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Risk factors for mental retardation

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SUMMARY Risk factors for mental retardation were studied prospectively in 12 000 children born in northern Finland in 1966 and followed to the age of 14 years. The number of untraced children was less than 2 per 1000. Altogether 326 children had an IQ less than 86, and the incidence of severe retardation (IQ less than 50) was especially high. An incidence figure for children with mental retardation, a separate figure for healthy children, and also the death rate were calculated for each disease. Only in the cases of Down's syndrome and some hereditary diseases were all the exposed children mentally retarded; in other diseases some children did not seem to suffer any sequelae. A risk factor could be found for 50·6% of the total number of children with mental retardation, the percentage decreasing from the severest to the mildest form (86·7%, 45·4%, and 30·9%). Some 9·4% of the healthy children and 77·7% of those who died had had one or more of these conditions. Prenatal conditions were most often associated with severe mental retardation (64%), and perinatal conditions with mild retardation, (IQ 50 to 70; 27%) and mental subnormality (IQ 71 to 85; 18%). Cases with no known risk factor were more common among boys than girls.

A detailed knowledge of the risk factors for mental retardation in a community forms the natural basis for preventive measures. The facilities for exact diagnosis depend on the medical information available on aetiological factors and therefore alter with time. Thus MacKay[1] noted that in 1965 a definite aetiological diagnosis for severe mental retardation could be made in only one third of all patients;[2] but this could be achieved in 65% of cases by 1973[3] and in 86% by 1977.[4] Even though the aetiological diagnoses in many instances are specific to an eponymous entity, there are many conditions, such as anoxia, which result in retardation in some children only, and studies of this type are seldom epidemiological population studies from which we can also find out what proportion of the children exposed to a specific disease will be mentally retarded, what proportion will be perfectly healthy, and what proportion will die.

The risk factors for mental retardation were studied in a birth cohort for 1966 in northern Finland.[5] The investigation was started during pregnancy and the children were followed up to the age of 14 years. Morbidity during the 14 years, and particularly that for the newborn period, was carefully recorded prospectively. In the follow up, special emphasis was laid on recording the incidence of mental retardation, a figure which was found to be unusually high, especially for severe retardation.[6]

Series and methods

The series comprised 12 058 live births from the two northern-most provinces of Finland, Oulu and Lapland, and covered 96% of all children born in the region in 1966.[5] Because the study was started during pregnancy, all the cases with an expected date of delivery in 1966 were included, so that a small number of preterm infants were born in 1965 and post-term infants in 1967. Twin infants numbered 314 and singletons 11 744. Perinatal mortality was 26·0 per 1000 and 278 liveborn children had died before the age of 14 years.

Follow up data on the mental and physical development of the children were collected at various ages, the latest follow up being in 1980 and 1981, representing an age of 14 years. Only 14 children, 1·2 per 1000, could not be traced at that age, all having emigrated to other countries. A total of 82·9% of the original cohort were still living in northern Finland in 1980, 10·7% were living in other parts of Finland, and 6·5% had emigrated to other countries, mainly to Sweden.

Data were collected during the period 1966 to 1983 in the following ways:

(1) In 1965 and 1966, 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 clinics for 12 068 mothers.[5]

(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 628 children, 81·2% of the total of 773 who were admitted at that age and for whom diagnoses were available (see below). The following details were used for this study: the blood sugar and bilirubin concentrations, body temperature on hospital admission, and the diagnoses for the mother and child.

(3) Diagnoses on admission to the four children's hospitals in the area between 1966 and 1972 were collected by the research group, (n=3528).[7]

(4) A questionnaire on the children's health and development at the age of 1 year was filled in by the public health nurses at the child welfare centres, (n=10 822).[8]

(5) Hospital records and special forms were filled in for those children who visited neurological outpatient clinics in the area either because of their symptoms or when requested to do so for study purposes (n=1841).[9 10]

(6) All existing protocols for IQ tests and psychologist's evaluations were collected from the child guidance centres, hospitals, and institutions for mentally retarded children (n=483).[6 11]

(7) Data were also collected from national registers of—death certificates (from 1965, n=278); hospital discharge register (from 1972, n=5454); child subsidies for chronically sick children (from 1967, n=349); mentally retarded children (from 1979, n=82).

