"ASSESSMENT AND COMPARISON OF COGNITIVE FUNCTION IN OFFSPRINGS OF PATIENTS DIAGNOSED WITH SCHIZOPHRENIA AND ALCOHOL DEPENDENCE SYNDROME"

By

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B.L.D.E (DEEMED TO BE UNIVERSITY)

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In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

PSYCHIATRY

Under the guidance of

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ABSTRACT

Background: Cognitive functioning is essential for academic, social, and adaptive success. Offspring of individuals with psychiatric illnesses, particularly schizophrenia and alcohol dependence syndrome (ADS), are at increased risk of cognitive impairments due to genetic and environmental influences. Understanding the nature and extent of these deficits is critical for early identification and targeted interventions.

Aim: To assess and compare cognitive functioning in the offspring of patients diagnosed with schizophrenia and alcohol dependence syndrome.

Methods: This cross-sectional study included 62 participants (31 in each group) aged 12 years and above, comprising offspring of patients with either schizophrenia or ADS. Cognitive functions were evaluated using the Mini-Mental State Examination (MMSE), Wechsler Intelligence Scale for Children (WISC-IV), and Wechsler Adult Intelligence Scale (WAIS-IV). Statistical analysis was conducted using SPSS version 20, employing Mann-Whitney U tests and Pearson's correlation analyses.

Results: The schizophrenia-offspring group exhibited significantly lower scores in multiple cognitive domains compared to the ADS-offspring group. These included working memory (mean 56.09, $p = 0.001$), perceptual reasoning (mean 89.36, $p = 0.003$), and verbal comprehension (mean 90.64, $p = 0.026$). Full-Scale IQ also showed a near-significant difference ($p = 0.058$). Freedom from distractibility was notably lower in the schizophrenia group (mean 75.35,$p = 0.005$). MMSE scores did not differ significantly between the groups. Correlation analyses revealed that the duration of parental illness moderately influenced cognitive outcomes.

Conclusion: Offspring of schizophrenia patients exhibit more pronounced cognitive deficits—especially in working memory, verbal comprehension, and executive function—than those of alcohol-dependent parents, indicating a greater heritable neurodevelopmental risk. Early screening with tools like WISC or WAIS-IV and targeted interventions are essential to reduce long-term academic and functional impairments in these high-risk groups.

Keywords: Cognitive function, schizophrenia, alcohol dependence syndrome, offspring, WAIS, WISC, MMSE, neurodevelopment, working memory, verbal comprehension.

1 INTRODUCTION:

Cognitive functioning includes core mental processes such as attention, perception, memory, language, learning, problem-solving, and executive skills [1], all of which are vital for adapting to one's environment, achieving academic goals, managing behavior, and building social relationships [2]. While cognitive development typically follows a standard progression, early disruptions—especially those linked to parental psychiatric conditions—can interfere significantly with this trajectory. Children of individuals with schizophrenia or alcohol dependence syndrome are particularly vulnerable. Genetic studies have identified several chromosomal regions associated with the heritability of cognitive deficits seen in offspring of alcohol-dependent parents. Notably, chromosome 4q has been repeatedly linked to variations in P300 amplitude, a neurophysiological indicator of attention and working memory, as shown in studies by Hill et al. (1998) and Porjesz et al. (2002). Chromosome 2p14–q21 has also been associated with externalizing behaviors and related endophenotypes in research by Dick et al. (2004). Additionally, Kendler et al. (2011) found connections between chromosome 6q and both early-onset alcohol use and cognitive difficulties. These findings indicate that specific genetic regions may influence not only susceptibility to alcohol dependence but also the neurocognitive and behavioral characteristics that increase risk in affected offspring. [3].

Schizophrenia is a complex and chronic neuropsychiatric disorder affecting approximately 1% of the global population. Characterized by persistent psychotic symptoms such as hallucinations, delusions, and disorganized thinking, the disorder also significantly impacts social functioning and emotional regulation [4]. One of the less visible but increasingly recognized aspects of schizophrenia is cognitive dysfunction. These deficits span many domains, including attention, memory, processing speed, and executive functioning. Importantly, cognitive impairments are not episodic like psychosis; instead, they remain relatively stable and represent a core feature of the disorder [5]. A study by Snitz (2006 assessed 140 patients with schizophrenia and found that 89.3% experienced significant cognitive impairment, with impairments observed in attention (60%), memory (65.7%), fluency (55%), language (61.4%), and visuospatial skills (63.6%) (43).

Crucially, these cognitive abnormalities are not restricted to individuals already diagnosed with schizophrenia. A growing body of evidence highlights the presence of cognitive deficits in their biological offspring, even in the absence of clinical symptoms [6]. These deficits often become apparent during key

developmental periods such as school-age and adolescence. Research by Alloway et al. (2009) indicates that 55% to 60% of these children perform below average in neuropsychological tasks assessing working memory, attention span, and verbal reasoning. These impairments can interfere with academic progression and adaptive functioning. Approximately 52% of these offspring show reduced academic achievement compared to their age-matched peers, often necessitating individualized educational plans, remedial instruction, or exceptional learning accommodations in formal school settings [7].

“Schizophrenia has a heritable component (60–80%), and early-life cognitive impairments in offspring are increasingly considered genetically linked traits, or endophenotypes (Tsuang et al., 1999).”“Endophenotypes refer to internal, genetically inherited features that are not immediately evident but suggest increased vulnerability to psychiatric disorders.” signal a heightened risk of developing a disorder. In schizophrenia, endophenotypes help connect genetic predisposition to observable clinical features (Gottesman & Gould, 2003). “Oculomotor irregularities like poor performance in antisaccade tasks are commonly seen in both patients and unaffected relatives, marking potential neurocognitive traits of risk.” (Nuechterlein et al., 2004). Additionally, neurophysiological markers like reduced P300 amplitude, lower mismatch negativity (MMN), and impaired pre-pulse inhibition (PPI) are frequently documented (Jeon & Polich, 2003; Javitt et al., 2008). Oculomotor irregularities, such as impaired smooth pursuit and antisaccade performance, are also found in patients and their close relatives, reinforcing their significance as endophenotypes (Calkins et al., 2008). These traits help understand schizophrenia’s biological roots and identify vulnerable individuals before symptom onset.

Similarly, “Children of alcohol-dependent parents often face challenges in working memory, impulse control, and sustained attention (Hill et al., 2000; Iacono & Malone, 2011).”“Lower P300 responses in event-related potential studies may reflect inherited vulnerabilities to alcohol use disorders (Begleiter et al., 1984; Porjesz & Begleiter, 2003).” Other neurophysiological features, such as increased resting EEG beta activity and diminished error-related negativity (ERN), suggest abnormalities in arousal regulation and cognitive control (Iacono et al., 2002). Furthermore, “Traits such as impulsivity, difficulty regulating emotions, and early behavioral issues are common among these children, indicating heightened risk for later psychopathology.”(Sher et al., 1991; Tarter et al., 2003). These findings underscore the value of endophenotypes as early markers that can guide preventive interventions before the full onset of illness. (8)

A comprehensive meta-analysis of neuropsychological assessments conducted on first-degree relatives of schizophrenia patients by Snitz et al. (2006) strongly supports the hypothesis that cognitive dysfunction serves as a widespread and reliable indicator of familial psychiatric risk [(43).] The findings revealed that sustained attention deficits were observed in approximately 57% of these individuals, impairments in executive functioning were present in 54%, and verbal learning deficits affected 49%. These figures are significantly elevated compared to baseline estimates in the general population, which typically report much lower incidence rates for such impairments. This disparity reinforces the understanding that these cognitive deficits are not incidental but are closely tied to a heritable neurodevelopmental vulnerability associated with schizophrenia. These cognitive markers often remain present even without clinical symptoms, underscoring their value as early risk indicators. Thus, these findings advocate for the routine use of cognitive screening tools in the offspring of schizophrenia patients to enable early detection, preventive mental health support, and academic interventions [10].

Additionally, children raised in families affected by schizophrenia are not only influenced by genetic factors but are also frequently exposed to a range of environmental challenges that can exacerbate cognitive vulnerability [[11].] Chen et al. (2025) highlighted that early life stress can significantly disrupt the development and plasticity of prefrontal cortical circuits, which aligns with findings that children of individuals with schizophrenia, many of whom experience adverse childhood events, are at heightened risk for impaired cognitive and emotional regulation. [(11).] Moreover, it is estimated that 44% of these children live in economically marginalized households, and 51% have limited or no access to early childhood education and structured learning environments.

These socioeconomic constraints further hinder the cognitive stimulation necessary for optimal development, placing this population at a compounded risk of long-term neurodevelopmental deficits [[12].]

In parallel, alcohol dependence syndrome (ADS) represents a chronic, progressive, and relapsing condition that is characterized by compulsive alcohol intake, increased tolerance, withdrawal symptoms, and a noticeable decline in occupational and social functioning [[13]]. While a wealth of research has documented the direct neurotoxic effects of chronic alcohol use on individuals, a growing body of literature now emphasizes the significant intergenerational impacts of Alcohol Dependence Syndrome (ADS). For instance, Gierski et al. (2013) found that adult offspring of alcohol-dependent probands exhibit notable impairments

in executive functions, highlighting the lasting neurocognitive consequences associated with parental alcohol dependence. [29]. Alarmingly, approximately 45% to 50% of children of alcohol-dependent parents demonstrate moderate to severe cognitive impairments across various domains. The most frequently affected areas include attention regulation, processing speed, inhibitory control, and working memory. These deficits can manifest early in childhood and tend to persist into adolescence unless addressed through targeted interventions [14].

Children of alcohol-dependent parents often exhibit significant cognitive and academic difficulties due to a complex interplay of genetic, neurobiological, and environmental factors. Prenatal alcohol exposure and inherited vulnerabilities may impair the development of brain regions responsible for executive functioning, attention, and memory. These impairments are often compounded by chronic exposure to household stress, emotional neglect, and inconsistent caregiving, all of which disrupt healthy neurodevelopment and interfere with learning processes. In academic settings, these challenges manifest as difficulties with tasks requiring sustained attention, sequencing, and cognitive flexibility—skills essential for reading comprehension, problem-solving, and classroom participation. Teachers and psychologists frequently observe increased rates of inattention, impulsivity, and academic disengagement in such children, often necessitating remedial education, individualized education plans (IEPs), or special education support. Moreover, the emotional burden of growing up in a substance-affected environment can further reduce motivation, increase school absenteeism, and lead to behavioral disturbances, thereby compounding the cognitive and educational impact [15].

Moreover, the impact of ADS extends beyond biological mechanisms. Children raised in alcohol-affected households are disproportionately exposed to family dysfunction, unstable living conditions, and increased stress levels. According to family studies, around 59% of such children report exposure to parental conflict, and 42% experience neglect or inconsistent caregiving [16].

These factors adversely affect brain maturation, particularly in the prefrontal cortex, crucial for executive function, decision-making, and self-regulation. Developmental psychologists have reported that nearly 39% of children with an alcohol-dependent parent display significant emotional and behavioral regulation issues by the age of 12, often accompanied by observable cognitive lag [17].

Notably, prenatal alcohol exposure remains another contributing factor. Even in cases where the parent ceased alcohol consumption postnatally, exposure during pregnancy has lasting effects. Among children exposed to alcohol in utero, an estimated 35–40% show signs of cognitive impairment, including difficulties in language processing, learning, retention, and abstract thinking. These effects are often persistent and can affect social adaptability and self-esteem during adolescence and early adulthood [18].

While both groups—offspring of schizophrenia patients and offspring of ADS patients—are at a high risk of cognitive deficits, the specific profiles of impairment may differ. The schizophrenia group tends to show broader and more pervasive impairments across several cognitive domains, with nearly 58% affected in three or more areas [19]. In contrast, cognitive dysfunction in the ADS group is often more localized to attention and impulse control, though still prevalent in 45–50% of cases. These distinctions are clinically relevant, as they may reflect differences in underlying neurodevelopmental pathways and inform group-specific intervention strategies [20].

Importantly, the long-term implications of these cognitive deficits extend into adulthood. Longitudinal cohort studies have demonstrated that approximately 37% of children of schizophrenia patients and 33% of children of alcohol-dependent individuals go on to develop a psychiatric disorder by the age of 25, including mood disorders, anxiety, and substance use disorders [21]. Furthermore, children with early cognitive difficulties are significantly more likely to drop out of school, experience social isolation, and struggle to maintain employment. According to public health data, nearly 42% of at-risk offspring with cognitive impairments require mental health intervention during adolescence—a rate three times higher than in the general population.

Given the significant public health implications, early identification and comparison of cognitive functioning in these two at-risk groups is paramount. Evaluating the similarities and differences in cognitive profiles can help in understanding the etiological distinctions between schizophrenia and ADS and may facilitate the development of early preventive strategies. Plassman et al. (2010), in a comprehensive systematic review published in the *Annals of Internal Medicine*, identified several modifiable and non-modifiable factors associated with cognitive decline in later life. The study highlighted that lower educational attainment, which limits cognitive reserve, significantly increases vulnerability to cognitive deterioration. Additionally, the presence of cardiovascular risk factors, such as hypertension, diabetes, and smoking, was found to affect

cerebral blood flow adversely and contribute to neurodegeneration. Depression emerged as another significant contributor, likely due to its association with hippocampal atrophy and poor neurocognitive outcomes. The authors also noted that physical inactivity, social isolation, and limited engagement in mentally stimulating activities accelerate cognitive decline. Furthermore, genetic predisposition, particularly the presence of the APOE ε4 allele, was identified as a strong non-modifiable risk factor for age-related cognitive impairment. These findings underscore the multifactorial nature of cognitive decline and the importance of early intervention through lifestyle modifications and management of medical comorbidities. Furthermore, insights into domain-specific impairments, such as attention deficits in ADS offspring or executive dysfunction in schizophrenia offspring, can guide tailored educational and clinical interventions that address the unique challenges faced by each group [20]

This study seeks to systematically assess and compare cognitive functioning among offspring of individuals diagnosed with schizophrenia and alcohol dependence syndrome. The study will use established neuropsychological assessment tools to examine key domains such as attention, memory, executive function, and cognitive flexibility. Through a comparative approach, this research aims to illuminate the scope and specificity of cognitive impairments in each group, contributing to a more nuanced understanding of intergenerational psychiatric risk and laying the foundation for future cognitive and behavioral interventions.

2 REVIEW OF LITERATURE:

The quest to understand how schizophrenia and alcohol dependence uniquely affect cognitive functioning led Dixit et al. (2020) to undertake a comparative neuropsychological investigation. Driven by the need to distinguish the cognitive impact of these disorders, the researchers designed a hospital-based cross-sectional study that carefully recruited 30 patients with chronic schizophrenia, 30 alcohol-dependent individuals, and 30 healthy matched controls. The participants were evaluated using well-established instruments: "the Positive and Negative Syndrome Scale (PANSS)" for schizophrenia and "the Severity of Alcohol Dependence Questionnaire (SADQ-C)" for alcohol use disorders. Cognitive functioning was assessed via the "AIIMS Comprehensive Neuropsychological Battery in Hindi (Adult Form)", allowing for a comprehensive evaluation across cognitive domains. The findings were telling: 83.3% of chronic schizophrenia patients demonstrated neuropsychological dysfunction, compared to 36.7% in the alcohol-dependent group, while no impairments were noted in the healthy controls. Schizophrenia patients showed marked deficits in motor skills, tactile functions, visual and speech processing, reading, writing, arithmetic, memory, and intellectual capabilities. Alcohol-dependent individuals also exhibited significant, albeit less severe, impairments in many of these domains. Correlations between PANSS/SADQ scores and cognitive dysfunction emphasized that the severity of clinical symptoms was closely tied to the extent of cognitive impairment. Dixit concluded that while both conditions involve cognitive deficits, schizophrenia results in more severe and widespread impairments, particularly in tactile perception, memory, arithmetic, and general intellectual functions [22].

In an effort to shed light on the concept of resilience, often overlooked in neurocognitive research, Deng et al. (2018) sought to compare adaptive resilience levels among individuals with "schizophrenia, bipolar disorder, and healthy controls", while also exploring the relationship between resilience and cognitive performance. The study involved 81 patients with schizophrenia, 34 with bipolar disorder, and 52 healthy individuals. Each participant completed the Connor-Davidson Resilience Scale (CD-RISC) and underwent cognitive assessments focused on verbal comprehension, executive function, and working memory. Using group comparisons and linear regression, Deng observed significantly lower resilience and cognitive performance in both clinical groups compared to healthy controls, with schizophrenia patients scoring the lowest. Interestingly, although resilience correlated positively with cognitive performance across the full

sample, the association did not hold within each diagnostic group when examined independently. Regression analysis further revealed that cognitive performance could not fully account for differences in resilience, implying that variables, perhaps emotional, social, or environmental, might contribute. The study concluded that while schizophrenia and bipolar disorder impair both resilience and cognitive functioning, resilience appears to be influenced by broader factors beyond the cognitive domains assessed [23].

Exploring the biological underpinnings of cognitive dysfunction in schizophrenia, Epstein et al. (2014) focused on white matter abnormalities in adolescents with early-onset schizophrenia (EOS). Their study also aimed to determine whether these structural changes could be linked to neurocognitive deficits. To do this, they compared four groups: 55 adolescents with EOS, 21 identified as "clinically high risk (CHR) for schizophrenia, 31 with cannabis use disorder (CUD), and 55 healthy controls. Participants underwent diffusion tensor imaging (DTI) and tractography to assess fractional anisotropy (FA)—a marker of white matter integrity—across six major white matter tracts, including the cingulum bundle, corticospinal tract (CST), and inferior fronto-occipital fasciculus (IFOF). The EOS and CHR groups exhibited reduced FA in bilateral CST, left ILF, and left IFOF compared to healthy controls. Notably, the CUD group only showed reduced FA in the left IFOF. Furthermore, reduced FA in the left ILF and IFOF among EOS participants was linked to poorer neurocognitive performance, suggesting that structural changes in language and visual processing tracts may contribute to cognitive deficits in schizophrenia. Epstein concluded that white matter abnormalities, especially in the left hemisphere, could serve as early biomarkers of schizophrenia risk and help explain associated cognitive dysfunction [24].

Taking a life-course approach, Meier et al. (2014) asked whether schizophrenia involves a specific pattern of neuropsychological decline from childhood to adulthood, and how this compares with other disorders. They followed a representative birth cohort of 1,037 individuals from Dunedin, New Zealand, born in 1972–1973, and assessed them from childhood through age 38, achieving an impressive 95% retention rate. Cognitive testing, including IQ and domain-specific assessments, was conducted at ages 7, 9, 11, 13, and again at 38. The study found that individuals who developed schizophrenia showed clear declines in IQ and in specific cognitive areas like processing speed, learning, executive functioning, and motor abilities over time. Notably, verbal abilities and delayed memory remained relatively intact. This decline was unique to schizophrenia and was not observed in those with persistent depression or mild cognitive impairment. Furthermore, informant reports

highlighted more real-world cognitive challenges in the schizophrenia group. Meier concluded that schizophrenia involves a distinct pattern of domain-specific cognitive decline, likely rooted in early developmental neurobiological changes [25].

To disentangle the overlapping yet distinct manifestations of inattention in schizophrenia and adult ADHD, Hwang et al. (2025) explored how inattention differs between schizophrenia and adult ADHD—two disorders that share this symptom despite contrasting clinical features. Hwang et al. aimed to determine whether attention deficits and disrupted connectivity in these disorders reflect a common pathophysiological mechanism or are distinct in pattern and severity, which could inform differential diagnosis and targeted treatment strategies. In a study involving 20 participants from each group (schizophrenia, ADHD, and healthy controls), attention tests, IQ assessments, and resting-state fMRI were used to examine brain activity, particularly within the default mode network (DMN). The results revealed opposing patterns: schizophrenia patients showed reduced DMN connectivity linked to slower processing speed, while ADHD patients had heightened connectivity associated with more divided-attention errors. These findings suggest that although both disorders involve inattention, the underlying neural mechanisms differ, reflecting disorder-specific patterns of DMN dysregulation [21].

Meanwhile, Malisza et al. (2012) investigated whether children with alcohol-related neurodevelopmental disorder (ARND) and those with ADHD, despite similar outward behaviors, exhibited different brain activity during cognitive tasks. The study included 63 children aged 10 to 14 years across three groups: ARND, ADHD, and typically developing (TD) controls. During a 1-back spatial working memory task, fMRI and DTI were used to measure brain activation and white matter integrity. TD children activated posterior brain regions, ARND children showed heightened activation in both frontal and parietal regions, and ADHD children engaged frontal areas alone. Interestingly, ARND children had the highest overall brain activity but showed reduced accuracy and increased variability in response times. DTI results showed reduced white matter integrity in ARND participants, correlating with poorer task performance. The study concluded that while ARND and ADHD children may appear behaviorally similar, the neurological mechanisms driving their behavior differ significantly [26].

Turning to familial vulnerability, Gierski et al. (2013) explored whether executive dysfunction and impulsivity, commonly observed in alcohol dependence, might also be present in non-alcoholic adult offspring. Involving 155 adults screened for psychiatric and substance use conditions, the study compared those with (FHP) and

without (FHN) a family history of alcohol dependence. Participants were assessed using the Barratt Impulsiveness Scale (BIS-11) and a neuropsychological test battery for executive functioning (EF). The results showed that FHP individuals had significantly higher impulsiveness and lower EF performance than FHN participants. Regression analysis revealed that the number of alcohol-dependent family members predicted EF impairment, while impulsiveness was independent of family history. Gierski concluded that EF impairments may serve as neurocognitive endophenotypes—heritable risk markers—for alcohol dependence, whereas impulsivity reflects a separate vulnerability [27].

Investigating the compounded burden of comorbidity, Manning et al. (2007) set out to determine whether individuals with dual diagnoses—schizophrenia and alcohol dependence—experience greater cognitive impairment than those with either condition alone. Drawing from a community psychiatric sample of 120 patients, Manning divided them into three groups: schizophrenia only, alcohol dependence only, and both conditions. Participants completed the Mini-Mental State Examination (MMSE) alongside psychiatric and substance use evaluations. Results showed that those with both schizophrenia and alcohol dependence exhibited significantly greater impairments in language, reading, writing, and visuospatial tasks. Interestingly, MMSE global scores failed to capture these domain-specific deficits, prompting Manning to recommend age-adjusted MMSE scoring for more accurate detection. The study emphasized that dual diagnosis patients carry a heavier cognitive burden, requiring nuanced assessment tools [28].

Lastly, Green et al. (2009) explored how prenatal alcohol exposure affects executive functioning in children diagnosed with "fetal alcohol spectrum disorders (FASD)". The study included 89 children aged 8 to 15 years with "FAS, partial FAS (pFAS), or alcohol-related neurodevelopmental disorder (ARND), compared to 92 healthy controls. Using the Cambridge Neuropsychological Tests Automated Battery (CANTAB), the team assessed reaction time, decision-making, planning, and spatial working memory." Results showed that FASD children had significantly longer reaction and decision times, with increasing deficits as task complexity grew. Spatial working memory emerged as particularly impaired, with an effect size of Cohen's d = 1.1. Though minor differences existed among the FASD subtypes, all were significantly more impaired than controls. Green concluded that CANTAB is a sensitive tool for detecting cognitive deficits in children prenatally exposed to alcohol, even in the absence of visible facial anomalies [29].

3 AIMS

To assess and compare the cognitive functions in the offspring of patients diagnosed with schizophrenia and Alcohol Dependence syndrome.

OBJECTIVES

- To assess the cognitive function in the offspring of patients diagnosed with schizophrenia.
- To assess cognitive function in the offspring of patients diagnosed with alcohol dependence syndrome.
- To compare the cognitive function of offspring of schizophrenia and alcohol dependence syndrome

MATERIAL AND METHOD

4.1 SOURCE OF DATA:

The study will be performed at BLDE (DU) Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura.

