American Journal of Epidemiology
Vol. 147, No. 3
Printed in U.S.A.

Prevalence and Correlates of Mental Retardation among Children in Karachi, Pakistan

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This paper reports estimates of the prevalence of mental retardation and associated factors based on a population survey of 2- to 9-year-old children in Greater Karachi, Pakistan. A two-phase survey was implemented during the years 1988–1989. In the first phase, a cluster sample of 6,365 children (5,748 from urban areas and 617 from rural areas) was screened for disabilities using a parental report known as the Ten Questions instrument. In the second phase, all children with positive screening results and a 10% sample of those with negative results were referred for structured medical and psychological assessments. Estimates of the prevalence of mental retardation were 19.0/1,000 children (95% confidence interval (CI) 13.5–24.4) for serious retardation and 65.3/1,000 children (95% CI 48.9–81.8) for mild retardation. Both estimates were considerably higher than respective prevalence estimates obtained in industrialized countries and in selected less developed countries. In this population, lack of maternal education was strongly associated with the prevalence of both serious (odds ratio = 3.26, 95% CI 1.26–8.43) and mild (odds ratio = 3.08, 95% CI 1.85–5.14) retardation. Other factors that were independently associated with mental retardation in Karachi included histories of perinatal difficulties, neonatal infections, postnatal brain infections, and traumatic brain injury, as well as current malnourishment. Further research is needed to assess the contribution of consanguineous marriage, improvements in child survival, and other factors to the unusually high prevalence of mental retardation in this population. *Am J Epidemiol* 1998;147:281–8.

child development; developing countries; developmental disabilities; mental retardation; prevalence

Mental retardation is one of the most common disabilities occurring in childhood. Studies of the frequency of and risk factors for cognitive disorders in children have been almost entirely restricted to developed countries, where service records and registries provide a feasible means of case identification (1–3). Defining serious mental retardation in childhood as an intelligence quotient below 50 with deficits in adaptive behavior, prevalence is remarkably constant between 3 and 5 per 1,000 children, with relatively little variation over time, across socioeconomic conditions, or across populations. By contrast, defining mild mental retardation as an intelligence quotient in the range of 50–70 with deficits in adaptive behavior, prevalence in developed countries is highly variable, ranging across populations from as low as 2/1,000 to as high as 40/1,000. It is strongly associated with low socioeconomic status (1, 3). Epidemiologic and clinical studies of mental retardation in developed countries also show that severe retardation is much more commonly associated than is mild retardation with both known causes of retardation (including genetic, nutritional, infectious, toxic, traumatic, and other factors) and comorbid brain disorders (including cerebral palsy, seizures, vision impairments, and hearing impairments) (4, 5).

Pilot studies of severe mental retardation conducted in selected populations in Pakistan and India have reported extraordinarily high prevalence estimates in the range of 12–24 /1,000 (6–8). Because some of the specific causes of and risk factors for mental retardation that are now uncommon in developed countries remain highly prevalent in less developed countries, and because child survival is beginning to improve in some countries (a situation which in developed countries appears to have resulted in increases in the prevalence of childhood disability (9)), the possibility of an elevated frequency of severe mental retardation in less developed countries is plausible and requires confirmation. This paper reports estimates of the prevalence

Received for publication February 4, 1997, and in final form August 15, 1997.

Abbreviations: CI, confidence interval; OR, odds ratio.

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of both serious and mild mental retardation and potential risk factors for them based on a population survey of 2- to 9-year-old children in Greater Karachi, Pakistan.