The children with mental retardation were thus identified from data collected by the study group or from national registers. Careful inquiries were carried out on all children not attending school at a level appropriate for their age in order to check that all cases had been included. The 326 children with an IQ less than 86, were divided into severe cases, IQ less than 50 (n=98); mild, IQ 50 to 70 (n=66); and mentally subnormal, IQ 71 to 85 (n=162). Some 25 children with an IQ less than 86 died before the age of 14 years, and except for one they all belonged to the most retarded group.[6] Altogether 13 different types of tests for 472 children were used, the predominant test, Terman-Merrill was standardised for Finnish children in the 1950s,[12 13] whereas the other tests were translations and used according to the original instructions. Each child underwent an average of 2·4 tests, frequently of different types. The most current and complete test was chosen if there were several available. For this study the IQ was assessed using the Terman-Merrill test for 217 children and using the Wechsler intelligence scale[14] for 205 children. For the remaining 50 children the IQ was obtained using one of the less common tests.

The risk factors were subdivided into two types:

(a) A prenatal or perinatal causal factor was considered to have been present whenever such a diagnosis of evident clinical importance had been given to the child during the period or later findings clearly showed a prenatal or perinatal origin.

(b) A postnatal risk factor was considered to be present whenever a clinically important diagnosis which could be assumed to affect development was present.

All types of haemolytic diseases indicated as diagnoses for the child in the hospital records and all cases of perinatal jaundice in which the bilirubin concentration was over 20·1 mg/100 ml were counted as cases of haemolytic disease (Table 2). A diagnosis of hypoglycaemia was given if this diagnosis was recorded in his medical records or if the blood sugar value in any test had been below 1·1 mmol/l (Table 2). A diagnosis of hypothermia was given when the body temperature was less than 35°C on admission to the children's hospital (Table 2).

The incidence of risk diagnoses was calculated for the cases of mental retardation;[6] the healthy children without this, cerebral palsy, or epilepsy up to the age of 14 years (n=11 320);[9 10] and for deaths (n=278).[11] For each case of mental retardation only one diagnosis—that which was thought to be the most likely causal risk factor—is indicated in Tables 1 to 4. All existing diagnoses were counted for the healthy children and those who had died. The analysis was carried out in this manner because a disease cannot be given as a risk for health. Furthermore, the basic aim of the study was to analyse to what extent the risk factors for mental retardation were also connected with a fatal outcome, whereas a detailed analysis of the causes of death per se was beyond the scope of this study. Exposed cases of cerebral palsy and epilepsy were also included in the divisor when calculating the death rate. The cases which did not have any risk diagnosis for mental retardation were distributed into groups with an Apgar score of 7 or less at 1 and 15 minutes, with birthweight less than 2500 g, with length of gestation less than 37 weeks, and with birthweight less than 2 SDS below the mean, each case being assigned only to the first of these groups for which it qualified. The relative risks for mental retardation and for mortality are calculated in Tables 2 to 4, that is the ratio of the rate of mental

retardation (or mortality) among those who had the disease to the rate among those who did not have this disease.

Results

Table 1 shows the numbers of children with mental retardation, the numbers of healthy children, and the death rates among those with congenital malformation syndromes, chromosomal aberrations, and hereditary diseases. There were no healthy children among the Down's syndrome cases or those with hereditary diseases (2 leucodystrophy, 1 progressive muscular dystrophy, 1 Werding-Hoffmann's disease, 1 unspecified cerebral degeneration). Altogether 19 healthy children had or had had one of the other diseases studied here, but the difference between the numbers with mental retardation and healthy children was highly significant for all the diseases except for chondrodystrophia. Mortality was highest among children with multiple anomalies and second highest for those with spina bifida (Table 1).