4.2 METHOD OF COLLECTION OF DATA:

4,3 INCLUSION CRITERIA

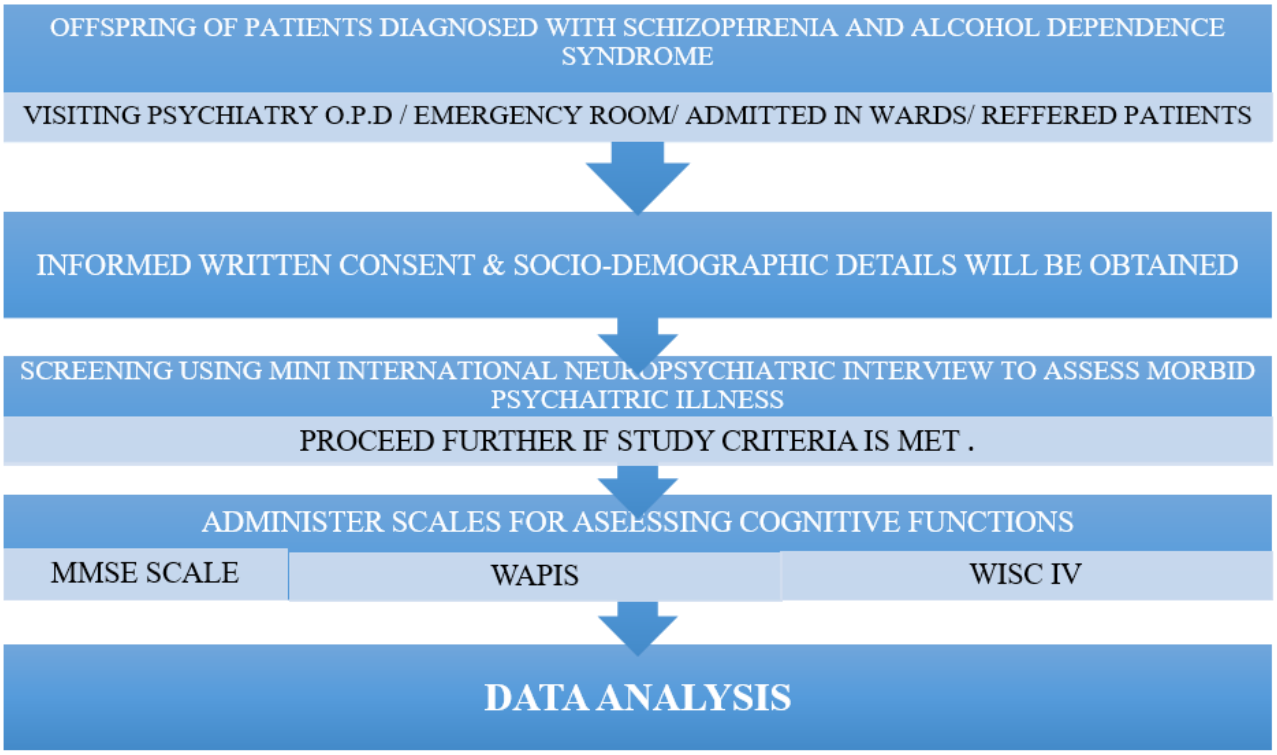
Any Offspring of patients diagnosed with either schizophrenia or alcohol dependence syndrome, aged 12 and above.

4.4 EXCLUSION CRITERIA

1. Offspring already diagnosed with any psychiatric illness.
2. Offspring with a history of alcohol use.
3. Offspring diagnosed with Intellectual Disability Disorder.
4. Any major medical illness that affects cognitive function.

4.5 STUDY DURATION: 24 MONTHS (May 2023- MAY 2025)

4.6 METHODOLOGY:



4.7 SAMPLING:

With the anticipated Prevalence of both illnesses, the study would require a sample size of 62(for each group 31, assuming equal group sizes), so to achieve a power of 85% for detecting a difference in Means: Inequality, t-tests - Means: Difference between two independent means (two groups) with 5% level of significance.

Statistical Analysis:

The data obtained will be entered into a Microsoft Excel sheet, and regression analyses cognitive performance will be performed. Using a statistical package for the social sciences (SPSS) (Version 20).

Results will be presented as Mean, SD, counts, percentages, and diagrams. The two groups will be compared for normally distributed continuous variables using an independent sample t-test. For not normally distributed variables, the Mann-Whitney U test is used. For Categorical variables, the two groups will be compared using the Chi-square test/Fisher's exact test. If $p<0.05$, it will be considered statistically significant. All statistical data will be analyzed using a two-tailed test.

4.8 SCALES USED FOR ASSESSMENT:

1. Mini-Mental Status Examination
2. Wechsler intelligent scale for children IV
3. Wechsler Adult Intelligence Scale.

1 MINI-MENTAL STATE EXAMINATION (MMSE):

The **Mini-Mental State Examination (MMSE)** is one of the most widely used cognitive screening tools for detecting **global cognitive impairment**, particularly in elderly populations. Developed by Folstein et al. in 1975, the MMSE evaluates functions such as orientation, attention, memory, language, and visuospatial construction. It demonstrates acceptable **construct and concurrent validity**, with significant correlations observed between MMSE scores and clinical diagnoses of dementia and other standardized neuropsychological tests. Studies report **sensitivity ranging from 70% to 85%** and **specificity from 70% to 80%,** depending on the chosen cutoff score and patient population. When administered by trained professionals, the tool also shows strong test-retest reliability ($r = 0.80–0.95$) and high inter-rater reliability ($\kappa = 0.83–0.99$). However, its performance can be influenced by **educational level, language, and cultural background**, necessitating localized adaptations such as the Hindi MMSE for better accuracy in non-Western populations. Despite its limitations, the MMSE remains a practical, time-efficient, and clinically valuable instrument in both research and routine cognitive assessment.

2 WECHSLER INTELLIGENCE SCALE FOR CHILDREN (WISC)

The **Wechsler Intelligence Scale for Children – Fifth Edition (WISC-V)** is a comprehensive, standardized tool used to assess the intellectual functioning of children aged **6 to 16 years**. It evaluates five primary cognitive domains: **Verbal Comprehension**, **Visual Spatial**, **Fluid Reasoning**, **Working Memory**, and **Processing Speed**, contributing to the **Full-Scale IQ (FSIQ)**. The WISC-V exhibits excellent **construct validity**, with factor analyses supporting its five-factor model, aligning with contemporary theories of cognitive development. It also demonstrates strong **concurrent validity**, showing robust correlations with other standardized measures such as

the Stanford-Binet Intelligence Scales and the Woodcock-Johnson Tests. Regarding **reliability**, the WISC-V yields high internal consistency (Cronbach's α ranging from **0.84 to 0.95** across index scores) and **test-retest reliability** coefficients typically ranging from **0.82 to 0.93**, indicating strong stability over time. Due to its robust psychometric properties and clinical utility, the WISC-V is widely employed in educational, psychological, and neurodevelopmental evaluations for identifying learning disorders, intellectual disability, ADHD, and other cognitive impairments in children.

3. THE WECHSLER ADULT INTELLIGENCE SCALE –

Fourth Edition (WAIS-IV) is a widely used standardized instrument designed to assess the intellectual functioning of individuals aged 16 to 90 years. It provides a comprehensive profile of cognitive abilities through four primary index scores: Verbal Comprehension, Perceptual Reasoning, Working Memory, and Processing Speed, along with a Full-Scale IQ (FSIQ). The WAIS-IV demonstrates strong psychometric properties, with high construct and concurrent validity, as evidenced by its alignment with theoretical intelligence models and strong correlations with other established cognitive measures. Its well-established predictive validity makes it helpful in identifying cognitive deficits across a range of neurological and psychiatric conditions. The scale also shows excellent reliability, with test-retest coefficients for composite scores ranging from 0.82 to 0.94 and internal consistency coefficients (Cronbach's α) ranging from 0.88 to 0.98, indicating stable and consistent measurement. Because of its clinical precision and diagnostic utility, the WAIS-IV remains a cornerstone in adult cognitive assessment across psychological, educational, and neuropsychiatric contexts.

RESULTS

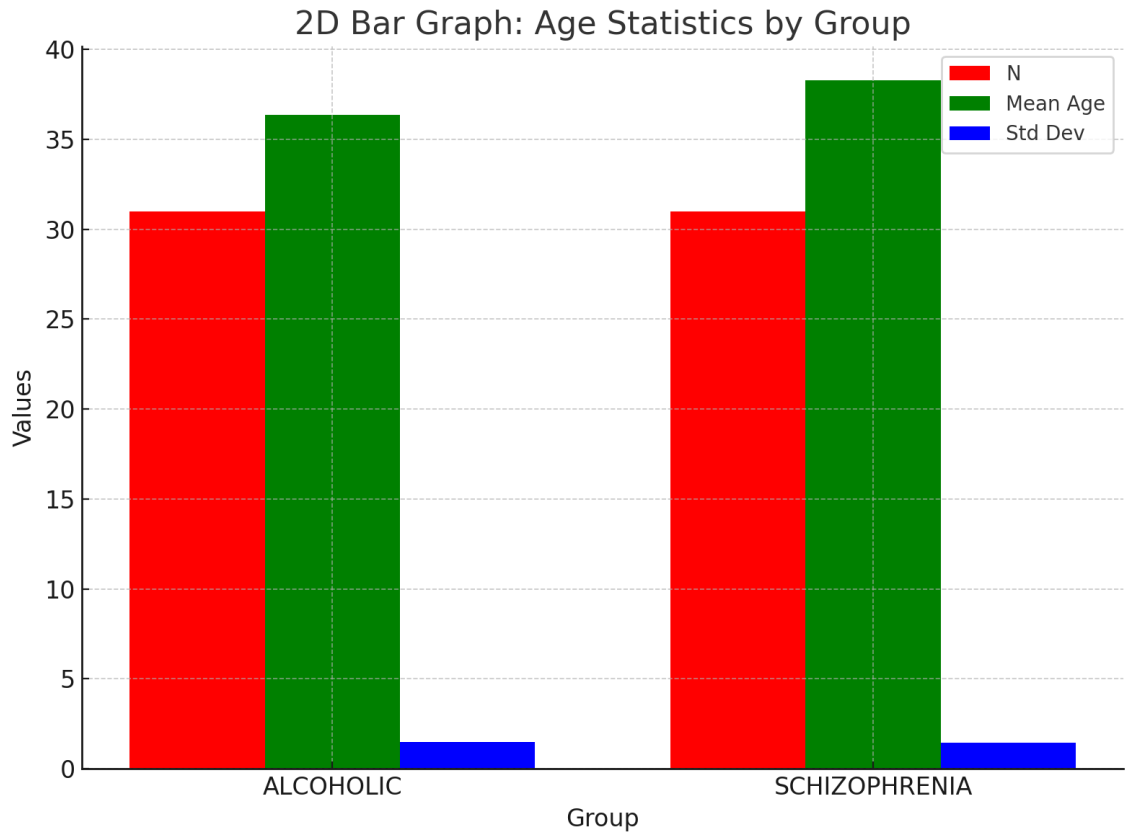
SOCIODEMOGRAPHIC PROFILE

AGE

Table 1: AGE

AGE	N	MEAN AGE	STD DEVIATION
OFFSPRINGS OF ALCOHOLIC	31	36.38	1.480
OFFSPRINGS OF SCHIZOPHRENIA	31	38.27	1.451

Graphical representation 1: AGE



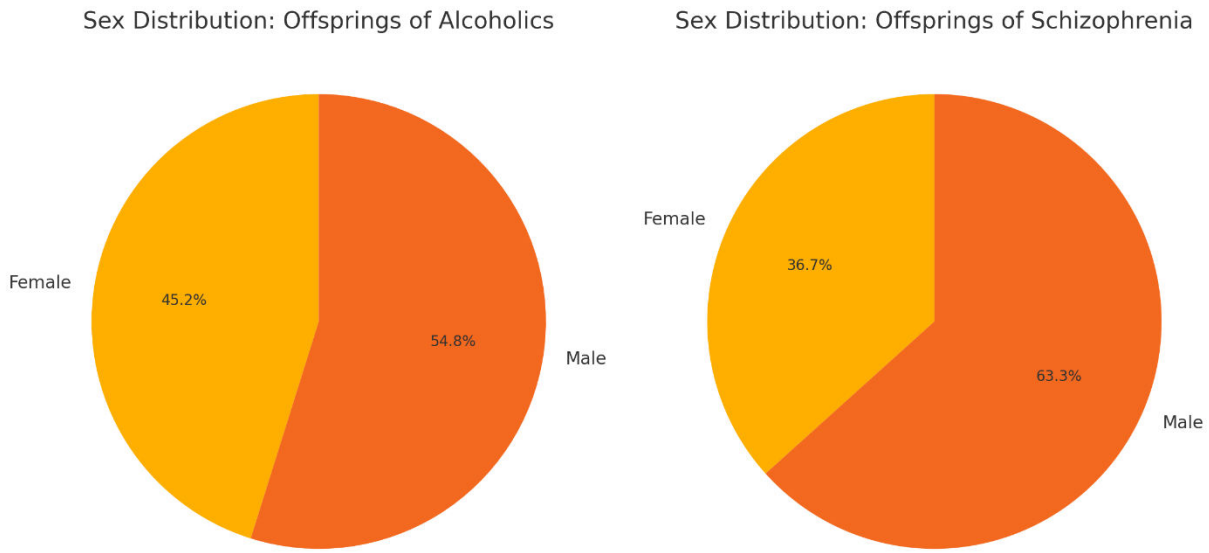
Graph 1: AGE

SEX

Table 2: SEX

SEX	OFFSPRINGS OF ALCOHOLICS	OFFSPRINGS OF SCHIZOPHRENIA	TOTAL
FEMALE	14	11	25
MALE	17	19	20
TOTAL	31	31	62

Graphical representation 2: SEX



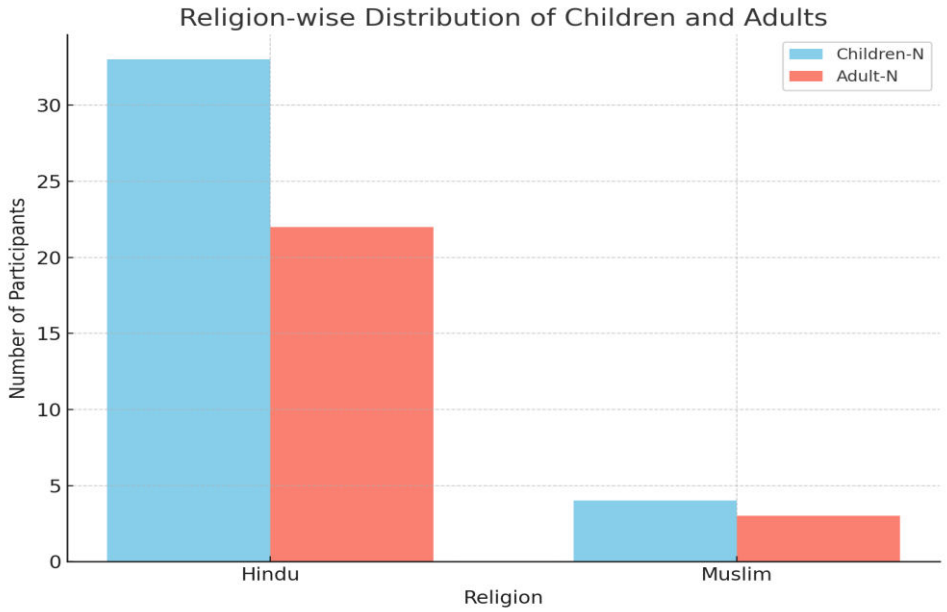
Graph 2: SEX

RELIGION

Table 3: RELIGION

RELIGION	CHILDREN-N	ADULT-N	TOTAL
HINDU	33	22	55
MUSLIM	4	3	7
TOTAL	37	25	62

Graphical representation 3: RELIGION

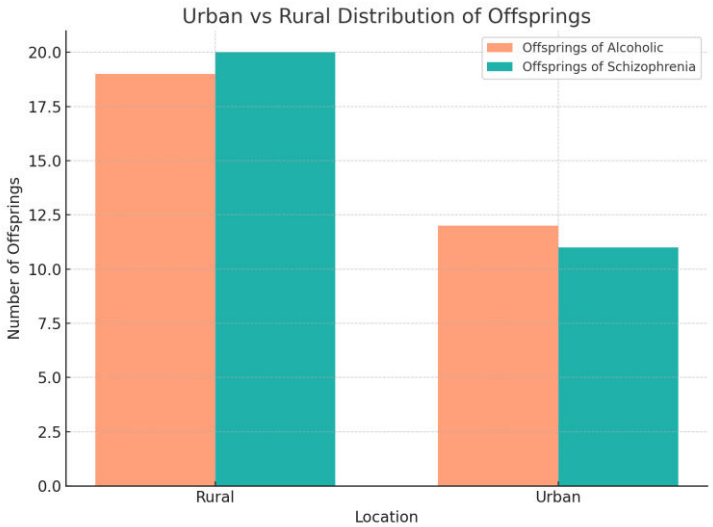


URBAN AND RURAL

Table 4: URBAN AND RURAL

	OFFSPRINGS OF ALCOHOLIC	OFFSPRINGS OF SCHIZOPHRENIA	TOTAL
RURAL	19	20	39
URBAN	12	11	23
TOTAL	31	31	62

Graphical representation 4: URBAN AND RURAL

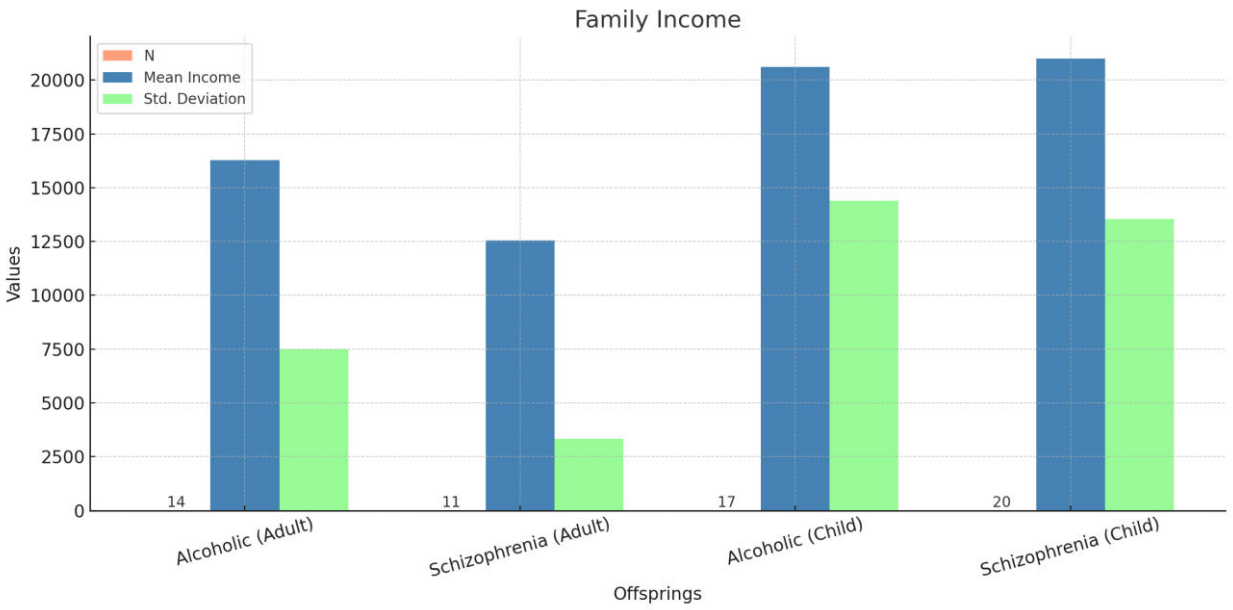


FAMILY INCOME

Table 5: FAMILY INCOME

Offsprings	N	Mean Income	Std. Deviation
Alcoholic (Adult)	14	16,285.71	7,487.72
Schizophrenia (Adult)	11	12,545.45	3,327.57
Alcoholic (Child)	17	20,617.65	14,393.50
Schizophrenia (Child)	20	21,000.00	13,549.13

Graphical representation 5: FAMILY INCOME

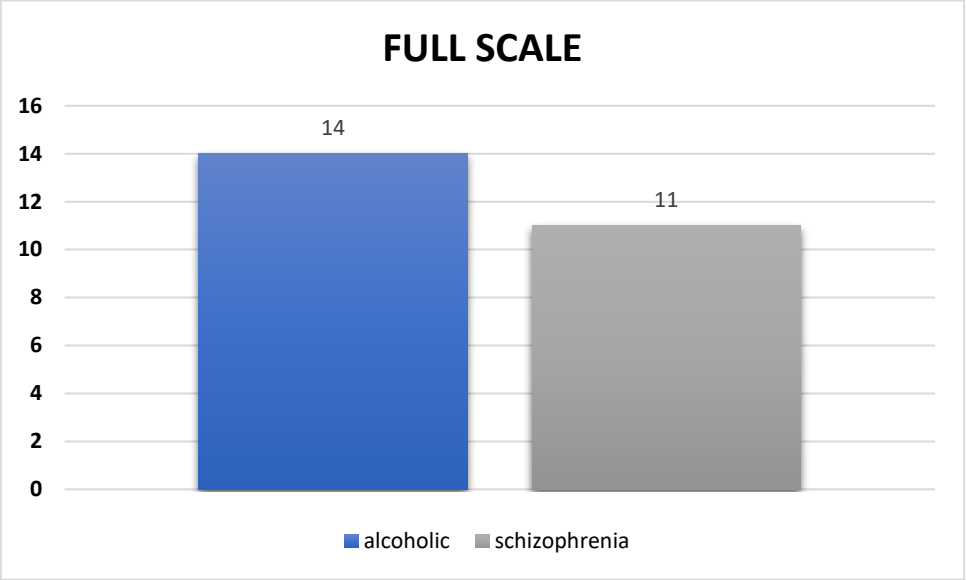


Graph 5: FAMILY INCOME

WAIS 4: Wechsler Adult Intelligence Scale 4 GROUP STATISTICS OF OFFSPRINGS OF ALCOHOLIC PARENTS VERSUS OFFSPRINGS OF SCHIZOPHRENIA(ADULT)

TABLE 6 Group **Statistics**

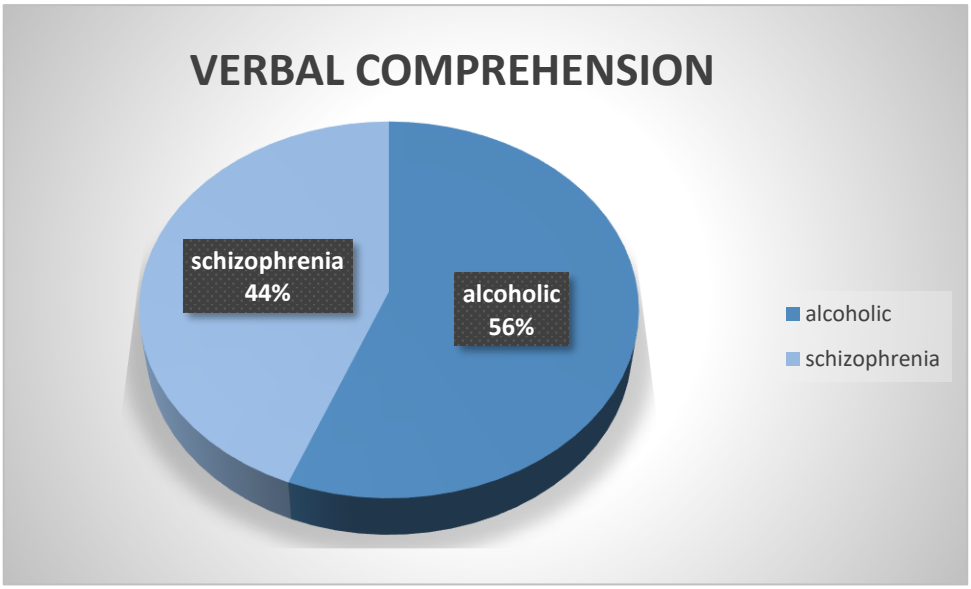
ADULT		N	MEAN	St. Deviation
FULL SCALE	OFFSPRINGS OF ALCOHOLIC	14	93.79	8.972
	OFFSPRINGS OF SCHIZOPHRENIA	11	87.27	6.857
VERBAL COMPREHENSION	OFFSPRINGS OF ALCOHOLIC	14	100.29	11.789
	OFFSPRINGS OF SCHIZOPHRENIA	11	90.64	7.379
PERCEPTUAL REASONING	OFFSPRINGS OF ALCOHOLIC	14	107.00	12.070
	OFFSPRINGS OF SCHIZOPHRENIA	11	89.36	10.984
WORKING MEMORY	OFFSPRINGS OF ALCOHOLIC	14	87.93	7.898
	OFFSPRINGS OF SCHIZOPHRENIA	11	56.09	30.517
PROCESSING SPEED	OFFSPRINGS OF ALCOHOLIC	14	101.86	11.818
	OFFSPRINGS OF SCHIZOPHRENIA	11	95.18	11.643
MMSE	OFFSPRINGS OF ALCOHOLIC	14	24.21	1.578
	OFFSPRINGS OF SCHIZOPHRENIA	11	23.82	1.328



Graph 6: Full Scale IQ

- Compares cognitive functioning; OFFSPRINGS OF ALCOHOLIC has a higher average IQ (93.79) than OFFSPRINGS OF SCHIZOPHRENIA (87.27).
- Near-significant difference (p = 0.058).

