

Risk factors for psychopathology in children with intellectual disability: a prospective longitudinal population-based study

J. L. Wallander,¹ M. C. Dekker² & H. M. Koot³

¹ Sociometrics Corporation, Los Altos, CA, USA

² Erasmus Medical Center, Department of Child and Adolescent Psychiatry, Rotterdam, the Netherlands

³ Vrije Universiteit Amsterdam, Amsterdam, the Netherlands

Abstract

Background This study examined risk factors for the development of psychopathology in children with intellectual disability (ID) in the developmental, biological, family and social-ecological domains.

Methods A population sample of 968 children, aged 6–18, enrolled in special schools in the Netherlands for educable and trainable ID were assessed at Time 1. A random 58% were re-contacted about 1 year later, resulting in a sample of 474 at Time 2.

Results Psychopathology was highly consistent over 1 year. Risk factors jointly accounted for significant, but small, portions of the variance in development of psychopathology. Child physical symptoms, family dysfunction and previous parental mental health treatment reported at Time 1 were uniquely associated with new psychopathology at Time 2.

Conclusions Prevention and early intervention research to find ways to reduce the incidence of psychopathology, possibly targeting family functioning, appear important.

Keywords children, intellectual disability, longitudinal design, psychopathology, risk factors

Correspondence: Jan L. Wallander, Sociometrics Corp., 170 State St., #260, Los Altos, CA 94022-2812, USA (e-mail: janw@socio.com).

Introduction

Studies conducted prior to mid-1990s reported an inconsistent prevalence of psychopathology in children with intellectual disability (ID), from 9% to 87% (e.g. see Einfeld & Tonge 1996a; Dykens 2000; Wallander *et al.* 2003). This wide range may be explained by the use of different definitions of both ID and psychopathology, measurements of psychopathology, ranges of IQ defining the study population and samples (e.g. general population vs. referred sample). More recent studies have improved with standardized assessments of psychopathology and clearly defined samples (Einfeld & Tonge 1996b; Wallander *et al.* 1996; Dekker *et al.* 2002; Emerson 2003). They report a prevalence between 35% and 49% when applying standardized criteria for psychopathology, primarily using the general population instruments developed by Achenbach (1991). These prevalences can be compared with the 10% to 15% commonly reported for the general population of children (Verhulst & Koot 1995). Therefore, recent literature indicates that children with ID are a group at risk for psychopathology, experiencing about a three-fold increase compared with the general population.

Assigning risk status to a group implies that not all members of the group realize the negative outcome. In fact, the majority of children with ID do not experience significant psychopathology. This raises

the question, what differences are there between those who do and do not display psychopathology? Identifying a more refined profile of risk factors has three goals: identify potential aetiological processes; identify subgroups of children with ID that should be targeted for prevention or early intervention services; and suggest how such services may best be implemented. Until recently (e.g. Tonge *et al.* 2001; Wallander *et al.* 2001; Dekker & Koot 2003a; Emerson 2003), there had been few studies on risk factors for psychopathology in children with ID. However, there is a substantial body of evidence regarding risk factors for psychopathology in the general child population (cf. Rutter 1999; Masten 2001), which can guide examination of such factors for children with ID. We assume some similarity in risk factors between children in the general population and with ID.

It is of heuristic value to organize risk factors across domains, such as the biological, individual psychological and family, as well as the broader social ecology domains. In the case of children with a range of intellectual deficits and chronological ages, however, it becomes difficult to measure most individual psychological factors (e.g. emotional reactivity, self-perceptions, individual competencies) because they require self-report. Moreover, laboratory-based assessments are not feasible when surveying a large population sample. For these methodological reasons, we excluded the individual psychological risk domain from this study.

We focused within the *biological risk* domain on the current and past physical health of the child. Children with ID experience biological dysfunction at a higher rate than children in general (cf. Horowitz & Haritos 1998; Bryant & Maxwell 1999). This is due to, for example, the pre- and neo-natal insults that are among the causes of ID and a higher exposure to poverty (e.g. Bryant & Maxwell 1999; Ramey & Ramey 1999). Numerous *family* risk factors have been implicated for development of psychopathology. Past or current parental psychopathology is a commonly identified one (e.g. Chassin *et al.* 1999; Mesman & Koot 2000). Family dysfunction also is a significant risk factor for the general population (e.g. Dowling & Gorell-Barnes 1999; Ingoldsby *et al.* 2001). *Social-ecological* stratification, capturing typical experiences of a defined group of individuals or families, is associated with psychopathology, such as fam-

ily structure, socio-economic and ethnic minority status (e.g. Rutter & Sandberg 1992).