MATERIALS AND METHODS

Study design and sampling

A two-phase, cross-sectional survey procedure was implemented in 1988–1989. In phase 1, a cluster sampling strategy modeled after that of the World Health Organization's Expanded Programme on Immunization (10) was used to obtain a sample of 6,365 children between the ages of 2 and 9 years (inclusive) living in Greater Karachi, an area inhabited by nearly eight million people (94 percent in urban households and 6 percent in rural households). Nine of the area's 43 urban census zones and three of its rural census zones were selected, with the probability of selection being proportional to the size of the zone's population. Within each of the zones selected, a cluster of contiguous households was chosen at random. This was achieved by randomly selecting a number less than 100 and counting consecutive households from the first one in a corner of the zone to the number selected at random (walking toward the center of the zone). This household served as the index household. The cluster included a contiguous series of households, beginning with the index one. All 2- to 9-year-old children residing in each cluster were screened for disabilities during phase 1 of the survey. Nearly all (98.7 percent) of the selected households were successfully contacted and agreed to participate in the study. In phase 2, all children who were screened positive and a systematic sample of 10 percent of those who were screened negative were referred for clinical evaluations. The sample that was screened negative was included in phase 2 to allow ascertainment of false negative screening results and estimation of the prevalence of disability in children screened negative (11). Although inclusion in the survey of more than one child per household was not optimal for statistical purposes (due to nonindependence of observations), it was considered necessary for practical and ethical reasons, since the screening was followed by referral for indicated assessments and treatment. The mean number of children screened per household was two (range, 1–4).

Phase 1: the Ten Questions screen for disability

The Ten Questions instrument is a brief questionnaire designed to screen for serious cognitive, motor, seizure, vision, and hearing disabilities among young children in surveys of culturally diverse populations (12–16) (see Appendix). Five of the questions focus specifically on cognitive development; two questions relate to movement disability; and one question each focuses on seizures, vision, and hearing, respectively. Using a global rather than a disability-specific interpretation of the Ten Questions (16), a child was considered to have screened positive for any disability if a response to any one question indicated potential disability. The questionnaire was translated into Urdu and administered during a personal interview with a parent or guardian. The interviewers were social work students from Karachi whose training and supervision was overseen by the study's principal investigator (Z. M. H.). Using the global definition, the Ten Questions screen has been shown to have good reliability ($\kappa = 0.67$ in a test-retest study of the Urdu translation (15)) and validity (sensitivity = 85 percent) for detecting severe neurodevelopmental disabilities (14). In addition to the Ten Questions, the interviewers administered a structured form to collect demographic information about each child and household.

Phase 2: the clinical evaluation

Clinical evaluations of children referred to phase 2 were performed (without knowledge of the screening results) by a team of local psychologists and physicians. The diagnosis of mental retardation was made consensually by a psychologist and physician after they had independently examined the child and discussed their findings. Psychological assessment of mental retardation was based on nonverbal scales from the 1985 revision of the Stanford-Binet intelligence test (17) and an adaptive behavior scale developed for (and normative for) children in Pakistan (18). The physician's assessment of mental retardation was based on the child's developmental history and a structured observation of the child's functioning in language, in following instructions, and in motor skills and behavior. Classification of a child as mentally retarded implied significant deficits in both cognitive function and adaptive behavior. Severity of retardation was classified by the psychologist according to intelligence quotient: 50–70 for mild, 35–49 for moderate, and <35 for severe. Standard procedures and criteria were used in the medical assessment (which included a medical history, physical examination, and neurologic examination in addition to the observation of function) to make diagnoses and to assess levels of disability (mild, moderate, and severe) in four areas other than cognition: motor skills, seizures, vision, and hearing (19). In this paper, moderate and severe disabilities are combined into a single category called "serious."

Data management

All data from the household survey, the screening, and the psychological and medical evaluations were recorded on precoded forms, entered into a computerized database, and linked by study identification numbers (with all personal identifiers removed). Accuracy checks and necessary corrections were made both before and after the data were entered into the database.

