ORIGINAL ARTICLE

# School Performance in Finnish Children and Later Development of Schizophrenia

## *A Population-Based Longitudinal Study*

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**Background:** We examined whether children who are diagnosed as having schizophrenia in adulthood could be distinguished from their peers on performance in elementary school.

**Methods:** We used a case-control study design nested within a population-based birth cohort of all individuals born in Helsinki, Finland, between January 1, 1951, and December 31, 1960. Case ascertainment was from 3 national health care registers. Elementary school records were obtained for 400 children who were diagnosed as having schizophrenia in adulthood and for 408 controls. Results were analyzed for the 4 years of schooling (ages 7-11 years) that were common to all pupils. School subjects were entered into a principal components analysis and produced 3 factors: academic, nonacademic, and behavioral. These factors were compared between cases and controls after adjusting for sex and social group. Eligibility for high school and progression to high school were investigated among cases and controls.

**Results:** Cases performed significantly worse than controls only on the nonacademic factor (which loaded sports and handicrafts). There were no differences between the groups on the academic or behavioral factors, and there were no significant clinical correlates of factor scores. Cases were significantly less likely than controls to progress to high school, despite similar eligibility.

**Conclusions:** Poor performance in sports and handicrafts during elementary school, which may indicate a motor coordination deficit, appears to be a risk factor for later schizophrenia. Poor academic performance in elementary school was not a risk factor for schizophrenia in this study. Lack of expected progression to high school among cases, despite good academic grades, provides evidence for deteriorating premorbid functional adjustment in schizophrenia.

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THE ORIGINS of many adult mental disorders can be detected in childhood characteristics,[1] and subtle differences in development and behavior have been found among children who later develop schizophrenia, as far back as prenatal and perinatal life.[2-4] Longitudinal developmental research relies heavily on prospective cohort studies, but this design is not entirely suitable for schizophrenia research because of the low incidence of the illness and the long follow-up period required. One solution is to use cohorts that were set up for other reasons. This "opportunistic" strategy has already produced important results,[5-9] but the major disadvantage is that it yields relatively few cases of schizophrenia with consequent low statistical power.[10,11]

The "nested" case-control design includes all cases within a cohort but only a selection of comparison subjects, and is efficient of resources while combining the advantages of both cohort and case-control studies.[12,13] Our study uses such a design to examine the elementary school records of children who develop schizophrenia in adulthood, compared with general population controls. We hypothesized that children who later developed schizophrenia would have poorer performance on a range of scholastic measures than controls, and that examination of the exact nature of their educational difficulties would provide clues to the developmental processes affected in schizophrenia.

## RESULTS

There was a significant excess of males among the cases compared with the controls (odds ratio, 1.6; 95% confidence interval, 1.2-2.1). Cases and controls did not differ significantly on social class distribution. The mean (±SD) age at onset of schizophrenia was 25.1 ± 5.5 years (range, 13.0-40.1 years). The mean annual duration of hospitalization for schizophrenia was 50.2 ± 65.4 days (range, 0.0-309.4 days). The mean familial loading score for schizophrenia among the cases was 0.36 ± 1.30 (range, –0.61 to 5.8).



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## SUBJECTS AND METHODS

### STUDY POPULATION AND ASCERTAINMENT OF CASES

The study population consisted of all individuals who were born in Helsinki, Finland, between January 1, 1951, and December 31, 1960. Cases of schizophrenia born during this 10-year period were ascertained from 3 national computerized databases: the Finnish Hospital Discharge Register (FHDR) and 2 registers of the Social Insurance Institution: the Pension Register and the Free Medicine Register. The data in all registers were linked by means of the unique social security number for each individual. Information from these registers was available for the period January 1, 1969, through December 31, 1991. Before 1987, the *International Classification of Diseases, Eighth Revision* (*ICD-8*)[14] diagnostic system was used, after which diagnoses were coded according to *International Classification of Diseases, Ninth Revision (ICD-9)*,[15] with the use of *DSM-III-R* criteria.[16,17] The Social Insurance Institution indexes only the first 3 digits of diagnostic codes. Therefore, we defined as cases all individuals with a "295" diagnosis according to *ICD-8* and *ICD-9* numbering schemes; this included individuals with schizophrenia, schizoaffective disorder, or schizophreniform disorder. The FHDR covers all public and private hospitals in Finland, and the discharge diagnoses for each admission are made by the attending physician. The Free Medicine Register and the Pension Register give primary diagnoses for individuals receiving state-subsidized outpatient medication and disability pensions, respectively. All Finnish citizens have free access to inpatient and outpatient health care and are entitled to a state-funded disability pension, and more than 90% of psychotic patients in Finland come into contact with the health care system in at least one of those ways.[18] The diagnostic validity of the FHDR has been examined against *DSM-III-R* criteria and has been found to have excellent (92%-100%) specificity for diagnoses of schizophrenia.[19-21]

We identified 928 individuals from the registers who had received a "295" diagnosis and who were born in Helsinki between 1951 and 1960. Child health cards were located for 486 of these individuals (52%) in the city archives, indicating that they had attended school in Helsinki; the remainder had attended school elsewhere. Since the 2 main obstetric hospitals are based in Helsinki, women come from outlying areas to give birth there, but the children attend the school nearest their home. In addition, during the 1960s and 1970s there was a large expansion of the suburban areas around Helsinki, and families with young children, from all social groups, moved out of the city at that time.

