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EPIDEMIOLOGICAL STUDY OF INTELLECTUAL DISABILITY IN DISTRICT LOWER DIR, KHYBER PAKHTUNKHWA, PAKISTAN

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ABSTRACT

Background: Intellectual disability (ID) is a significant population health issue in Pakistan, particularly in rural communities where the healthcare system has few infrastructures, and the rate of consanguinity is high. This research had the objective of determining the prevalence in epidemiological terms, risk factors and etiological determinants of ID in District Lower Dir, Khyber Pakhtunkhwa.

Procedures: A cross-sectional study was conducted using population based on multistage stratified random sampling of all the seven tehsils of District Lower Dir. The response rate was 67 percent and 4,099 participants aged 5-90 years were enrolled (out of 390 households). The full-scale tests were made, such as demographic profiling, clinical testing, anthropometric testing, and cognitive testing with standardized tools (Universal Nonverbal Intelligence Test, Stanford-Binet Intelligence Scales and Vineland Adaptive Behavior Scales). The diagnosis was according to DSM-5. The univariate results were compared by means of statistical tests comprising of chi-square tests and penalized Firth logistic regression in order to establish the multivariate results of the sociodemographic predictors, and the etiological history was evaluated descriptively.

Findings: The total prevalence of ID was 2.8% (115/4,099) with mild ID taking 2.1% of the total population. Males had more univariate prevalence compared to females (3.5% vs. 2.0%; $p=0.003$). The prevalence of ID was almost twice in consanguinity parentage than in non-consanguinity unions (4.6% vs. 2.3; $p=0.001$). Etiological disaggregation showed that genetic causes were the most common (68.7, 79/115), then postnatal factors (13.9, 16/115), perinatal factors (12.2, 14/115) and prenatal factors (3.5, 4/115). Congenital onset was an independent predictor of ID in multivariate Firth penalized logistic regression that controlled on sociodemographic factors ($B=8.05$, $SE=3.53$, $p=0.023$), and gender ($p=0.241$), consanguinity ($p=0.922$) and family structure (nuclear: $p=0.236$; extended: $p=0.371$) lost their statistically significant values after adjustment.

Conclusions: This is a first systematic epidemiological study in Lower Dir that presents an ID prevalence of 2.8% of which genetic factors describe 68.7% of the occurrences. The findings indicate the urgency of genetic counseling services in communities, premarital screening programs, and better perinatal care in high consanguinity communities. The research contains the necessary baseline information in the development of evidence-based health policy in rural Pakistan.

Keywords: Intellectual disability, epidemiology, prevalence, consanguinity, risk factors, Pakistan, Firth logistic regression.

INTRODUCTION

Intellectual disability (ID) is a neurodevelopmental disorder that is marked by pronounced impairments in intellectual functioning, as well as in adaptive functioning, which comprises conceptual, social, and practical areas, that occur during the neurodevelopmental period before 18



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years of age [1,2]. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), ID is a problem with reasoning, problem-solving, abstract thinking, judgment, academic learning and experience learning and at the same time, adaptive functioning problems resulting in inability to meet developmental and sociocultural expectations of personal autonomy and social responsibility [2]. The current definition of ID is very different as compared to the historical methods which marginalized and institutionalized the affected individuals to the current definition of ID as a developmental disorder with multifactorial biological, environmental and social factors [3,4]. At the international level, ID is estimated at approximately 200 million individuals and it is one of the most widespread developmental disorders globally with a prevalence of 1-3 percent of the total population yet the prevalence of this disorder is very variable due to the difference in the diagnostic criteria, sampling methods and sociocultural influences [5-7]. World Health Organization documents that prevalence in the low- and middle-income nations (LMICs) typically exceeds 3% though such statistics are greatly augmented by preventable elements like malnutrition, birth injuries and pathogenic infections that have yet to be effectively contained, and high-income nations have already achieved advancement in minimizing prevalence through early intervention programmes, superior maternal health services, and universal genetic screening [5]. In Pakistan, which has specific studies (most of which are hospital based with some community surveys) estimate a prevalence of 1.9-2.5, but is likely to be much higher due to poor disease registries, stigma and the absence of standard diagnostic tools [11,12]. In Khyber Pakhtunkhwa (KP) province at least, and in the mountainous districts within KP in particular, and in Lower Dir in particular, there is actually very little reliable epidemiological information, and the health records are overwhelmed by communicable diseases and developmental disabilities which are simply underreported due to cultural influences and limited public awareness [13]. The etiology of ID is highly varied and it encompasses genetic factors, environmental factors and sociocultural factors. The genetic factors account an approximation of 30-60 percent in outbred populations with the remaining instances being attributed to prenatal, perinatal, or postnatal insults like chromosomal abnormalities, maternal infections, birth asphyxia, meningitis, extreme malnutrition and exposure to environmental toxins [14-16]. In Pakistan, consanguinity marriage is a very powerful culture dimension that the national estimates indicate that 60-70 percent of all marriages are between blood relatives, which is nearly 80 percent in some districts resulting into high homozygosity and consequently autosomal recessive diseases including ID [12]. Pakistan Genetic Mutation Database estimates that a population of approximately 29 million Pakistanis is suspected to be afflicted with genetic diseases as a result of consanguinity but has little knowledge of genetic risk in absence of genetic counseling facilities [12]. Despite enormous genetic disease burden in Pakistan, there is a desperate lack of molecular and epidemiological research in rural Khyber Pakhtunkhwa, and Lower Dir offers a desperate knowledge gap, severely geographically isolated, high-consanguinity, mountainous region with a severely limited supply of healthcare access where systematic epidemiological data are completely absent [13]. In this way, the study was carried out to find out the prevalence of ID, the risk factors of the disease, and to explain the etiological determinants of the disease in District Lower Dir.