Table 2 depicts the number of children exposed to various perinatal conditions, the incidences of these

Table 1 *Incidence of congenital malformation syndromes, chromosomal aberrations, and hereditary diseases among children with mental retardation (MR) and healthy children. For the children with MR, only one (the most likely) causal diagnosis is indicated, but for the others all the diagnoses given are counted*

Diagnosis	*Cases of MR (n=326)*		*Healthy children (n=11 320)*		*P*	*Deaths*		
	No	*/1000*	*No*	*/1000*		*No*	*Underlying cause*	*/1000 exposed*
Down's syndrome (n=39)	39	119·6	0	0·0	0·000	15	(7)	384·6
Spina bifida (n=14)	5	15·3	5	0·4	0·000	6	(2)	428·6
Hydrocephalus (n=17)	6	18·4	5	0·4	0·000	6	(4)	352·9
Other CNS anomaly (n=14)	10	30·7	1	0·1	0·000	2	(0)	142·9
Multiple anomalies (n=20)	4	12·3	5	0·4	0·000	10	(5)	500·0
Metabolic disorders (n=9)	5	15·3	1	0·1	0·000	2	(1)	222·2
Hereditary diseases (n=5)	5	15·3	0	0·0	0·000	1	(1)	200·0
Chondrocystrophy (n=5)	1	3·1	4	0·4	0·020	0	(0)	·0
Total (n=123)	75	230·1	21	1·9	0·000	42	(20)	350·4
No of children	75		19			39	(20)	

CNS=central nervous system.

Table 2 *Incidence of perinatal diseases among children with mental retardation (MR) and healthy children. For the children with MR, only one (the most likely) causal diagnosis is indicated, but for the others all the diagnoses given are counted*

Diagnosis	*Cases of MR (n=326)*		*Healthy children (n=11 320)*		*Relative risk for MR (%)*	*P*	*Deaths*			*Relative risk for death*
	No	*/1000*	*No*	*/1000*			*No*	*Underlying cause*	*/1000 exposed*	
Intraventricular or cerebral haemorrhage (n=30)	2	6·1	4	0·4	12·0	0·000	24	(11)	800·0	37·9
Brain injury (n=75)	7	21·5	25	2·2	8·0	0·000	26	(8)	346·7	16·5
Asphyxia or anoxia (n=155)	11	33·7	69	6·1	5·0	0·000	59	(45)	380·6	20·7
Premature separation of placenta (n=15)	3	9·2	8	0·7	9·8	0·000	3	(0)	214·3	9·4
Haemolytic disease (n=97)	6	18·4	75	6·6	2·7	0·005	6	(3)	61·9	2·7
Hypocalcaemia (n=10)	1	3·1	4	0·4	7·2	0·020	0	(0)	·0	0·0
Cephalhaematoma (n=9)	1	3·1	7	0·6	4·5	0·096	0	(0)	·0	0·0
Toxaemia of pregnancy (n=61)	3	9·2	48	4·2	2·1	0·181	5	(0)	82·0	3·6
Diabetes mellitus in mother (n=15)	1	3·1	11	1·0	3·0	0·245	3	(1)	200·0	8·8
Hypoglycaemia (n=49)	2	6·1	32	2·8	2·1	0·275	10	(0)	204·1	9·1
Hypothermia (n=109)	2	6·1	53	4·7	1·3	0·706	45	(0)	412·8	21·2
Haemorrhagic disease (n=43)	1	3·1	24	2·1	1·4	0·716	15	(4)	348·8	15·9
Apgar score <8 at 1′ and/or 15′* (n=170)	3	9·2	20	1·8	4·7	0·003	43	(0)	252·9	12·8
Birthweight <2500 g* (n=524)	11	33·7	268	23·7	1·4	0·241	113	(14)	215·6	15·1
Length of gestation* <37 weeks (n=696)	6	18·4	326	28·8	0·6	0·266	104	(0)	149·4	9·8
Birthweight <mean—2 SD* (n=247)	1	3·1	71	6·3	0·5	0·467	33	(0)	133·6	6·4
Total (n=2305)	61	187·1	1045	92·3	2·2	0·000	489	(86)	212·1	21·6
No of children (n=1284)	61		958				173	(86)		