### SELECTION OF CONTROLS AND TRACING OF SCHOOL CARDS

The next Helsinki-born child with a different surname listed after each case in the Child Health Clinic archives was taken as a control. The record cards are stored in alphabetical order by year of birth. If the next card also belonged to a case, the previous card was taken as the control. All individuals with "295" diagnoses were included among our cases, but other psychiatric diagnoses were not excluded from the control group. The names of the schools attended by all cases and controls were noted. School record cards from the state elementary school system were traced for 400 cases and 408 controls in the Helsinki City Archives (83.1% of those who attended school in Helsinki). Twenty cards had been destroyed during school fires, and 23 children had attended foreign-language schools, which are outside the state system. We have no information on the remaining untraced cards (54 cases and 67 controls). It is possible that these subjects had moved away from Helsinki after starting their schooling and the card had been transferred with them. Information from the school record cards was entered onto a specially designed computerized database, blind to diagnosis. Subjects had attended 60 different elementary schools in Helsinki. We obtained information from at least 1 year of schooling on 400 cases and 408 controls, but numbers fluctuate slightly from year to year because of migration to and from the city and emigration. Actual numbers of cases in years 1 to 4 were 384, 391, 380, and 367, respectively. Actual numbers of controls in years 1 to 4 were 382, 396, 376, and 361, respectively. There were no significant differences between numbers of cases and controls who had repeated a grade, and when these repeaters were excluded from the analysis, the results did not change.

### INFORMATION ON CORRELATES OF ILLNESS AND CONFOUNDERS

The FHDR provided information on age at first admission and duration of hospitalization. Onset was considered to be the age at first diagnosis with schizophrenia. Nineteen cases (4.7%) had onset of illness at or before 18 years of age. The main analyses were conducted with and without these cases, and since the results were not significantly different, these early-onset cases were retained in subsequent analyses. Paternal occupation was recorded on the school record cards. Four socioeconomic groups were identified on the basis of the City of Helsinki Social Group Classification[22]: (1) professionals, managers, and higher administrative or clerical employees; (2) lower clerical employees; (3) skilled workers; and (4) unskilled workers. In the analysis these were collapsed into 2 groups: professional/clerical and skilled/unskilled workers.

### FAMILIAL LOADING SCORE

Record linkage between the health care registers and the National Population Register gave data on psychiatric diagnoses in first-degree relatives. A familial loading score was calculated for each case to estimate genetic risk of schizophrenia.[23] This score, which takes account of family size and age structure, was based on the following assumptions: the

### COMPARISON OF SCHOOL PERFORMANCE (FACTOR SCORES) BETWEEN CASES AND CONTROLS

**Table 2** shows the results of multilevel modeling of school performance in the 3 factor scores in cases compared with controls. There was a significant main effect for case-control status for the nonacademic factor only: cases performed significantly worse than controls. There was no significant main effect for case-control status for the academic or the behavior scores. There were no significant main effects for sex, but there was a trend toward better perfor-



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lifetime risk of schizophrenia in a first-degree relative is 10% for probands with familial schizophrenia and 0.5% for probands with sporadic illness; and the age range at risk is 15 to 50 years, with a linear increase in risk from zero to lifetime risk. The likelihood ratio of a proband's illness being familial or sporadic, given that a relative of age $x$ is affected, is $[(0.1)(x-15)/(50-15)]/[(0.005)(x-15)/(50-15)] = 20$, and the likelihood ratio if a relative of age $x$ is unaffected is 1 minus this. Such a likelihood ratio was calculated for each relative, and an overall likelihood ratio for whether the proband's illness was familial or sporadic was obtained by multiplying together the individual likelihood ratios. The loading score was obtained by taking the logarithm of the product. A loading score of 0 indicates equal support for the proband's illness to be familial or sporadic. A positive score indicates greater support for familiality, while a negative score indicates greater support for the proband's illness to be sporadic. This loading score has been used previously on a Finnish register-based sample.[24]

### FINNISH ELEMENTARY SCHOOL SYSTEM AND SCHOOL RECORD CARDS

During the period of this study, children in Finland attended an elementary school from the age of 7 years.[25,26] Children attended the school that was closest to their home. Children who did not attend the state educational system included severely deaf children, blind children, severely brain-damaged children, and some children in institutional care. Children who were educationally retarded (1.3%-1.9%) or who suffered from emotional or conduct disorder (0.3%-0.8%) were cared for within the state system in special classes.[27] Within the state system there were separate schools for children whose first language was Swedish (approximately 10%), but the same curriculum was followed. All children in the state system studied the same subjects for the first 4 years of schooling. At the end of grade 4, when children were aged 11 years, each child was given a ranking score based on the results of their summer examinations. This score helped to determine whether the child went on to high school, which gave a more academic education, or remained at the elementary school for an additional 4 years.