METHODS

Design and Set-up of the Study.

The study used the population-based cross-sectional design to establish the prevalence and the factors of intellectual disability among the residents in District Lower Dir, Khyber Pakhtunkhwa, Pakistan. The mountainous terrain, rural settlement structure, and consanguinity marriages were all common characteristics of the purposely selected district that was believed to be the appropriate place to investigate the factors of genetic and environmental influence that determine ID.



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Sampling Strategy

Among seven tehsils (Adenzai, Timergara, Khall, Balambat, Lal Qala, Munda, and Samarbagh), multistage stratified random sampling was adopted in order to have a representative geographic and demographic coverage of the stratum by means of systematic selection of households with references to four cardinal directions. A total of 390 households were recruited which gave a final sample of 4,099 people aged between 5-90 years. The sample size was calculated by the use of a population proportion formula with confidence level of 95 percent and margin of error of 5 percent using population of the district which was approximately 1.65 million [17].

Ethical Considerations

The Institutional Ethics Committee of the Islamia College, Peshawar (Protocol No. ICP/IRB/2022/114) gave the ethical approval. Informed consent in a written form was taken and, in all participants, or its representatives of the law, and secrecy and protection of data were well noted. Where possible, consent was obtained among the participants who had ID, and in cases of minors, legal guardians gave consent.

Data Collection

A structured questionnaire was used to gather the demographic variables (gender, age, marital status, family structure), the type of parental marriage (consanguinity vs. non-consanguinity), and the comprehensive history of medical history such as prenatal and perinatal and postnatal events. The anthropometric measurements (height, weight, head circumference), physical and neurological examination as a system, and dysmorphic features were performed tropically in accordance with the WHO standards [18]. The standardized tests on cognitive functioning that were culturally appropriate were used, including the Universal Nonverbal Intelligence Test (UNIT), Stanford-Binet Intelligence Scales (Fifth Edition), and Woodcock-Johnson Tests of Cognitive Abilities. Vineland Adaptive Behavior Scales (VABS) [19] were used to measure the scales of adaptive behavior.

Diagnostic Criteria

Diagnosis and severity were determined by using criteria of DSM-5 [2] that required (1) impairment of intellectual functions, which were determined by clinical examination and standardized testing (IQ less than 70), (2) impairment of adaptive functioning in the conceptual, social, or practical domains, and (3) onset of the impairment during development (before age 18). Severity was classified as mild (IQ 50-69), moderate (IQ 35-49), severe (IQ 20-34), or profound (IQ <20).

Etiological Classification

Genetic cause: Family history of the same conditions, known genetic syndromes or affected siblings in the past.

Prenatal history Maternal infections (rubella, CMV, toxoplasmosis), maternal malnutrition, or antenatal complications.

History of perinatal: Asphyxia of birth, preterm birth, low birth weight, obstetrics, or protracted labor.

Postnatal history: Meningitis/encephalitis, traumatic brain injury, severe malnutrition, exposure to environmental toxins, or childhood infections.

Statistical Analysis

Statistical analysis was done using SPSS version 27.0 and Stata version 14. Descriptive statistics was used to summarize base line characteristics. Univariate relationships between ID and categorical variables (gender, parental marriage type, family structure) were investigated by chi-square tests. Because the measure of outcomes (2.8) is small, Firth penalized logistic regression [20] has been used in the multivariate analysis in order to reduce bias in the parameter estimates. The multivariate model consisted of gender, type of parental marriage, family structure, and the



onset of ID. The etiological history was not included in the regression model since it has a deterministic correlation to the outcome (complete separation). The results are in the form of regression coefficients (B), standard errors (SE), 95% confidence interval, and p-values, with the statistical significance being $p < 0.05$.