Statistical analysis

Because only a sample of those children who were screened negative was clinically evaluated, it was necessary to compute adjusted estimates of prevalence and its variance as described by Shrout and Newman (11). Thus, because only 10 percent of children with negative screening results were evaluated, the data for these children were weighted by a factor of 10 in the analysis. Logistic regression was used to evaluate associations between mental retardation and potential risk factors (20). PC CARP (21), a computer program developed for analysis of data from multiphase studies, was used to obtain standard errors for constructing confidence intervals around the prevalence estimates and regression coefficients (exponentiated to obtain odds ratios). Variables were entered into the logistic regression models if the p values associated with their regression coefficients were less than 0.25; they were retained if their respective p values were less than 0.10 and/or their removal substantially affected the magnitude of the regression coefficients for other variables in the model.

RESULTS

A total of 936 (14.7 percent) of the 6,365 children surveyed had positive screening results on the Ten Questions instrument (table 1); 818 (87 percent) of these children and 545 (10 percent) of those children screening negative were clinically evaluated in phase 2. Serious mental retardation was diagnosed in 90 children, including two with false negative screening results. Mild mental retardation was diagnosed in 140 children, including 29 with negative results on the Ten Questions screen. The overall prevalence estimates obtained from the weighted analysis were 18.97/1,000 (95 percent confidence interval (CI) 13.54–24.40) for serious retardation and 65.33/1,000 (95 percent CI 48.87–81.78) for mild retardation. For both serious and mild retardation, the point estimates of prevalence were higher in rural areas than in urban areas and in children whose mothers had no formal education versus some formal education (table 2). No notable sex differences were observed for either serious or mild retardation. The prevalence of mild retardation appeared to increase with age (table 2).

Among children with serious mental retardation, 57 percent had other neurodevelopmental disabilities (including motor, seizure, vision, and/or hearing disorders), as compared with 10 percent of children with mild retardation and 2 percent of those with normal cognition. Among children with serious retardation, specific causes were identified by the assessment team in only 17 (19 percent). These included 12 cases attributed to brain infection during infancy or childhood (i.e., postnatal), three cases attributed to cretinism, and two cases attributed to traumatic brain injury during childhood. Only 12 (9 percent) of the mild cognitive disabilities were attributed to a specific

TABLE 1. Demographic and medical characteristics of 2- to 9-year-old children screened* for cognitive disabilities in Karachi, Pakistan, by urban/rural residence, 1988–1989†

	Urban (n = 5,748)‡	Rural (n = 617)	Total (n = 6,365)
Screened positive	14.3	18.7	14.7
Male sex	54.0	51.1	53.7
Age group			
2–5 years	52.4	58.9	53.1
6–9 years	47.6	41.1	46.9
In school (ages 6–9 years)	76.3	52.2	74.1
Maternal education			
None	54.5	90.9	58.3
Primary	21.8	7.6	20.3
Secondary	20.8	1.5	18.8
Higher	2.9	0	2.6
No family ownership of land or home	51.7	78.8	54.5
Siblings			
Mean no. ever born	4.4	4.7	4.4
Mean no. living	3.9	3.8	3.9
Sibling death	30.4	54.1	32.8
Consanguinity§	57.8	88.1	60.9
No prenatal care	31.1	89.1	36.6
Child born in a hospital or clinic	54.1	30.6	51.7
Attended birth			
Doctor	40.8	12.2	38.2
Trained midwife	25.3	20.2	24.8
Untrained midwife	32.4	67.4	35.7
Other	1.5	0.2	1.4
Prematurity/small size at birth	19.1	26.0	19.7
Perinatal difficulty	38.7	61.0	40.6
Neonatal infection	8.0	5.9	5.4
Goiter in mother or child	11.1	0.4	10.2
Child not fully immunized	38.3	50.9	39.5
Postnatal brain infection	0.9	3.3	1.1
Head injury with loss of consciousness	3.0	3.8	3.1
Child appeared malnourished	2.0	4.5	2.2

* Using the Ten Questions screen (see Appendix).
† All data are percentages unless otherwise indicated.
‡ Numbers in parentheses, number screened.
§ Child's parents were related by blood as first cousins or as uncle–niece.