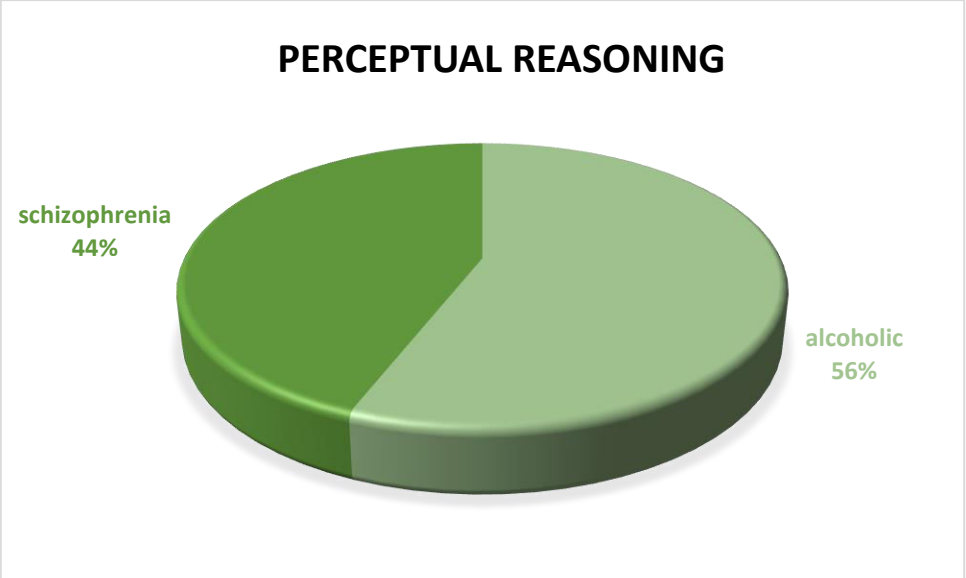
Graphical representation 7: Verbal Comprehension



Graph 7: Verbal Comprehension

- Significant difference (p = 0.026); OFFSPRINS OF ALCOHOLIC performed better verbally.

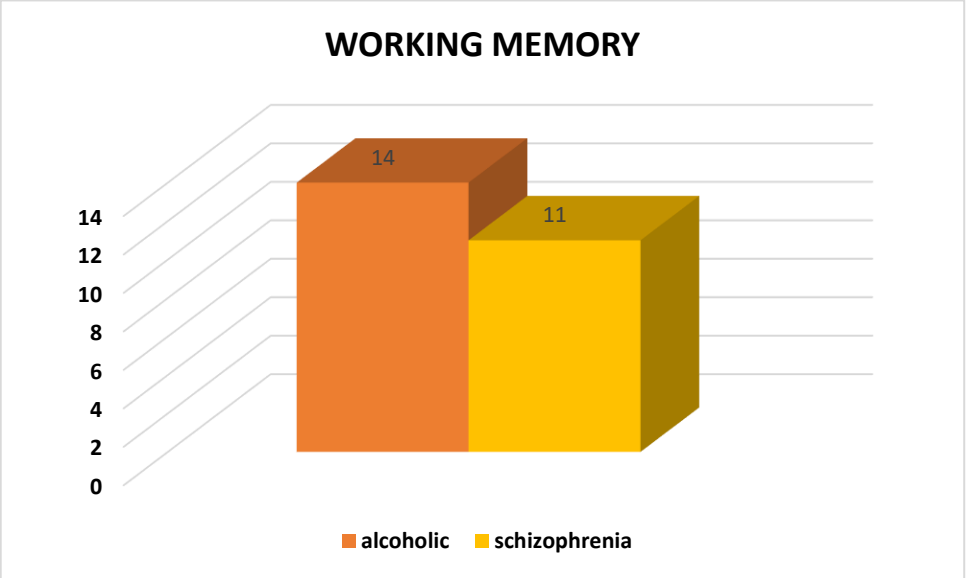
Graphical representation 8: Perceptual Reasoning



Graph 8: Perceptual Reasoning

- OFFSPRINGS OF ALCOHOLIC shows much stronger reasoning skills; highly significant (p = 0.003)

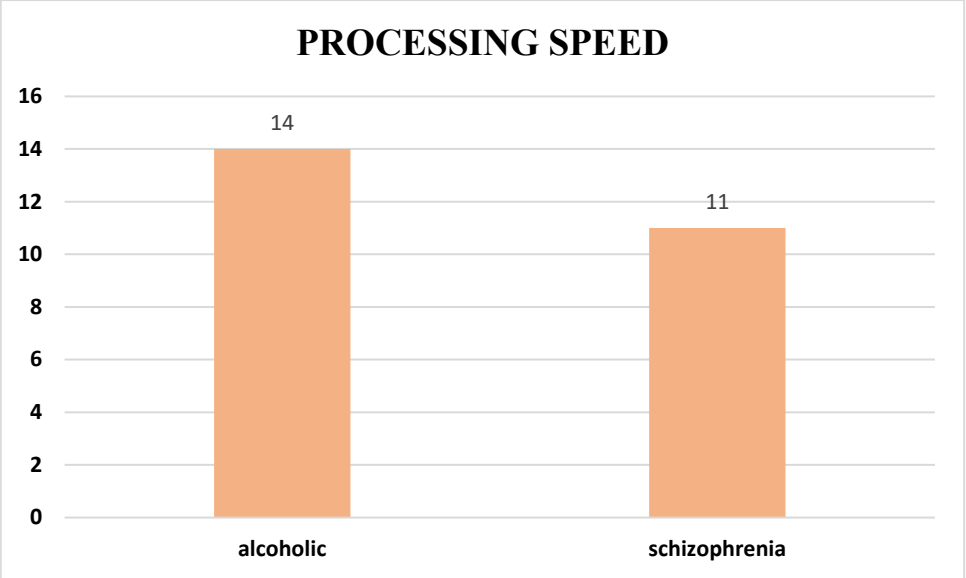
Graphical representation 9: Working Memory



Graph 9: Working Memory

Significant disparity in scores; OFFSPRINGS OF SCHIZOPHRENIA performed much worse (p = 0.001), indicating cognitive challenges.

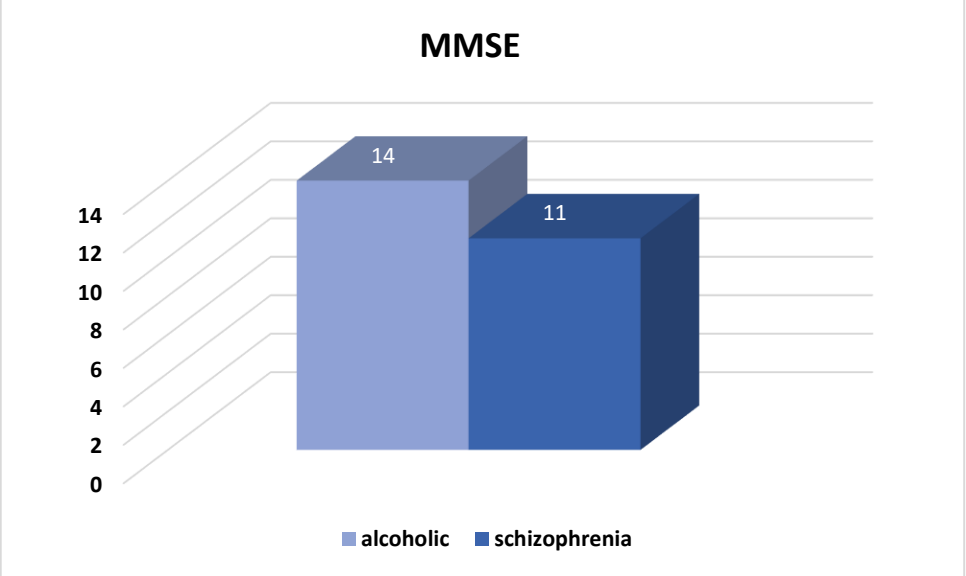
Graphical representation 10: Processing Speed



Graph 10: Processing Speed

No significant difference (p = 0.236), although OFFSPRINS OF ALCOHOLIC has a slight edge.

- **Graphical representation 11: MMSE**



Graph 11: MMSE

Alcoholics' offspring showed better results.

WISC-Wechsler Intelligence Scale for Children(Children)

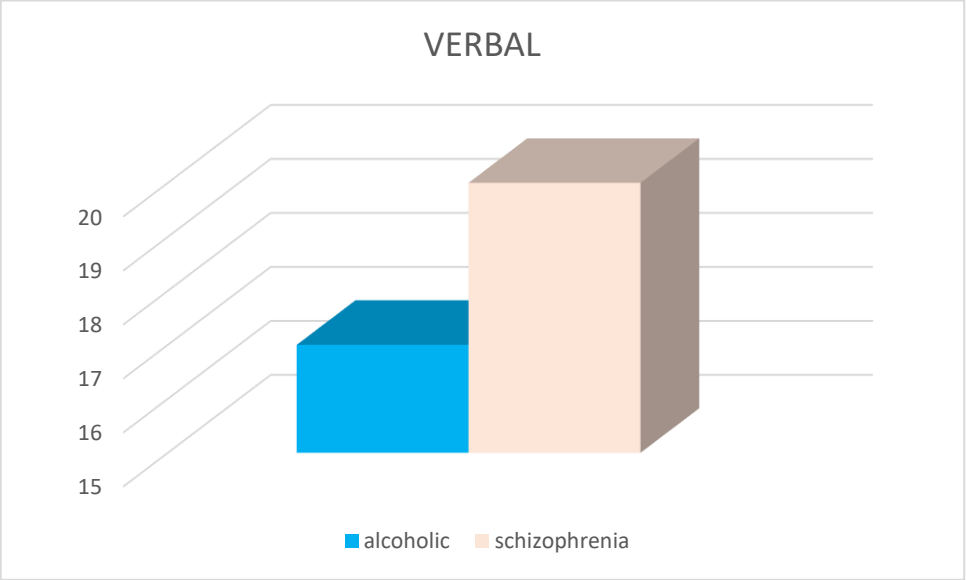
Group Statistics: OFFSPRINGS OF ALCOHOLIC VERSUS OFFSPRINGS OF SCHIZOPHRENIA(Children)

Table 7: group statistics

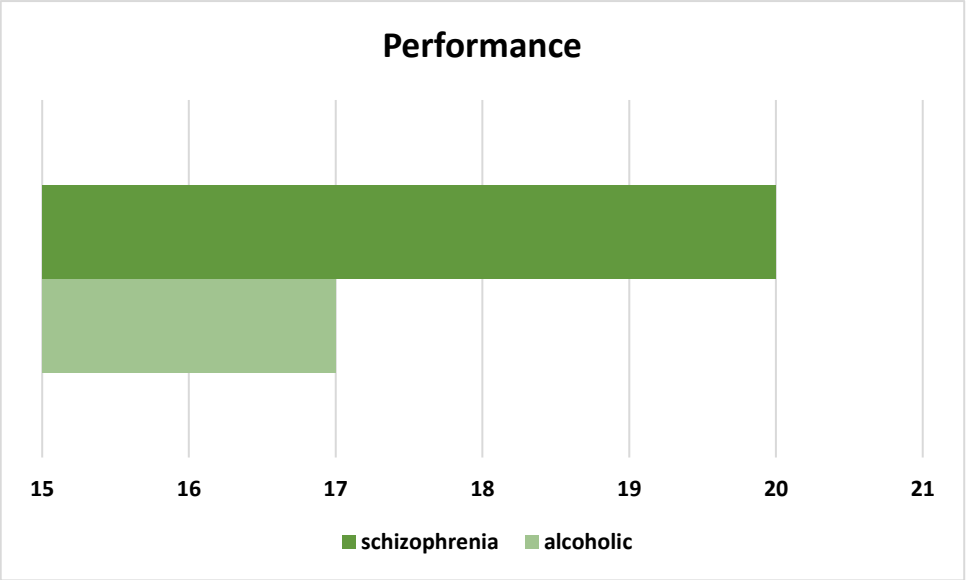
CHILDREN		N	Mean	Std. Deviation
VERBAL	OFFSPRINGS OF ALCOHOLIC	17	91.59	8.704
	OFFSPRINGS OF SCHIZOPHRENIA	20	85.95	9.976
PERFORMANCE	OFFSPRINGS OF ALCOHOLIC	17	93.94	7.941
	OFFSPRINGS OF SCHIZOPHRENIA	20	96.45	8.959
FULL SCALE	OFFSPRINGS OF ALCOHOLIC	17	91.94	7.949
	OFFSPRINGS OF SCHIZOPHRENIA	20	89.55	8.420
VERBAL COMPREHENSION	OFFSPRINGS OF ALCOHOLIC	17	84.12	6.244
	OFFSPRINGS OF SCHIZOPHRENIA	20	79.65	16.620
PERCEPTUAL ORGANISATION	OFFSPRINGS OF ALCOHOLIC	17	75.88	5.085
	OFFSPRINGS OF SCHIZOPHRENIA	20	77.05	4.685
FREEDOM FOR DISTRACTION	OFFSPRINGS OF ALCOHOLIC	17	84.29	8.387
	OFFSPRINGS OF SCHIZOPHRENIA	20	75.35	8.543
PROCESSING SPEED	OFFSPRINGS OF ALCOHOLIC	17	84.06	4.322
	OFFSPRINGS OF SCHIZOPHRENIA	20	84.40	5.082
MMSE	OFFSPRINGS OF ALCOHOLIC	17	23.88	3.180

	OFFSPRINGS OF SCHIZOPHRENIA	**20**	**25.05**	**2.089**

Graphical Representation 12: Verbal



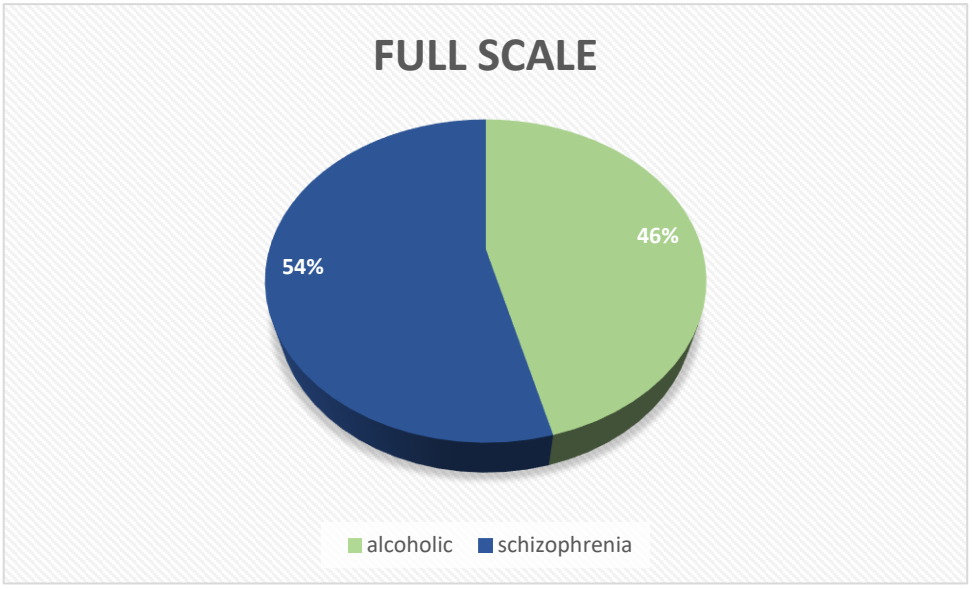
Graphical Representation 13: Performances



13. Graph: Performances

OFFSPRINGS OF ALCOHOLIC outperforms OFFSPRINGS OF SCHIZOPHRENIA (p = 0.053, near significance)

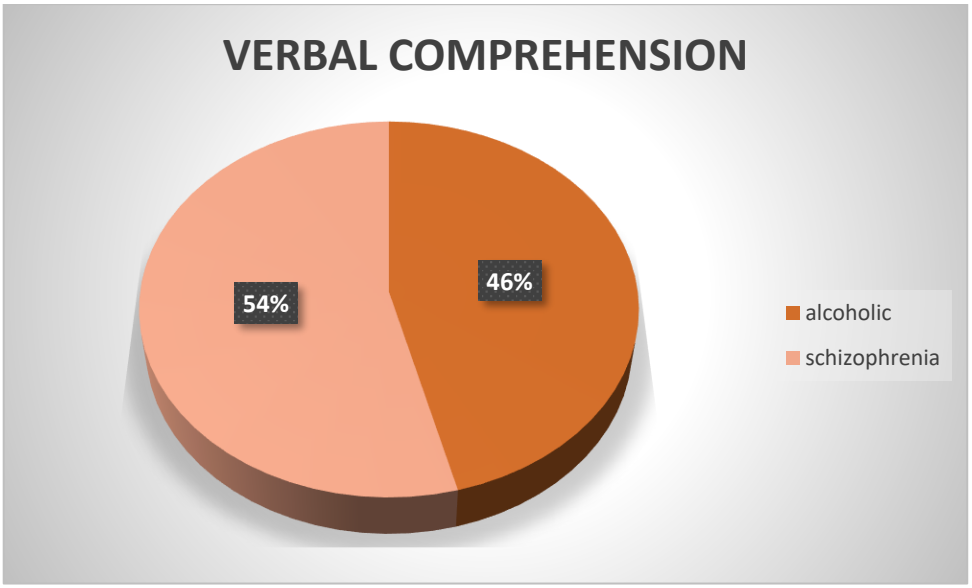
Graphical Representation 14: Full Scale



Graph 14: Full Scale IQ

Minimal difference; not statistically significant.

- **Graphical Representation 15: Verbal Comprehension**



Graph 15: Verbal Comprehension

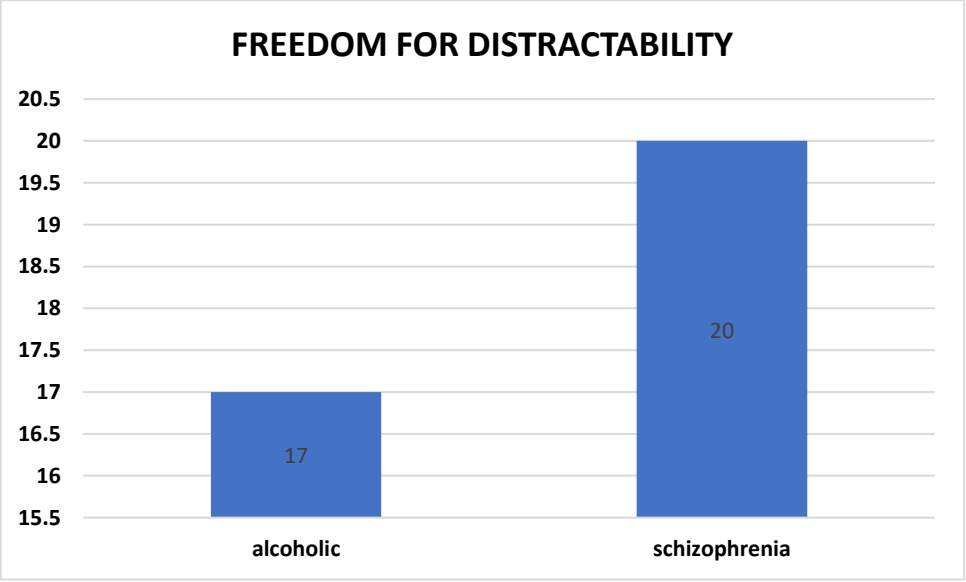
- OFFSPRINGS OF ALCOHOLIC has slightly higher scores, which are not significant.

Graphical Representation 16: Perceptual organization

Graph 16: Perceptual Organisation

- **Close values; not significantly different.**

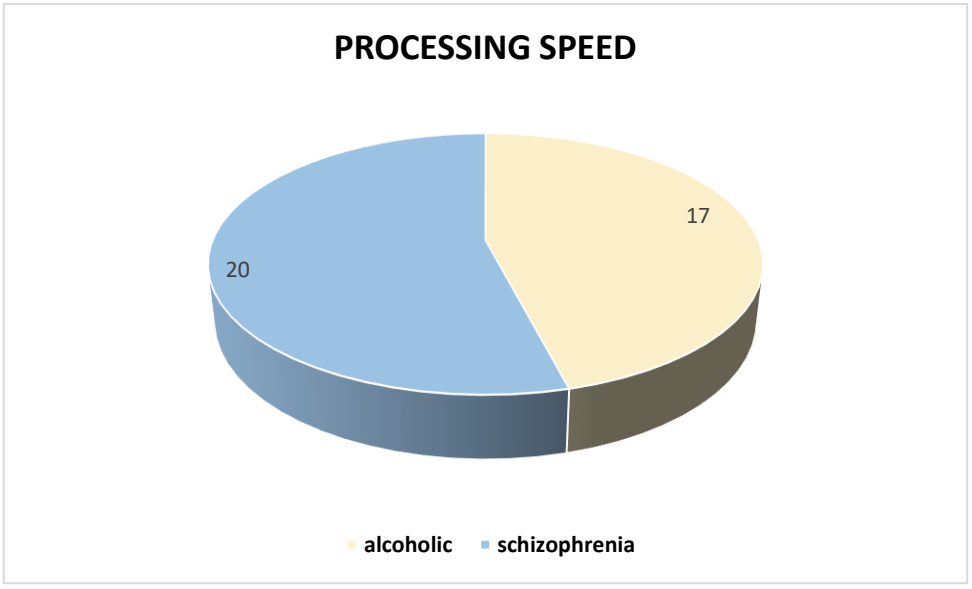
Graphical Representation 17: Freedom For Distractibility



Graph 17: Freedom from Distractibility

- **Significant (p = 0.005); OFFSPRINGS OF ALCOHOLIC performs better, indicating better attention regulation.**

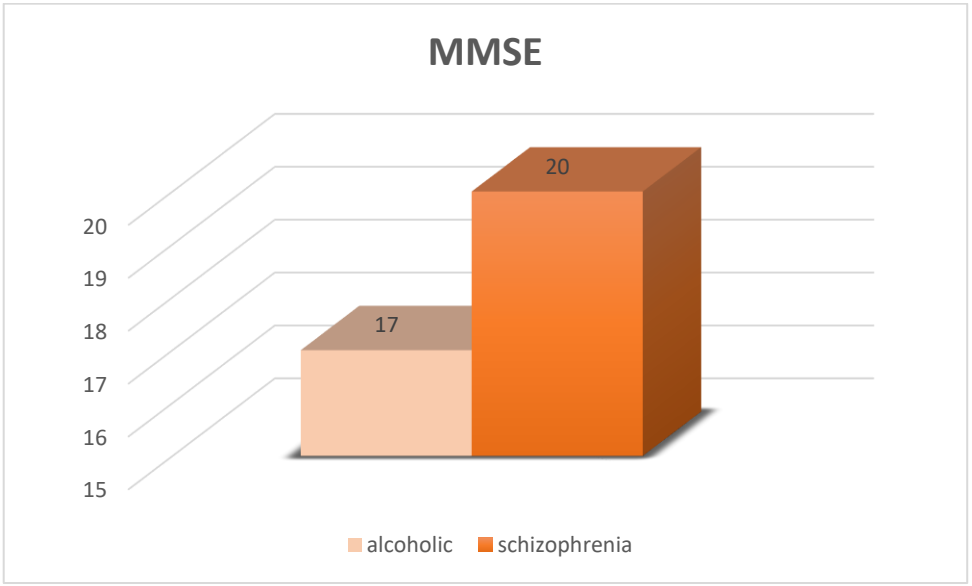
Graphical Representation 18: Processing Speed



Graph 18: Processing Speed

- **Nearly identical scores in both groups.**

Graphical Representation 19: MMSE



Graph 19: MMSE

- **Slightly higher in Group OFFSPRINGS OF SCHIZOPHRENIA; not significant.**
- **Mann-Whitney Test: Independent sub-scale between offspring of alcoholics vs. offspring of schizophrenia (Adult)**

Table 8: Sub-test comparison between offspring (ADULT)

Test Statistics: Offsprings of Alcoholics vs. Offsprings of Schizophrenia

Variable	P Value
Full Scale IQ	0.058
Verbal Comprehension	0.026
Perceptual Reasoning	0.003
Working Memory	0.001
Processing Speed	0.236
MMSE	0.508

Correlations

CORRELATIONS: DURATION OF ILLNESS IN THE PARENT VERSUS FULL-SCALE IQ(ADULTS)

TABLE 9: DURATION OF ILLNESS IN PARENTS VERSUS FULL-SCALE IQ(ADULT)

Variable	N	r (Correlation Coefficient)	P-VALUE
Duration of illness in the parent	25	0.360	0.077

CORRELATIONS: DURATION OF ILLNESS IN THE PARENT VERSUS FULL-SCALE IQ(CHILDREN)

TABLE 10: DURATION OF ILLNESS VERSUS FULL-SCALE IQ(CHILDREN)

Variable	N	Correlation Coefficient (r)	P Value
Full-Scale IQ (Children)	37	0.035	0.839

CORRELATION: FULL-SCALE IQ OF OFFSPRING OF ALCOHOLIC VERSUS OFFSPRING OF SCHIZOPHRENIA (BOTH)

TABLE 11

FULL SCALE	N	FULL-SCALE MEAN	SDT DEVIATION	P VALUE
ALCOHOLIC	31	92.77	8.334	
SCHIZOPHRENIA	31	88.74	7.861	
				0.43

MMSE Comparison Between Groups

TABLE 12

Group	N	Mean	Standard Deviation	P VALUE
ALCOHOLIC	**31**	**92.77**	**8.334**	
SCHIZOPHRENIA	**31**	**88.74**	**7.861**	
				0.43

5 DISCUSSION:

Cognitive functioning plays a vital role in shaping an individual's ability to process, interpret, and respond to the world around them. Core domains such as attention, memory, executive functioning, verbal comprehension, and reasoning are essential for academic and occupational success and overall quality of life [30]. Over the years, increasing attention has been given to how psychiatric illnesses in parents may influence the cognitive outcomes of their children. When parents are affected by severe mental health conditions like schizophrenia or alcohol dependence syndrome, their offspring are often exposed to a combination of genetic predisposition and environmental adversity, placing them at greater risk of cognitive disruption [31]. Understanding the extent and nature of these impairments is crucial for early detection, prevention, and intervention strategies aimed at at-risk populations.

