al*. 2003). The use of medication is changing so rapidly that the results of infrequent use in the 1990's are not comparable to the latest statistics of medication (Social Insurance Institution of Finland and National Agency for Medicines, 2003). However, medication does not 'cure' autism, and therapy and other supportive arrangements should therefore be available during medication.

Though autism cannot be cured, because the original difficulty or deficit remains, the individual's and family's quality of life can be improved remarkably. One hundred and seventy-eight subjects (95.2%) out of 187 showed some improvement on the Childhood Autism Rating Scale (CARS) in this study. This strengthens the consensus that early, effective and regular interventions in autism really helped most individuals with AD and should be provided as early as possible to children with autism. Sometimes the optimal habilitation or treatment model may be difficult to find, as was the case in the challenging group of AD (Study IV). In fact, after the follow-up period in 1997 and after the implementation of a structured intervention program at school and at home, many subjects even in this challenging group had fewer and less severe problems in adolescence. The therapies or interventions last for a long time, and some persons may have several therapeutic interventions going on at the same time. It may hence be very hard to determine the real cause for a superior outcome (Study III). In some cases, a structured daily program and communication-based programs aimed at the child's level were not available at school age. On the other hand, natural progress of getting older might at least partially implicate the improvement in adolescence in some cases. However, there is always hope to improve the situation even if there has been no specific habilitation of autism in childhood. If parents cannot accept their child's autism, the outcome of AD will not be optimal (Study IV). The parental counselling provided locally has supported families to cope with their everyday life. A new project was launched in Northern Finland in 1999 to support families, professionals and persons with autism spectrum disorders.

6.3 Implications for practice

The detection and understanding of AD in Finland has recently improved with the specialised instruction in interview methods arranged for the staff of Oulu University Hospital in 2001 and 2003: Autism Diagnostic Interview Schedule – Revised (ADI-R) for interviewing the parents (about the child) and Autism Diagnostic Observation Schedule (ADOS) for observation of the autistic persons themselves. However, it was not possible to use these instruments in the diagnostic evaluation of AD in the 90's. These instruments are very useful in research but may be too time-consuming in clinical use. If I had a possibility to do this research again, I would include these interview methods in the research plan.

This survey from Northern Finland supports the assumption that medical disorders do have a more important role in autism than previously believed and should be taken into account more carefully in the diagnosis, treatment and habilitation planning. In practice, the possibility of autism should also be taken into account even when the person already has some other diagnosis or diagnoses. Furthermore, what makes autism different is the wide range of skills and deficits and the high rate of associated behavioural, mental health and learning problems and, hence, the need for a wide range of professionals. However, a new clinical neuropsychiatry practice with a pedagogical approach is under construction in Finland to take into account this broader psychophysical view of individuals with autism.

This study showed that if AD was detected early in life and an adequate intervention was carried out, remarkable improvements of the symptomatology were sometimes achieved. There is good evidence to show that the most effective intervention programmes begin at an early age, i.e. between two to four years (Rogers, 1996). With modern technologies and study designs, the risk factors initiating the causal chain that culminates in this profoundly disabling disorder will soon be identified. The great hope is that this understanding will help us to develop more definitive treatments in order to improve the long-term outcomes for the majority of individuals with autism. However, behaviour will always be determined by many factors, and by different factors at different times, and it is sometimes not possible to determine the function underlying a particular challenging behaviour. Outcome studies in autism suggest that the disabling features and cognitive style are lifelong (Gillberg & Billstedt 2000, Baird *et al.* 2003). The manifestation of autism changes over time and depending on the other developmental impairments, personality and the onset of medical or mental health problems, and the care, treatment and habilitation of AD should thus be taken into account in all age groups. Other strategies, including cognitive behavioural therapy, are less well researched, but some evidence shows that both cognitive behaviour therapy and the use of social stories can be of benefit, as can social skills training (Baird *et al.* 2003). There is evidence that social skills groups specifically designed for children or adults with autism can improve certain aspects of social functioning (Howlin 1998). Additionally, Campbell (1996) suggested that there is little reliable evidence to show that psychoanalytically based interventions are helpful for individuals with AD.

It also seems that establishing appropriate management strategies in the early years can help to minimise, or even avoid, many behavioural problems (Schopler *et al.* 1980, Lovaas 1987, Rogers 1996, Mesibov 1997). It is now well established that early intervention, care and treatment, appropriately adapted to each individual child's pattern of strengths and weaknesses, can significantly help to minimise or avoid problems and to ensure that the child is able to develop his or her existing skills to the full extent. And if parents, professionals of education (teachers, school assistants) and other professionals (medical doctors, psychologist, nurses, therapists) work together, this will markedly improve the consistency of management techniques and help to ensure the generalisation and maintenance of newly acquired behaviours. Computer-aided language and reading training as well as interactive training have been developed recently to improve the communication of children with autism. Computer-based interventions designed for facial affect recognition and the identification of social deficits also yielded promising results in a recent study (Bölte *et al.* 2002). Unfortunately, computer-based interventions

were not a well-known method in the 1990's in this study group of children with AD. In addition, the lack of Finnish-language computer programs and skilled teaching professionals has resulted in scant use of computer-based interventions in Northern Finland. However, new Finnish computer programs are currently under construction.