In addition to these generally applicable risk factors, there are also other factors associated with the compromised developmental trajectory inherent in ID. That is, there are wide-ranging developmental differences among children within this group that can be included in a *developmental risk* domain. First, ID encompasses a range of intellectual levels. It is unclear whether those with mild ID experience more psychopathology than those with moderate ID (e.g. Koller *et al.* 1982; Gillberg *et al.* 1986). Moreover, there are individual differences in other adaptive deficits co-occurring with ID, such as in the daily living, communication and social skills areas that may contribute to psychopathology (Sparrow *et al.* 1984).

Our aim was thus to test associations in youth with ID between the development of psychopathology and candidate risk factors in four domains. We ordered the risk domains from more proximal to distal, starting with characteristics of the individual (Developmental and Biological Domains), followed by the family (Family domain) and finally the broader social ecology (Social-ecological domain). Additional aims were to examine the stability of psychopathology and to distinguish between variables that may be associated with psychopathology concurrently and those that can be construed as risk factors in that they are associated with the *development* of psychopathology over time and precede the outcome (Kraemer *et al.* 1997).

Methods

Participants and procedures

Sampling and recruitment

The sampling frame was the registers of schools for children with educable and trainable ID in the province of Zuid-Holland in the Netherlands. When this study was initiated in 1996, the main criterion to enter a school for students with educable ID was an IQ between 60 and 80, and for those with trainable ID between 30 and 60, and that there was no severe physical or sensory disability nor need for constant supervision. Of school-age students in the Netherlands, 1.6% attended a school for educable ID and 0.4% for trainable ID (CBS 1993). A 20% random

sample of children was drawn from each of the 115 schools for educable and trainable ID that participated in this study (out of a total of 132 possible schools). Parents/guardians (hereafter, both are referred to as parents) of the sampled children were mailed information about the study with a form to mail back to permit staff to contact them. Reminder notes followed for parents who did not respond to the initial mailing. Children were included in the study if they were at the ages of 6–18, lived at home for four or more days per week and had at least one parent who had enough comprehension of the Dutch language to be interviewed. Most measures were completed in parent interviews at home. A subset were administered to parents by mail and collected during home visits, and archival data were collected from the schools.

Enrolled sample

Of the 1615 sampled children, 141 (8.7%) were excluded because of parental language deficits, 7 (0.4%) because of being outside the age range when a visit could be made and 71 (4.4%) because they had left school or moved since the sample roster was formed. Of the 1396 eligible, 164 (11.8%) refused to participate, 231 (16.6%) could not be contacted by the research staff and 33 (2.4%) returned incomplete research material. The final sample size was $n = 968$ at Time 1, which is 83.1% of those who could be contacted by the research staff and 69.3% of those eligible. This sample ($M = 11.8$ year, $SD = 3.0$) is described in Table 1. Prevalence of psychopathology has been reported by Dekker & Koot (2003b).

Follow-up sample

About 1 year later, resources allowed for a random sample $n = 557$ (58% of the Time 1 participants) to be re-contacted. The mean interval between Times 1 and 2 assessments was 410 days ($SD = 80$). Six families were excluded because they did not meet the language requirements for the more complicated interview at Time 2 and five children were no longer living at home, yielding an eligible $n = 546$. Of these eligible, 11 (2.0% of eligible for follow-up) parents could not be contacted and 61 (11.2%) refused to participate, resulting in Time 2 $n = 474$ (86.8% of eligible). The follow-up sample was administered the measure of psychopathology described below. No sig-

Table 1 Sample characteristics

	%
ID level	
Educable	69
Trainable	31
Sex	
Male	60
Female	40
SES	
Low	57
Medium to high	43
Family	
Single parent	16
Two parents	84
Ethnicity	
At least one parent is non-Dutch	20
No parent is non-Dutch	80

ID, intellectual disability; SES, socio-economic status.

nificant ($P > 0.05$) differences were found between the original eligible Time 1 and Time 2 sample in the distribution of sex [$\chi^2_{(1)} = 3.1$], parental educational level [$\chi^2_{(4)} = 4.1$], socio-economic status (SES) [$\chi^2_{(1)} = 2.2$] or year of birth [$\chi^2_{(14)} = 7.5$]. There was no significant difference in the percentage of children scoring in the deviant range of the Total [$\chi^2_{(1)} = 0.8$], Externalizing [$\chi^2_{(1)} = 0.32$] and Internalizing [$\chi^2_{(1)} = 2.4$] Behavior Problems Scales of the Child Behavior Checklist (CBCL). However, those parents who did not participate in any phase and who gave information on SES ($n = 122$) were more likely to have lower SES (68.9%) compared with Time 1 participants [55.4%; $\chi^2_{(1)} = 11.0$].