This article examines only the results from the 4 years when all children studied a common program. The core curriculum subjects were mathematics, religion, reading, writing, handicrafts, physical education, and music.[25,26] There were 2 examinations per year, in December and in May. We recorded only the results of the summer examinations. Marks given for each subject ranged from 4 (fail) to 10 (excellent). All pupils were given marks for conduct and attentiveness each year, and number of hours of absence without leave was recorded. Most children scored 10 for conduct, but a mark was deducted for transgressing school rules. It was considered a particular disgrace to score less than full marks for conduct and was an indicator of disruptive behavior.

### STATISTICAL ANALYSIS

Principal components analysis was used to reduce the school variables, including the behavioral measures, to a smaller number of underlying factors. Varimax rotation was used to make the results more interpretable. The criterion for significant loadings, by individual items, was set at 0.5. The principal components analysis yielded 3 factors with eigenvalues greater than 1.0: an academic factor, a behavioral factor, and a nonacademic factor. The subjects that loaded onto each factor are shown in **Table 1**. The results in relevant individual subjects were summed to derive separate academic and nonacademic scores, and the factor scores were used to create the behavior score. High scores in the academic and nonacademic factors indicate better performance. A high score in the behavioral factor indicates poor behavior. There was a strong positive correlation (0.47) between the academic and the nonacademic factors. The correlations between the academic and the behavioral factors and between the nonacademic and the behavioral factors did not exceed 0.1.

The dependence of the 3 factors, academic, nonacademic, and behavioral, on case-control status and other covariates was investigated by multilevel modeling.[28] The method is a form of multiple linear regression that takes account of the hierarchical nature of the dataset, where individual measurement occasions are nested within pupils who in turn are nested within schools. Items nested within a higher-level unit (eg, occasions within the same subject) tend to be intercorrelated because they are all affected by the same set of (unmeasured) influences specific to the higher-level unit. Multilevel models allow for these correlations and deal efficiently with missing data. The method partitions the overall variance for each factor into a different component for each level in the hierarchy. The components in this analysis were a between-school variance (level 3), a between-subject variance (level 2), and a within-subject (over time) variance (level 1). Case-control status, sex, and social group were entered as fixed effects. To examine interactions with case-control status, school year was included as a main effect (both continuous and categorical), and 2 interaction terms, case by grade and case by sex, were included in the model.

To examine the clinical correlates of the 3 factor scores among the cases, age at onset of schizophrenia, mean annual number of days of hospitalization, and familial loading score were each divided into quintiles. Mean factor scores were computed for each quintile. Linear trends were examined by entering quintile as a continuous variable into a multiple linear regression adjusted for sex and social group. $P$ values were corrected for clustering within person by robust methods.[29] Ranking scores at the end of grade 4 were compared between cases and controls by means of the $t$ test and adjusted for sex and social group by regression analysis. Logistic regression was used to examine progression to high school, adjusting for the confounding effects of sex and social group. Analyses were performed with the statistical programs Stata[28] and MLwiN.[30]

mance in academic subjects by girls. There were significant effects for social group on all factors. Social groups 1 and 2 performed better than social groups 3 and 4 on the academic and nonacademic factor scores, but worse in the behavior factor. The interaction terms, case by sex and case by grade, were not significant for any of the 3 factors. There was no significant variance between schools on the academic, nonacademic, or behavioral factor scores. The percentage of residual variance resulting from between-subject variation (intraclass correlation) was 77% of the variance in the academic and behavioral factor scores and 60% of the variance in the nonacademic factor.



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## CORRELATES OF FACTOR SCORES

**Table 3** presents the correlates of the factor scores among the cases. There was no influence of age at onset, severity of illness, or genetic risk of schizophrenia on results for any of the 3 factors.

## RANK IN CLASS AND PROGRESSION TO HIGH SCHOOL

In Finland, a child's rank in his or her class at the end of grade 4 was used to determine eligibility for entry to high school. Ranking scores were standardized for class size and ranged from 0.0 (last) to 1.0 (first). There was no difference between cases and controls on mean rank in their class at age 11 years, either before or after controlling for sex and social group (0.48 ± 0.45 vs 0.52 ± 0.29; $t_{629} = -1.3$; $P = .19$). There was no difference in the proportion of cases compared with controls who came first in their class (4.8% vs 4.4%; $\chi^2_1 = 0.03$; $P = .86$) or last in their class (7.3% vs 8.6%; $\chi^2_1 = 0.36$; $P = .55$). However, despite similar ranking scores, cases were only about half as likely as controls to proceed to high school after grade 4 (adjusted odds ratio, 0.6; 95% confidence interval, 0.44-0.82).