Schizophrenia is widely recognized as a complex neurodevelopmental disorder marked by not just psychotic symptoms, but also significant and persistent cognitive impairments [32]. These impairments often precede the onset of the illness and can remain even when clinical symptoms are controlled. Research has shown that first-degree relatives of individuals with schizophrenia, particularly offspring, often exhibit subtle yet measurable cognitive deficits across various domains, including working memory, processing speed, and executive functioning [33]. Such findings suggest the presence of cognitive endophenotypes—heritable traits that signal increased vulnerability to developing the disorder. These deficits may interfere with academic performance, social development, and adaptive functioning, making early cognitive monitoring essential in these individuals.

In contrast, alcohol dependence syndrome exerts its effects on offspring through a more complex interaction of genetic vulnerability and environmental exposure [34]. Children raised in environments where alcohol misuse is prevalent may experience neglect, inconsistent parenting, or emotional trauma—all of which have independently been linked to poor cognitive outcomes [35].

Furthermore Children of alcohol-dependent parents often face severe disruptions in their developmental trajectory due to a combination of biological, psychological, and environmental factors. In some cases, alcohol exposure during pregnancy can directly harm brain development, while in others, genetic and epigenetic changes passed from the parents may predispose the child to difficulties with memory, attention, and emotional control. These children are also more likely to grow up in unstable, stressful environments where emotional neglect, inconsistent

parenting, and family conflict are common. As a result, they frequently struggle with learning, have trouble focusing in school, and may show signs of anxiety, depression, or behavioural problems. Over time, these challenges can interfere with academic progress, social relationships, and emotional growth, increasing the risk of future mental health issues or substance use, thus continuing the cycle of vulnerability across generations.

While cognitive impairments in these offspring may not mirror those seen in the children of schizophrenia patients, difficulties in attention, impulse control, and learning are commonly reported, indicating a different yet significant pattern of disruption [36].

Although cognitive deficits are present in the offspring of both groups, emerging evidence points to greater and more widespread impairment in the children of individuals with schizophrenia [37]. Domains such as working memory, perceptual reasoning, and verbal comprehension tend to be more severely affected, suggesting a broader neurodevelopmental vulnerability [38]. The presence of these deficits, often before the appearance of any clinical symptoms, underscores the importance of proactive cognitive assessment in these at-risk individuals. Identifying such patterns helps in early intervention and opens avenues for designing cognitive rehabilitation and support systems to reduce long-term functional impairment. This understanding reinforces the need for comparative research to explore the differential impact of parental psychiatric conditions on cognitive development in offspring [39].

Children of parents with schizophrenia or alcohol dependence often show lower IQ scores compared to those from unaffected families, but the reasons behind these effects differ. In the case of schizophrenia, the reduction in IQ is thought to be mainly due to genetic and neurodevelopmental factors. These children may inherit a vulnerability that affects their brain development, especially in areas responsible for thinking, memory, and problem-solving. Even if they do not show signs of mental illness themselves, they often struggle with tasks involving attention, verbal reasoning, and working memory (Snitz et al., 2006; Meier et al., 2014). On the other hand, children of alcohol-dependent parents may experience slightly less severe but still noticeable IQ difficulties. These are often linked to environmental factors such as poor parenting, emotional neglect, and in some cases, exposure to alcohol before birth, which can harm brain development (Malisza et al., 2012; Gierski et al., 2013; AlSaad et al., 2023). While both groups are at risk for cognitive challenges, children of individuals with schizophrenia typically show broader and more persistent impairments in IQ In our study, the Full-Scale IQ scores among adults revealed a higher average in the alcohol dependence group (M = 93.79, SD = 8.97) compared to the schizophrenia group (M

= 87.27, SD = 6.85), with a near-significant difference (p = 0.058). This suggests that individuals exposed to parental alcohol dependence may have better preserved overall cognitive functioning than those with a parental history of schizophrenia. The lower scores in the schizophrenia group may reflect the broader neurodevelopmental burden and potential genetic liability associated with this disorder. Supporting this interpretation, Belon-Hercilla et al. (2020) reported that patients with schizophrenia exhibited more severe impairments in general intelligence and executive function compared to those with alcohol use disorder, emphasizing schizophrenia's more global impact on cognition [40]. Several genes have been implicated in schizophrenia-related cognitive dysfunction and reduced IQ. Notably, the *COMT* (Catechol-O-methyltransferase) gene, which affects dopamine metabolism in the prefrontal cortex, has been associated with working memory deficits and lower IQ. Variants in the *DISC1* (Disrupted-in-Schizophrenia 1) gene have also been linked to neurodevelopmental abnormalities and cognitive impairments. Genome-wide association studies (GWAS) have also identified polygenic risk scores involving multiple loci, such as those near the *ZNF804A* and *NRG1* genes, correlated with schizophrenia susceptibility and reduced cognitive performance. [40].

The observed discrepancy in IQ scores aligns with the concept that schizophrenia affects multiple brain systems crucial to intelligence, including those responsible for abstract reasoning, memory, and processing speed. In our study, the schizophrenia group had significantly lower scores in several cognitive domains, particularly in working memory and perceptual reasoning, contributing to an overall lower Full-Scale IQ. This may reflect the greater neurodevelopmental burden and structural brain abnormalities commonly associated with schizophrenia, particularly in prefrontal and temporal regions. Tolmacheva et al. (2021) similarly found that individuals with schizophrenia showed pronounced deficits in visual memory, verbal learning, and executive control, all of which contribute to lower IQ. The authors noted that even in stabilized patients, these deficits persist and may reflect underlying disruptions in neural connectivity and synaptic function .[41]

In addition to executive dysfunction and memory decline, schizophrenia-related cognitive deficits may be more deeply embedded in early neurodevelopmental processes, which manifest even before illness onset. Cannon et al. (2000) examined childhood cognitive functioning in individuals at high risk for schizophrenia and found consistent reductions in IQ from a young age, particularly in verbal and nonverbal reasoning. These findings support the notion that cognitive decline in schizophrenia is not solely a consequence of symptomatic illness or treatment

effects but may instead reflect a core trait-like characteristic of the disorder, which could explain the lower IQ observed in our schizophrenia-offspring group [42].

Furthermore, while alcohol dependence is also associated with cognitive impairment, especially in areas like working memory and decision-making, its impact may depend more on the severity and chronicity of use, with some cognitive functions remaining intact in the absence of prolonged abuse. Ventriglio et al. (2015) showed that outpatients with alcohol dependence had relatively preserved IQ scores compared to schizophrenia patients, even when controlling for mood and anxiety symptoms. This supports our study's findings, where alcohol-offspring demonstrated better full-scale cognitive functioning than their schizophrenia-offspring counterparts, possibly due to less severe neurodevelopmental disruption [43].

In the current study, verbal comprehension scores were significantly higher in the offspring of alcohol-dependent individuals (M = 100.29, SD = 11.79) compared to the offspring of schizophrenia patients (M = 90.64, SD = 7.37), with the difference reaching statistical significance (p = 0.026). This domain reflects core language functions such as vocabulary, verbal reasoning, and abstract thinking, all essential for effective communication and learning. The markedly lower scores in the schizophrenia group suggest disruptions in the development of language-related cognitive processes, likely influenced by genetic and neurodevelopmental vulnerabilities. These findings closely align with those of Ravindran et al. (2020), who observed significantly lower verbal IQ scores in children of schizophrenia patients compared to control groups, reinforcing the notion that verbal impairments can be early indicators of inherited risk .[44}

In the present study, the offspring of alcohol-dependent parents (COAs) exhibited moderate impairments in cognitive domains, particularly in working memory (mean = 87.93), verbal comprehension (mean = 91.59), and freedom from distractibility (mean = 84.29). These findings are consistent with previous research demonstrating that COAs, even in the absence of direct substance use, experience significant but domain-specific cognitive deficits. For instance, Gierski et al. (2013) reported that adult offspring of alcohol-dependent individuals showed impairments in executive functioning and impulse control, similar to the reduced attention regulation observed here. Addington & Addington (1997) also found that children of alcoholics had diminished executive abilities and attention, while verbal intelligence remained relatively preserved, mirroring the verbal performance patterns seen in the current findings. Although this study did not directly examine fetal alcohol exposure, the results are consistent with studies such as Green et al. (2009) and Malisza et al. (2012), which documented that even subclinical prenatal

alcohol exposure can lead to impairments in planning, processing speed, and spatial memory. Interestingly, processing speed in the COA group remained relatively intact in the current analysis, potentially indicating lower levels of prenatal exposure or protective postnatal environments. Nonetheless, the observed deficits support the involvement of both genetic and neurodevelopmental mechanisms, as emphasized by Hill et al. (2000) and Porjesz et al. (2002), who highlighted electrophysiological anomalies such as reduced P300 amplitude among COAs, indicative of impaired attention and cognitive control.

Verbal comprehension deficits in schizophrenia are often interpreted as stemming from structural and functional brain abnormalities, particularly in the left temporal and frontal lobes. These areas are heavily involved in semantic processing and language production. Faye et al. (2022) found that patients with schizophrenia, regardless of whether they had comorbid alcohol dependence, consistently performed worse on verbal reasoning and comprehension tasks when compared to healthy individuals. The findings suggest that language-related deficits in schizophrenia may be relatively independent of other clinical variables and may represent a core cognitive marker of the illness [45].

The disparity in verbal comprehension may also be rooted in early developmental delays in language acquisition, commonly observed among individuals at familial risk for schizophrenia. Zhang et al. (2013) conducted a study on adolescents with schizophrenia and their unaffected siblings, revealing that both groups exhibited impairments in verbal learning and comprehension when compared to controls. These deficits were especially evident in verbal reasoning and fluency tasks, suggesting that language difficulties may represent a stable trait marker of genetic vulnerability to schizophrenia. Our study's findings support this pattern, indicating that lower verbal comprehension scores among schizophrenia-offspring likely reflect neurodevelopmental disruptions present well before clinical symptom onset [46].

On the other hand, verbal abilities in the alcohol-offspring group remained relatively intact, which may be due in part to the less pervasive and more environmentally modifiable nature of alcohol-related cognitive risks. Addington & Addington (1997) found that while substance abuse does contribute to cognitive decline in some domains, its impact on verbal IQ was less pronounced than that observed in schizophrenia, and concluded that children of alcoholics exhibit mild but significant impairments in attention, executive functioning, memory, and academic skills, with a complex interplay between genetic vulnerability and environmental adversity contributing to these deficits. Their results suggest that while alcohol dependence may impair attention and memory, it does not

consistently disrupt language-based cognition, explaining the comparatively higher verbal comprehension scores seen in our alcohol-offspring group [47].

The present study revealed that working memory scores were drastically lower in the schizophrenia group (M = 56.09, SD = 30.51) compared to the alcohol group (M = 87.93, SD = 7.89) a highly significant result (p = 0.001). This finding underscores the critical impairment in retaining and manipulating information, a core function of working memory. These results are echoed by Diwadkar et al. (2011), who examined adolescents with a familial risk of schizophrenia. They found that only the schizophrenia-offspring group demonstrated working memory impairments, particularly under longer delay intervals. This highlights a unique vulnerability in the cognitive circuitry of schizophrenia-risk youth, likely tied to dysfunctions in the dorsal prefrontal cortex 48]

Working memory deficits have been consistently reported among offspring of individuals with alcohol dependence. Hill et al. (2013) investigated this relationship by examining neuropsychological performance and brain structure, particularly focusing on the caudate nucleus, in children and young adults at high familial risk for alcohol dependence. The study found that high-risk offspring exhibited significantly poorer working memory performance than low-risk controls. These deficits were not solely attributed to personal substance use but appeared to reflect underlying vulnerabilities associated with familial risk. Moreover, genetic variations in dopamine-related genes (COMT and DRD2) influence working memory functioning, suggesting a neurobiological basis for the cognitive impairments observed. Hill and colleagues emphasized that poor working memory in these children might serve as an early neurocognitive marker for later development of externalizing behaviors and substance use disorders .

Further supporting our findings, Jeon et al. (2012) reported working memory impairments in both schizophrenia patients and individuals at ultra-high risk for psychosis. Using the n-back task, they found that verbal and spatial working memory performance declined significantly at higher task loads (2-back and 3-back), reinforcing that increasing cognitive demand disproportionately affects those at risk. Our study's use of WAIS working memory measures aligns with this, as both high-load tasks and daily executive challenges are especially taxing for individuals with familial schizophrenia risk [49].

Underlying structural and functional brain alterations may also explain working memory deficits in schizophrenia. A recent fMRI meta-analysis by Ding et al. (2024) found consistent abnormalities in the dorsolateral prefrontal cortex and parietal regions of schizophrenia patients, key areas responsible for working memory processing. These neuroimaging results support our interpretation that our observed cognitive deficits may be biologically embedded and not merely behavioral symptoms. The authors concluded that these neurofunctional markers could be diagnostic biomarkers for identifying working memory dysfunction in schizophrenia [50].

The genetic basis for working memory impairments also finds support in recent research. Zhang et al. (2016) conducted a meta-analysis of fMRI studies involving unaffected relatives of schizophrenia patients and reported altered activation patterns in prefrontal and parietal cortices during working memory tasks. These relatives, who had not developed psychosis themselves, still showed abnormal brain activity, particularly reduced activation in the right middle frontal gyrus (BA9), indicating a genetically modulated vulnerability. This further reinforces our conclusion that the substantial working memory deficits in schizophrenia-offspring are likely driven by inherited neurological dysfunction rather than environmental exposure alone [51].

In the present study, processing speed was higher in the alcohol-offspring group (M = 101.86, SD = 11.81) than in the schizophrenia-offspring group (M = 95.18, SD = 11.64). However, the difference did not reach statistical significance ($p = 0.236$). Despite this, the trend aligns with longstanding research indicating that processing speed is consistently reduced in individuals with schizophrenia or genetic risk. Thuaire et al. (2020) demonstrated that reduced processing speed mediates nearly all executive function impairments in schizophrenia, suggesting that slower information processing is a fundamental bottleneck in cognitive performance. Their study also emphasized that age interacts with these impairments, showing amplified slowing in older schizophrenia patients [52].

Further refining this perspective, Angerville et al., (2023) examined different components of processing speed in schizophrenia—behavioral execution, response processing, and accuracy—and found that each subcomponent was differentially associated with symptoms, illness duration, and overall intelligence. Their results indicate that processing speed in schizophrenia is not a single, uniform deficit but involves multiple systems that are variably disrupted. Our study's general slowing in the schizophrenia-offspring group likely reflects such a multifaceted breakdown, though WAIS measures may not capture all subcomponents individually [53].

Adding further nuance, Manning et al (2009) conducted a confirmatory factor analysis to determine which cognitive domains were most influenced by processing speed. They concluded that when processing speed was

statistically controlled, differences between schizophrenia patients and controls in other domains (e.g., executive functioning, working memory, attention) diminished significantly. This supports our interpretation that reduced processing speed may be a foundational impairment in schizophrenia, contributing to downstream deficits in multiple areas. The findings from our schizophrenia-offspring group thus likely reflect both inherited neurological inefficiencies and their broad cognitive implications [54].

In our study, children in the alcohol-offspring group (M = 91.59, SD = 8.70) scored higher in verbal IQ than children in the schizophrenia-offspring group (M = 85.95, SD = 9.97). Although the difference approached but did not reach statistical significance (p = 0.053), the trend points to a consistent verbal disadvantage in the schizophrenia-risk group. This observation aligns with findings by Dickson et al. (2014), who demonstrated that children with a first-degree relative diagnosed with schizophrenia exhibited significantly reduced performance in verbal comprehension, scholastic achievement, and verbal working memory. These deficits were most pronounced in children with high familial loading, indicating that multiple affected relatives compound the cognitive burden [55].

A longitudinal perspective further supports our results. In a prospective high-risk study, Pin et al. (2009) assessed WISC verbal subtests in children later diagnosed with schizophrenia spectrum disorders. Their results showed that verbal IQ, particularly vocabulary and similarities scores, were significantly lower in children who developed schizophrenia compared to controls. These findings emphasize the predictive value of early verbal deficits, reinforcing the idea that impairments in expressive and abstract verbal reasoning may precede clinical symptoms and serve as early cognitive markers of schizophrenia risk [56].

The current study revealed that while both groups offspring of alcohol-dependent parents (COAs) and offspring of individuals with schizophrenia—demonstrated cognitive impairments, the **nature and severity of these deficits varied significantly**. Offspring of schizophrenia patients exhibited **more pronounced and widespread impairments**, particularly in **working memory (mean = 56.09), perceptual reasoning (mean = 89.36), and verbal comprehension (mean = 90.64)**, consistent with findings from **Meier et al. (2014)** and **Snitz et al. (2006)**, who reported that cognitive deficits in schizophrenia-offspring are often **persistent, generalized, and indicative of underlying neurodevelopmental vulnerability**. In contrast, COAs showed **moderate impairments**, notably in **freedom from distractibility (mean = 84.29)** and **working memory (mean = 87.93)**, with relatively **preserved processing speed and perceptual reasoning**, aligning with the results of **Gierski et**

al. (2013) and **Addington & Addington (1997)** These patterns suggest that while schizophrenia-offspring may be affected by **heritable disruptions in brain development**, COAs are more likely influenced by a **combination of genetic predisposition, environmental adversity, and in some cases, prenatal alcohol exposure**. Supporting this, **Porjesz et al. (2002)** and **Hill et al. (2000)** identified electrophysiological abnormalities in COAs, whereas **Cannon et al. (2000)** and **Nuechterlein et al. (2004)** emphasized structural and functional brain deficits in schizophrenia-offspring. Despite differences in etiology, both groups are at elevated risk for **academic difficulties, emotional dysregulation, and long-term psychiatric morbidity**, reinforcing the need for **early cognitive assessment and tailored intervention strategies** based on the specific risk profile of each population.[56]

Neuroanatomical evidence further supports the verbal deficits observed in at-risk youth. In a structural MRI study, Bhojraj et al. (2009) reported that adolescents with a high genetic risk for schizophrenia exhibited verbal fluency impairments, abnormal gray matter volume, and reversed asymmetry in the pars triangularis, a key region for expressive language. Their findings indicate that disrupted lateralization in language-related brain areas may underlie these individuals' poor verbal fluency and comprehension. This neurobiological disruption helps explain why our schizophrenia-offspring group showed lower verbal IQ scores even in the absence of psychiatric symptoms, suggesting that such deficits are rooted in early brain development rather than acquired through environment alone [57].

In present study, freedom from distractibility—reflecting sustained attention and mental control—was significantly higher in the alcohol-offspring group ($M = 84.29$, $SD = 8.38$) than in the schizophrenia-offspring group ($M = 75.35$, $SD = 8.54$), a statistically significant difference ($p = 0.005$). This suggests that children and young adults with a parental history of schizophrenia experience more difficulty managing distractions and maintaining attention on cognitive tasks. This pattern was also observed in Burton et al. (2018), who studied 7-year-old children with a familial risk of schizophrenia and found deficits in both sustained attention and interference control compared to healthy controls and those at risk for bipolar disorder. These early attentional deficits may represent foundational vulnerabilities that impair academic and cognitive functioning over time [30].

Supporting this, Demeter et al. (2013) demonstrated that patients with schizophrenia exhibited higher distractor vulnerability using the Sustained Attention Task (SAT). Unlike healthy participants, schizophrenia patients showed specific difficulty with selection-control when distractors were introduced, indicating poor ability to filter irrelevant

stimuli. Notably, vigilance was preserved, suggesting that distraction sensitivity, rather than general alertness, is a core deficit. Our findings similarly indicate that the schizophrenia-offspring group struggles more with distractibility, even if their overall task performance remains otherwise intact [31].

Neuroimaging evidence further supports these behavioral findings. In a study of healthy siblings of schizophrenia patients, Antonucci et al. (2016) found altered functional connectivity in the thalamus and medial prefrontal cortex during attentional tasks. These brain regions are critical for filtering distractions and engaging task-relevant control networks. The fact that similar anomalies were seen in non-diagnosed siblings suggests a biological underpinning to the distractibility we observed in our schizophrenia-offspring sample, pointing to inherited disruption of attention-regulating brain networks [32].

Finally, Franke et al. (1994) examined attention in schizophrenia patients, their siblings, and controls using the Continuous Performance Test (CPT). While both patients and siblings showed general attentional impairments, only patients exhibited elevated distractibility under distraction conditions. This suggests that while attentional control issues may be partially inherited, the full expression of distractibility may require genetic and illness-related factors. Our study supports this distinction: offspring of schizophrenia patients may possess latent attention vulnerabilities that manifest behaviorally as distractibility, even in the absence of full-blown psychosis [33].