Measures

Psychopathology domain (three dimensions)

This domain was assessed with the Behavior Problem Scales of the CBCL (Achenbach 1991) completed by the parent. The CBCL consists of 120 problem behaviours that are rated from 'not true' (0) to 'very true or often true' (2) for the past 6 months. Norms have been established on 2227 children for the Dutch translation (Verhulst *et al.* 1996). Extensive reliability and validity analyses indicate that the scales have

satisfactory psychometric properties, which have been confirmed for the Dutch translation (e.g. De Groot *et al.* 1994; Verhulst *et al.* 1996). It might be suggested that the CBCL, as a general population instrument, is not optimal for assessing psychopathology in children with ID. However, both US and Dutch versions have been used with ID samples (e.g. Curfs *et al.* 1991; Floyd & Phillippe 1993; Wallander *et al.* 1996, 2001; Van Lieshout *et al.* 1998; Dekker *et al.* 2002). Analysis with the current sample have shown reasonable cross-informant correlations between parent and teacher report, and both good convergent validity with a psychopathology instrument specifically developed for children with ID and predictive validity for DSM (Diagnostic and Statistical Manual of Mental Disorder)-IV diagnosis (Dekker *et al.* 2002; Dekker & Koot 2003a). Our analyses used the raw scores from the (1) Total (2) Internalizing, and (3) Externalizing Behavior Problem Scales to focus on broad dimensions of psychopathology and reduce the use of multiple dependent tests that would have resulted if subscales had been employed. Cronbach's alpha ranged 0.88–0.95 in the current sample for these broad-band scales, which are comparable (0.90–0.97) with those reported for the Dutch general population (Verhulst *et al.* 1996).

Developmental risk domain (four factors)

(1) *Intellectual Disability* was indexed by school placement. Children had been assigned to a school for educable (IQ generally between 60 and 80) or trainable ID (IQ generally between 30 and 60), coded 0 and 1, respectively. Recent scores on recognized IQ tests could be obtained from the school records for 55% of the enrolled children. Children at educable ID schools had a mean IQ = 71.9 (SD = 8.5, $n = 387$) vs. 56.2 (SD = 9.7, $n = 149$) for those at trainable ID schools. The additional developmental risk variables of (2) *communication* (3) *socialization* and (4) *daily living disability* were assessed with subscales (15 items each) of the Dutch translation of the Vineland Screener (VS; Sparrow *et al.* 1993). Administered to the parent by a trained interviewer, the VS measures personal and social abilities of children. From a pool of 261 items from the Vineland Adaptive Behavior Scales (VABS; Sparrow *et al.* 1984), 45 were selected for the VS based on ease of administration, reliability, domain coverage and strength of correlation with the

VABS. Correlations between the VS and the VABS domain scores range from 0.92 to 0.95 for 6- to 18-year-olds (Sparrow *et al.* 1993). Cronbach's alpha in the present sample was 0.87 for the communication, 0.71 for socialization and 0.84 for daily living disability domain.

Biological risk domain (three factors)

(1) *Physical Symptoms* were assessed with the Wahler Physical Symptom Checklist (WPSI; Wahler 1983). The WPSI measures how often each of 41 somatic complaints (e.g. nausea, headaches, skin trouble, difficulty in swallowing) is bothersome (almost never, once a year, once a month, once a week, twice a week, nearly every day). It has shown high internal consistency (KR20 0.85–0.93 across samples), and good differentiation among healthy individuals, those who manifest complaints in conjunctions with psychiatric disorders, and disability claimants. Cronbach's alpha was 0.78 for parent report in the present sample. Total scores were dichotomized into high physical complaints (highest 25%, total WPSI score >18) vs. low (coded 1 vs. 0). (2) *Chronic Disease History* was measured by asking the parent whether the child had a physical condition which impaired the child's daily life for at least 3 months per year (coded Yes = 1 and No = 0). (3) *Lengthy Hospitalization* was measured by asking the parent whether the child has a physical condition that has caused hospitalization of at least 1 month or will cause in the near future (coded Yes = 1 and No = 0).