## COMMENT

The nested case-control design of this study affords many advantages: the general population base minimizes selection bias, the use of standardized prospectively recorded childhood data minimizes information and recall bias, and the large number of cases gives high statistical power to examine effects. The unexpected negative finding in our study was that preschizophrenic children performed just as well as their peers in academic subjects throughout the school grades. Previous studies, mainly of case-control design, have found lower childhood IQ[31] among schizophrenic patients, and cohort studies have shown an inverse linear relationship between low IQ in childhood and adolescence and risk of schizophrenia.[5,8,32]

We have considered several possible explanations for this discrepancy:

1. The Finnish school system during the 1950s and 1960s was very structured, with standardized teaching methods, rigid adherence to the curriculum, and strong social pressure to conform to behavioral and social norms. The preschizophrenic child may perform well academically in such an ordered and predictable environment. A recent analysis of school performance as a predictor of later psychiatric illness in the 1966 North Finland birth cohort supports our results: there was no difference in examination results at age 16 years between the preschizophrenic children and a nonhospitalized general population comparison group.[9]

2. Our sample included some cases with schizoaffective disorder and schizophreniform disorder; these are putatively less severe illnesses than schizophrenia; and such children may have performed better in school. However, we found no relationship between academic ability and severity of illness, so it is unlikely that these cases influenced the results.

3. Most other studies examining this issue have used specialized educational tests[5,6,8] or IQ tests,[31] which were administered by trained personnel. Such tests are likely to pick up subtler cognitive abnormalities than can be detected from routine school grades. However, school examination results have the advantage of being a practical, "working" measure of intelligence among schoolchildren in their normal environment, rated by their own teachers. Preschizophrenic children may perform better in such familiar circumstances than in an artificial test situation.

4. There is evidence that high-risk children who experience nonoptimal rearing environments show greater childhood impairments.[33-35] Our sample included only

**Table 1. Principal Components Analysis of School Subject Scores (With Varimax Rotation)***

| School Subjects | Factor 1 (Academic) | Factor 2 (Behavioral) | Factor 3 (Nonacademic) |
|---|---|---|---|
| Conduct | 0.14 | **–0.73** | –0.15 |
| Absences, h | 0.04 | **0.69** | –0.24 |
| Attentiveness | **0.68** | –0.27 | 0.16 |
| Mathematics | **0.79** | –0.08 | 0.13 |
| Reading | **0.79** | 0.11 | 0.03 |
| Writing | **0.87** | –0.01 | 0.11 |
| Religion | **0.75** | –0.08 | 0.18 |
| Handicrafts | 0.44 | –0.21 | **0.51** |
| Sports | 0.13 | –0.20 | **0.82** |
| Eigenvalue | 3.6 | 1.13 | 1.01 |
| % Variance | 40.5 | 12.5 | 11.0 |

**Bold face type indicates variables with loadings greater than 0.5 or less than –0.5.*

**Table 2. Multilevel Modeling Results for Performance in the Academic, Nonacademic, and Behavioral Factors Among Cases and Controls**

| | Academic | | | Nonacademic | | | Behavioral | | |
|---|---|---|---|---|---|---|---|---|---|
| | Regression Coefficient | SE | *P* | Regression Coefficient | SE | *P* | Regression Coefficient | SE | *P* |
| Case/control status* | –0.36 | 0.36 | .29 | –0.35 | 0.10 | <.001 | –0.01 | 0.05 | .90 |
| Sex* | 0.59 | 0.35 | .09 | 0.08 | 0.10 | .46 | –0.01 | 0.05 | .92 |
| Social group* | –2.61 | 0.36 | <.001 | –0.33 | 0.11 | .002 | –0.15 | 0.05 | .003 |
| Within-subject variance | 4.57 | 0.18 | . . .† | 0.81 | 0.03 | . . . | 0.17 | 0.02 | . . . |
| Between-subject variance | 14.71 | 0.98 | . . . | 1.19 | 0.09 | . . . | 0.57 | 0.02 | . . . |

**Baseline categories against which dummy variables are estimated: control, male, and social groups 1 and 2, respectively.*
*†Ellipses indicate not applicable.*



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**Table 3. Relationship Between School Performance (Factors) and Indexes of Illness Severity and Genetic Risk Among Schizophrenic Patients**