RESULTS

This study examined 4,099 participants (51.7% male, 48.3% female) with a mean age of 27.95 years (range: 5–90 years). The majority of the respondents resided in nuclear (49.8), extended (32.2), and single-family (18.0) and consanguinity parental marriage was at 21.2. The overall prevalence of intellectual disability (ID) was 2.8 (115/4,099), the most prevalent level of intellectual disability was the mild intellectual disability (ID) (2.1 percent of the total population, representing 75.7 percent of the cases of intellectual disability), followed by the moderate intellectual disability (ID), the severe intellectual disability (ID), and the profound intellectual disability (ID). In terms of onset, there were 1.5% congenital cases (62/4,099) and the acquired cases 0.4% (18/4,099). The etiological disaggregation of the 115 ID cases showed that genetic causes included the most significant proportion (68.7 per cent, 79/115), then postnatal factors (13.9 per cent, 16/115), perinatal factors (12.2 per cent, 14/115) and prenatal factors (3.5 per cent, 4/115). Univariate analysis revealed that some factors were related to the presence of ID: males had a much higher prevalence of ID than females (3.5% vs. 2.0%; $\chi^2 = 8.85$, $p = 0.003$), and children of consanguineous marriages were more prevalent with ID than those of non-consanguineous marriages (4.6% vs. 2.0%). The independent sociodemographic predictors of ID and control low outcome prevalence were done using the Firth multivariate penalized logistic regression. After adjusting for all covariates, sex (female: $B = -0.25$, $p = 0.241$), consanguineous marriage ($B = -0.22$, $p = 0.922$), and family structure (nuclear: $B = -2.00$, $p = 0.236$; extended: $B = -1.59$, $p = 0.371$) were not significant predictors. However, it was found that disease congenital onset ($B = 8.05$, $p = 0.023$) and not acquired onset ($p = 0.905$) made a big prediction of ID. These results indicate that although demographic variables like sex and consanguinity are significantly bivariately related with ID, congenital onset proves to be the most significant independent sociodemographic predictor in the present population with the etiological factors especially genetic causing the majority of the cases when considered descriptively.

Table 1. Selected Baseline Characteristics of Study Participants (N = 4,099)

Characteristic	Category	n (%)
Gender	Male	2,119 (51.7%)
	Female	1,980 (48.3%)
Age range & mean (years)		5-90 (27.95)
Marital status	Single	2,104 (51.3%)
	Married	1,995 (48.7%)
Age group (years)	5 - 19	1,583 (38.6%)
	20 - 34	1,250 (30.5%)
	35 - 49	714 (17.4%)
	50 - 64	355 (8.7%)
	65 - 79	175 (4.3%)
	80 - 94	22 (0.5%)
Family structure	Single	739 (18.0%)
	Nuclear	2,041 (49.8%)
	Extended	1,319 (32.2%)
Physical disability (any kind)	No	4,059 (99.0%)
	Yes	40 (1.0%)



<i>Marriage type of parents</i>	Non-consanguineous	3,229 (78.8%)
	Consanguineous	870 (21.2%)
<i>Prevalence of ID</i>	Normal	3,984 (97.2%)
	Intellectually disabled	115 (2.8%)
<i>Severity levels of ID</i>	Normal	3,984 (97.2%)
	Mild	87 (2.1%)
	Moderate	15 (0.4%)
	Severe	9 (0.2%)
	Profound	4 (0.1%)
<i>Onset of disease</i>	Normal	4,019 (98.0%)
	Congenital	62 (1.5%)
	Acquired	18 (0.4%)
<i>Etiological history of ID</i>	No history of ID	3,986 (97.2%)
	Prenatal history	4 (0.1%)
	Perinatal history	14 (0.3%)
	Postnatal history	16 (0.4%)
	Genetic cause	79 (1.9%)
<i>Participants by facial features</i>	Normal	4,049 (98.8%)
	Broad forehead	7 (0.2%)
	Short philtrum	1 (0.0%)
	Coarse facial features	2 (0.0%)
	Long chin	10 (0.2%)
	Dysmorphic facial features	30 (0.7%)