*For the first 6 columns only cases which are not included in the previous diagnoses in this Table or in Tables 1, 3 and 4 are indicated.

diseases among mentally retarded and healthy children, and the relative risk for retardation or death resulting from such exposure. The relative risk for both was greatest if the child had intraventricular or cerebral haemorrhage, while the difference in incidence between the children with mental retardation and healthy children was also highly significant in the case of brain injury, premature separation of placenta, and asphyxia or anoxia. The incidence was significant for the children who had had haemolytic disease or hypocalcaemia. In the cases of maternal toxaemia and diabetes, neonatal haemorrhagic disease, hypothermia, hypoglycaemia, and cephalhaematoma the difference between the group with retardation and the healthy children was not significant, even though mortality was clearly increased in most of these diseases. Table 2 also shows those cases with low Apgar scores, low birthweight or short gestational age, or both, for whom no other causal diagnosis was found, each case being assigned to the first of these categories to which it belongs. Only in the cases with low Apgar scores was the difference between children with mental retardation and healthy children significant.

Data on the children who had suffered from intrauterine or neonatal infection are presented in Table 3, which covers altogether 12 children with mental retardation and 82 healthy children. Congenital rubella and cytomegalovirus infections, unspecified prenatal infection, septicaemia, and bacterial meningitis were highly significantly more common among the mentally retarded than the healthy children, but for viral meningitis and neonatal pneumonia, the difference was not significant, even though mortality was very high in the latter condition.

Table 4 gives the risk diagnoses for the children whose mental retardation was acquired after the

Table 3 *Incidence of intrauterine and neonatal infectious diseases among children with mental retardation (MR) and healthy children. For the children with MR, only one (the most likely) causal diagnosis is indicated, but for the others all the diagnoses given are counted*

Diagnosis	*Cases of MR (n=326)*		*Healthy children (n=11 320)*		*Relative risk for MR (%)*	*P*	*Deaths*			*Relative risk for death*
	No	*/1000*	*No*	*/1000*			*No*	*Underlying cause*	*/1000 exposed*	
Congenital rubella (n=2)	1	3·1	1	0·1	17·9	0·000	0	(0)	0·0	0·0
Congenital cytomegalovirus (n=6)	2	6·1	4	0·4	12·0	0·000	0	(0)	0·0	0·0
Prenatal infection (n=3)	1	3·1	1	0·1	17·9	0·000	0	(0)	0·0	0·0
Septicaemia (n=13)	2	6·1	5	0·4	10·3	0·000	6	(4)	461·5	20·4
Bacterial meningitis (n=2)	1	3·1	0	0·0	35·8	0·000	1	(0)	500·0	21·8
Pneumonia (n=110)	4	12·3	57	5·0	2·4	0·074	46	(26)	418·2	21·5
Viral meningitis (n=19)	1	3·1	17	1·5	2·0	0·478	0	(0)	0·0	0·0
Total (n=155)	12	36·8	85	7·5	4·6	0·000	53	(30)	341·9	18·1
No of children (n=145)	12		81				48	(30)		

Table 4 *Cumulative incidence of diseases which occurred after the first month of life up to the age of 14 years among children with mental retardation (MR) and healthy children. For the children with MR, only one (the most likely) causal diagnosis is indicated, but for the others all the diagnoses given are counted*