| | Quintile (No.) | Range | Mean ± SD Score | | |
|---|---|---|---|---|---|
| | | | Academic* | Nonacademic* | Behavioral Factor* |
| Age at onset† | 1 (79) | 13.0 to 20.5 y | 38.7 ± 4.1 | 14.7 ± 1.4 | −0.011 ± 0.7 |
| | 2 (79) | 20.6 to 23.1 y | 38.2 ± 4.5 | 14.9 ± 1.2 | −0.0009 ± 1.3 |
| | 3 (79) | 23.1 to 26.1 y | 37.8 ± 4.7 | 14.9 ± 1.3 | −0.006 ± 1.2 |
| | 4 (79) | 26.1 to 29.9 y | 38.9 ± 4.6 | 14.8 ± 1.4 | 0.006 ± 0.8 |
| | 5 (79) | 29.9 to 40.1 y | 38.1 ± 3.7 | 14.7 ± 1.3 | 0.115 ± 0.8 |
| Familial loading score‡ | 1 (78) | −0.61 to −0.31 | 37.8 ± 4.8 | 15.0 ± 1.4 | 0.18 ± 1.5 |
| | 2 (78) | −0.31 to −0.26 | 38.6 ± 4.2 | 14.9 ± 1.3 | −0.10 ± 0.5 |
| | 3 (77) | −0.26 to −0.22 | 38.7 ± 4.3 | 14.7 ± 1.3 | −0.06 ± 0.5 |
| | 4 (78) | −0.22 to −0.10 | 38.5 ± 4.0 | 14.8 ± 1.2 | −0.05 ± 0.8 |
| | 5 (78) | −0.10 to 5.90 | 38.2 ± 4.3 | 14.7 ± 1.3 | 0.35 ± 1.2 |
| Severity of illness§ | 1 (77) | 0.0 to 6.5 d | 39.3 ± 3.9 | 14.9 ± 1.3 | 0.03 ± 1.2 |
| | 2 (76) | 6.5 to 17.3 d | 38.0 ± 4.4 | 14.8 ± 1.3 | −0.003 ± 0.7 |
| | 3 (76) | 17.3 to 37.3 d | 38.6 ± 4.5 | 14.9 ± 1.3 | −0.09 ± 0.6 |
| | 4 (76) | 38.2 to 70.3 d | 37.9 ± 4.6 | 14.8 ± 1.3 | −0.25 ± 0.8 |
| | 5 (76) | 70.7 to 309.4 d | 38.0 ± 4.1 | 14.8 ± 1.4 | 0.08 ± 1.4 |

** Tests for linear trend not significant.*
*† Age at first diagnosis with schizophrenia.*
*‡ See Verdoux et al[23] for details.*
*§ Average number of days in hospital per year.*

cases who were both born and educated in Helsinki, thus excluding some children with unstable family environments and residential instability. However, we would still have captured those children who moved frequently but remained within Helsinki, and we have attempted to control, as far as possible, for factors that might influence migration by using nonmigratory controls.

Our main "positive" finding is that children who develop schizophrenia in adulthood perform significantly worse than their peers on sports and handicrafts (the nonacademic factor). One explanation is that sports and handicrafts test coordination skills, and that preschizophrenic children show deficits in motor coordination when compared with their peers. This explanation is supported by evidence from high-risk studies showing that children at genetic risk for schizophrenia are distinguished by impairments of motor development and fine motor coordination,[36-43] which appear to predict schizophrenia-spectrum disorders in adulthood.[39,43] Further evidence comes from general population birth cohort studies that show delayed motor milestones,[5] clumsiness,[6] and poor sports ability[44] in childhood as predictors of later schizophrenia, and from studies of childhood home-movie footage of schizophrenic patients showing left-sided neuromotor abnormalities during the first 2 years of life.[45,46] Such abnormalities are not confined to childhood: 33% to 60% of schizophrenic patients,[47-52] including neuroleptic-naive patients,[53,54] show "soft" neurological signs in adulthood.

However, an alternative explanation is that poor performance in sports and handicrafts may result from personality or motivational factors. In structured settings (such as mathematics class), individual differences may be minimized by situational pressures to conform. However, sports and handicrafts are "softer" subjects that represent the more social, unstructured aspects of the curriculum and reflect other abilities, such as artistic ability and teamwork. It may be these aspects of school life that preschizophrenic children find particularly difficult, and in which they express early schizoid tendencies. Previous case-control[55-57] and cohort[58-60] studies have described poor premorbid adjustment and poor social functioning in childhood among individuals with schizophrenia and significant behavioral differences from peers.

Our finding that preschizophrenic children were less likely than controls to progress to high school also provides support for childhood personality or motivational problems in schizophrenia. Many cases who were eligible to move into the academic stream by virtue of their ranking score in the grade 4 examination chose instead to stay on at the elementary school for an additional 4 years to finish their education. We do not know whether the preschizophrenic children were themselves reluctant to proceed, or were discouraged from doing so by parents or teachers. The high school was perceived at that time as a more stressful and demanding environment than the elementary school. Failure to finish school[61] and "lack of academic or vocational ambition"[62] have been noted previously as risk factors for schizophrenia. In the 1966 North Finland birth cohort, the proportion of preschizophrenic children "not in normal class" at age 14 years was about 3 times higher than in the comparison group.[9] Although we did not find any significant differences in the behavioral factor between cases and controls, it was a relatively crude measure that indexed mainly disruptive behaviors and unauthorized absences. It is possible that, had more detailed or specially designed teacher's ratings of behavior been available, as in the birth cohort and high-risk studies,[7,63] subtle behavioral differences would have emerged.