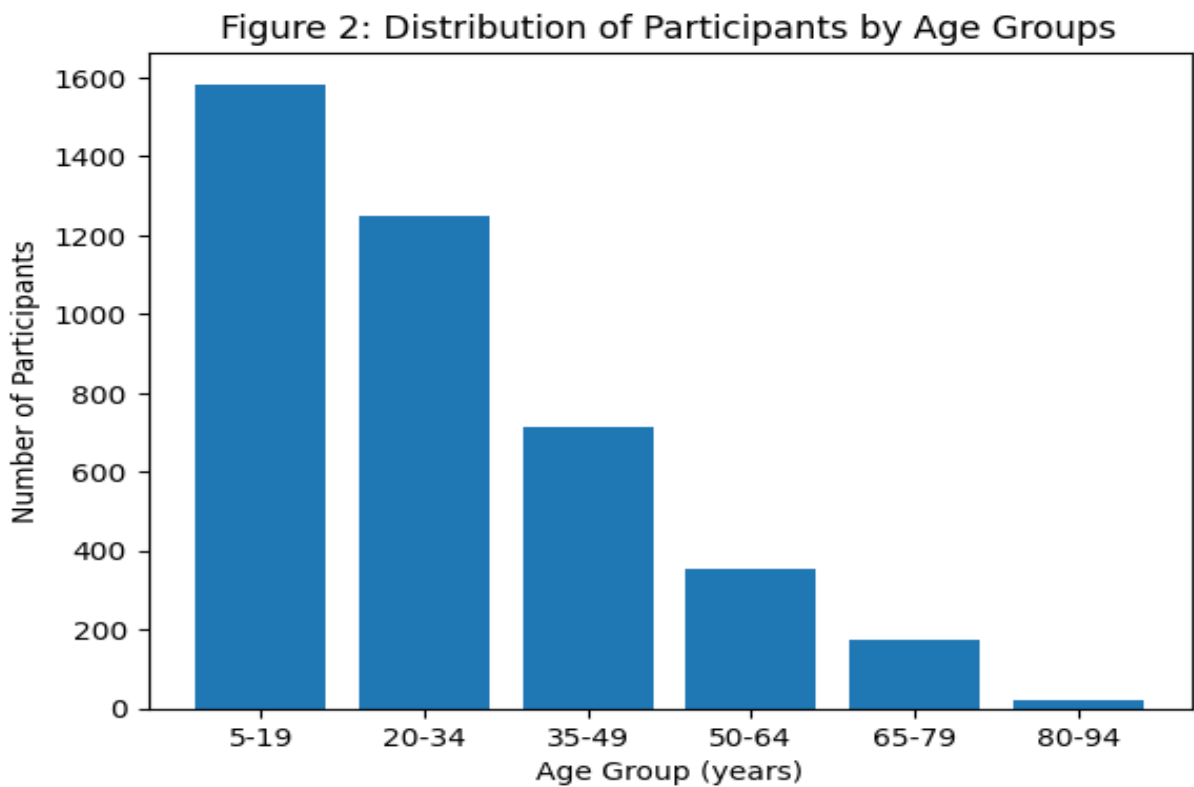




Figure 3: Severity Levels of Intellectual Disability

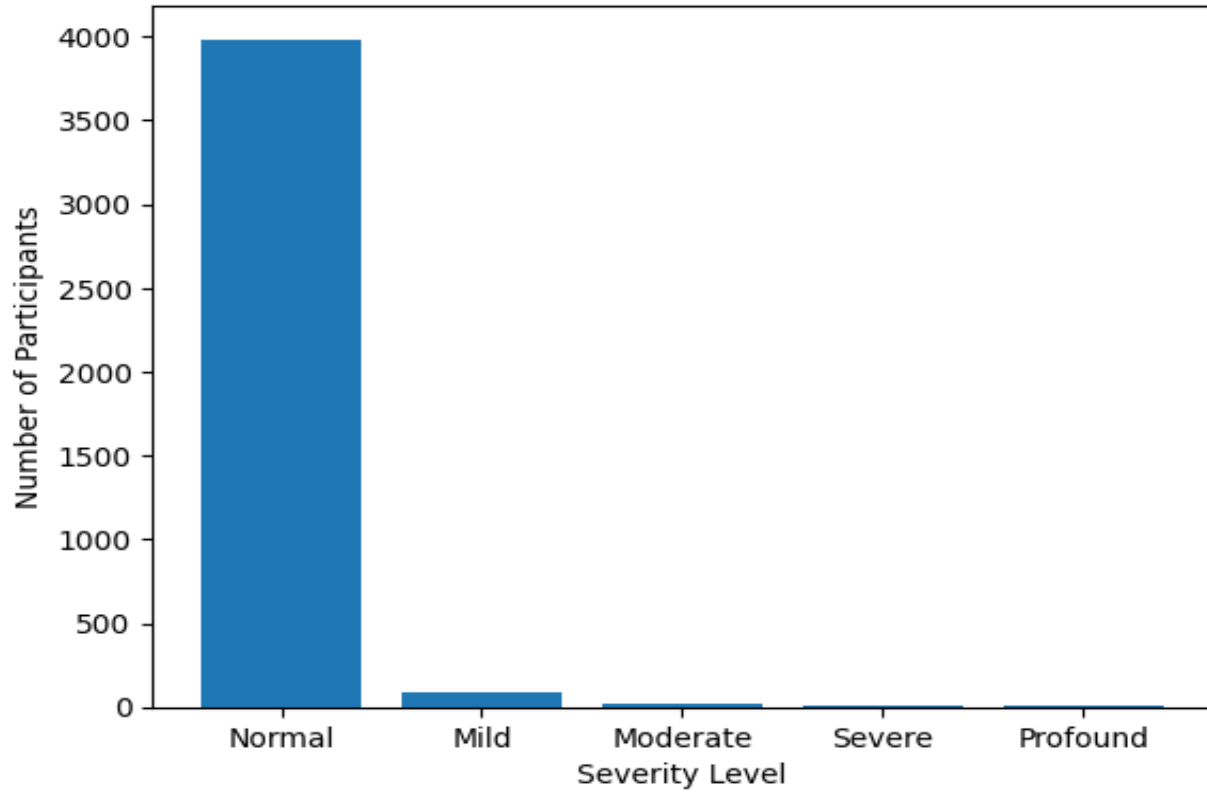


Figure 1: Distribution of Participants by Gender

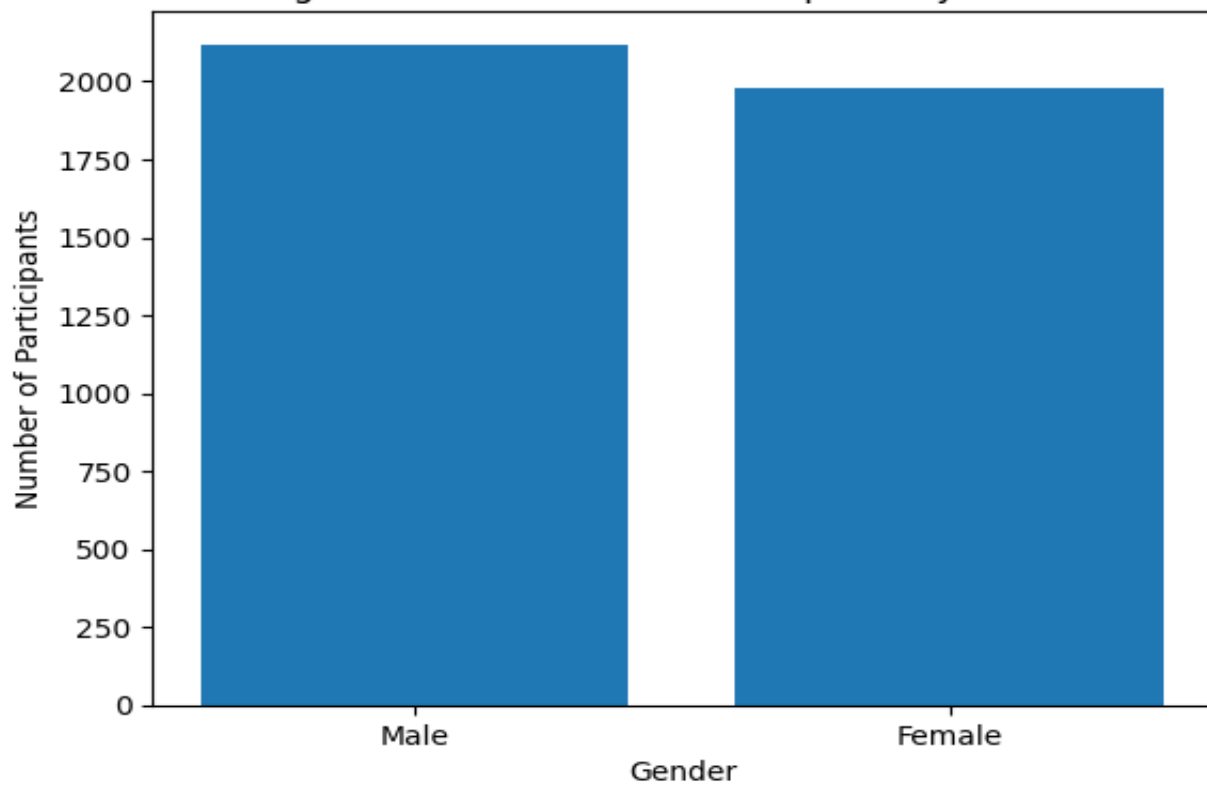




Table 2. Univariate Analysis: Differences in ID Prevalence by Demographic and Clinical Factors (N = 4,099)

Variable	Category	Normal n (%)	Intellectually Disabled n (%)	χ^2	p-value
Sex	Male	2,044 (96.5%)	75 (3.5%)	8.85	0.003
	Female	1,940 (98.0%)	40 (2.0%)		
Parental Marriage	Non-consanguineous	3,154 (97.7%)	75 (2.3%)	13.01	0.001
	Consanguineous	830 (95.4%)	40 (4.6%)		
Family Structure	Single	723 (97.8%)	16 (2.2%)	4.19	0.123
	Nuclear	1,973 (96.7%)	68 (3.3%)		
	Extended	1,288 (97.6%)	31 (2.4%)		
Etiological History*	No history	3,984 (100%)	2 (0.05%)	NA**	<0.001
	Prenatal	0 (0.0%)	4 (100%)		
	Perinatal	0 (0.0%)	14 (100%)		
	Postnatal	0 (0.0%)	16 (100%)		
	Genetic	0 (0.0%)	79 (100%)		

Etiological history is presented descriptively and was not included in multivariable regression due to deterministic relationship with ID (complete separation).