TABLE 2. Prevalence of serious and mild mental retardation and related odds ratios among 2- to 9-year-old children in Karachi, Pakistan, by selected demographic factors, 1988–1989

	Prevalence (per 1,000)	Odds ratio	95% CI*
Age (years)			
2–5			
Serious	20.14	1.00	
Mild	54.21	1.00	
6–9			
Serious	17.40	0.88	0.43–1.83
Mild	77.75	1.47	0.98–2.87
Sex			
Female			
Serious	19.35	1.00	
Mild	58.74	1.00	
Male			
Serious	18.71	0.99	0.48–2.04
Mild	71.05	1.22	0.82–1.83
Residence			
Urban			
Serious	17.18	1.00	
Mild	58.92	1.00	
Rural			
Serious	34.74	2.21	0.87–5.57
Mild	120.85	2.23	1.33–3.75
Maternal education			
Some			
Serious	8.40	1.00	
Mild	29.52	1.00	
None			
Serious	26.59	3.26	1.26–8.43
Mild	90.80	3.08	1.85–5.14

* CI, confidence interval.

cause: one to prenatal difficulties and 11 to postnatal brain infections.

Only 3.7 percent of the children with mental retardation had been previously evaluated for retardation or had received any services for cognitive disability or learning problems. The frequency of previous evaluation or receipt of services was lower in rural areas (1.9 percent) than in urban areas (4.1 percent) and was lower for mild retardation (2.4 percent) than for serious retardation (8.3 percent). Among school-age children (ages 6–9 years), the percentage of children attending school was much lower for children with mental retardation than for others: 8.4 percent for children with serious retardation, 48.2 percent for children with mild retardation, and 77.3 percent for children without cognitive disability.

Table 1 shows data on demographic and medical factors in the children screened, stratified by urban/rural residence. Most of the factors shown in table 1 can be viewed as indicators of socioeconomic disadvantage and/or risk factors for mental retardation. They show children in rural areas to be at greatest risk.

We identified 13 of these factors a priori as potential antecedents to mental retardation. Dichotomously defined, they are:

- lack of maternal education,
- consanguinity (i.e., child's parents related by blood as first cousins or as uncle–niece),
- history of goiter in the mother or child,
- no prenatal medical care,
- medically unattended birth (birth not attended by a trained health care provider),
- home birth,
- prematurity or small size at birth,
- perinatal difficulties (including prematurity or small size at birth, cesarean section, breech presentation, or other difficulty),
- neonatal infection,
- lack of immunization (child not fully immunized against tetanus, pertussis, diphtheria, poliomyelitis, and tuberculosis),
- postnatal brain infection,
- postnatal traumatic brain injury, and
- current malnourishment of the child.

Five of these factors (consanguinity, goiter, unattended birth, prematurity or small size at birth, and lack of immunization) were not associated with mental retardation (mild or serious) in either urban or rural areas. We found the remaining eight variables, in addition to rural residence, to be associated with mental retardation. Stratification of these eight variables (lack of maternal education, lack of prenatal care, perinatal complications, home birth, neonatal infection, postnatal brain infection, traumatic brain injury, and malnourishment), dichotomously defined, by urban/rural residence indicated no apparent effect modification by residential area in terms of their associations with mental retardation. In evaluating statistical associations between each of the potential risk factors, we did not adjust for urban/rural residence, because this factor may best be viewed as distal or antecedent to the other risk factors being evaluated, rather than as a confounder. Unadjusted odds ratios indicated strong associations between lack of maternal education, perinatal complications, postnatal brain infection, and malnourishment and both serious and mild mental retardation (table 3). Neonatal infections were strongly associated with serious retardation but not mild retardation, and lack of prenatal care, home birth, and traumatic brain injury showed significant associations with mild retardation but not serious retardation (table 3).