On balance, we favor the motor coordination explanation, rather than the personality or motivational explanation, for the poor performance in nonacademic sub-



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**Table 4. Finnish Elementary School Curriculum: Sports and Physical Exercise**

| Grade | Period | Curriculum |
|---|---|---|
| 1, 2 | Autumn and spring outdoor sports period | Athletics exercises; catching and throwing exercises as part of children's games |
| | Autumn and spring indoor sports period | Gymnastics and physical exercises according to schedule; games involving music and physical exercise |
| | Winter sports period | Skiing and skating |
| 3, 4 | Autumn and spring outdoor sports period | *Boys:* Athletics training including triathlon and relay races; Finnish baseball and football; cross-country races and orienteering |
| | | *Girls:* Relay races and athletics exercises; Finnish baseball; cross-country races and orienteering |
| | Autumn indoor sports period | Gymnastics and physical exercises according to schedule |
| | | *Boys:* 20-30 min must be spent on gymnastic apparatus |
| | | *Girls:* Separate classes for sports and for music exercises and traditional dance |
| | Winter sports period | Exercises as before, skiing, skating; ice hockey *(boys only)* |

jects among the preschizophrenic children. The Finnish elementary school curriculum during the 1950s and 1960s placed a great deal of emphasis on sports and handicrafts; at least 4 hours per week was devoted to these activities, and there was a rigorous schedule of skills and crafts to be mastered (**Table 4**).[25,26] The emphasis was on the acquisition of skills (such as catching, skiing, skating, and gymnastics) rather than team sports, particularly in the early years of schooling. Children were graded on their athletic ability or the quality of their handicrafts rather than on perceived effort or team spirit. Of course, the most parsimonious explanation, and the most difficult to disprove, is that our findings represent both motor coordination deficits and poor psychosocial adjustment in children who later develop schizophrenia. It is unclear how specific our findings are for schizophrenia; there is some evidence that patients with affective illness also show poor premorbid social adjustment and childhood developmental abnormalities.[57,64] Further longitudinal studies of other disordered groups would help elucidate this issue.

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## REFERENCES

1. Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders: longitudinal evidence from a birth cohort. *Arch Gen Psychiatry.* 1996;53:1033-1039.
2. Jones P. The early origins of schizophrenia. *Br Med Bull.* 1997;53:135-155.
3. Cannon M, Murray RM. Neonatal origins of schizophrenia. *Arch Dis Child.* 1998; 78:1-3.
4. Jones PB, Rantakallio P, Hartikainen A-L, Isohanni M, Sipila P. Schizophrenia as a long-term outcome of pregnancy, delivery and perinatal complications: a 28-year follow-up of the 1966 North Finland birth cohort. *Am J Psychiatry.* 1998; 155:355-364.
5. Jones PB, Rodgers B, Murray RM, Marmot MG. Child developmental risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet.* 1994;344: 1398-1402.
6. Crow TJ, Done DJ, Sacker A. Childhood precursors of psychosis as clues to its evolutionary origins. *Eur Arch Psychiatry Clin Neurosci.* 1995;245: 61-69.
7. Done DJ, Crow TJ, Johnstone EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ.* 1994; 309:699-703.
8. David A, Malmberg A, Brandt L, Allebeck P, Lewis G. IQ and risk for schizophrenia: a population-based cohort study. *Psychol Med.* 1997;27:1311-1323.
9. Isohanni I, Järvelin M-R, Nieminen P, Jones P, Rantakallio P, Jokelainen J, Isohanni M. School performance as a predictor of psychiatric disorder in adult life: a 28-year follow-up in the Northern Finland 1966 birth cohort. *Psychol Med.* 1998; 28:967-974.
10. Cannon M, Jones P. Schizophrenia: neuroepidemiology review series. *J Neurol Neurosurg Psychiatry.* 1996;61:604-613.
11. Jones P, Done DJ. From birth to onset: a developmental perspective of schizophrenia in two national birth cohorts. In: Keshavan KS, Murray RM, eds. *Neurodevelopmental Models of Psychopathology.* Cambridge, England: Cambridge University Press; 1997:119-136.
12. Rothman K, Greenland S. Case-control studies. In: Rothman K, Greenland S, eds. *Modern Epidemiology.* 2nd ed. Philadelphia, Pa: Lippincott-Raven Publishers; 1998:93-114.
13. Langholz R, Thomas S. Nested case-control and case-cohort methods of sampling from a cohort: a critical comparison. *Am J Epidemiol.* 1990;31:169-176.
14. World Health Organization. *International Classification of Diseases, Eighth Revision (ICD-8).* Geneva, Switzerland: World Health Organization; 1969.
15. World Health Organization. *International Classification of Diseases, Ninth Revision (ICD-9).* Geneva, Switzerland: World Health Organization; 1977.
16. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition.* Washington, DC: American Psychiatric Association; 1987.
17. Kuoppasalmi K, Lönnqvist J, Pylkkänen K, Huttunen M. Classification of mental disorders in Finland: a comparison of the Finnish classification of mental disorders in 1987 with *DSM-III-R* criteria. *Psychiatr Fennica.* 1989;20:65-81.
18. Lehtinen V, Joukamaa M, Lahtela K, Raitasalo R, Jyrkinen E, Maatela J, Aromaa A. Prevalence of mental disorders among adults in Finland: basic results from the Mini-Finland Health Survey. *Acta Psychiatr Scand.* 1990;81:418-425.
19. Isohanni M, Mäikikyrö T, Moring J, Rasanen P, Hakko H, Partanen U, Koiranen M, Jones PB. A comparison of clinical and research *DSM-III-R* diagnoses of schizophrenia in a Finnish national birth cohort. *Soc Psychiatry Psychiatr Epidemiol.* 1997;32:303-308.
20. Cannon TD, Kaprio J, Lönnqvist J, Huttunen M, Koskenvuo M. The genetic epidemiology of schizophrenia in a Finnish twin cohort: a population-based modeling study. *Arch Gen Psychiatry.* 1998;55:67-74.
21. Mäikikyrö T, Isohanni M, Moring J, Hakko H, Hovatta I, Lönnqvist J. Accuracy of register-based schizophrenia diagnoses in a genetic study. *Eur Psychiatry.* 1998; 13:57-62.
22. Central Statistics Office. *Alphabetical Coding Manual for Social Class Based on Occupations: Population Census 1970.* Helsinki, Finland: Central Statistics Office; 1971.