In the Present study, the MMSE scores showed minimal differences across groups, both in adults (schizophrenia: 23.82, alcohol: 24.21) and children (schizophrenia: 23.88, alcohol: 25.05), suggesting that the MMSE may not effectively capture domain-specific or subtle cognitive deficits present in these populations. This observation aligns with the findings of Oudman et al. (2014), who directly compared the MMSE and MoCA in patients with Korsakoff's Syndrome—a condition also characterized by complex cognitive impairments. Their analysis revealed that the MMSE misdiagnosed nearly half of the impaired patients, while the MoCA correctly identified all cases. The authors concluded that MoCA offered superior diagnostic sensitivity and discriminative power, especially in cognitive domains such as executive function, attention, and memory, which are similarly compromised in schizophrenia. This reinforces our interpretation that MMSE may inadequately assess early or subtle deficits in high-risk psychiatric populations [34].

Furthermore, Dong et al. (2010) conducted a comparative analysis following stroke. They found that 32% of patients who passed the MMSE still showed impairment on MoCA, suggesting that MMSE may miss executive and visuospatial deficits. These domains are especially relevant to psychiatric populations. In contrast, the MoCA,

which includes attention, abstraction, and executive control assessments, was much more successful in differentiating cognitive subtypes. The result underscores the need to use more nuanced tools like MoCA when evaluating early or mild cognitive dysfunction, such as in children or young adults at genetic risk for schizophrenia [35]

Complementary to this, Pendlebury et al. (2012) showed that over 50% of individuals with normal MMSE scores (≥27) had impairments on the MoCA when assessed post-stroke or during memory decline. The key advantage of MoCA in this context was its ability to detect early-stage or multi-domain mild cognitive impairment (MCI), including in executive function and attention. These findings are critical for schizophrenia research, where early intervention is paramount and domain-specific impairments are often present even before clinical symptoms emerge. Our study's nearly indistinguishable MMSE scores between alcohol and schizophrenia-offspring groups may reflect this blind spot in the MMSE's diagnostic capacity [36].

Lastly, Rademeyer and Joubert (2016) conducted a direct comparison between MMSE and MoCA in outpatient schizophrenia patients and found a statistically significant difference between the scores on the two assessments. The average MMSE score was 27.17, while the average MoCA score was substantially lower at 22.53 ($p = 0.000008$), indicating that MMSE consistently overestimated cognitive functioning. Their findings revealed that the MoCA captured impairments in domains like executive functioning, abstraction, and memory that the MMSE routinely missed. This supports our conclusion that MMSE lacks the specificity and sensitivity required to detect the early and domain-specific cognitive vulnerabilities present in psychiatric populations, including schizophrenia-offspring [37].

The findings indicate that offspring of patients with schizophrenia suffer more widespread and severe cognitive impairments than those with alcohol-dependent parents. These impairments are not limited to a single cognitive domain but span across memory, reasoning, attention, and language. The evidence from both our study and the literature suggests that while alcohol dependence can impair cognitive function, schizophrenia has a more pervasive and heritable impact on neurocognitive development.

Even in the absence of direct alcohol consumption, children of alcohol-dependent parents are at increased risk for cognitive impairments due to a combination of genetic vulnerability, epigenetic influences, and environmental adversity. Research suggests that certain cognitive functions—such as attention, working memory, executive functioning, and processing speed—are commonly affected in these children. Genetically, they may inherit subtle

alterations in brain structure and function that predispose them to problems with self-regulation, learning, and problem-solving (Gierski et al., 2013). Additionally, epigenetic changes caused by the parents' chronic alcohol use can alter gene expression in the offspring, impacting brain development. Furthermore, these children often grow up in environments characterized by emotional neglect, inconsistent caregiving, financial stress, or parental conflict. This can disrupt normal brain maturation, especially in areas like the prefrontal cortex and hippocampus (AlSaad et al., 2023). The combination of inherited and environmental factors contributes to the cognitive difficulties observed, even in children who have never consumed alcohol themselves

. These insights call for early cognitive screening and interventions in children of schizophrenia patients to mitigate long-term educational and functional challenges.

7 LIMITATIONS OF STUDY

- The small sample size reduces the generalizability of the findings to broader populations.
- The cross-sectional design prevents conclusions about the progression or causality of cognitive deficits over time.
- The study lacked a control group of offspring from parents without psychiatric illness, limiting the context for interpreting cognitive differences.
- Sociodemographic variables such as education quality, parental support, and prenatal exposures were not fully controlled or examined.
- The use of MMSE, a global cognitive screener, may have underestimated specific deficits, particularly in executive and abstract functions.
- Environmental influences like trauma history, family stress, and parenting style, which could significantly impact cognitive development, were not comprehensively measured.

5.2 STRENGTH OF THE STUDY

- The study is among the few that directly compare cognitive functioning in offspring of schizophrenia and alcohol-dependent patients.
- Standardized tools like WAIS-IV and WISC were used, providing robust and comprehensive cognitive

profiling across age groups.

- Inclusion of both children and adults allowed for developmental comparisons and insights into when cognitive impairments emerge.
- The analysis included multiple cognitive domains, enabling a more detailed understanding of specific strengths and weaknesses in each group.
- The focus on familial psychiatric history highlights genetic and transgenerational influences on cognitive development.

Appropriate statistical methods, such as the Mann-Whitney U test and correlation analysis, enhanced the reliability and interpretability of the results.

9 Conclusion

- The current study aimed to assess and compare the cognitive functioning of offspring of individuals diagnosed with schizophrenia and those with alcohol dependence syndrome. Drawing on standardized cognitive tools such as the WAIS-IV and WISC for adult and child participants, the study evaluated multiple cognitive domains, including working memory, processing speed, verbal comprehension, perceptual reasoning, and freedom from distractibility. The rationale behind this comparative approach lies in the emerging understanding that psychiatric disorders exert not only intrapersonal consequences but also significant transgenerational effects, particularly on cognitive development in offspring. The study's findings lend support to this growing body of literature and help further delineate the differential cognitive risk profiles associated with schizophrenia and alcohol-related psychiatric backgrounds.
- The results showed that while both groups—offspring of schizophrenia patients and offspring of alcohol-dependent individuals—displayed cognitive impairments, the nature and severity of those impairments differed meaningfully between the two groups. Notably, the schizophrenia-offspring group consistently underperformed in almost all cognitive domains assessed. This group showed markedly lower scores in working memory, verbal comprehension, and perceptual reasoning, and significantly lower freedom from distractibility, indicating sustaining attention and mental control challenges. These findings suggest that schizophrenia, a disorder known for its neurodevelopmental underpinnings, may transmit a broader and deeper cognitive vulnerability to the next generation, independent of the presence of psychiatric symptoms in offspring themselves.
- Among the most striking observations was the significant difference in working memory scores, with the schizophrenia group showing a drastic reduction compared to the alcohol group. This may be attributed to structural and functional brain differences, which are structural or functional brain differences inherited or shaped by early developmental influences. Literature supports that working memory deficits are strongly associated with familial risk for schizophrenia, pointing toward their potential as cognitive endophenotypes. Similarly, perceptual reasoning, which involves non-verbal problem-solving and abstract spatial reasoning,

was another domain where schizophrenia-offspring underperformed. These findings align with existing neurocognitive research suggesting that the offspring of schizophrenia patients may experience early and enduring deficits that are not merely reflections of environmental exposure or lifestyle factors but likely reflect inherited neurocognitive disruption.

- In contrast, the alcohol-offspring group demonstrated relatively preserved cognitive abilities in most domains. Though some impairments were observed, particularly in areas such as attention regulation and verbal comprehension, they were consistently less severe and less widespread than those seen in the schizophrenia-offspring group. This distinction suggests that while alcohol dependence can impact cognitive development in offspring, primarily through environmental instability or prenatal exposure, its effect may not be as deeply rooted in neurodevelopment as schizophrenia. This comparative finding is clinically meaningful because it emphasizes the different mechanisms by which parental psychiatric illness influences cognitive outcomes in offspring—one more heritable and neurodevelopmental, the other more environmentally mediated.
- Moreover, the study evaluated general cognitive functioning using the Mini-Mental State Examination (MMSE). While the MMSE did not reveal substantial differences between the groups, this finding underscores its limitations as a tool for detecting nuanced cognitive impairment. Research consistently shows that MMSE may fail to identify executive function deficits, working memory limitations, or verbal reasoning issues—all of which were prominent in the schizophrenia-offspring group in this study. This reinforces the importance of using domain-specific assessments when investigating at-risk populations, especially for early identification and targeted intervention planning.
- including children and adults in the study also allowed for a valuable developmental comparison. While cognitive impairments were present in both age groups, the consistency of these findings across lifespan stages suggests that the cognitive vulnerability in schizophrenia-offspring may emerge early and persist over time. In contrast, the cognitive outcomes in the alcohol-offspring group appeared more variable, possibly reflecting the interaction of environmental, psychosocial, and educational factors. The implication here is that early cognitive screening and intervention may be especially crucial for the schizophrenia-offspring population to mitigate long-term academic and functional challenges.

In conclusion, the study adds to the growing evidence that familial psychiatric illness, especially schizophrenia, carries a significant cognitive burden for offspring. These cognitive difficulties span multiple domains, are evident even in clinical symptoms, and may serve as early vulnerability indicators. The contrast between the schizophrenia and alcohol-offspring groups also highlights the heterogeneity of psychiatric inheritance and the importance of tailoring preventive and therapeutic strategies accordingly. This study paves the way for more personalized approaches to intervention, risk assessment, and developmental monitoring in high-risk youth by identifying specific cognitive domains that are disproportionately affected. Ultimately, these findings reinforce the importance of integrating cognitive assessments into clinical protocols for children and adolescents with a family history of severe psychiatric illness.

10 SUMMARY:

Working memory was significantly impaired in the schizophrenia-offspring group (M = 56.09, SD = 30.51), compared to the alcohol-offspring group (M = 87.93, SD = 7.89; p = 0.001), indicating a profound difficulty in retaining and manipulating information, suggestive of a potential neurocognitive endophenotype of schizophrenia.

Perceptual reasoning scores were markedly lower in the schizophrenia-offspring group (M = 89.36, SD = 10.98) than in the alcohol-offspring group (M = 107.00, SD = 12.07; p = 0.003), highlighting impairments in non-verbal reasoning, spatial organization, and abstract problem-solving.

Verbal comprehension was significantly better in the alcohol-offspring group (M = 100.29, SD = 11.79) than in the schizophrenia-offspring group (M = 90.64, SD = 7.37; p = 0.026), reflecting possible early developmental language deficits in children of schizophrenia patients.

Freedom from distractibility was significantly compromised in the schizophrenia-offspring group (M = 75.35, SD = 8.54) relative to the alcohol group (M = 84.29, SD = 8.38; p = 0.005), suggesting poor attention control and increased vulnerability to cognitive interference.

Processing speed was lower in the schizophrenia group (M = 95.18, SD = 11.64) compared to the alcohol group (M = 101.86, SD = 11.81), though the difference was not statistically significant (p = 0.236). Nonetheless, the trend supports previous findings of cognitive slowing in schizophrenia.

Full Scale IQ scores were lower in the schizophrenia group (M = 87.27, SD = 6.85) versus the alcohol group (M = 93.79, SD = 8.97), with a near-significant difference (p = 0.058), reinforcing a pattern of global cognitive decline in the schizophrenia-offspring group.

Verbal IQ in children assessed via WISC was higher in the alcohol-offspring group (M = 91.59, SD = 8.70) than in the schizophrenia group (M = 85.95, SD = 9.97; p = 0.053), showing early language delays in high-risk children for schizophrenia.

MMSE scores showed minimal differences between groups in children and adults, reflecting its limited sensitivity in detecting domain-specific impairments such as working memory, executive function, and verbal

abstraction.

Cognitive impairments in schizophrenia-offspring were consistent across both children and adults, indicating that these deficits are likely early-emerging and persistent, not age-dependent or context-specific.

Alcohol-offspring showed comparatively preserved cognition, though some deficits were still observed, suggesting that environmental factors like home instability or parental neglect may influence outcomes rather than direct neurodevelopmental transmission.

Statistical significance was strongest in domains related to memory and attention, supporting literature that identifies these as key vulnerability areas in individuals with a familial history of schizophrenia.

Environmental and psychosocial factors such as socioeconomic status, trauma exposure, or parental involvement were not the primary focus. However, they remain important variables for future studies given their known influence on cognitive outcomes.

The study supports the hypothesis that schizophrenia transmits a broader and more severe cognitive risk to offspring compared to alcohol dependence, possibly due to its neurodevelopmental and genetic basis.

The use of WAIS and WISC allowed for a detailed profile of domain-specific cognitive abilities, helping distinguish subtle but clinically relevant differences between the two high-risk groups.

Implications include the need for early cognitive screening and interventions in children with a family history of schizophrenia, to support educational planning, social functioning, and psychological development.

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ETHICAL CLEARANCE

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 870/2022-23 1/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "ASSESSMENT OF PATHWAY TO CARE AMONG PATIENTS WITH DHAT SYNDROME: A CROSS SECTIONAL OBSERVATIONAL STUDY".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR NISHANTH REDDY A.

NAME OF THE GUIDE: DR. SANTOSH RAMDURG, PROFESSOR & HOD, DEPT. OF PSYCHIATRY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

ANNEXURE –VI

BLDE (DU), SHRI BM PATIL MEDICAL COLLEGE HOSPITAL & RC VIJAYAPURA - 586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned, _____, S/O D/O W/O _____, aged ___ years, ordinarily resident of _____ do hereby state/declare that Dr. SIDDHARTH PATIL of Shri. B. M. Patil Medical College Hospital and Research Centre have explained to me in my own language that he is conducting a dissertation/research titled "ASSESSMENT AND COMPARISON OF COGNITIVE FUNCTION IN OFFSPRINGS OF PATIENTS DIAGNOSED WITH SCHIZOPHRENIA AND ALCOHOL DEPENDENCE SYNDROME" under the guidance of Dr.Santosh Ramdurg, and requesting my participation in the study. Apart from routine treatment procedures, follow-up observations will be utilized for the study as reference data. Further Doctor has informed me that my participation in this study helped in the evaluation of the results of the study, which is a useful reference for the treatment of other similar cases in the near future. The Doctor has also informed me that information given by me, observations made photographs, and video graphs taken upon me by the investigator will be kept confidential and not assessed by a person other than my legal hirer or me except for academic purposes.

The Doctor informed me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during the course of treatment/study related to diagnosis, procedure of treatment, the result of treatment or prognosis.

At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want, or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of the dissertation or research, the diagnosis made, mode of treatment, I the undersigned Shri/Smt _____ under my fully conscious state of mind agree to participate in the said research/dissertation.

Signature of the patient:

Signature of Doctor:

Dr. SIDDHARTH PATIL

Witness: 1.

ANNEXURE VII

PROFOMA FOR GENERAL DETAILS COLLECTION

BLDE'S SHRI BM PATIL MEDICAL COLLEGE HOSPITAL & RC, VIJAYAPURA.

Name: CASE NO:

Age: IP NO:

Sex:

Religion:

Occupation:

Residence: Urban/ Rural

Family income:

Address and Mobile number:

Educational status: No schooling, 1-5 class, 5-10 class, 11-12 class, Degree, Post Degree

Socio-Economic status: L-SES / M-SES / U-SES

Details of Illness

Diagnosed with any Psychiatric illness in past-

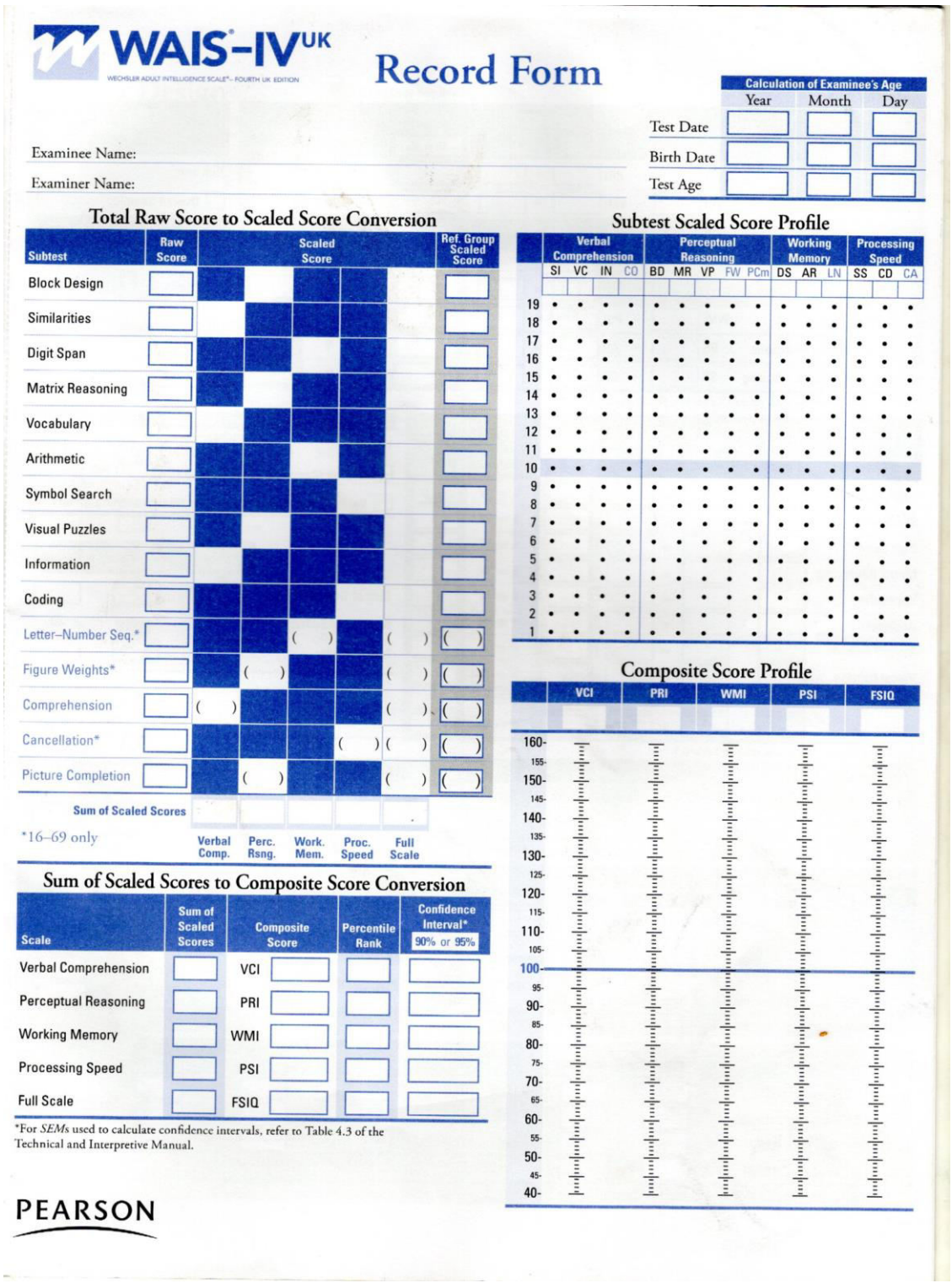
YES / NO Duration of illness in a parent-

Whether you were conceived before or after the illness developed in your

parent- BEFORE/ AFTER

Substance use- alcohol/tobacco/others/ NIL

Wechsler Adult Intelligence Scale (WAIS)



WAIS®-IV UK
WECHSLER ADULT INTELLIGENCE SCALE®- FOURTH UK EDITION

Record Form

	Calculation of Examinee's Age		
	Year	Month	Day
Test Date			
Birth Date			
Test Age			

Examinee Name:

Examiner Name:

Total Raw Score to Scaled Score Conversion

Subtest	Raw Score	Scaled Score	Ref. Group Scaled Score
Block Design			
Similarities			
Digit Span			
Matrix Reasoning			
Vocabulary			
Arithmetic			
Symbol Search			
Visual Puzzles			
Information			
Coding			
Letter–Number Seq.*		( )	( )
Figure Weights*		( )	( )
Comprehension		( )	( )
Cancellation*		( )	( )
Picture Completion		( )	( )
Sum of Scaled Scores			

*16–69 only

Verbal Comp. | Perc. Rsng. | Work. Mem. | Proc. Speed | Full Scale

Sum of Scaled Scores to Composite Score Conversion

Scale	Sum of Scaled Scores	Composite Score	Percentile Rank	Confidence Interval* 90% or 95%
Verbal Comprehension		VCI		
Perceptual Reasoning		PRI		
Working Memory		WMI		
Processing Speed		PSI		
Full Scale		FSIQ		

*For *SEMs* used to calculate confidence intervals, refer to Table 4.3 of the Technical and Interpretive Manual.

PEARSON

Subtest Scaled Score Profile

	Verbal Comprehension				Perceptual Reasoning					Working Memory			Processing Speed		
	SI	VC	IN	CO	BD	MR	VP	FW	PCm	DS	AR	LN	SS	CD	CA
19	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
18	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
17	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
16	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
15	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
14	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
13	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
12	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
11	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
10	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
9	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
1	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•

Composite Score Profile

	VCI	PRI	WMI	PSI	FSIQ
160					
155					
150					
145					
140					
135					
130					
125					
120					
115					
110					
105					
100					
95					
90					
85					
80					
75					
70					
65					
60					
55					
50					
45					
40					

Analysis

Discrepancy Comparison

	Comparison	Score 1		Score 2		Difference	Critical Value .15 or .05	Significant Difference	Base Rate
Index Level	VCI – PRI	VCI	-	PRI	=			Y or N	
	VCI – WMI	VCI	-	WMI	=			Y or N	
	VCI – PSI	VCI	-	PSI	=			Y or N	
	PRI – WMI	PRI	-	WMI	=			Y or N	
	PRI – PSI	PRI	-	PSI	=			Y or N	
	WMI – PSI	WMI	-	PSI	=			Y or N	
Subtest Level	Digit Span – Arithmetic	DS	-	AR	=			Y or N	
	Symbol Search – Coding	SS	-	CD	=			Y or N	

Basis for Comparison

Tick one:

- ☐ Overall Sample
- ☐ Ability Level

For discrepancy comparisons, refer to Tables B.1, B.2, B.3, and B.4.

Determining Strengths and Weaknesses

Subtest	Subtest Scaled Score		Mean Scaled Score		Difference From Mean	Critical Value .15 or .05	Strength or Weakness	Base Rate
Block Design		-		=			S or W	
Similarities		-		=			S or W	
Digit Span		-		=			S or W	
Matrix Reasoning		-		=			S or W	
Vocabulary		-		=			S or W	
Arithmetic		-		=			S or W	
Symbol Search		-		=			S or W	
Visual Puzzles		-		=			S or W	
Information		-		=			S or W	
Coding		-		=			S or W	

Basis for Comparison

Tick one:

- ☐ Overall Mean of 10 Core Subtests
- ☐ Verbal Comprehension & Perceptual Reasoning Means

	10 Core Subtests	3 Verbal Comprehension	3 Perceptual Reasoning
Sum of Scaled Scores			
Number of Subtests	÷10	÷3	÷3
Mean Score			

For strengths and weaknesses, refer to Table B.5.

Process Analysis

Total Raw Score to Scaled Score Conversion

Process Score	Raw Score	Scaled Score	Process Score	Raw Score	Scaled Score
Block Design No Time Bonus			Digit Span Backwards		
Digit Span Forwards			Digit Span Sequencing		

For scaled scores, refer to Table C.1.

Scaled Score Discrepancy Comparison

	Comparison	Score 1		Score 2		Difference	Critical Value .15 or .05	Significant Difference	Base Rate
Process Level	Block Design – Block Design No Time Bonus	BD	-	BDN	=			Y or N	
	Digit Span Forwards – Digit Span Backwards	DSF	-	DSB	=			Y or N	
	Digit Span Forwards – Digit Span Sequencing	DSF	-	DSS	=			Y or N	
	Digit Span Backwards – Digit Span Sequencing	DSB	-	DSS	=			Y or N	

For discrepancy comparisons, refer to Tables C.2 and C.3.

Raw Score to Base Rate Conversion

Process Score	Raw Score	Base Rate
Longest DS Forwards (LDSF)		
Longest DS Backwards (LDSB)		
Longest DS Sequence (LDSS)		
Longest LN Sequence (LLNS)		

For base rate information, refer to Tables C.4 and C.5.