In multivariate analyses, we identified four models: two models each of independent associations between potential predictors and serious and mild mental retar-

TABLE 3. Frequency (%) of potential risk factors for mental retardation and unadjusted odds ratios for associations with serious and mild retardation among 2- to 9-year-old children in Karachi, Pakistan, 1988–1989*

	Seriously mentally retarded children (n = 90)	Mildly mentally retarded children (n = 140)	Cognitively normal children (n = 1,133)	Association with serious mental retardation		Association with mild mental retardation	
				OR†	95% CI†	OR	95% CI
Lack of maternal education	81.8	81.3	56.4	3.26	1.26–8.43	3.08	1.85–5.14
Consanguinity‡	56.7	69.0	60.4	0.85	0.41–1.77	1.46	0.95–2.25
Goiter	7.1	9.7	10.3	0.67	0.16–2.87	0.93	0.45–1.91
Lack of prenatal care	40.2	50.5	35.6	1.21	0.57–2.55	1.85	1.22–2.81
Untrained birth attendant	49.1	53.5	35.6	1.74	0.83–3.65	2.08	1.38–3.13
Home birth	55.5	64.4	47.1	1.39	0.67–2.88	2.03	1.34–3.09
Prematurity/small size at birth	28.2	26.5	19.1	1.67	0.74–3.78	1.53	0.96–2.44
Any perinatal complication	57.0	55.9	39.2	2.07	0.93–4.61	1.97	1.28–3.02
Neonatal infection	24.2	5.4	5.0	5.95	2.48–14.24	1.09	0.44–2.70
Child not immunized	45.2	45.2	39.0	1.31	0.62–2.74	1.28	0.85–1.94
Postnatal brain infection	9.7	4.9	0.7	16.20	4.11–63.93	7.48	2.46–22.76
Traumatic brain injury	4.1	8.7	2.7	1.44	0.22–9.55	3.41	1.60–7.27
Current malnutrition	14.0	9.4	1.5	10.92	3.62–32.97	6.82	3.11–14.92

* Children with missing information on a given variable were excluded from analyses involving that variable. The following variables had missing information for a percentage of children: goiter in the mother or child (9.4%); lack of prenatal care (2.3%); untrained birth attendant (4.4%); home birth (0.9%); prematurity/small size at birth (1.8%); any perinatal complications (12.0%); neonatal infection (1.7%); postnatal brain infection (1.9%); and traumatic brain injury (1.1%).

† OR, odds ratio; CI, confidence interval.

‡ Child's parents were related by blood as first cousins or as uncle–niece.

dation (table 4). Variables included in the first two models were restricted to those that would almost certainly have preceded the development of cognitive disability in the child—namely, lack of maternal education, lack of prenatal care, home birth, perinatal difficulties, and neonatal infections. In the remaining two models, we entered these variables as well as factors that could have occurred or developed subsequent to the onset of cognitive disability—namely, postnatal brain infection, traumatic brain injury, and malnutrition.

All four models showed strong independent effects of maternal education. The first two models showed that history of neonatal infection was also an independent predictor of serious retardation, and lack of prenatal care and history of perinatal complications were independently predictive of mild retardation once maternal education was controlled (table 4). When factors that could have followed rather than preceded the onset of cognitive disability were included (models 3 and 4), the variables that had significant independent associations with serious retardation included, in addition to lack of maternal education: neonatal infection, postnatal brain infection, and current malnutrition. Those showing independent associations with the prevalence of mild retardation included, in addition to lack of maternal education: history of perinatal difficulties, history of postnatal brain infection, history of traumatic brain injury, and current malnutrition (table 4). Inclusion of an indicator variable for rural residence in these models did not affect the magnitude of the odds ratios. Moreover, the odds ratios for rural residence were not significantly different from 1.0 once the other variables had been included, which suggests that rural residence is not an independent risk factor for mental retardation.