** χ^2 not applicable due to expected cell counts <5 in multiple cells. Data shown as column percentages.

Figure 4: Association of Demographic Variables with Intellectual Disability

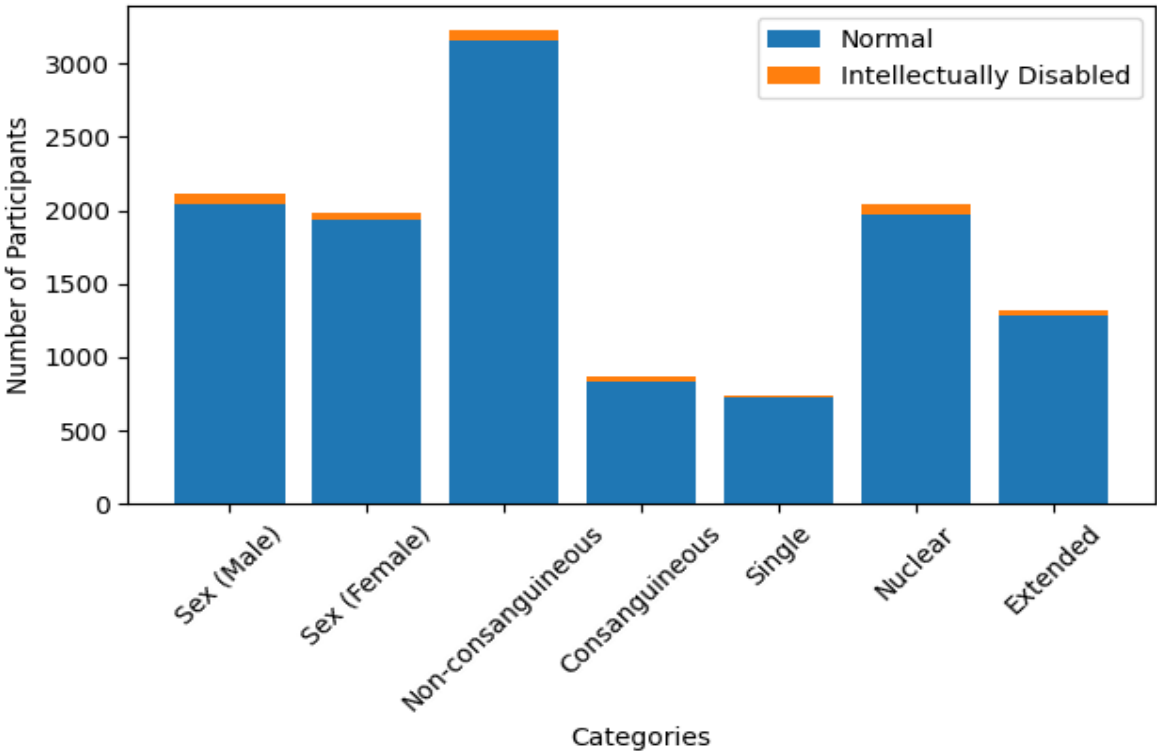
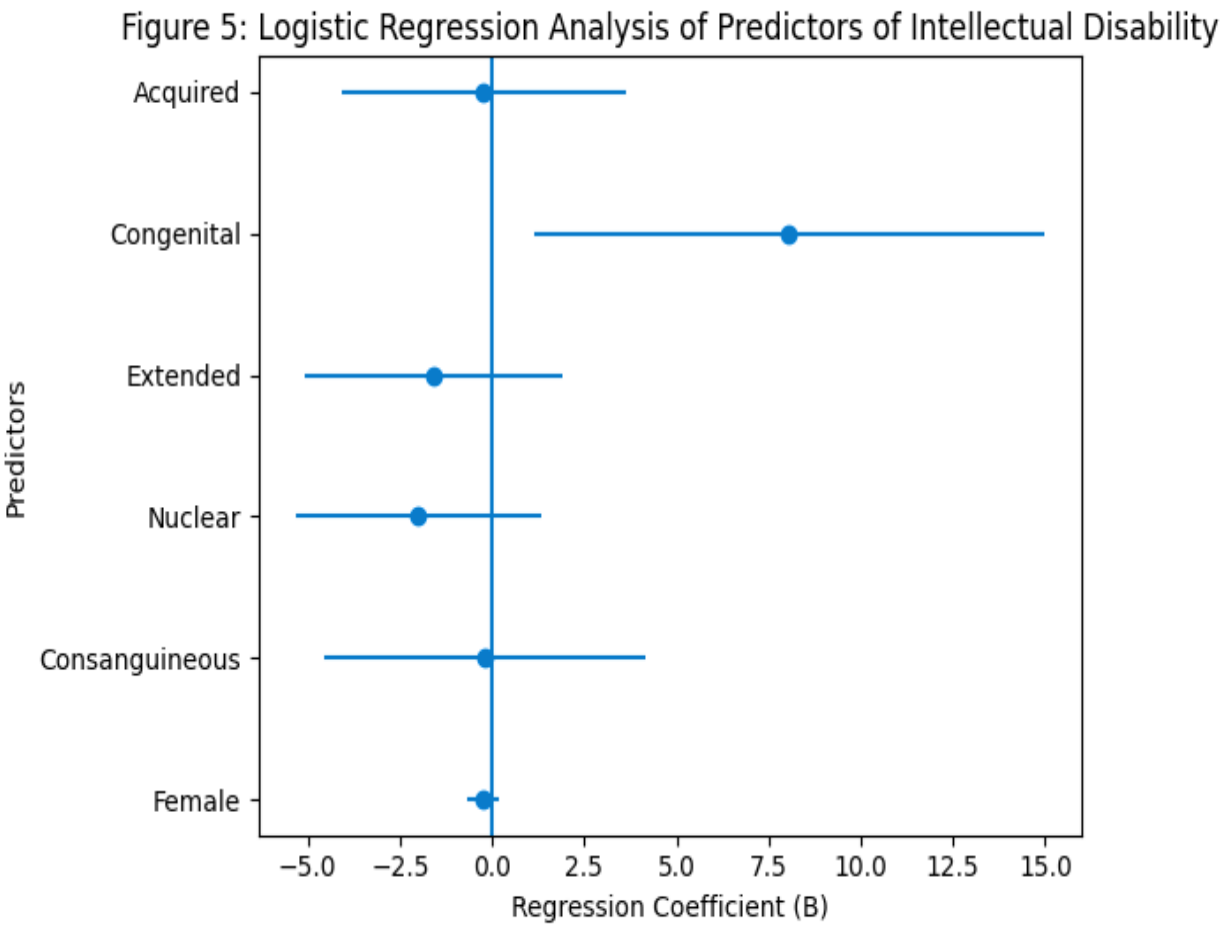