Family risk domain (three factors)

(1) *Parental Distress* was assessed by parents completing a short form of the Young Adult Self-Report (YASR; Achenbach 1997), which uses the 29 of the 110 items that discriminated best between psychiatric referred and non-referred subjects (Verhulst & van der Ende 1997). These items mainly represent internalizing problems. The 29-item problem score yielded a Cronbach's alpha of 0.90 in the present sample. (2) *Parental Mental Health Treatment History* was measured by asking the parent whether she/he or the other parent, as applicable, had ever received in- or out-patient treatment for a psychiatric disorder or emotional and behavioural problems (coded Yes = 1 and No = 0). (3) *Family Dysfunction* was assessed with the 12-item general functioning subscale of the

McMaster Family Assessment Device (FAD-GF; Byles *et al.* 1988). The items reflect behaviours believed to be essential for the functioning of families, such as problem solving, communication, roles, affective responsiveness and affective involvement. Byles *et al.* (1988) reported a Cronbach's alpha of 0.86 and a test-retest reliability of 0.71 for the FAD-GF subscale. Subscales of the FAD have been found to discriminate between psychiatric families and non-clinical families (Byles *et al.* 1988). Cronbach's alpha was 0.85 in the present sample.

Social-ecological risk domain (four factors)

(1) *Life Events Exposure* was assessed with 15 items [e.g. a parent leaving the family, death of a family member, new (born) children in the family] from the Life Events Questionnaire (Berden *et al.* 1990). The parent indicated which events had occurred to the child in the past 2 years, which were summed. Demographic status was assessed with a typical checklist from which dichotomies were coded (Yes = 1, No = 0) for: (2) *Low SES*, ascribed when the parent indicated being unemployed, unskilled worker or a worker with low vocational training; (3) *Single Parent*; and (4) *Ethnic Minority*, which was attributed to when at least one parent had immigrated to the Netherlands.

Data analysis

Multiple regression analysis to test the associations between candidate risk factors and psychopathology were conducted separately with the three dimensions of psychopathology as dependent variables. Controlling for Time 1 psychopathology enables the testing of risk factors associated with development of psychopathology from Time 1 to Time 2 (Kraemer *et al.* 1997). Sex and age were entered as additional control variables. All analyses were completed on the 'total' and 'internalizing' scores, calculated in the standard fashion as well as without including the items constituting the Somatic narrow-band scale. The latter modified scores were used to remove the possible overlap when considering associations between 'biological risk' factors and psychopathology scores based in part on items addressing physical symptoms. Because there were no substantive differences in the findings between these two approaches, only those pertaining

to scores without the contribution of somatic items are reported here. Risk domains were entered hierarchically in the regression models representing their proximity in potentially influencing the development of psychopathology by Time 2 (see the aims). The change in variance accounted for by the set of variables in a risk domain was inspected at each step. If significant, the regression weights associated with specific risk factors within that domain were inspected for significance. For comparative purposes, these analyses were repeated with the Time 1 psychopathology dimensions as the dependent variables.

Results

Consistency in psychopathology

Psychopathology was found to decrease statistically significantly on average over the 1-year interval (all $r_s \geq 3.24$, all $P_s < 0.001$), although absolute changes in raw scores must be considered small [M (SD) total: Time 1 = 33.77 (22.35), Time 2 = 30.10 (22.07); internalizing: Time 1 = 9.15 (7.71), Time 2 = 8.25 (7.58); externalizing: Time 1 = 11.23 (9.31), Time 2 = 10.23 (9.59)]. Moreover, individuals' levels of psychopathology were ordered highly consistently from Time 1 to Time 2 (all $r_s \geq 0.75$, $P_s < 0.001$).

Longitudinal analysis

Table 2 presents the findings for the longitudinal analyses in the top panel. Significant portions (3–5%, $P < 0.01$) of the variance in the development of all dimensions of psychopathology from Time 1 to Time 2 was accounted for by the joint risk domains. Only the Biological Risk and Family Risk domains contributed to significant portions of the variance in new Total and Internalizing psychopathology. Only Biological Risk contributed to Externalizing psychopathology. Neither Developmental Risk, entered first following control variables, nor Social-ecological Risk, entered last in the hierarchical regression models, contributed significantly to the development of psychopathology in any of the three dimensions. Inspection of significant individual risk factors, reported in Table 2, indicates that Physical Symptoms and Parental Mental Health Treatment contributed to unique variance in all dimensions of psychopathology. In addition, Family Dysfunction

Table 2 Multiple regression analysis of associations between risk domains and dimensions of psychopathology