DISCUSSION

The results of this epidemiologic survey carried out in Karachi, Pakistan, are in agreement with those of pilot studies conducted in Pakistan and India (6–8) showing estimates of the prevalence of mental retardation that are much higher than those observed in developed countries. Unlike the previous pilot studies, which were restricted to serious mental retardation, the present study shows elevated prevalence rates in Karachi for both serious and mild retardation. Though in agreement with those of earlier pilot studies, these prevalence estimates contrast with estimates from an epidemiologic survey in Bangladesh (22) which used a study design and diagnostic procedures virtually identical to those of the present study. In Bangladesh, the prevalence of serious mental retardation in 2- to 9-year-old children was found to be 5.9/1,000 (95 percent CI 3.4–8.4), only slightly higher than the prevalence range observed in developed countries, and the prevalence of mild retardation was found to be within the range observed in developed countries: 14.4/1,000 (95 percent CI 7.8–21.1) (22). Because a

TABLE 4. Factors independently associated with the prevalence of serious and mild mental retardation among 2- to 9-year-old children in Karachi, Pakistan, 1988–1989

	Odds ratio*	95% CI†
Model 1: serious mental retardation (restricted to factors that most likely preceded onset of cognitive disability)		
Lack of maternal education	3.33	1.30–8.51
Neonatal infection	5.49	2.28–13.22
Model 2: mild mental retardation (restricted to factors that most likely preceded onset of cognitive disability)		
Lack of maternal education	3.07	1.77–5.34
No prenatal care	1.58	1.01–2.46
Perinatal difficulty	1.92	1.24–2.96
Model 3: serious mental retardation (not restricted to factors that necessarily preceded onset of cognitive disability)		
Lack of maternal education	3.23	1.23–8.48
Neonatal infection	5.14	2.03–13.06
Postnatal brain infection	14.02	3.07–63.93
Malnourishment	10.19	3.19–32.58
Model 4: mild mental retardation (not restricted to factors that necessarily preceded onset of cognitive disability)		
Lack of maternal education	3.57	2.05–6.22
Child's age (6–9 years vs. 2–5 years)	1.78	1.14–2.80
Perinatal difficulty	1.94	1.24–3.03
Postnatal brain infection	6.31	1.82–21.91
Traumatic brain injury	3.42	1.44–8.15
Malnourishment	4.23	1.64–10.90

* Adjusted for all other variables in the model.
† CI, confidence interval.

common case-finding procedure was used in the Bangladeshi study and the present study, it is likely that the observed differences in prevalence are real rather than due to a methodological artifact.

Children with mental retardation are generally at increased risk of mortality (23). It is possible that the mortality of children with disabilities is higher in Bangladesh than in Pakistan, since child mortality rates overall are higher in Bangladesh than in Pakistan (24). Thus, a mortality differential between Pakistan and Bangladesh may explain the lower prevalence of mental retardation in Bangladesh relative to Pakistan. The cross-sectional data available from these studies do not allow ascertainment of the mortality experienced by disabled and nondisabled children in these populations.

Another possible explanation for the high prevalence of mental retardation in Karachi is the extraordinarily high prevalence of consanguineous marriage in this population. Sixty percent of the children screened for disability in the present study were offspring of consanguineous unions (mostly first cousins, a preferred marriage pattern among many Pakistanis (25, 26)). It has been pointed out that in populations where consanguinity is a dominant marriage pattern, consanguinity may not be associated at an individual level with the occurrence of disorders resulting from recessive genes, including many of the specific causes of mental retardation. This is because even "unexposed" children (i.e., offspring of nonconsanguineous marriages) in such a population would be expected to have high inbreeding coefficients due to consanguinity in previous generations (i.e., their grandparents and great-grandparents) (27). Thus, the absence of an individual-level association between consanguinity and mental retardation in this study is not inconsistent with the possibility that the excess prevalence of mental retardation is due, at least in part, to a high frequency in this population of recessive genes for neurodevelopmental disorders resulting from frequent consanguinity. The excess prevalence of mental retardation observed in this population, along with the high prevalence of consanguinity, calls for genetic epidemiologic studies to identify the contribution of specific recessively inherited forms of mental retardation.