The criticism of medicalisation reminds us that we may too easily forget the child or adolescent behind the diagnosis. Many persons with autism and their families are constantly searching for the best possible education, treatment or care to help them cope with everyday life and to find the potentials of each individual. Coping with a child who has a chronic illness or a severe disorder is a highly individualised process, and there is evidence to suggest that some families may never fully adjust to the situation (Koller *et al.* 1992, Taanila 1997, Sivberg 2002). The family's ability to acquire and allocate resources to meet the demands is a critical aspect of adjustment and adaptation. The family may acquire new resources as a result of good adjustment and adaptation, and good outcomes serve as a feedback to the family system and increase their repertoire of capacities. Conversely, poor outcomes increase the demands and may lead to maladaptation (Antonovsky 1979, Pattersson 1988, Jordan 1999). Support from other parents and individuals with ASD may give courage to cope with everyday life. A clinical review of parental needs by Baird *et al.* (2003) have shown that parents are often very delighted to meet other parents of children with AD and to find that their children have a lot in common in terms of behaviour and mannerisms. Meetings with other parents and individuals with ASD should also be sponsored more by municipalities, hospitals and The Social Insurance Institution of Finland (SIIF, KELA), even though this has not been considered therapy in the traditional sense. SIIF sponsors many therapies, e.g. speech therapy, for children and adolescents in Finland, but there is no reimbursement for parents' meetings.

The essential question should be stated and also studied more specifically: what really helps in the habilitation of and coping with ASD? Parents are usually very happy when they get some help for their children, and they do not even know of any other possibilities. Many parents become frustrated and give up seeking alternative treatments because they are simply unable to get the information they need about locally available facilities. Therefore, we should encourage the professionals working in this field to guide parents and also to seek for the best solutions through consultation if their own knowledge of ASD is inadequate. In research and interventions, it is also important to meet the parents and to keep up regular contacts with them. Without this, both the staff and the parents might speculate and doubt about what is really going on. Working with the parents is essential for a positive outcome. Also when planning the intervention, the parents should have an opportunity to speak with the professionals who frequently have contacts with the child, including the attending doctor, psychologist, social worker, teacher and therapists. It was also clear in this study that the parents had usually detailed and excellent long-term knowledge about their own child, much of which can be useful for those professionals who are working with an individual with ASD.

The long distances in rural areas are a big problem in the availability of treatment, care and habilitation. The treatment programs and therapies varied in the study area, depending on the availability of trained staff. Though many children had some therapy (usually speech or physiotherapy) at an early age, it was not sufficient to alleviate the severity of autism. It is therefore necessary also to develop locally operating practices for

individual demands. Other aspects of communicative development were also very poor in some cases. In Northern Finland, the lack of professionals (especially speech therapists with knowledge of autism) also had a paralysing effect on the quality of habilitation. These are important factors when considering the need to finance the intervention, treatment and care of AD. In the northern parts of the circumpolar area, long distances from home to services constitute a major problem, which is being increasingly solved by the use of telematics (Pesämaa *et al.* 2004). In the case of autism, this might essentially help the health care and educational staff by providing opportunities for consultation. Local social, public health and educational administrators would be able to use this information, which would be very valuable for assessing specific needs and financing.

7 Conclusions

The total prevalence (± 5% CI) of AD in Northern Finland was 13.9 (12.0–15.7)/10 000. The prevalence in boys was 20.8 (17.8–24.3)/10 000 and that in girls 6.8 (4.9–8.6)/10 000. The prevalence of ”classic” autism in the area was 5.6 (4.4–6.8)/10 000, being 8.2 (6.6–10.8)/10 000 in boys and 2.5 (1.5–3.7)/10 000 in girls. The age specific prevalences obtained in this study showed the prevalence to be lowest, 6.1 per 10 000, in the oldest age group of 15- to 18-year-old children, and highest, 20.7 per 10 000, in the age group of 5–7 years, when the criteria of ICD-10 and DSM-IV were used. According to this study, we have about three- to fourfold prevalence of AD in Northern Finland now compared to 16 years ago.

Associated medical disorders or associated disorders of known or suspected genetic origin were found in 12.3%, including tuberous sclerosis, Down syndrome, fragile-X-syndrome, Klinefelter syndrome, XYY syndrome, chromosome 17 deletion, chromosome 46,XX,dup(8)(p) and mitochondriopathy. Other associated medical disorders were epilepsy, hydrocephalus, fetal alcohol syndrome and cerebral palsy. Severe impairment of vision was found in 3.7% of the individuals with autistic disorder. Epilepsy was found in 34 (18.2%), cerebral palsy in 8 (4.3%) and other associated neurological disorders (such as hydrocephalus, fetal alcoholic syndrome, Sotos syndrome, neonatal meningitis/encephalitis). Medical disorders seem to have a special impact on the genesis of autistic disorder and need to be thoroughly examined in each child with autistic disorder.

One-hundred and fifty-two (82.9%) children and adolescents with autism received more than one therapeutic intervention or specific training program. The most common therapies were physiotherapy as well as speech, occupational and music therapy. 43.9% of the children and adolescents with autism received specific training according to TEACCH (Treatment and Education of Autistic and related Communication-Handicapped Children), 10.2% according to Lovaas and 30.5% according to the Portage program. Antiepileptic medication had been prescribed to 23.9% and psychopharmacological interventions to 14.9% of the individuals with autistic disorder (AD). One hundred and seventy-eight subjects out of 187 showed some improvement on the Childhood Autism Rating Scale (CARS), even if no statistically significant difference was found between the outcome of the available habilitation methods. Good outcome is

often achieved by understanding the child's fundamental difficulties in communication and social interaction and also by understanding the ritualistic and obsessional tendencies that are characteristic of autism.

One aim of this study was to elicit reliable information of intervention, treatment and care of AD for decision-makers as well as ideas for giving support and, because of the presumed better outcome, saving resources in the long run. The important message of this study is that early, effective and regular interventions in autism have a good impact within a short period of time (two to four years) and should be provided as early as possible to children with autism. However, we still lack specific information and research regarding the effectiveness of the different programs and the interventions.

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