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23. Verdoux H, van Os J, Sham P, Jones P, Gilvarry K, Murray R. Does familiality predispose to both emergence and persistence of psychosis? *Br J Psychiatry*. 1996;168:620-626.
24. Suvisaari J, Haukka J, Tanskanen A, Lönnqvist J. Distinct age of onset and outcome in familial and sporadic schizophrenia. *Br J Psychiatry*. 1998;173:494-500.
25. Helsinki City Finnish Language Elementary Schools. Elementary school curriculum. Helsinki, Finland: State Printing Office; 1955.
26. Helsinki City Finnish Language Elementary Schools. Elementary school curriculum. Helsinki, Finland: State Printing Office; 1966.
27. Somerkivi U. The *History of Helsinki's Elementary Schools*. Helsinki, Finland: Finnish Literature Press Ltd; 1977.
28. Goldstein H. *Multilevel Statistical Models*. 2nd ed. London, England: Edward Arnold; 1995.
29. *Stata Statistical Software Release 5.0*. College Station, Tex: Stata Corp; 1995.
30. Goldstein H, Rasbash J, Plewis I, Draper D, Browne W, Yang M, Woodhouse G, Healy M. *A User's Guide to MLwiN (Version 1.0)*. London, England: Institute of Education; 1998.
31. Aylward E, Walker E, Bettes B. Intelligence in schizophrenia: meta-analysis of the research. *Schizophr Bull*. 1984;10:430-459.
32. Jones P. Childhood motor milestones and IQ prior to schizophrenia: results from a 43 year old British birth cohort. *Psychiatr Fennica*. 1995;26:63-80.
33. Mednick SA, Schulsinger F. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. *J Psychiatr Res*. 1968;6:267-291.
34. Parnas J, Teasdale TW, Schulsinger F. Institutional rearing and diagnostic outcome in children of schizophrenic mothers: a prospective high-risk study. *Arch Gen Psychiatry*. 1985;42:762-769.
35. Tienari P. Interaction between genetic vulnerability and family environment: the Finnish adoptive family study of schizophrenia. *Acta Psychiatr Scand*. 1991;84:460-465.
36. Reider RO, Nichols PL. Offspring of schizophrenics, III: hyperactivity and neurological soft signs. *Arch Gen Psychiatry*. 1979;36:665-674.
37. Hans SL, Marcus J. Neurobehavioral development of infants at risk for schizophrenia. In: Walker EF, ed. *Schizophrenia: A Life-Course Developmental Perspective*. New York, NY: Academic Press; 1991:35-53.
38. Marcus J, Hans SL, Auerbach JG, Auerbach AG. Children at risk for schizophrenia: the Jerusalem Infant Development Study, II: neurobehavioral deficits at school age. *Arch Gen Psychiatry*. 1993;50:797-809.
39. Fish B. Infant predictors of the longitudinal course of schizophrenic development. *Schizophr Bull*. 1987;13:395-409.
40. Fish B, Marcus J, Hans SL, Auerbach JG, Perdue S. Infants at risk for schizophrenia: sequelae of a genetic neurointegrative defect: a review and partial replication of pandysmaturation in the Jerusalem Infant Development Study. *Arch Gen Psychiatry*. 1992;49:221-235.
41. Erlenmeyer-Kimling L, Kestenbaum C, Bird H, Hilldoff U. Assessment of the New York High-Risk Project subjects in sample A who are now clinically deviant. In: Watt NF, Anthony EJ, Wynne LC, Roll JE, eds. *Children at Risk for Schizophrenia*. New York, NY: Cambridge University Press; 1984:227-240.
42. Marcus J, Hans SL, Nagler S, Auerbach JG, Mirsky AF, Aubrey A. A review of the NIMH Israeli Kibbutz-City Study and the Jerusalem Infant Development Study. *Schizophr Bull*. 1987;13:425-438.
43. Olin S-CS, Mednick SA. Risk factors of psychosis: identifying vulnerable populations premorbidly. *Schizophr Bull*. 1996;22:223-240.
44. Jones P, Murray R, Rodgers B. Childhood risk factors for adult schizophrenia in a general population birth cohort at age 43 years. In: Mednick SA, Hollister JM, eds. *Ne ural Development and Schizophrenia*. New York, NY: Plenum Press; 1995: 151-176.