Using lack of maternal education as an indicator for socioeconomic disadvantage, it is notable that the prevalence rates of both mild and serious retardation in Karachi were significantly associated with low socioeconomic status. A similar pattern was observed in Bangladesh, though the association was weaker for serious retardation than for mild retardation (22). This finding suggests that a fundamental feature of the epidemiology of mental retardation as observed in industrialized countries—the association between socioeconomic disadvantage and mild but not serious retardation—may not apply under conditions that currently prevail in some less developed countries. The conditions in these settings that could plausibly give rise to an excess of both mild and serious retardation in the poorest segment of the population include the relatively undeveloped level of prenatal and perinatal services and the high prevalence of brain infections and nutritional deficiencies. These factors, which are associated with socioeconomic disadvantage, were shown here to be major risk factors for both mild and serious mental retardation in Karachi.

The strikingly low percentages of retarded children in this population who had been previously evaluated, had received services, or had attended school point to a need for improved recognition of and provision of

services for children with cognitive disabilities in less developed countries.

The relatively large number of false negative screening results for children diagnosed with mild mental retardation (29 of 140) is not surprising, given that the Ten Questions screen is designed for and has been validated for identification of serious but not mild neurodevelopmental disabilities (14). An important advantage of the two-phase study design used here, in which a random sample of those screening negative is referred for diagnostic evaluations, is that it allows estimation of prevalence even of conditions for which the sensitivity of the screen is low (11).

An important limitation of this study is the cross-sectional nature of the data, which limits their utility for causal inference. It is not possible to determine the directionality of many of the relations observed. For example, the strong associations between the prevalence of mental retardation and malnutrition, traumatic brain injury, and postnatal brain infection could result from these variables themselves being causes of brain disorder or from children with cognitive impairments being at greater risk for these exposures. Moreover, variables associated with the prevalence of mental retardation could be etiologic factors or they could simply be factors associated with the survival of mentally retarded children—two explanations that cannot be distinguished using the cross-sectional data available from this study. In addition, the retrospective nature of the data collected on variables such as perinatal difficulties, neonatal and postnatal infection, and head injury raises the possibility that recall bias could explain all or part of the observed associations between these variables and the occurrence of mental retardation.

Despite these limitations, the prevalence estimates and associations reported here from one population in the less developed world begin to fill a major gap in our information about the epidemiology of mental retardation. In doing so, they begin to provide a needed epidemiologic basis for indices of quality of life and disability in low income populations. The demand for such indices is increasing, along with recognition of the limitations of mortality as the primary or sole basis for planning and evaluating public health interventions (28).

ACKNOWLEDGMENTS

This work was supported by the BOSTID Program of the National Academy of Sciences, the Epilepsy Foundation of America, the National Institute of Neurological Disorders and Stroke (grant R29 NS27971–01–05), and the New York State Psychiatric Institute.

The authors gratefully acknowledge the contributions of Drs. Zena Stein, Farooq Anwer, Lillian Belmont, Leslie Davidson, Naila Khan, Patrick Shrout, Molly Thorburn, and Sultana Zaman to the design and conduct of this research.

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APPENDIX

The Ten Questions Screen

1. Compared with other children, did the child have any serious delay in sitting, standing, or walking?
2. Compared with other children, does the child have difficulty seeing, either in the daytime or at night?
3. Does the child appear to have difficulty hearing?
4. When you tell the child to do something, does he/she seem to understand what you are saying?
5. Does the child have difficulty in walking or moving his/her arms, or does he/she have weakness and/or stiffness in the arms or legs?
6. Does the child sometimes have fits, become rigid, or lose consciousness?
7. Does the child learn to do things like other children his/her age?
8. Does the child speak at all (can he/she make himself/herself understood in words; can he/she say any recognizable words)?
9. *For 3- to 9-year-olds, ask:* Is the child's speech in any way different from normal (not clear enough to be understood by people other than his/her immediate family)? *For 2-year-olds, ask:* Can he/she name at least one object (for example, an animal, a toy, a cup, a spoon)?
10. Compared with other children of his/her age, does the child appear in any way mentally backward, dull, or slow?