Table 3. Multivariable Firth Penalized Logistic Regression Analysis of Sociodemographic Predictors of Intellectual Disability (N = 4,099)

Predictor	Category	B	SE	p-value	95% CI
Sex	Male (ref)	—	—	—	—
	Female	-0.25	0.21	0.241	-0.67 to 0.17
Parental Marriage	Non-consanguineous (ref)	—	—	—	—
	Consanguineous	-0.22	2.22	0.922	-4.58 to 4.14
Family Structure	Single (ref)	—	—	—	—
	Nuclear	-2.00	1.69	0.236	-5.32 to 1.32
	Extended	-1.59	1.77	0.371	-5.07 to 1.89
Onset of ID	No onset (ref)	—	—	—	—
	Congenital	8.05	3.53	0.023	1.13 to 14.97
	Acquired	-0.23	1.96	0.905	-4.08 to 3.61
Constant		-8.90	2.65	0.001	-14.09 to -3.70

Etiological history was not included in this model due to complete separation (perfect prediction of ID). B = regression coefficient; SE = standard error; CI = confidence interval; ref = reference category. Firth penalized logistic regression was used to reduce bias in parameter estimates for rare events.





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DISCUSSION

The sample intellectual disability prevalence of 2.8 in the District Lower Dir samples has this population at the higher end of the global estimates, but it is also the first systematic epidemiological study of this part of the world which was not previously studied [5-7]. This prevalence is very much consistent with the 2.9% that is shown in meta-analyses of studies in low- and middle-income countries and the 3 percent upper value that the World Health Organization indicates in resource-limited settings [5,7]. This was low relative to the previous Pakistani results of 1.9-2.5% which was probably due to the community based methodology that was employed which consequently revealed the cases that were concealed in the hospital based studies due to stigma, finances and access to healthcare as is the case with the mountainous rural dwellers [11,12]. The most prevalent type of ID is the mild ID (75.7 per cent of individuals with ID, 2.1 per cent of the total population), representing the trends of the world population in general, meaning that with certain educational intervention, a substantial percentage of individuals with ID may be in a position to work independently [21]. The low number of severe and profound ID (0.3% combined) may be as a result of under-ascertainment of the most severely affected who are frequently housebound and less accessible to community surveys. The ID rates between the males were very high (3.5% vs. 2.0; $p=0.003$), which has been recorded in the literature all over the world showing a male preponderation with a ratio of approximately 1.5:1 [5,22]. This trend may be due to X-linked genetic diseases like Fragile X syndrome, higher susceptibility of males to perinatal insults, and diagnostic procedures that prefer to identify males who have externalizing behaviors [23,24]. The loss of the statistical significance of sex in the multivariate analysis, however, is a pointer that excess in males in the univariate analysis may be a result of confounding factors in association with other risk factors rather than an independent biological effect. The greatest measure of ID was consanguinity (4.6 vs. 2.3; $p=0.001$) which has been found to align with literature that documented a rise of risk of autosomal recessive diseases in high cousin marriages [25,26]. The implication of this finding carries a massive health implication to the society bearing in mind that consanguinity unions in Pakistan form 60-70 percent of marriages in the nation [27]. However, the fact that this correlation weakens when there are additional variables indicates that consanguinity could potentially work by increasing the probability of the occurrence of genetic disorders but not independently, and this has profound implications on the development of interventions [28]. The public health plans to prevent consanguinity in marriages and issues surrounding genetic counseling should focus on making genetic counseling more accessible to consanguineous couples and premarital screening programs other than just discouraging the activities being practiced by the culture [29]. Genetic causes (68.7% of the cases of ID) are overwhelmingly high in comparison with the 30-60% range of the literature on the world of the predominantly outbred groups [14,23].