45. Walker EF, Lewine RJ. Prediction of adult-onset schizophrenia from childhood home-movies of the patients. *Am J Psychiatry*. 1990;47:1052-1056.
46. Walker EF, Savoie T, Davis D. Neuromotor precursors of schizophrenia. *Schizophr Bull*. 1994;20:441-451.
47. Woods BT, Kinney DK, Yurgelun-Todd D. Neurologic abnormalities in schizophrenic patients and their families, I: comparison of schizophrenic, bipolar, and substance abuse patients and normal controls. *Arch Gen Psychiatry*. 1986;43: 657-663.
48. Rossi A, DeCatalado S, DiMichele V, Manna V, Ceccoli S, Stratta P, Casacchia M. Neurological soft signs in schizophrenia. *Br J Psychiatry*. 1990;157:735-739.
49. Lane A, Colgan K, Moynihan F, Burke T, Waddington JL, Larkin C, O'Callaghan E. Schizophrenia and neurological soft signs: gender differences in clinical correlates and antecedent factors. *Psychiatry Res*. 1996;64:105-114.
50. Heinrichs DW, Buchanan RW. Significance and meaning of neurological signs in schizophrenia. *Am J Psychiatry*. 1988;145:11-18.
51. Walker E, Shaye J. Familial schizophrenia: a predictor of neuromotor and attentional abnormalities in schizophrenia. *Arch Gen Psychiatry*, 1982;32:1153-1156.
52. Griffiths TD, Sigmundsson T, Takei N, Rowe D, Murray RM. Neurological abnormalities in familial and sporadic schizophrenia. *Brain*. 1998;121:191-203.
53. Gupta S, Andreasen NC, Arndt S, Flaum M, Schultz SK, Hubbard WC, Smith M. Neurological soft signs in neuroleptic-naive and neuroleptic-treated schizophrenic patients and in normal controls. *Am J Psychiatry*. 1995;152:191-196.
54. Sanders RD, Keshavan MS, Schooler N. Neurological examination abnormalities in neuroleptic-naive patients with first-break schizophrenia: preliminary results. *Am J Psychiatry*. 1994;151:1231-1233.
55. Offord DR, Cross LA. Behavioral antecedents of adult schizophrenia: a review. *Arch Gen Psychiatry*. 1969;21:267-283.
56. Rutter M. Psychopathology and development: childhood antecedents of adult psychiatric disorder. *Aust N Z J Psychiatry*. 1984;18:225-234.
57. Cannon M, Jones P, Gilvarry C, Rifkin L, McKenzie K, Foerster A, Murray RM. Premorbid social functioning in schizophrenia and bipolar disorder: similarities and differences. *Am J Psychiatry*. 1997;154:1544-1550.
58. Malmberg A, Lewis G, David A, Allebeck P. Premorbid adjustment and personality in people with schizophrenia. *Br J Psychiatry*. 1998;172:308-313.
59. Watt NF. Patterns of childhood social development in adult schizophrenics. *Arch Gen Psychiatry*. 1978;35:160-165.
60. Watt NF. Childhood and adolescent routes to schizophrenia. In: Ricks DF, Roff M, Thomas A, eds. *Life History Research in Psychopathology*. Minneapolis, Minn: Minnesota Press; 1974:194-211.
61. Keith SJ, Regier DA, Rae RS. Schizophrenic disorders. In: Robins LN, Regier DA, eds. *Psychiatric Disorders in America: The Epidemiological Catchment Area Study*. New York, NY: Free Press; 1991:33-55.
62. Hartmann E, Milofsky E, Vaillant G, Oldfield M, Falke R, Ducey C. Vulnerability to schizophrenia: prediction of adult schizophrenia using childhood information. *Arch Gen Psychiatry*. 1984;41:1050-1056.
63. Olin SS, John RS, Mednick SA. Assessing the predictive value of teacher reports in a high risk sample for schizophrenia: an ROC analysis. *Schizophr Res*. 1995; 16:53-66.
64. van Os J, Jones P, Lewis G, Wadsworth M, Murray R. Developmental precursors of affective illness in a general population birth cohort. *Arch Gen Psychiatry*. 1997;54:625-631.



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