	Total (excl. somatic)	Internalizing (excl. somatic)	Externalizing
Longitudinal analysis (<i>n</i> = 474)			
Predictors (<i>R</i> ² -change)			
(Sex, age)	(0.01)	(0.02 ^{**})	(0.03 ^{**})
(Time 1 pathology)	(0.62 ^{***})	(0.58 ^{***})	(0.56 ^{***})
Developmental risk	0.00	0.01	0.00
Biological risk	0.02 ^{***}	0.02 ^{***}	0.02 ^{***}
Family risk	0.01 ^{***}	0.02 ^{***}	0.01 [*]
Social-ecological risk	0.00	0.00	0.00
Total for risk domains (<i>R</i> ²)	0.03 ^{**}	0.05 ^{***}	0.03 ^{**}
Significant risk factors (b)	Physical Symptoms (0.13 ^{***}) Family Dysfunction (0.07 [*]) Parental Treatment (0.09 ^{**})	Physical Symptoms (0.15 ^{***}) Parental Distress (0.08 [*]) Family Dysfunction (0.07 [*]) Parental Treatment (0.10 ^{**})	Physical Symptoms (0.14 ^{***}) Parental Treatment (0.08 [*])
Concurrent analysis (<i>n</i> = 968)			
Predictors (<i>R</i> ² -change)			
(Sex, age)	(0.00)	(0.01 [*])	(0.02 ^{**})
Developmental risk	0.14 ^{***}	0.11 ^{***}	0.12 ^{***}
Biological risk	0.19 ^{***}	0.18 ^{***}	0.09 ^{***}
Family risk	0.07 ^{***}	0.06 ^{***}	0.06 ^{***}
Social-ecological risk	0.01 ^{**}	0.01 ^{**}	0.01 ^{**}
Total for risk domains (<i>R</i> ²)	0.41 ^{***}	0.36 ^{***}	0.28 ^{***}
Significant risk factors (b)	Social Disability (0.28 ^{***}) Physical Symptoms (0.33 ^{***}) Parental Distress (0.27 ^{***}) Ethnic Minority (−0.09 ^{**})	Social Disability (0.23 ^{***}) ID Level (−0.12 ^{**}) Physical Symptoms (0.27 ^{***}) Parental Distress (0.26 ^{***})	Social Disability (0.30 ^{***}) Physical Symptoms (0.22 ^{***}) Parental Distress (0.21 ^{***}) Family Dysfunction (0.10 ^{***})

P* < 0.05; *P* < 0.01; ****P* < 0.001.

The variables in each predictor domain were entered as a set in the indicated order. Internalizing and total behavioural problem scores were calculated excluding the items on the Somatic narrow-band scale.

ID, intellectual disability; excl., excluding.

contributed uniquely to the variance in Total and Internalizing problems, while Parental Distress did so for Internalizing problems only.

Given the well-documented association between maternal distress and the reporting of behavioural problems in children (e.g. Velez *et al.* 1989; Williams *et al.* 1990), Parental Distress was entered as a control variable in an additional set of multiple regression analyses like the previous set. The total amount of variance contributed by the risk domains in each dimension of psychopathology was reduced less than 0.5%. Thus, the substantive findings were replicated.

Concurrent analyses

Table 2 presents the findings for the concurrent analyses in the bottom panel. Significant

(*P* < 0.001) and large portions (28–41%) of the variance in all three dimensions of psychopathology are accounted for jointly by the four risk domains. Each risk domain contributes to additional unique variance when entered in the predetermined order, with particularly large portions being associated with the most proximal domains of Developmental Risk and Biological Risk. Social Disability, Physical Symptoms and Parental Distress were individual variables being associated with concurrent psychopathology in all dimensions. When controlling for Parental Distress in the concurrent analysis, the contribution by the risk domains in each dimension of psychopathology was reduced substantially (15–25% less variance accounted for). However, the risk domains still accounted for significant portions of the variance (11–18%) in concurrent psychopathol-

ogy discounting the contribution of Parental Distress.

Discussion

Assessing psychopathology over a year-long period highlights the substantial consistency in problem behaviours being reported for children with ID. The 1-year stability observed herein ($rs > 0.75$) is higher than reported for the general population [e.g. Achenbach (1991) reported 7-month stability $rs = 0.49-0.56$], and the absolute level of psychopathology does not dissipate much over this period. Clinicians must realize that children with ID are at significant risk for psychopathology, which does not typically disappear over time. This has been demonstrated in numerous studies (e.g. Einfeld & Tonge 1996b; Wallander *et al.* 1996; Linna *et al.* 1999; Stromme & Diseth 2000; Dekker *et al.* 2002).

However, within this group some children are at higher risk than others. The most important risk factor for psychopathology at any given time is earlier psychopathology. Risk factors identified for internalizing and externalizing problems were not differentiated. Rather, physical symptoms, together with parental distress and family dysfunction, uniquely predicted the development of psychopathology in all dimensions. Individual differences in developmental competencies or social-ecological context did not predict new psychopathology.