Raw Score Discrepancy Comparison

Comparison	Score 1		Score 2		Difference	Base Rate
LDSF – LDSB		-		=		
LDSF – LDSS		-		=		
LDSB – LDSS		-		=		

For discrepancy comparisons, refer to Tables C.6, C.7, and C.8.

1. Block Design

(Time limit: See item)

Start
Ages 16–90: Sample Item, then Item 5

Reverse
Score of **0** on *either* Item 5 or Item 6, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **2** consecutive scores of 0

Score
Items 1–4: Score **0**, **1**, or **2** points.
Items 5–8: Score **0** or **4** points.
Items 9–14: Score **0**, **4**, **5**, **6**, or **7** points.
BDN
Items 1–4: Score **0**, **1**, or **2** points.
Items 5–14: Score **0** or **4** points.

	Design	Presentation Method	Time Limit	Completion Time		Constructed Design		Score				
16–90	S. Examinee / Examiner	Model and Picture	30"	Trial 1	Trial 2	Trial 1	Trial 2					
	1.	Model and Picture	30"	Trial 1	Trial 2	Trial 1	Trial 2	0	1	2		
	2.	Model and Picture	30"	Trial 1	Trial 2	Trial 1	Trial 2	0	1	2		
	3.	Model and Picture	30"	Trial 1	Trial 2	Trial 1	Trial 2	0	1	2		
	4.	Model and Picture	30"	Trial 1	Trial 2	Trial 1	Trial 2	0	1	2		
16–90	5. Examinee / Examiner	Picture	60"					0		4		
	6.	Picture	60"					0		4		
	7.	Picture	60"					0		4		
	8.	Picture	60"					0		4		
	9.	Picture	60"					0		31–60: 4	21–30: 5	11–20: 6	1–10: 7
	10.	Picture	60"					0		31–60: 4	21–30: 5	11–20: 6	1–10: 7
	11.	Picture	120"					0		76–120: 4	61–75: 5	31–60: 6	1–30: 7
	12.	Picture	120"					0		76–120: 4	61–75: 5	31–60: 6	1–30: 7
	13.	Picture	120"					0		76–120: 4	61–75: 5	31–60: 6	1–30: 7
	14.	Picture	120"					0		76–120: 4	61–75: 5	31–60: 6	1–30: 7

Block Design No Time Bonus (BDN) Total Raw Score (Maximum = 48) ☐

Block Design Total Raw Score (Maximum = 66) ☐

2. Similarities

Start
Ages 16–90:
Sample Item, then Item 4

Reverse
Score of **0** or **1** on *either* Item 4 or Item 5, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0**, **1**, or **2** points.
See the Administration and Scoring Manual for sample responses.

	Item	Response	Score
16–90	S. Two – Seven		
	1. Fork – Spoon		0 1 2
	2. Yellow – Green		0 1 2
	3. Carrots – Broccoli		0 1 2
16–90	†4. Horse – Tiger		0 1 2
	†5. Piano – Drum		0 1 2
	6. Boat – Car		0 1 2
	7. Nose – Tongue		0 1 2
	8. Food – Petrol		0 1 2
	9. Badge – Crown		0 1 2
	10. Bud – Baby		0 1 2
	11. Music – Tides		0 1 2
	12. Poem – Statue		0 1 2
	13. Anchor – Fence		0 1 2
	14. Wish – Expect		0 1 2
	15. Acceptance – Denial		0 1 2
	16. Always – Never		0 1 2
	17. Enemy – Friend		0 1 2
	18. Allow – Restrict		0 1 2

†If the examinee does not obtain a perfect score, provide corrective feedback as instructed in the Administration and Scoring Manual.

Similarities Total Raw Score
(Maximum = 36) ☐

3. Digit Span *(continued)*

Sequencing

Discontinue after scores of **0** on *both trials* of an item.

	Item	Trial	Correct Response	Response	Trial Score	Item Score
16–90	S.	2 – 3 – 1	1 – 2 – 3			
		5 – 2 – 2	2 – 2 – 5			
16–90	1.	1 – 2	1 – 2		0 1	0 1 2
		4 – 2	2 – 4		0 1	
	2.	3 – 1 – 6	1 – 3 – 6		0 1	0 1 2
		0 – 9 – 4	0 – 4 – 9		0 1	
	3.	8 – 7 – 9 – 2	2 – 7 – 8 – 9		0 1	0 1 2
		4 – 8 – 7 – 1	1 – 4 – 7 – 8		0 1	
	4.	2 – 6 – 9 – 1 – 7	1 – 2 – 6 – 7 – 9		0 1	0 1 2
		3 – 8 – 3 – 5 – 8	3 – 3 – 5 – 8 – 8		0 1	
	5.	2 – 1 – 7 – 4 – 3 – 6	1 – 2 – 3 – 4 – 6 – 7		0 1	0 1 2
		6 – 2 – 5 – 2 – 3 – 4	2 – 2 – 3 – 4 – 5 – 6		0 1	
	6.	7 – 5 – 7 – 6 – 8 – 6 – 2	2 – 5 – 6 – 6 – 7 – 7 – 8		0 1	0 1 2
		4 – 8 – 2 – 5 – 4 – 3 – 5	2 – 3 – 4 – 4 – 5 – 5 – 8		0 1	
	7.	5 – 8 – 7 – 2 – 7 – 5 – 4 – 5	2 – 4 – 5 – 5 – 5 – 7 – 7 – 8		0 1	0 1 2
		9 – 4 – 9 – 7 – 3 – 0 – 8 – 4	0 – 3 – 4 – 4 – 7 – 8 – 9 – 9		0 1	
	8.	5 – 0 – 1 – 1 – 3 – 2 – 1 – 0 – 5	0 – 0 – 1 – 1 – 1 – 2 – 3 – 5 – 5		0 1	0 1 2
		2 – 7 – 1 – 4 – 8 – 4 – 2 – 9 – 6	1 – 2 – 2 – 4 – 4 – 6 – 7 – 8 – 9		0 1	

LDSS (Max = 9)	**Digit Span Sequencing (DSS) Total Raw Score** (Maximum = 16)	

Digit Span Total Raw Score (Maximum = 48)

4. Matrix Reasoning

Start
Ages 16–90: Sample Items A & B, then Item 4

Reverse
Score of **0** on *either* Item 4 or Item 5, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0** or **1** point.
Correct responses are in **colour**.

	Item	Response					Score	
16–90	SA.	1	2	3	4	**5**		
	SB.	1	2	3	**4**	5		
	1.	1	2	**3**	4	5	0	1
	2.	1	**2**	3	4	5	0	1
	3.	**1**	2	3	4	5	0	1
16–90	4.	1	2	3	4	**5**	0	1
	5.	1	2	**3**	4	5	0	1
	6.	1	2	3	**4**	5	0	1
	7.	1	2	3	**4**	5	0	1
	8.	**1**	2	3	4	5	0	1
	9.	1	2	3	4	**5**	0	1
	10.	1	**2**	3	4	5	0	1
	11.	**1**	2	3	4	5	0	1
	12.	1	2	3	4	**5**	0	1

Item	Response					Score	
13.	**1**	2	3	4	5	0	1
14.	1	2	**3**	4	5	0	1
15.	1	2	3	4	**5**	0	1
16.	1	**2**	3	4	5	0	1
17.	1	2	**3**	4	5	0	1
18.	**1**	2	3	4	5	0	1
19.	1	2	3	**4**	5	0	1
20.	1	**2**	3	4	5	0	1
21.	**1**	2	3	4	5	0	1
22.	1	2	3	4	**5**	0	1
23.	1	2	3	**4**	5	0	1
24.	1	**2**	3	4	5	0	1
25.	1	2	**3**	4	5	0	1
26.	1	2	3	**4**	5	0	1

Matrix Reasoning Total Raw Score (Maximum = 26)

5. Vocabulary

Start
Ages 16–90: Item 5

Reverse
Score of **0** or **1** on *either* Item 5 or Item 6, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Items 1–3: Score **0** or **1** point.
Items 4–30: Score **0**, **1**, or **2** points.
See the Administration and Scoring Manual for sample responses.

	Item	Response	Score
	1. Book		0 1
	2. Aeroplane		0 1
	3. Basket		0 1
	4. Bed		0 1 2
16–90	†5. Apple		0 1 2
	†6. Glove		0 1 2
	7. Breakfast		0 1 2
	8. Curious		0 1 2
	9. Assemble		0 1 2
	10. Consume		0 1 2
	11. Terminate		0 1 2
	12. Tranquil		0 1 2
	13. Ponder		0 1 2
	14. Reluctant		0 1 2
	15. Confide		0 1 2

†If the examinee does not obtain a perfect score, provide corrective feedback as instructed in the Administration and Scoring Manual.

continue

5. Vocabulary *(continued)*

Discontinue after **3** consecutive scores of **0**.

Item	Response	Score
16. Remorse		0 1 2
17. Plagiarise		0 1 2
18. Acute		0 1 2
19. Generate		0 1 2
20. Compassion		0 1 2
21. Tangible		0 1 2
22. Evolve		0 1 2
23. Diverse		0 1 2
24. Fortitude		0 1 2
25. Ominous		0 1 2
26. Encumber		0 1 2
27. Audacious		0 1 2
28. Tirade		0 1 2
29. Pragmatic		0 1 2
30. Palliate		0 1 2

Vocabulary Total Raw Score (Maximum = 57) ☐

6. Arithmetic

(Time limit: 30 seconds)

Start
Ages 16–90:
Sample Item, then Item 6

Reverse
Score of **0** on *either* Item 6 or Item 7, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0** or **1** point.

	Item	Completion Time	Correct Response	Response	Score
16–90	S. Footballs		3		
	†1. Flowers		Counts to 3		0 1
	†2. Apples		Counts to 10		0 1
	3. Bats		6		0 1
	4. Birds		9		0 1
	5. Leads		2		0 1
16–90	6. Blankets		8		0 1
	7. Pens		5		0 1
	8. Toys		5		0 1
	9. Older		17		0 1
	10. Books		5		0 1
	11. Tickets		3		0 1
	12. Packs		200		0 1
	13. Cards		38		0 1
	14. Run		140		0 1
	15. Line		30		0 1
	16. Cakes		186		0 1
	17. Maps		600		0 1
	18. Hours		47		0 1
	19. Pies		49½		0 1
	20. Laps		51		0 1
	21. Machines		96		0 1
	22. Mail		23,100		0 1

†If the examinee does not give a correct response, provide corrective feedback as instructed in the Administration and Scoring Manual.

Arithmetic Total Raw Score
(Maximum = 22)

7. Symbol Search

(Time limit: 120 seconds)

Start
Ages 16–90:
Demonstration Items, Sample Items, then Test Items

Discontinue
After **120** seconds

Score
Use the Symbol Search Scoring Key to score the examinee's responses.
Subtract Number Incorrect from Number Correct.
If the total raw score is <0, enter 0 as the total raw score.

Completion Time		Number Correct		Number Incorrect		Symbol Search Total Raw Score (Maximum = 60)
			−		=	

8. Visual Puzzles

(Time limit: See item)

Start
Ages 16–90:
Demonstration Item, Sample Item, then Item 5

Reverse
Score of **0** on *either* Item 5 or Item 6, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0** or **1** point.
Correct responses are in **colour**.

	Item	Time Limit	Completion Time	Response Choices	Score
16–90	D.			**1** 2 **3** 4 5 **6**	
16–90	S.			**1** 2 **3** 4 5 **6**	
	1.	20"		1 **2** **3** 4 **5** 6	0 1
	2.	20"		**1** **2** 3 4 **5** 6	0 1
	3.	20"		**1** 2 3 **4** 5 **6**	0 1
	4.	20"		1 **2** **3** 4 5 **6**	0 1
16–90	5.	20"		1 2 **3** 4 **5** **6**	0 1
	6.	20"		**1** 2 **3** 4 5 **6**	0 1
	7.	20"		1 **2** 3 4 **5** **6**	0 1
	8.	30"		**1** 2 **3** **4** 5 6	0 1
	9.	30"		**1** 2 **3** 4 5 **6**	0 1
	10.	30"		**1** **2** 3 4 **5** 6	0 1
	11.	30"		**1** **2** 3 4 **5** 6	0 1
	12.	30"		**1** 2 3 **4** **5** 6	0 1
	13.	30"		1 2 **3** **4** 5 **6**	0 1
	14.	30"		**1** **2** 3 4 5 **6**	0 1
	15.	30"		1 **2** **3** **4** 5 6	0 1
	16.	30"		1 2 **3** **4** 5 **6**	0 1
	17.	30"		**1** **2** 3 4 5 **6**	0 1
	18.	30"		**1** 2 3 4 **5** **6**	0 1
	19.	30"		1 **2** **3** 4 **5** 6	0 1
	20.	30"		**1** 2 **3** **4** 5 6	0 1
	21.	30"		**1** 2 3 4 **5** **6**	0 1
	22.	30"		1 **2** **3** 4 **5** 6	0 1
	23.	30"		1 2 **3** **4** 5 **6**	0 1
	24.	30"		1 2 **3** **4** **5** 6	0 1
	25.	30"		**1** **2** **3** 4 5 6	0 1
	26.	30"		1 2 **3** **4** 5 **6**	0 1

Visual Puzzles Total Raw Score
(Maximum = 26)

9. Information

Start
Ages 16–90:
Item 3

Reverse
Score of **0** on *either* Item 3 or Item 4, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0** or **1** point.
See the Administration and Scoring Manual for sample responses.

	Item	Response	Score
	*1. Monday		0 1
	*2. Shape		0 1
16–90	†3. Thermometer		0 1
	†4. Seconds		0 1
	5. Hamlet		0 1
	6. Line		0 1

*Responses requiring specific query are identified in the Administration and Scoring Manual.
†If the examinee does not give a correct response, provide corrective feedback as instructed in the Administration and Scoring Manual.

continue

9. Information *(continued)*

Discontinue after **3** consecutive scores of 0.

Item	Response	Score
*7. Brazil		0 1
8. Cleopatra		0 1
9. World War II		0 1
10. Water		0 1
11. Sahara		0 1
12. Italy		0 1
13. Olympics		0 1
14. MLK Jr.		0 1
15. Relativity		0 1
16. Gandhi		0 1
17. Boil		0 1
18. Marie Curie		0 1
*19. Vessels		0 1
20. Language		0 1
21. Organ		0 1
22. Catherine		0 1
23. Sherlock Holmes		0 1
24. Alice		0 1
*25. Circumference		0 1
*26. Minutes		0 1

*Responses requiring specific query are identified in the Administration and Scoring Manual.

Information Total Raw Score
(Maximum = 26) ☐

10. Coding

(Time limit: 120 seconds)

Start
Ages 16–90: Demonstration Items, Sample Items, then Test Items

Discontinue
After **120** seconds

Score
Use the Coding Scoring Template to score the examinee's responses.
Score **1** point for each correct response.

Completion Time ☐

Coding Total Raw Score
(Maximum = 135) ☐

11. Letter–Number Sequencing

Start
Ages 16–69:
Demonstration Item A, Sample Item A, then Item 1
Ages 70–90:
Do not administer

Discontinue
After scores of **0** on *all three trials* of an item

Score
Score **0** or **1** point for each trial.
LLNS
Number of letters and digits recalled on last trial scored 1 point.

	Item	Trial	Correct Responses		Response	Trial Score	Item Score
16–69	DA.	C – 1	1 – C				
16–69	SA.	A – 4	4 – A				
		2 – B	2 – B			0 1	0 1
16–69	†1.	D – 1	1 – D			0 1	2 3
		4 – C	4 – C			0 1	
		E – 5	5 – E			0 1	0 1
	†2.	3 – A	3 – A			0 1	2 3
		C – 1	1 – C			0 1	

†If the examinee does not say the number first, say, **Remember to say the number first, then say the letter.**

	DB. 2 – B – 1	1 – 2 – B				
	SB. D – 5 – A	5 – A – D				
	SB. 2 – B – 4	2 – 4 – B				
	5 – C – A	5 – A – C	A – C – 5		0 1	0 1
	3. F – E – 1	1 – E – F	E – F – 1		0 1	2 3
	3 – 2 – A	2 – 3 – A	A – 2 – 3		0 1	
	1 – G – 7	1 – 7 – G	G – 1 – 7		0 1	0 1
	4. H – 9 – 4	4 – 9 – H	H – 4 – 9		0 1	2 3
	3 – Q – 7	3 – 7 – Q	Q – 3 – 7		0 1	
	Z – 8 – N	8 – N – Z	N – Z – 8		0 1	0 1
	5. M – 6 – U	6 – M – U	M – U – 6		0 1	2 3
	P – 2 – N	2 – N – P	N – P – 2		0 1	
	V – 1 – J – 5	1 – 5 – J – V	J – V – 1 – 5		0 1	0 1
	6. 7 – X – 4 – G	4 – 7 – G – X	G – X – 4 – 7		0 1	2 3
	S – 9 – T – 6	6 – 9 – S – T	S – T – 6 – 9		0 1	
	8 – E – 6 – F – 1	1 – 6 – 8 – E – F	E – F – 1 – 6 – 8		0 1	0 1
	7. K – 4 – C – 2 – S	2 – 4 – C – K – S	C – K – S – 2 – 4		0 1	2 3
	5 – Q – 3 – H – 6	3 – 5 – 6 – H – Q	H – Q – 3 – 5 – 6		0 1	
	M – 4 – P – 7 – R – 2	2 – 4 – 7 – M – P – R	M – P – R – 2 – 4 – 7		0 1	0 1
	8. 6 – N – 9 – J – 2 – S	2 – 6 – 9 – J – N – S	J – N – S – 2 – 6 – 9		0 1	2 3
	U – 6 – H – 5 – F – 3	3 – 5 – 6 – F – H – U	F – H – U – 3 – 5 – 6		0 1	
	R – 7 – V – 4 – Y – 8 – F	4 – 7 – 8 – F – R – V – Y	F – R – V – Y – 4 – 7 – 8		0 1	0 1
	9. 9 – X – 2 – J – 3 – N – 7	2 – 3 – 7 – 9 – J – N – X	J – N – X – 2 – 3 – 7 – 9		0 1	2 3
	M – 1 – Q – 8 – R – 4 – D	1 – 4 – 8 – D – M – Q – R	D – M – Q – R – 1 – 4 – 8		0 1	
	6 – P – 7 – S – 2 – N – 9 – A	2 – 6 – 7 – 9 – A – N – P – S	A – N – P – S – 2 – 6 – 7 – 9		0 1	0 1
	10. U – 1 – R – 9 – X – 4 – K – 3	1 – 3 – 4 – 9 – K – R – U – X	K – R – U – X – 1 – 3 – 4 – 9		0 1	2 3
	7 – M – 2 – T – 6 – F – 9 – A	2 – 6 – 7 – 9 – A – F – M – T	A – F – M – T – 2 – 6 – 7 – 9		0 1	

LLNS (Max = 8)

Letter–Number Sequencing Total Raw Score
(Maximum = 30)

12. Figure Weights

(Time limit: See item)

Start
Ages 16–69:
Demonstration Items A & B, Sample Item, then Item 4
Ages 70–90:
Do not administer

Reverse
Score of **0** on *either* Item 4 or Item 5, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0** or **1** point.
Correct responses are in **colour**.

	Item	Time Limit	Completion Time	Response					Score	
16–69	DA.			1	**2**	3	4	5		
	DB.			**1**	2	3	4	5		
16–69	S.			1	2	**3**	4	5		
	1.	20"		1	2	3	4	**5**	0	1
	2.	20"		**1**	2	3	4	5	0	1
	3.	20"		1	**2**	3	4	5	0	1
16–69	4.	20"		1	2	**3**	4	5	0	1
	5.	20"		1	2	3	**4**	5	0	1
	6.	20"		**1**	2	3	4	5	0	1
	7.	20"		1	2	3	**4**	5	0	1
	8.	20"		1	2	3	**4**	5	0	1
	9.	20"		1	2	3	4	**5**	0	1
	10.	20"		**1**	2	3	4	5	0	1
	11.	20"		1	**2**	3	4	5	0	1
	12.	20"		1	2	3	4	**5**	0	1
	13.	40"		1	2	**3**	4	5	0	1
	14.	40"		**1**	2	3	4	5	0	1
	15.	40"		1	2	3	**4**	5	0	1
	††16.	40"		**1**	2	3	4	5	0	1
	17.	40"		1	2	**3**	4	5	0	1
	18.	40"		1	2	**3**	4	5	0	1
	19.	40"		1	**2**	3	4	5	0	1
	20.	40"		1	**2**	3	4	5	0	1
	21.	40"		1	**2**	3	4	5	0	1
	22.	40"		1	2	3	4	**5**	0	1
	23.	40"		1	2	**3**	4	5	0	1
	24.	40"		1	2	3	4	**5**	0	1
	25.	40"		**1**	2	3	4	5	0	1
	26.	40"		1	**2**	3	4	5	0	1
	27.	40"		1	2	3	**4**	5	0	1

††Give verbatim as instructed in the Administration and Scoring Manual.

Figure Weights Total Raw Score (Maximum = 27)

13. Comprehension

Start
Ages 16–90:
Item 3

Reverse
Score of **0** or **1** on *either* Item 3 or Item 4, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **3** consecutive scores of 0

Score
Score **0**, **1**, or **2** points.
See the Administration and Scoring Manual for sample responses.

	Item	Response	Score
	1. Watches		0 1 2
	2. Clothes		0 1 2
16–90	†*3. Envelope		0 1 2

†If the examinee does not obtain a perfect score, provide corrective feedback as instructed in the Administration and Scoring Manual.

*Responses requiring specific query are identified in the Administration and Scoring Manual.

continue

3. Comprehension *(continued)*

Discontinue after **3** consecutive scores of 0.

Item	Response	Score
†4. Money		0 1 2
§5. Foods		0 1 2
6. Licence		0 1 2
7. History		0 1 2
§8. Countries		0 1 2
§9. Job		0 1 2
§10. Outer space		0 1 2
11. Fall		0 1 2
12. Animals		0 1 2
13. Land		0 1 2
14. Teeth		0 1 2
15. Winter		0 1 2
16. Democracy		0 1 2
17. Crime		0 1 2
18. Waters		0 1 2

he examinee does not obtain a perfect score, provide corrective feedback as instructed in the Administration and Scoring Manual.
he examinee responds with only one general concept, say, **Tell me some more reasons why** *[rephrase item appropriately]*.

Comprehension Total Raw Score
(Maximum = 36) ☐

14. Cancellation

(Time limit: 45 seconds)

Start
Ages 16–69:
Demonstration Item A, Sample Item A, then Item 1
Ages 70–90:
Do not administer

Discontinue
After **45** seconds for each item

Score
Use the Cancellation Scoring Template to score the examinee's responses.
Subtract Number Incorrect from Number Correct for each item score.
If the item score is <0, enter 0 as the item score.
The total raw score is the sum of the item scores.

	Item	Completion Time	Number Correct		Number Incorrect		Item Score
16–69	1.			—		=	
	2.			—		=	

Cancellation Total Raw Score
(Maximum = 72)

15. Picture Completion

(Time limit: 20 seconds)

Start
Ages 16–90:
Sample Item, then Item 4

Reverse
Score of **0** on *either* Item 4 or Item 5, administer preceding items in **reverse** order until two consecutive perfect scores are obtained.

Discontinue
After **4** consecutive scores of 0

Score
Score **0** or **1** point.
See the Administration and Scoring Manual for sample responses.

Each of the following prompts can be provided ***once only*** during subtest administration.