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It is probable that this genetic burden results because of high rates of consanguinity which concentrate recessive mutations, geographic isolation which causes a founder effect and the extensive clinical trials that have defined syndromic forms [30,31]. They played an important role of perinatal (12.2) and postnatal (13.9) factors, which are preventable conditions that ought to be addressed with definite intervention strategies including enhanced obstetric care, increased vaccination, and enhanced nutrition programs [32,33]. The contribution of prenatal factors (maternal infections, teratogen exposure) of 3.5 percent suggests the option of maternal health education and infection control programs. Congenital onset in isolation was also a predictor of ID in multivariate analysis ($B=8.05$, $p=0.023$), which again argues that most of the ID in this group is developmental, and not acquired. The data that the cases counted as acquired onset (postnatal) was merely 18 of 115 (15.7) suggests that the postnatal interventions would contribute less to the total burden of ID compared to the primary prevention of the genetic and perinatal factors.

Study Limitations

It has several limitations that are to be taken into account. To start with, the cross-sectional design does not allow establishing cause-effect relationships; these relationships should be viewed as correlates instead of causal. Second, etiological assignment was based on clinical history and family reports as opposed to molecular confirmation, which created the possibility of recall bias and misclassification. Third, the investigation was carried out in one district that has distinctive demographic and cultural features, which can restrict the extrapolation to other areas of Pakistan. Fourth, the response rate of 67 percent is also satisfactory though it introduces the risk of having non-response bias in case the non-participating households were not comparable to the participants. Fifth, individuals with severe ID in an institutional environment (rare in this case) or individuals who were confined to their homes and inaccessible may have been under-represented.

Strengths

Despite these weaknesses, there are strong points in this paper. The multistage stratified random sampling on population-based design gives representative estimates of LowerDir. The wide sample size ($N=4,099$) and the inclusion of the entire age range (5-90 years old) represent the entire range of ID. Diagnostic validity is met by means of thorough diagnostic assessments in the assistance of standardized tests (UNIT, Stanford-Binet, VABS) and DSM-5 criteria. Logistic regression is used by Firth appropriately to deal with the rare events issue inherent in ID epidemiology.

Implications of Policy and Practice.

The conclusions have a number of implications. To start with, the high percentage rate of genetic causes (68.7) in a high end consanguinity population urges the need to have a regional genetic counseling centre and come up with culturally appropriate premarital screening programs. Second,



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the role of perinatal factors (12.2) is high and it means that additional obstetric training and neonatal resuscitation training should be provided in the peripheral health facilities. Third, the attributable proportion of 13.9 to postnatal factors (meningitis, malnutrition, trauma) suggests that there can be improved vaccination, nutritional programs, and trauma prevention. Fourth, the data of mild ID (75.7% of cases) justifies the inclusion policy of education and the necessity to employ rehabilitation services within the community rather than institutionalization.

Future Research

The case identification that has been identified should be subject to further research in whole-genome sequencing to identify new autosomal recessive intellectual disability genes, which are specific to this group. Here, longitudinal follow-up designs of developmental trends and the natural history of ID are needed. The cost-effectiveness analysis of genetic counseling and perinatal interventions would inform resource allocation. Finally, these quantitative results would be strengthened by qualitative studies on how the community views ID, stigma, and the access to healthcare services.

CONCLUSIONS

It is the first systematic epidemiological survey in District Lower Dir, Khyber Pakhtunkhwa and the findings show that the prevalence of ID is 2.8 and genetic factors account to 68.7 percent of the prevalence which is very high as compared to the outbred populations all over the world. In univariate analysis, consanguinity and male sex are linked with increased prevalence of ID, whereas congenital onset becomes the most significant independent sociodemographic predictor. The findings highlight the pressing need of community-based genetic counseling services, premarital screening programs, enhanced perinatal care, and enhanced postnatal preventive interventions of high-consanguinity settings with limited resources. The study will provide valuable background data of evidence-based development of health policy in rural Pakistan.

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