The association between physical symptoms and psychopathology exists when somatic indications of psychopathology are excluded from consideration, and may suggest a biological vulnerability for psychopathology. Biological dysfunction exists in children with ID at a higher rate than in the general population (cf. Horowitz & Haritos 1998; Bryant & Maxwell 1999), even in the absence of clearly identified organic causes for their disability. In addition to jeopardizing physical health, such biological dysfunction may increase the risk for psychopathology.

This study replicated in children with ID (cf. Emerson 2003) the often reported association between parental and family dysfunction and psychopathology in children in general (e.g. Chassin *et al.* 1999; Dowling & Gorell-Barnes 1999; Mesman & Koot 2000; Ingoldsby *et al.* 2001). This finding does not identify whether parental and family dysfunction

is among the causes or consequences of psychopathology. Likely, this association exists due to a transactional process, including genetic and environmental transmissions of psychological dysfunction in the child, which may in turn increase the stress on the family members and impair their functioning. A number of studies have shown that people with ID are at risk of psychological stress (e.g. Bramston *et al.* 1999; Emerson 2003), a great deal of which may result from negatively perceived interpersonal relationships (e.g. Bender *et al.* 1999; Bramston & Fogarty 2000). It may well be that parental and family dysfunction further taxes the coping capacities of these children.

A longitudinal analysis of correlates of psychopathology provides different information from a concurrent analysis. While candidate risk factors jointly appear to be strongly related to psychopathology, when examined concurrently, the prediction of new psychopathology is much less powerful. Moreover, developmental deficiencies are correlated with concurrent psychopathology, yet do not predict new psychopathology. Physical symptoms and parental distress are associated with both concurrent and new psychopathology. It seems that the potential predictive power of several cross-sectionally related factors is reduced to zero by the very strong consistency of the children's psychopathology. It is only the longitudinal correlates that can be construed as true risk factors in that they precede the outcome (Kraemer *et al.* 1997).

Limitations of this study include the reliance of school placement as an index of ID level. Whereas mean IQs were different between children at educable and trainable schools, there was some overlap between their distributions. Also, there was an underrepresentation of low-SES families (albeit still at 57%) compared with the population of children with ID. The results do not inform about children residing in institutional settings, nor do they generalize to children with more severe ID or to children with severe additional sensory or physical conditions. Moreover, the measure of psychopathology used herein, the CBCL, was designed for typically developing children. It may not therefore capture certain problem behaviours that occur more commonly in people with lower IQ, such as stereotypic behaviour, self-injury and pica. Thus, this is expected to have been a lesser problem for youth from the schools for

educable ID compared with youth from the schools for trainable ID. Another noteworthy limitation is that the 1-year interval may not have been sufficient for some risk factors to exert their expected influence on the development of psychopathology.

No causal direction should be inferred from the present findings, as it could not be shown that changes in the risk factors caused changes in the outcomes. Although important significant associations between risk factors and psychopathology were detected, inclusion of other possible risk factors might improve predictive power. Examples might include early life or life time risk factors, familial ID, genetic deficiencies related to ID, inadequate psychological competencies and peer rejection. Future longitudinal studies are needed to examine factors predicting onset and prognosis of psychopathology in children with ID. Information is needed about developmental tracks of psychopathology in children with ID, the effects of major life transitions, and their associated risk and protective factors. These needs require longer-term longitudinal studies than the study conducted here.

Because of the strong stability in psychopathology in children with ID, it is imperative to stimulate prevention and early intervention research to find ways to reduce the incidence of psychopathology. Routine screening may be called for, given a typical prevalence of about 40% in this population. A general population instrument such as the CBCL may serve this need in children with mild to moderate ID. Problem behaviours in children with ID may otherwise go undetected, in part due to 'diagnostic overshadowing' (White *et al.* 1995). This occurs when behaviours indicative of psychopathology are assumed to exist as a function of the ID, and therefore not warrant clinical attention. Once identified, interventions mainly focused on the child's psychopathology and in part on the functioning of family members, especially parents, are suggested by the present results (cf. Emerson 2003).