If the examinee names the pictured object instead of referring to or pointing to the missing part, say, **Yes, but what is missing?**

If the examinee refers to or points to a part that is off the page, say, **A part is missing in the picture. What is it that is missing?**

If the examinee refers to or points to an unessential missing part, say, **Yes, but what is the most important part missing?**

	Item	Completion Time	Verbal Response	Pointing Response	Score
16–90	S. Comb			PC PX	
	1. Table			PC PX	0 1
	2. Face			PC PX	0 1
	3. Mirror			PC PX	0 1
6–90	†4. Glasses			PC PX	0 1
	†5. Jogging			PC PX	0 1
	6. Knife			PC PX	0 1
	7. Jug			PC PX	0 1
	8. Roses			PC PX	0 1
	9. Pie			PC PX	0 1
	10. Cow			PC PX	0 1
	11. Gate			PC PX	0 1
	12. Trees			PC PX	0 1
	13. Lockers			PC PX	0 1
	14. Karate			PC PX	0 1
	15. Barn			PC PX	0 1
	16. Walking			PC PX	0 1
	17. Puddles			PC PX	0 1
	18. Shoes			PC PX	0 1
	19. Tent			PC PX	0 1
	20. Car			PC PX	0 1
	21. Bookshelf			PC PX	0 1
	22. Basket			PC PX	0 1
	23. Plane			PC PX	0 1
	24. Cooker			PC PX	0 1

If the examinee does not give a correct response, provide corrective feedback as instructed in the Administration and Scoring Manual.

Picture Completion Total Raw Score
(Maximum = 24)

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Examinee Name: Age:

Sex: ☐ F ☐ M Handedness: ☐ R ☐ L ID:

Examiner Name:

Testing Site:

Record Form

Behavioural Observations

Referral source/Reason for referral/Presenting complaint(s)

Language (e.g. first/native language, other language, English fluency, expressive and receptive language ability, articulation)

Physical appearance

Visual/Auditory/Motor problems (Were problems corrected [e.g. with glasses, assistive listening device]?)

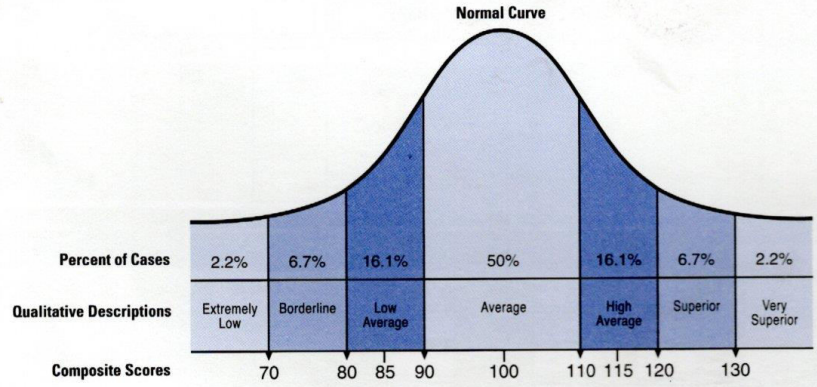
Attention and concentration

Attitude toward testing (e.g. rapport, eager to speak, working habits, interest, motivation, reaction to success/failure)

Affect/Mood

Unusual behaviours/Verbalisations (e.g. perseverations, stereotypic movements, bizarre and atypical verbalisations)

Other notes



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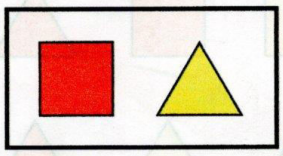
Response Booklet 2

Cancellation

Examinee Name: _____ Age: _____

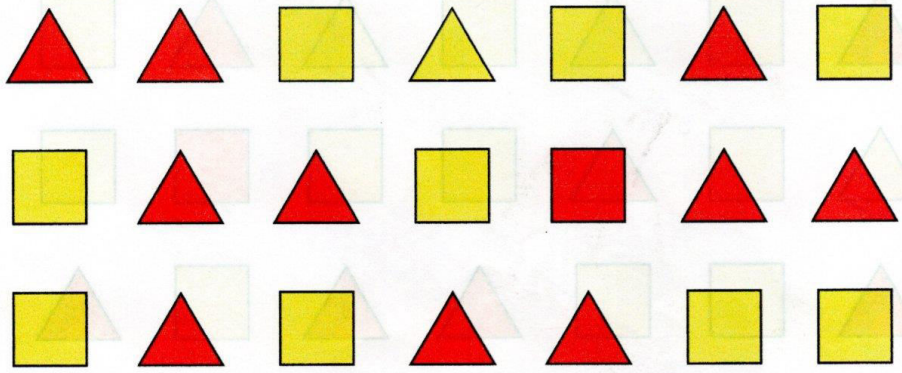
Examiner Name: _____

Test Date: _____



Demonstration Item A.

Sample Item A.



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Response Booklet 1

Symbol Search

Coding

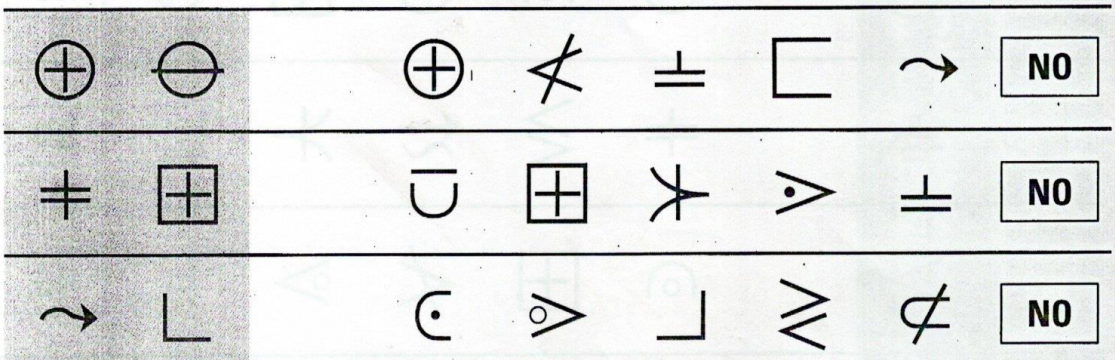
Examinee Name: _____ Age: _____

Examiner Name: _____

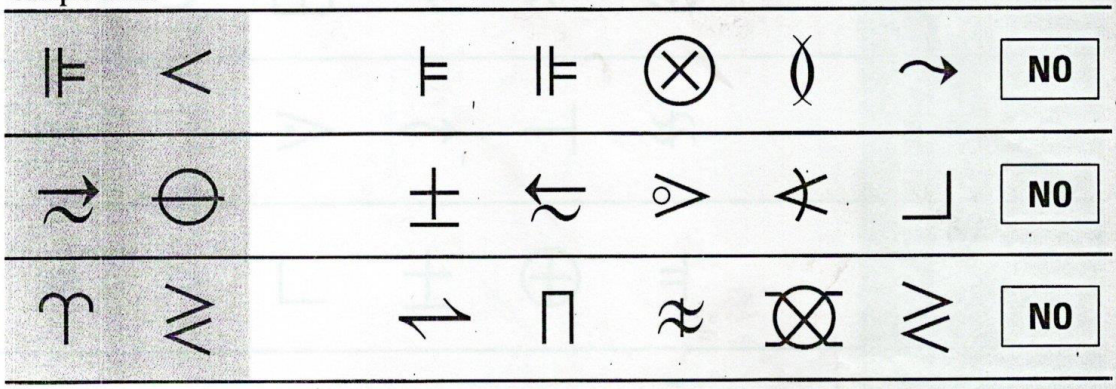
Test Date: _____

Symbol Search

Demonstration Items



Sample Items



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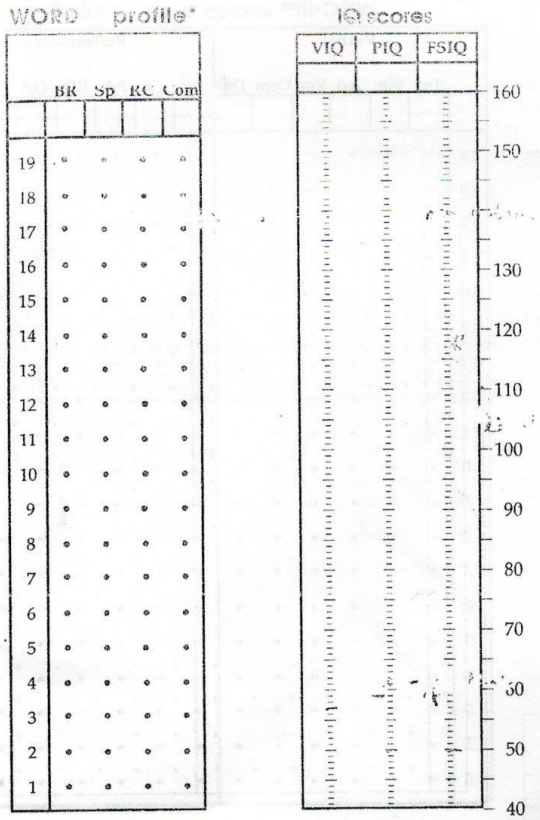
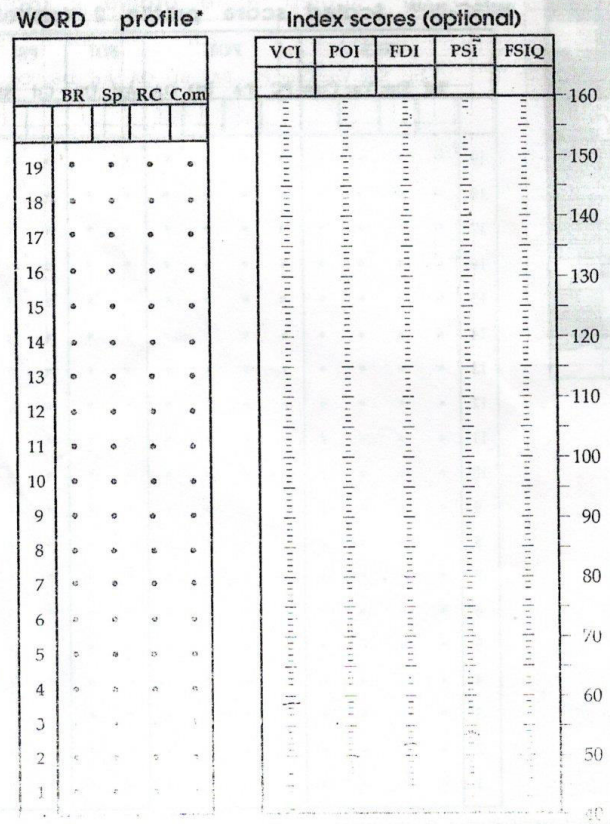
Wechsler Intelligence Scale for Children (WISC)





Subtest score evaluation

Tables 1a and 1b show the minimum difference betw any single subtest scaled score and the average scaled s of the group of subtests against which it is being compa that is required to achieve statistical significance. Di ences are presented for two levels of confidence: 0.15 0.05. For example, as indicated in Table 1a, a Picture C pletion scaled score that is at least 3.31 points abov below the child's average scaled score on five Performa subtests is significantly different from that mean scor the 0.05 level of confidence.

Table 2 provides similar information for interpreting difference between a single subtest scaled score and mean score on the subtests contributing to the two m factor-based indexes, Verbal Comprehension and Per tual Organisation. For a fuller discussion of these sco see Chapter 4 in the WISC-III[UK] Manual.

Table 1a

	Average of 5 subtests		Average of 6 subtests		Average 7 subtest	
Significance level	**0.15**	**0.05**	**0.15**	**0.05**	**0.15**	**0.0**
Information	2.42	2.88	2.54	2.99		
Similarities	2.52	3.00	2.65	3.12		
Arithmetic	2.69	3.19	2.83	3.34		
Vocabulary	2.21	2.62	2.30	2.70		
Comprehension	2.74	3.26	2.90	3.41		
Digit Span			2.44	2.87		
Picture Completion	2.79	3.31	2.95	3.47	3.08	3.6
Coding	2.76	3.28	2.91	3.43	3.04	3.5
Picture Arrangement	2.85	3.38	3.01	3.55	3.15	3.6
Block Design	2.33	2.76	2.42	2.85	2.51	2.9
Object Assembly	3.13	3.72	3.33	3.92	3.49	4.0
Symbol Search			3.01	3.55	3.15	3.6
Mazes					3.44	4.0

Table 1b

	Average of 10 subtests		Average of 12 subtests		Average 13 subtes	
Significance level	**0.15**	**0.05**	**0.15**	**0.05**	**0.15**	**0.0**
Information	2.88	3.32	2.97	3.41	3.02	3.4
Similarities	3.02	3.48	3.12	3.58	3.17	3.6
Arithmetic	3.24	3.74	3.36	3.86	3.42	3.9
Vocabulary	2.58	2.97	2.65	3.04	2.69	3.0
Comprehension	3.33	3.84	3.45	3.96	3.51	4.0
Digit Span			2.84	3.26	2.89	3.3
Picture Completion	3.31	3.81	3.43	3.94	3.49	3.9
Coding	3.26	3.77	3.39	3.89	3.44	3.9
Picture Arrangement	3.39	3.91	3.52	4.04	3.58	4.0
Block Design	2.63	3.04	2.72	3.12	2.76	3.1
Object Assembly	3.78	4.37	3.94	4.52	4.00	4.5
Symbol Search			3.52	4.04	3.58	4.0
Mazes					3.94	4.5

Table 2

	Average of 4 Verb. Comp. subtests		Average of 4 Per. Org. subtests	
Significance level	**0.15**	**0.05**	**0.15**	**0.05**
Information	2.24	2.69		
Similarities	2.33	2.79		
Arithmetic				
Vocabulary	2.07	2.48		
Comprehension	2.51	3.01		
Digit Span				
Picture Completion			2.59	3.11
Coding				
Picture Arrangement			2.04	3.17
Block Design			2.22	2.66
Object Assembly			2.88	3.45
Symbol Search				
Mazes				

1. Picture Completion

Time limit: 20" each item. Discontinue after 5 consecutive failures. For ages 8–16, reverse sequence of preceding items after failure on either of first two items administered.

	Item	Response	Score 0 or 1
All ages	Sample: Pencil		
6–7	1. Fox		
	2. Box		
	3. Cat		
	4. Hand		
8–9	5. Elephant		
	6. Man		
10–13	7. Door		
	8. Mirror		
	9. Clock		
	10. Chest of drawers		
14–16	11. Belt		
	12. Leaf		
	13. Stepladder		
	14. Woman's face		
	15. Dice		
	16. Bath		
	17. Light bulb		
	18. Whistle		
	19. Piano		
	20. Scissors		
	21. Male profile		
	22. Thermometer		
	23. Trellis		
	24. Orange		
	25. Goldfish		
	26. Supermarket		
	27. Telephone		
	28. Umbrella		
	29. House		
	30. Tennis shoe		

Total subtest score (maximum = 30)

2. Information

Discontinue after 5 consecutive failures.
For ages 8–16, reverse sequence of preceding items after failure on either of first two items administered.

	Item	Response	Score 0 or 1
6–7	1. Nose		1
	2. Ears		1
	3. Legs		1
	4. Thursday		1
8–10	5. Coins		1
	6. March		1
	7. Week		1
11–13	8. Boil		1
	9. Seasons		
	10. Hours		
14–16	11. Dozen		
	12. Stomach		1
	13. Leap year		
	14. Columbus		
	15. Oceans		
	16. Oxygen		
	17. Brazil		
	18. Sun		
	19. Telephone		
	20. Hieroglyphics		
	21. Population		
	22. Greece		
	23. Water		
	24. Anne Frank		
	25. Glass		
	26. Barometer		
	27. Rust		
	28. London		
	29. Darwin		
	30. Aluminium		

Total subtest score (maximum = 30)

Picture Completion cautions checklist (see Manual pp. 110–111)
The following cautions should be given, if necessary, but each caution may be given only once during the test.

1. *"Yes, but what's missing?"* ☐
2. *"A part is missing in the picture. What is it that is missing?"* ☐
3. *"Yes, but what is the most important part that is missing?"* ☐

3. Coding

Discontinue after 120 seconds.

	Time limit	Complet. time	Total subtest score
6–7 Part A	120"		Max. = 65
8–16 Part B	120"		Max. = 119

Part A
Score including time-bonus points for perfect performance

Time in seconds	120–116	115–111	110–106	105–101	100–96	95–86	85–0
Score	59	60	61	62	63	64	65

4. Similarities

Discontinue after 4 consecutive failures.

Item	Response	
All ages	Sample: Red–Blue	Score 0 or 1
	*1. Piano–Guitar	
	*2. Candle–Lamp	
	3. Shirt–Shoe	
	4. Wh	
	5. Milk–W	
	†6. Apple–	Score 0 1 or 2
	7. Cat–Mouse	
	8. Elbow–Knee	
	9. Anger–Joy	
	10. Telephone–Radio	
	11. Painting–Statue	
	12. Family–Tribe	
	13. Ice–Steam	
	14. Temperature–Length	
	15. Mountain–Lake	
	16. Rubber–Paper	
	17. First–Last	
	‡18. Numbers 9 and 25	
	19. Salt–Water	

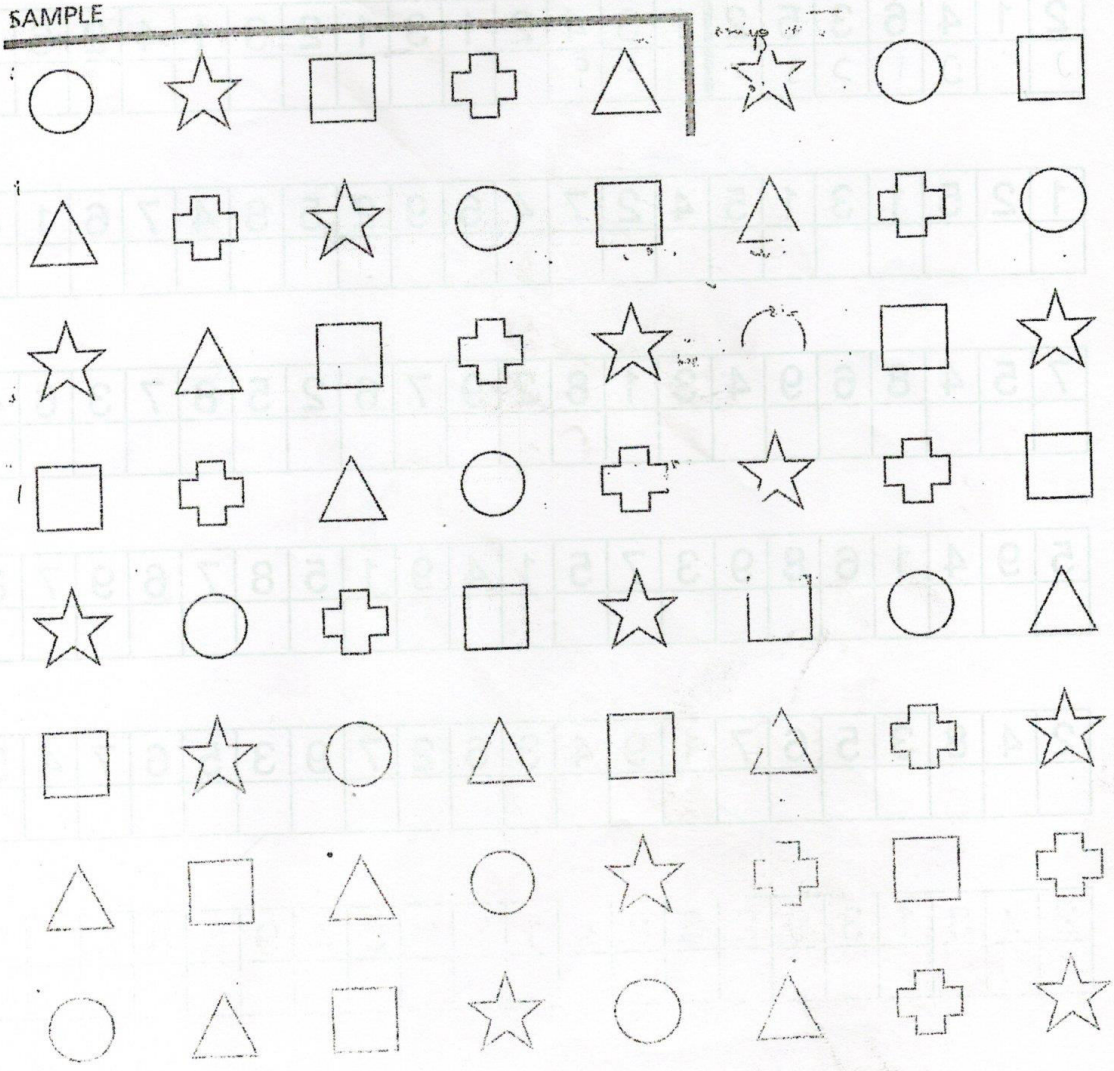
* If the child says that they are not alike, fails to respond, or gives an incorrect response, give an example of a 1-point response.
† If the child gives a 1-point response, give an example of a 2-point response.
‡ If the child gives a 1-point response, ask *"How else are the numbers 9 and 25 alike?"*

Total subtest score (maximum = 33) 11

4

A

SAMPLE



B

SAMPLE

2	1	4	6	3	5	2	1	3	4	2	1	3	1	2	3	1	4	2	6	3
)		)		)	)	)	c	c	c											

1	2	5	1	3	1	5	4	2	7	4	6	9	2	5	8	4	7	6	1	8

7	5	4	8	6	9	4	3	1	8	2	9	7	6	2	5	8	7	3	6	4

5	9	4	1	6	8	9	3	7	5	1	4	9	1	5	8	7	6	9	7	8

2	4	8	3	5	6	7	1	9	4	3	6	2	7	9	3	5	6	7	4	5

2	7	9	1	3	6	2	5	8	4	1	5	7	3	4	9	5	8	5	1	5

5. Picture Arrangement

Discontinue after 3 consecutive failures.
Items 1 and 2 are considered failed only if both trials are failed.
For ages 9–16, normal sequence of preceding items after failure on Item 3.

Note: Set out cards in sequence of dot patterns (right-hand corner of card) and record the child's card response order according to card number (left-hand corner).

	Item	Time limit	Complet. time	Response order	Score Circle the appropriate score	Score
All ages	Sample: Drinks machine					
6–8	1. Slide Trial 1	45″			0 2	
	1. Slide Trial 2	45″			0 1	
	2. Picnic Trial 1	45″			0 2	
	2. Picnic Trial 2	45″			0 1	
9–16	3. River crossing	45″			0 45–16 2 15–11 3 10–6 4 5–1 5	
	4. Snack time	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	5. Missing the boat	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	6. Hold-up	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	7. Gone fishing	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	8. House fire	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	9. Seeing stars	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	10. Ducks crossing	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	11. Rain shower	45″			0 45–21 2 20–16 3 15–11 4 10–1 5	
	*12. Walking the dog	60″			0 60–26 2 25–16 3 15–11 4 10–1 5	
	13. Ploughman's lunch	60″			0 60–26 2 25–16 3 15–11 4 10–1 5	
	†14. Snow scene	60″			0 654321 1 60–26 2 25–16 3 15–11 4 10–1 5	

* **456123** is an equally acceptable response.
† The response **654321** scores 1 point.

Total subtest score (maximum = 64)

6. Arithmetic

Discontinue after 3 consecutive failures.
For ages 7–16, reverse sequence of preceding items after failure on either of first two items administered.

	Problem	Time limit	Complet. time	Correct response	Response	Score Circle one
6	1. Count birds	30″		3		0 1
	2. Count trees	30″		12		0 1
	3. Leave 4	30″		4		0 1
	4. Leave 9	30″		9		0 1
	5. Ice cream	30″		2		0 1
7–8	6. Apple	30″		2		0 1
	7. Pence	30″		6		0 1
	8. Cakes	30″		3		0 1
	9. Books	30″		4		0 1
	10. Pencils	30″		5		0 1
	11. Chocolate	30″		7		0 1
9–12	12. Rulers	30″		6		0 1
	13. Crayons	30″	-	14	-	0 1
13–16	14. Newspapers	30″		7		0 1
	15. T-shirts	30″		£24		0 1
	16. Milk	30″		11		0 1
	17. Earn	30″		9		0 1
	18. Dozen	45″		10p		0 1
	19. Money	75″		£8.50		0 75–11 1 10–1 2
	20. Boxes	75″		£40		0 75–11 1 10–1 2
	21. Bicycle	75″		£42		0 75–11 1 10–1 2
	22. Pens	75″		3/10, 6/20 or 30%		0 75–11 1 10–1 2
	23. Journey	75″		45 mph		0 75–11 1 10–1 2
	24. Cars	75″		48		0 75–11 1 10–1 2

7

Total subtest score (maximum = 30)

7. Block Design

Discontinue after 2 consecutive failures.