Diagnosis	*Cases of MR (n=326)*		*Healthy children (n=11 320)*		*Relative risk for MR (%)*	*P*	*Deaths*			*Relative risk for death*
	No	*/1000*	*No*	*/1000*			*No*	*Underlying cause*	*/1000 exposed*	
Psychoses (n=12)	6	18·4	3	0·3	24·2	0·000	0	(0)	0·0	0·0
Intracerebral haemorrhage (n=5)	2	6·1	0	0·0	35·9	0·000	0	(0)	0·0	0·0
Unspecified cerebral disease (n=2)	1	3·1	1	0·1	17·9	0·000	0	(0)	0·0	0·0
Cerebral trauma (n=31)	5	15·3	19	1·7	7·5	0·000	2	(1)	64·5	2·8
Septicaemia (n=7)	1	3·1	3	0·3	9·0	0·007	2	(1)	285·7	12·5
Drowning (n=17)	1	3·1	5	0·4	6·0	0·039	11	(11)	647·1	29·2
Bacterial meningitis (n=32)	2	6·1	21	1·9	3·1	0·086	6	(4)	187·5	8·3
Encephalitis (n=20)	1	3·1	13	1·1	2·6	0·324	2	(0)	100·0	4·4
Total (n=120)	19	58·3	65	5·7	8·5	0·000	23	(17)	182·5	8·5
No of children (n=119)	19		65				23	(17)		

Table 5 *Incidence of risk diagnosis groups among children with severe (IQ <50) and mild (IQ 50–70) mental retardation, and mental subnormality (IQ 71–85), and also healthy children and deaths*

Diagnosis group	*IQ*						*Deaths up to 14 years (n=278)*		*Healthy children (n=11 320)*	
	<50 (n=98)		*50–70 (n=66)*		*71–85 (n=162)*					
	No	*/1000*	*No*	*/1000*	*No*	*/1000*	*No*	*/1000*	*No*	*/1000*
Prenatal (n=183)	63	642·9	4	60·6	14	86·4	44	240·4	72	6·4
Perinatal (n=1188)	18	183·7	18	272·7	29	179·0	151	127·1	932	82·3
Postnatal (n=106)	4	40·8	8	121·2	7	43·2	21	210·0	59	5·2
Total (n=1477)	85	867·4	30	454·5	50	308·6	216	777·0	1063	93·9

neonatal period, and the data on the healthy children who were exposed to a similar condition. Psychoses, intracerebral haemorrhage, unspecified cerebral disease, cerebral trauma, septicaemia, and drowning occurred significantly more often among the mentally retarded children than the healthy children, but the difference between these two groups was not significant in the case of meningitis and viral encephalitis.

Forty nine mentally retarded children had received altogether 70 diagnoses which were considered causal factors for other cases in addition to their own presumed causal diagnosis. For example, 24 children belonging to the group with congenital, chromosomal, or hereditary diseases (Table 1) had 39 additional diagnoses, 28 of which were perinatal (Table 2).

There were 36 mentally retarded children among whom the incidence of the presumed causal diagnosis or condition defined by the Apgar score, birthweight, and length of gestation did not differ significantly from that in the healthy children if only one such diagnosis was given for each case of retardation (Tables 2 to 4). When the incidences were counted for all the cases of mental retardation having had such a diagnosis or condition, it was only for neonatal serous meningitis, postneonatal viral encephalitis, and maternal diabetes that the incidence of diagnoses was not significantly higher among the mentally retarded. The child with postneonatal encephalitis had an additional diagnosis of psychosis, which was significantly more common for cases of mental retardation.

The incidence of various risk disease groups presented in Tables 1 to 4 are also calculated separately for the IQ groups less than 50, 50 to 70, and 71 to 85 in Table 5. The diagnoses were grouped into prenatal causes (hereditary, congenital, and chromosomal plus intrauterine infections), perinatal causes (diagnoses in Table 2 plus neonatal infections), and postnatal causes. The two cases in which the suspected risk diagnosis was not significantly more frequent for mental retardation (maternal diabetes, neonatal serous meningitis) were excluded from the conclusions.