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References

- Achenbach T. M. (1991) *Manual for the Child Behavior Checklist/4-18 and 1991 Profiles*. Department of Psychiatry, University of Vermont, Burlington, VT.
- Achenbach T. M. (1997) *Manual for the Young Adult Self-Report and Young Adult Behavior Checklist*. Department of Psychiatry, University of Vermont, Burlington, VT.
- Bender W. N., Rosenkrans C. B. & Crane M. (1999) Stress, depression, and suicide among students with learning disabilities: Assessing the risk. *Learning Disability Quarterly* **22**, 143-56.
- Berden G. F., Althaus M. & Verhulst F. C. (1990) Major life events and changes in the behavioural functioning of children. *Journal of Child Psychology and Psychiatry* **31**, 949-59.
- Bramston P. & Fogarty G. (2000) The assessment of emotional distress experienced by people with an intellectual disability: A study of different methodologies. *Research in Developmental Disabilities* **21**, 487-500.
- Bramston P., Fogarty G. & Cummins R. A. (1999) The nature of stressors reported by people with an intellectual disability. *Journal of Applied Research in Intellectual Disabilities* **12**, 1-10.
- Bryant D. M. & Maxwell K. L. (1999) The environment and mental retardation. *International Review of Psychiatry* **11**, 56-67.
- Byles J., Byrne C., Boyle M. H. & Offord D. R. (1988) Ontario Child Health Study: reliability and validity of the general functioning subscale of the McMasters Family Assessment Device. *Family Process* **27**, 97-104.
- CBS (1993) *Standaard Beroepen Classificatie 1992* [Standard Classification of Professions], 1992 edition 's-Gravenhage: sdu/uitgeverij/cbs-publikaties.
- Chassin L., Pitts S. C., DeLucia C. & Todd M. (1999) A longitudinal study of children of alcoholics: predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology* **108**, 106-19.
- Curfs L. M., Verhulst F. C. & Fryns J. P. (1991) Behavioral and emotional problems in youngsters with Prader-Willi syndrome. *Genetic Counseling* **2**, 33-41.
- De Groot A., Koot H. M. & Verhulst F. C. (1994) Cross-cultural generalizability of the Child Behavior Checklist cross-informant syndromes. *Psychological Assessment* **6**, 225-30.
- Dekker M. C. & Koot H. M. (2003a) DSM-IV disorders in children with borderline to moderate intellectual disability. II: child and family predictors. *Journal of the American Academy of Child and Adolescent Psychiatry* **42**, 923-31.