For ages 8–16, normal sequence of preceding items after failure on either trial of Design 3.

Child

Correct design	Time limit	Incorrect design	Complet. time	Correct design	Score Circle the appropriate score for each design	Score
1.	30"	Trial 1 Trial 2		Y N	0 Trial 1: 2 Trial 2: 1	
2.	45"	Trial 1 Trial 2		Y N	0 Trial 1: 2 Trial 2: 1	
3.	45"	Trial 1 Trial 2		Y N	0 Trial 1: 2 Trial 2: 1	
4.	45"			Y N	0 — 45–16: 4; 15–11: 5; 10–6: 6; 5–1: 7	
5.	45"			Y N	0 — 45–21: 4; 20–16: 5; 15–11: 6; 10–1: 7	
6.	75"			Y N	0 — 75–21: 4; 20–16: 5; 15–11: 6; 10–1: 7	
7.	75"			Y N	0 — 75–21: 4; 20–16: 5; 15–11: 6; 10–1: 7	
8.	75"			Y N	0 — 75–21: 4; 20–16: 5; 15–11: 6; 10–1: 7	
9.	75"			Y N	0 — 75–26: 4; 25–16: 5; 15–11: 6; 10–1: 7	
10.	120"			Y N	0 — 120–41: 4; 40–31: 5; 30–26: 6; 25–1: 7	
11.	120"			Y N	0 — 120–56: 4; 55–36: 5; 35–31: 6; 30–1: 7	
12.	120"			Y N	0 — 120–56: 4; 55–36: 5; 35–31: 6; 30–1: 7	

Examiner

Total subtest score (maximum = 69)

8. Vocabulary

Discontinue after 4 consecutive failures.

For ages 9–16, reverse sequence of preceding items after failure on either of first two items administered.

Item	Response	Score 0, 1 or 2
1. Clock		
2. Umbrella		
3. Hat		
4. Thief		
5. Cow		
6. Bicycle		

8. Vocabulary (continued)

Discontinue after 4 consecutive failures.
For ages 9–16, reverse sequence of preceding items after failure on either of first two items administered.

Item	Response	Score 0, 1 or 2
7. Donkey		
8. Alphabet		
9. Ancient		
10. Leave		
11. Brave		
12. Island		
13. Absorb		
14. Nonsense		
15. Precise		
16. Transparent		
17. Boast		
18. Migrate		
19. Fable		
20. Strenuous		
21. Mimic		
22. Rivalry		
23. Seclude		
24. Unanimous		
25. Amendment		
26. Compel		
27. Imminent		
28. Affliction		
29. Dilatory		
30. Aberration		

Total subtest score (maximum = 60)

9

9. Object Assembly

Do not discontinue. Administer *all* items.

Object	Time limit	Complet. time	No. of correct junctures	Multiply by	Score Circle the appropriate score for each object										Score
Sample: Apple															
1. Girl	120″		(0–6)	1	0	1	2	3	4	5	120–26 0	25–16 7	15–1 8		
2. Car	150″		(0–9)	1/2*	0	1	2	3	4	150–36 5	35–26 6	25–21 7	20–1 8		
3. Horse	150″		(0–5)	1	0	1	2	3	4	150–31 5	30–21 6	20–16 7	15–1 8		
4. Ball	180″		(0–7)	1	0	1	2	3	4	5	6	180–61 7	60–36 8	35–26 9	25–1 10
5. Face	180″		(0–13)	1/2*	0	1	2	3	4	5	6	180–81 7	80–56 8	55–41 9	40–1 10

* Round half scores upwards.

Total subtest score (maximum = 44)

10. Comprehension

Discontinue after 3 consecutive failures.

Item	Response	Score 0, 1 or 2
*1. Cut finger		
2. Find wallet		
3. Seat-belts		
†4. Smoke		
5. Lose ball		
6. Telephone book		
7. Fight		

* If the child does not give a 2-point response, illustrate with a few 2-point answers.
† If the child's response reflects only one general idea, ask for a second response.

10

10. Comprehension (continued)

Item	Response	Score 0, 1 or 2
†8. Lights		
†9. Rules		
10. Inspect meat		
†11. Number plates		
12. Stamps		
13. Promise		
†14. Newspaper		
15. Secret ballot		
†16. Paperback books		
†17. MPs (TDs)		
†18. Freedom of speech		

† If the child's response reflects only one general idea, ask for a second response.

Total subtest score (maximum = 36)

11. Symbol Search

Discontinue after 120 seconds.

	6–7 Part A	8–16 Part B
Time limit	120″	120″
Complet. time		
Number correct		
Number incorrect		
	Max. = 45	Max. = 45
Total subtest score		

(Total score = number correct minus number incorrect)

12. Digit Span

For both Digits Forward and Digits Backward, administer both trials of each item even if Trial 1 is passed.
Discontinue after failure of both trials of any item.
Administer Digits Backward even if Digits Forward score is 0.

Digits Forward		Trial score		Trial score	Item score 0,1 or 2
All ages	Trial 1/Response		Trial 2/Response		
1	2 - 9		4 - 6		
2	3 - 8 - 6		6 - 1 - 2		
3	3 - 4 - 1 - 7		6 - 1 - 5 - 8		
4	8 - 4 - 2 - 3 - 9		5 - 2 - 1 - 8 - 6		
5	3 - 8 - 9 - 1 - 7 - 4		7 - 9 - 6 - 4 - 8 - 3		
6	5 - 1 - 7 - 4 - 2 - 3 - 8		9 - 8 - 5 - 2 - 1 - 6 - 3		
7	1 - 6 - 4 - 5 - 9 - 7 - 6 - 3		2 - 9 - 7 - 6 - 3 - 1 - 5 - 4		
8	5 - 3 - 8 - 7 - 1 - 2 - 4 - 6 - 9		4 - 2 - 6 - 9 - 1 - 7 - 8 - 3 - 5		

Digits Forward score (maximum = 16)

Digits Backward		Trial score		Trial score	Item score 0,1 or 2
All ages	Trial 1/Response		Trial 2/Response		
	Sample 8 - 2		Sample 5 - 6		
1	2 - 5		6 - 3		
2	5 - 7 - 4		2 - 5 - 9		
3	7 - 2 - 9 - 6		8 - 4 - 9 - 3		
4	4 - 1 - 3 - 5 - 7		9 - 7 - 8 - 5 - 2		
5	1 - 6 - 5 - 2 - 9 - 8		3 - 6 - 7 - 1 - 9 - 4		
6	8 - 5 - 9 - 2 - 3 - 4 - 2		4 - 5 - 7 - 9 - 2 - 8 - 1		
7	6 - 9 - 1 - 6 - 3 - 2 - 5 - 8		3 - 1 - 7 - 9 - 5 - 4 - 8 - 2		

Digits Backward score (maximum = 14)

Total subtest score (maximum = 30)

13. Mazes

Discontinue after 2 consecutive failures.
For ages 8–16, normal sequence of Mazes 1–3 after partial credit on Maze 4; normal sequence of Sample and Mazes 1–3 after failure on Maze 4.

	Maze	Time limit	Complet. time	Number of errors	Score Circle the appropriate score for each maze						Score
6–7	Sample										
	1	30″						2+ errors 0	1 error 1	0 errors 2	
	2	30″						2+ errors 0	1 error 1	0 errors 2	
	3	30″						2+ errors 0	1 error 1	0 errors 2	
8–16	4	30″						2+ errors 0	1 error 1	0 errors 2	
	5	45″						2+ errors 0	1 error 1	0 errors 2	
	6	60″						2+ errors 0	1 error 1	0 errors 2	
	7	120″					3+ errors 0	2 errors 1	1 error 2	0 errors 3	
	8	120″				4+ errors 0	3 errors 1	2 errors 2	1 error 3	0 errors 4	
	9	150″				4+ errors 0	3 errors 1	2 errors 2	1 error 3	0 errors 4	
	10	150″			5+ errors 0	4 error 1	3 errors 2	2 errors 3	1 error 4	0 errors 5	

Mazes cautions checklist (see WISC-III^{UK} Manual p. 212)
The following cautions should be given, if necessary, but each caution may be given only once during the test.
1. *"You're not allowed to go through a wall."* ☐
2. *"Don't stop. Keep going until you find your way out."* ☐☐
3. *"You're not allowed to start again. Keep going from here* (point to the last place reached) *and try to find the right way out."* ☐
4. *"You should start here"* (point to the centre box). ☐
5. *"You must get right out."* ☐☐

Total subtest score (maximum = 28)

12

Name ..... Date .....

WISC-III^{UK}
Wechsler Intelligence Scale for Children Third Edition UK

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ISBN 0 7491 0205 5

11. Symbol Search

Discontinue after 120 seconds.

	6–7 Part A	8–16 Part B
Time limit	120″	120″
Complet. time		
Number correct		
Number incorrect		
	Max. = 45	Max. = 45
Total subtest score		

(Total score = number correct minus number incorrect)

12. Digit Span

For both Digits Forward and Digits Backward, administer both trials of each item even if Trial 1 is passed.
Discontinue after failure of both trials of any item.
Administer Digits Backward even if Digits Forward score is 0.

Digits Forward		Trial score		Trial score	Item score 0,1 or 2
All ages	Trial 1/Response		Trial 2/Response		
1	2 - 9		4 - 6		
2	3 - 8 - 6		6 - 1 - 2		
3	3 - 4 - 1 - 7		6 - 1 - 5 - 8		
4	8 - 4 - 2 - 3 - 9		5 - 2 - 1 - 8 - 6		
5	3 - 8 - 9 - 1 - 7 - 4		7 - 9 - 6 - 4 - 8 - 3		
6	5 - 1 - 7 - 4 - 2 - 3 - 8		9 - 8 - 5 - 2 - 1 - 6 - 3		
7	1 - 6 - 4 - 5 - 9 - 7 - 6 - 3		2 - 9 - 7 - 6 - 3 - 1 - 5 - 4		
8	5 - 3 - 8 - 7 - 1 - 2 - 4 - 6 - 9		4 - 2 - 6 - 9 - 1 - 7 - 8 - 3 - 5		

Digits Forward score (maximum = 16)

Digits Backward		Trial score		Trial score	Item score 0,1 or 2
All ages	Trial 1/Response		Trial 2/Response		
	Sample 8 - 2		Sample 5 - 6		
1	2 - 5		6 - 3		
2	5 - 7 - 4		2 - 5 - 9		
3	7 - 2 - 9 - 6		8 - 4 - 9 - 3		
4	4 - 1 - 3 - 5 - 7		9 - 7 - 8 - 5 - 2		
5	1 - 6 - 5 - 2 - 9 - 8		3 - 6 - 7 - 1 - 9 - 4		
6	8 - 5 - 9 - 2 - 3 - 4 - 2		4 - 5 - 7 - 9 - 2 - 8 - 1		
7	6 - 9 - 1 - 6 - 3 - 2 - 5 - 8		3 - 1 - 7 - 9 - 5 - 4 - 8 - 2		

Digits Backward score (maximum = 14)

Total subtest score (maximum = 30)

13. Mazes

Discontinue after 2 consecutive failures.
For ages 8–16, normal sequence of Mazes 1–3 after partial credit on Maze 4; normal sequence of Sample and Mazes 1–3 after failure on Maze 4.

	Maze	Time limit	Complet. time	Number of errors	Score Circle the appropriate score for each maze						Score
6–7	Sample										
	1	30″						2+ errors 0	1 error 1	0 errors 2	
	2	30″						2+ errors 0	1 error 1	0 errors 2	
	3	30″						2+ errors 0	1 error 1	0 errors 2	
8–16	4	30″						2+ errors 0	1 error 1	0 errors 2	
	5	45″						2+ errors 0	1 error 1	0 errors 2	
	6	60″						2+ errors 0	1 error 1	0 errors 2	
	7	120″					3+ errors 0	2 errors 1	1 error 2	0 errors 3	
	8	120″				4+ errors 0	3 errors 1	2 errors 2	1 error 3	0 errors 4	
	9	150″				4+ errors 0	3 errors 1	2 errors 2	1 error 3	0 errors 4	
	10	150″			5+ errors 0	4 error 1	3 errors 2	2 errors 3	1 error 4	0 errors 5	

Mazes cautions checklist (see WISC-III^{UK} Manual p. 212)
The following cautions should be given, if necessary, but each caution may be given only once during the test.
1. *"You're not allowed to go through a wall."* ☐
2. *"Don't stop. Keep going until you find your way out."* ☐☐
3. *"You're not allowed to start again. Keep going from here* (point to the last place reached) *and try to find the right way out."* ☐
4. *"You should start here"* (point to the centre box). ☐
5. *"You must get right out."* ☐☐

Total subtest score (maximum = 28)

12

Name ..... Date .....

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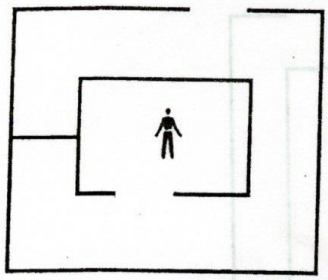
1



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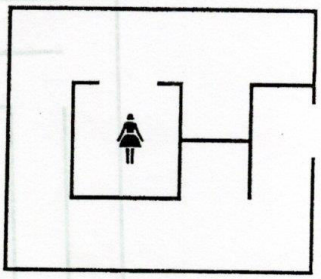

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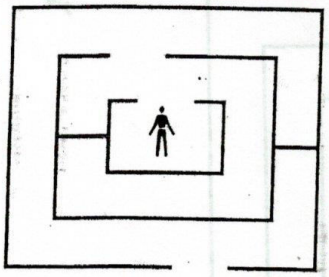

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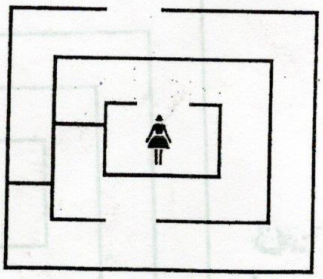

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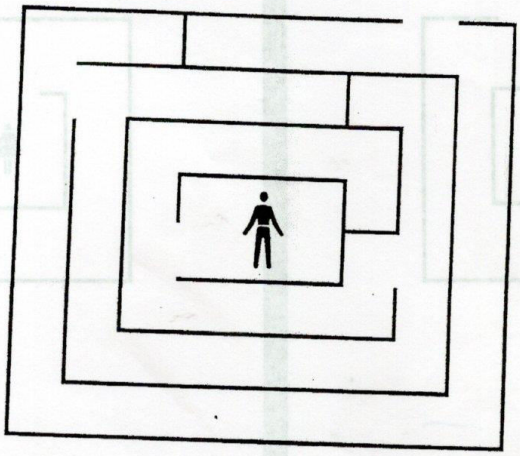

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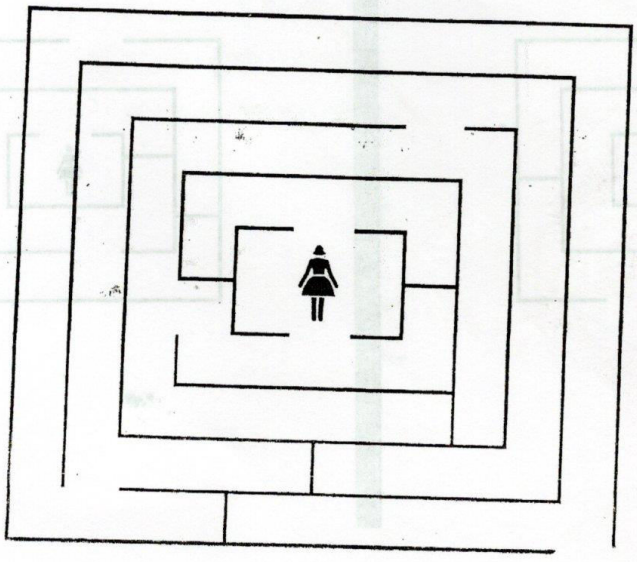
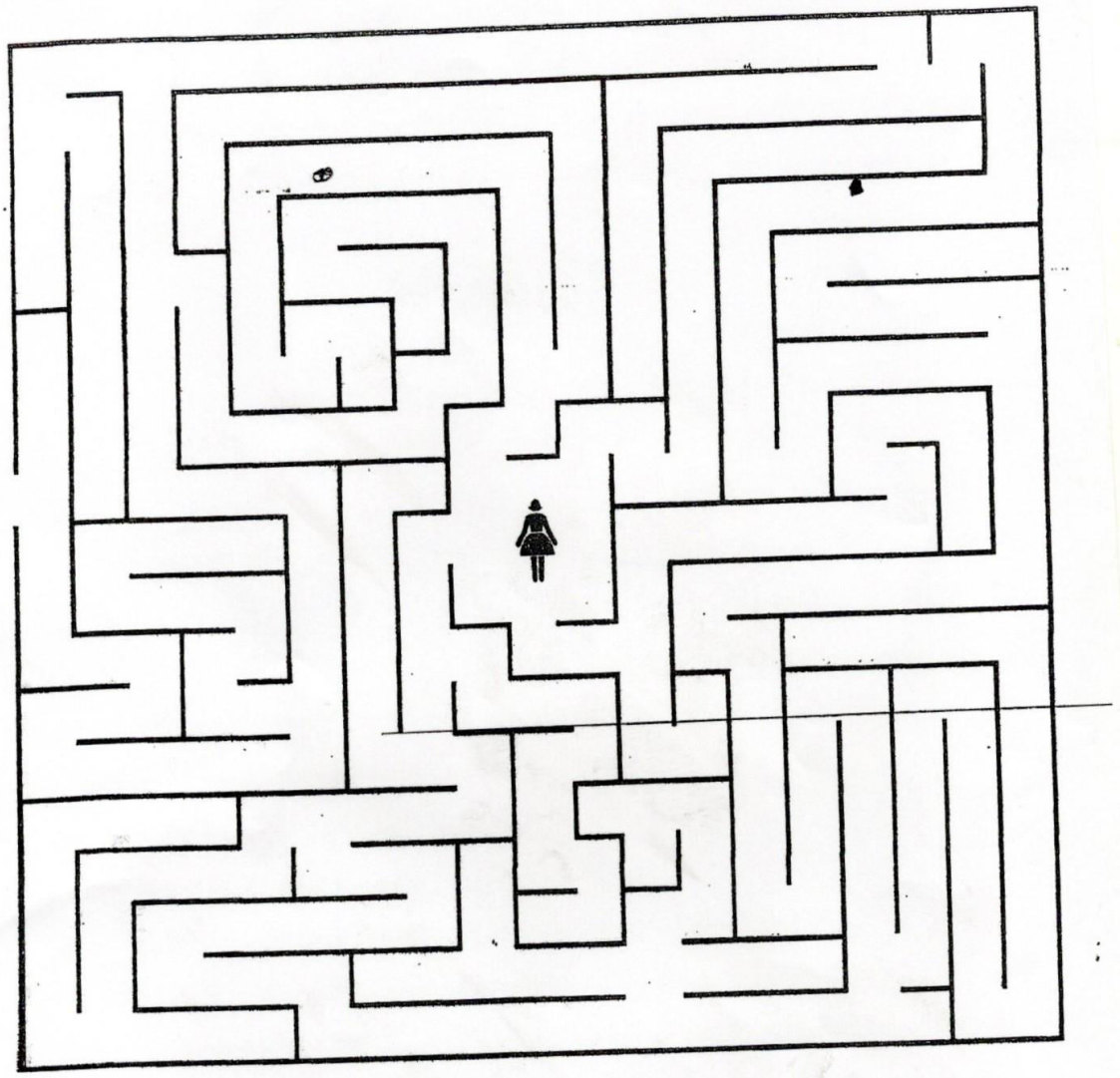


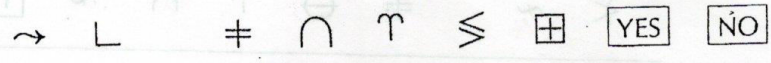


PART B

SAMPLE ITEMS

i

ii



PRACTICE ITEMS

B6

○ ⊕	≩ ↾ ⊖ ≉ ⌋	YES	NO
⊣ ⌊	⊣ ↛ ⊱ ∩ ⊗	YES	NO
∩ ∪	⇒ ⌐ ⊨ ⊞ ⋪	YES	NO
⊗ ≁	⊄ ⊗ ⊔ ‡ ⊖	YES	NO
⊅ ↾	⊪ ⇄ ⊙ ⌋ ⇝	YES	NO
⊱ ⊰	~ ∪ ≩ ≒ ≈	YES	NO
≈ ⊓	∩ ∪ ⌋ ⇜ ⇋	YES	NO
⊀ ≁	≠ ⊖ ↾ ⊓ ≁	YES	NO
⊏ ⊀	⊰ ⊙ ↾ ⊱ ∪	YES	NO
⊞ ~	≒ ⊢ ⊙ ⊞ ⌋	YES	NO
⊀ ⊰	⊱ ⊀ ⊰ ≒ ⊄	YES	NO
⇌ ↜	↛ ≩ ∪ ≁ ⩽	YES	NO
⊙ ⌝	⊣ ⊀ ⊰ ⊅ ≉	YES	NO
⇌ ⊗	⊞ ⊗ ⊀ ⌝ ⇜	YES	NO
⌋ ⊨	⊀ ⊢ ⇜ ⌝ ⇄	YES	NO

20

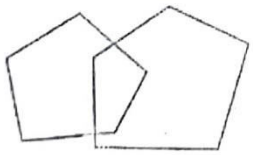
Mini mental status examination

SMART!

MINI MENTAL STATE EXAMINATION (MMSE)

Patient's name:

Hospital number:

ONE POINT FOR EACH ANSWER	DATE			
ORIENTATION				
Year Month Day Date Time	___/5	___/5	___/5	___/5
Country Town District Hospital Ward	___/5	___/5	___/5	___/5
REGISTRATION				
Examiner names 3 objects (eg apple, table, penny) Patient asked to repeat (1 point for each correct). THEN patient to learn the 3 names repeating until correct.	___/3	___/3	___/3	___/3
ATTENTION AND CALCULATION				
Subtract 7 from 100, then repeat from result. Continue 5 times: 100 93 86 79 65 Alternative: spell "WORLD" backwards - dlrow.	___/5	___/5	___/5	___/5
RECALL				
Ask for names of 3 objects learned earlier.	___/3	___/3	___/3	___/3
LANGUAGE				
Name a pencil and watch.	___/2	___/2	___/2	___/2
Repeat "No ifs, ands, or buts".	___/1	___/1	___/1	___/1
Give a 3 stage command. Score 1 for each stage. Eg. "Place index finger of right hand on your nose and then on your left ear".	___/3	___/3	___/3	___/3
Ask patient to read and obey a written command on a piece of paper stating "Close your eyes".	___/1	___/1	___/1	___/1
Ask the patient to write a sentence. Score if it is sensible and has a subject and a verb.	___/1	___/1	___/1	___/1
COPYING				
Ask the patient to copy a pair of intersecting pentagons: 	___/1	___/1	___/1	___/1
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CURRICULUM VITAE

PRIMARY GUIDE BIO-DATA

Name : Dr. SANTOSH RAMDURG

Age (in years) : 44 years

Date of birth : 25/06/1980

Educational Qualification:

Degree	Name of the College	Name of the University	Year of Passing
MBBS	JN MEDICAL COLLEGE, BELGAUM	RAJEEV GANDHI UNIVERSITY OF HEALTH SCIENCES BANGALORE	2003
M.D. PSYCHIATRY	AIIMS DELHI	AIIMS DELHI	2009

Present position : PROFESSOR AND HEAD

K.M.C Registration No : 69081

Teaching Experience : 8 YEARS 10 MONTHS

P.G Guide : 2 YEARS

U.G teacher : 7 YEARS 10 MONTHS

Publications: 22 published articles in international, national and state journals.

Research projects : 6 projects are ongoing.

BIO-DATA
INVESTIGATOR

Name : Dr. SIDDHARTH PATIL

Age : 30 years

Sex : Male

Qualification : M.B.B.S

Year of completion of internship : 2021

Medical Council number : KMC 142833