The healthy and dead children who had had one or more of the diseases that were taken as risk factors for mental retardation are also indicated in Table 5. To make these data more comparable with the figures for mental retardation, the children were counted only once, even if they had had two or more of these diseases, and were in such cases indicated in the first relevant group in Table 5. Only in some of the cases were these diseases the underlying causes of death (Tables 1–4).

A risk diagnosis was found in 86·7% of the cases of severe mental retardation, 45·4% of the mild cases, and 30·9% of those of mental subnormality. Some 9·4% of the healthy children and 77·7% of the children who died before the age of 14 years had been exposed to one or more of these conditions. A risk diagnosis was found for 94 of the 201 boys with an IQ less than 86 (46·8%), the corresponding figure for girls being 71 of 125 (56·8%).

Discussion

The method used here, in which only those diseases with a significantly higher incidence among the children with mental retardation were accepted as risk factors, may be too rigorous, since even in a population of 12 000 children the numbers for many of the diseases are not very high. For example, it may be wrong to exclude encephalitis as a causal factor and accept psychosis because of the difference in significance, because a psychosis is more likely to be related to foregoing encephalitis. This type of analysis nevertheless gives the order of importance of various diseases for mental retardation, and the exclusions do not greatly affect the results.

Eighty seven per cent of the cases of severe mental retardation had a known risk factor (Table 5), about the same figure as in a Swedish population[4] and in a British series[1]—86 and 88% respectively. Some 64% of the cases of severe retardation in this series were prenatal in origin, the

corresponding figure in the Swedish series being 73%[4] and that in the British series 55%,[1] while the figures for perinatal causes were 18%, 10%, and 20%, and those for postnatal causes 5%, 3%, and 10% respectively. In some conditions, the diagnosis may be confused by multiple factors. About one third of the cases with a known congenital chromosomal or hereditary cause in this series also had a perinatal diagnosis, and correspondingly some of the children whose mental retardation is assigned a perinatal cause may in fact have an unknown genetically determined condition. The higher percentage of cases for whom no risk diagnosis was found among the boys is probably also a sign of a genetically determined aetiology in some of the untraced cases.

The present results differ considerably from those reported for northern Sweden by Blomquist,[15] who gives the figure of 43% for prenatal aetiology. Since he includes multifactorial and unknown aetiological agents, the figure of 24% which includes mutant genes, chromosomal disorders, and acquired disorders corresponds better with our results, but the retrospective nature of the Swedish study make direct comparisons of the results difficult. The figures reached in an Aberdeen series[16] resembled those for northern Sweden, and differed from the Finnish figures. The corresponding figures from southern Sweden[17] differ less from the present results, prenatal causes being found in 23% of cases (including 5% genetic, 10% prenatal unknown, and 8% alcohol fetopathy). A large number of the differences are probably caused by discrepancies in data collection methods and in judging the diagnostic values of the various facts stated in the case histories. In addition, various obstetric factors are not considered here because only data obtained from a random sample of the study population is available for the healthy children. These factors would give a probably aetiology for more than 10% more cases of mild mental retardation and mental subnormality.

The aetiology of mental subnormality (IQ 71 to 85) is less commonly analysed in the published reports, and the risk factor more often remained undetected than in mild retardation in this series (69% and 55%).

The incidence of severe and mild mental retardation in this series is high when compared with other studies—severe retardation being 2·1 times and mild retardation 1·5 times more common here than in a Gothenburg series, for example.[6 17] Comparison of the proportions of the various risk factors in this series with those reported elsewhere does not give any clear evidence that the difference is attributable to any one specific cause. About one third of the cases of severe mental retardation had Down's syndrome, but this is not an uncommon finding.[18 19] The high incidence of Down's syndrome in the present series can be explained at least partly by the age distribution of the mothers,[6] but demographic background factors for the other conditions causing severe retardation have been less well studied to date.

Among the various risk diagnoses there were only a few causes (such as Down's syndrome) which gave rise to mental retardation in all the exposed children, whereas many children who suffered from anoxia during the newborn period were perfectly healthy afterwards.[20–23]

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