- Dekker M. C. & Koot H. M. (2003b) DSM-IV disorders in children with borderline to moderate intellectual disability, I: prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry* **42**, 915–22.
- Dekker M. C., Koot H. M., van der Ende J. & Verhulst F. C. (2002) Emotional and behavioral problems in children and adolescents with and without intellectual disability. *Journal of Child Psychology and Psychiatry* **43**, 1087–98.
- Dowling E. & Gorell-Barnes G. (1999) Children of divorcing families: a clinical perspective. *Clinical Child Psychology and Psychiatry* **4**, 39–50.
- Dykens E. M. (2000) Psychopathology in children with intellectual disability. *Journal of Child Psychology and Psychiatry* **41**, 407–17.
- Einfeld S. L. & Tonge B. J. (1996a) Population prevalence of psychopathology in children and adolescents with intellectual disability. I. Rationale and methods. *Journal of Intellectual Disability Research* **40**, 91–8.
- Einfeld S. L. & Tonge B. J. (1996b) Population prevalence of psychopathology in children and adolescents with intellectual disability. II. Epidemiological findings. *Journal of Intellectual Disability Research* **40**, 99–109.
- Emerson E. (2003) Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research* **47**, 51–8.
- Floyd F. J. & Phillippe K. A. (1993) Parental interactions with children with mild and moderate mental retardation. *Journal of Pediatric Psychology* **17**, 607–31.
- Gillberg C., Persson E., Grufman M. & Themner U. (1986) Psychiatric disorders in mildly and severely mentally retarded urban children and adolescents: epidemiological aspects. *British Journal of Psychiatry* **149**, 68–74.
- Horowitz F. D. & Haritos C. (1998) The organism and the environment: implications for understanding mental retardation. In: *Handbook of Mental Retardation and Development* (eds J. A. Burack, R. M. Hodapp & E. Ziegler), pp. 20–40. Cambridge University Press, New York.
- Ingoldsby E. M., Shaw D. S. & Garcia M. M. (2001) Intrafamily conflict in relation to boys' adjustment at school. *Development and Psychopathology* **13**, 35–52.
- Koller H., Richardson S. A., Katz M. & McLaren J. (1982) Behavior disturbance in childhood and the early adult years in populations who were and were not mentally retarded. *Journal of Preventive Psychiatry* **1**, 453–68.
- Kraemer H. C., Kazdin A. E., Offord D. R., Kessler R. C., Jensen P. S. & Kupfer D. J. (1997) Coming to terms with the terms of risk. *Archives of General Psychiatry* **54**, 337–43.
- Linna S., Moilanen I., Ebeling H., Piha J., Kumpulainen K., Tamminen T. & Almqvist F. (1999) Psychiatric symptoms in children with intellectual disability. *European Child and Adolescent Psychiatry* **8**, 77–82.
- Masten A. S. (2001) Ordinary magic: resilience processes in development. *American Psychologist* **56**, 227–38.
- Mesman J. & Koot H. M. (2000) Common and specific correlates of preadolescent internalizing and externalizing psychopathology. *Journal of Abnormal Psychology* **109**, 428–43.
- Ramey C. T. & Ramey S. L. (1999) Prevention of intellectual disabilities: early interventions to improve cognitive development. In: *The Nature–Nurture Debate: The Essential Readings* (eds S. J. Ceci & W. M. Williams), pp. 147–63. Blackwell Publishers, Malden, MA.
- Rutter M. (1999) Resilience concepts and findings: implications for family therapy. *Journal of Family Therapy* **21**, 119–44.
- Rutter M. & Sandberg S. (1992) Psychosocial stressors: concepts, causes and effects. *European Child and Adolescent Psychiatry* **1**, 3–13.
- Sparrow S. S., Balla D. A. & Cicchetti D. V. (1984) *Vineland Adaptive Behavior Scale*. American Guidance Service, Circle Pines, MN.
- Sparrow S. S., Carter A. S. & Cicchetti D. V. (1993) *Vineland Screener. Overview, Reliability, Validity, Administration and Scoring*. Yale University Child Study Center, New Haven, CT.
- Stromme P. & Diseth T. H. (2000) Prevalence of psychiatric diagnoses in children with mental retardation: data from a population-based study. *Developmental Medicine and Child Neurology* **42**, 266–70.
- Tonge B. J., Einfeld S. E. & Parmenter T. P. (2001) *A Follow Up Study of Emotional and Behavioural Problems in Young People with Intellectual Disability. Proceedings of the 34th Annual Gatlinburg Conference*. March, Charleston, SC.
- Van Lieshout C. F. M., de Meyer R. E., Curfs L. M., Koot H. M. & Fryns J.-P. (1998) Problem behaviors and personality of children and adolescents with Prader–Willi syndrome. *Journal of Pediatric Psychology* **23**, 111–20.
- Velez C. N., Johnson J. & Cohen P. (1989) A longitudinal analysis of selected risk factors for childhood psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry* **28**, 861–4.
- Verhulst F. C. & Koot H. M. (1995) *The Epidemiology of Child and Adolescent Psychopathology*. Oxford University Press, Oxford.
- Verhulst F. C. & van der Ende J. (1997) Factors associated with child mental health service use in the community. *Journal of the American Academy of Child and Adolescent Psychiatry* **36**, 901–9.
- Verhulst F. C., van der Ende J. & Koot H. M. (1996) *Handleiding Voor de CBCL/4–18 [Manual CBCL/4–18]*. Afdeling Kinder-en jeugdpsychiatrie, Sophia Kinderziekenhuis/Academisch Ziekenhuis Rotterdam/Erasmus Universiteit Rotterdam, Rotterdam.
- Wahler H. J. (1983) The Physical Symptom Inventory: measuring levels of somatic complaining behavior. *Journal of Clinical Psychology* **24**, 207–11.

- Wallander J. L., Dekker M. & Koot H. M. (2003) Psychopathology in children and adolescents with intellectual disability. In: *International Review of Research in Mental Retardation*, Vol. 26 (ed. L. M. Glidden), pp. 94–134. Academic Press, San Diego, CA.
- Wallander J. L., Frison S. & Rydvalova S. (2001) *Risk for Psychopathology in African American Adolescents with Mild Intellectual Disability*. Paper Presented at the 34th Gatlinburg Conference on Mental Retardation and Developmental Disabilities. March, Charleston, SC.
- Wallander J. L., Stankovic S. & Browne D. (1996) *Prevalence and Cumulative Incidence of Mental Health Problems in Minority Adolescents with Mild Intellectual Disability*. Paper presented at the 10th World Congress of the International Association for the Scientific Study of Intellectual Disabilities, Helsinki, Finland, July.
- White M. J., Nichols C. N., Cook R. S. & Spengler P. M. (1995) Diagnostic overshadowing and mental retardation: a meta-analysis. *American Journal on Mental Retardation* **100**, 293–8.
- Williams S., Anderson J., McGee R. & Silva P. A. (1990) Risk factors for behavioral and emotional disorder in pre-adolescent children. *Journal of the American Academy of Child and Adolescent Psychiatry* **29**, 413–19.

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