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NEWSLETTER

INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

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1.A STUDY ON THE DEMOGRAPHIC FACTORS AND AGE OF DIAGNOSIS FOR CHILDREN WITH AUTISM SPECTRUM DISORDERS

Introduction:

Children with autism spectrum disorders (ASD) typically exhibit behavioral and psychological issues. ASD is a challenging neurodevelopmental disorder. Due to their poor adaptive capabilities, these children become upset when their environment changes. Early childhood is when the symptoms first appear, and they have an impact on day-to-day activities. Co-occurring language difficulties, intellectual disabilities, and seizures are more common in children with ASD than in the general population.[1] ASD has developed into a chronic illness that may affect a person's wellbeing and quality of life. [2] In order to establish a scientific underpinning for the early identification, diagnosis, and intervention of autism spectrum disorders (ASD), the current study examined the age of diagnosis, treatment, and demographic variables of Chinese children with ASD.

Methods:

In order to assess their diagnosis, therapy, and basic family information, questionnaires were given to 1500 ASD children aged 2 to 7 from 13 cities. Children with ASD's symptoms and severity were assessed using the Childhood Autism Rating Scale (CARS), and their neurodevelopmental levels were assessed using the Children Neuropsychological and Behavior Scale-Revision 2016 (CNBS-R2016).

Results:

Researchers discovered that for children with ASD, the median (p25, p75) age for the first identification of a social-behavioral developmental delay was 24 (18, 30) months, the age for the first diagnosis was 29 (24, 36), and the age for the start of intervention was 33 (27, 42). Multiple linear regression (MLR) research revealed that the early detection of social behavioral developmental delay occurred later in children with ASD whose parents were divorced, separated, bereaved, or whose mothers were doing physically hard jobs. The age of the initial diagnosis was earlier among ASD children who resided in urbanized areas, had more severe ASD symptoms, or whose parents were not divorcing or living apart. The age for the start of intervention was shown to be earlier in ASD children who resided in cities or whose mothers had higher levels of education, while it was found to be later in ASD children whose mothers worked physically hard jobs.

Discussion:

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The primary aim of this study were to examine demographic parameters that can serve as a guide for the early identification, diagnosis, and intervention of ASD, as well as the age at which children with ASD were diagnosed and treated. Numerous research has recently proven that individual, families, and society are linked to the early identification of ASD, however the majority of the findings are debatable or haven't been further examined.[3] The current study discovered that the age of the initial diagnosis was earlier in ASD children whose parents were not divorcing or separated. These findings could be explained by the fact that stable relationships made it simpler for parents to notice their children's aberrant features and identify ASD symptoms early, allowing for timely treatment.[2]

Items	Age for the Initial Detection (Month)	Z	P	Age for the Initial Diagnosis (Month)	Z	P	Age for the Intervention (Month)	Z	P
Severity of ASD symptoms									
Mild-to-moderate (n=831)	24 (19,30)	−1.948	0.051	30 (24,37)	−2.639	0.008	34 (27,42)	−1.685	0.092
Severe (n=430)	24 (18,30)			28 (24,35)			32 (26,41)		
Comorbidity of DD or not									
Yes (n=720)	24 (20,30)	−1.779	0.075	30 (24,37)	−0.378	0.706	33 (27,42)	−0.949	0.343
No (n=330)	24 (18,30)			30 (24,36)			34 (27,42)		

Note: [2]Group comparisons were conducted using the rank sum tests.
Abbreviations: ASD, autism spectrum disorders; DD, developmental delay.

Relationship Between Age at Diagnosis and Treatment of ASD and Autism Symptom Severity and Diagnosis

Conclusion:

The severity of ASD symptoms and demographic characteristics influence the age at which children with ASD are diagnosed and treated . Missed diagnoses, delayed diagnoses, and delayed interventions for ASD are significant issues. Given the critical importance of early detection, diagnosis, and intervention, the following recommendations are necessary: actively carry out ASD promotion and education; improve people's understanding of ASD; increase the diagnostic and identification rates of domestic developmental-behavioral pediatricians for ASD; conduct ASD screening among children with suspected developmental retardation; strengthen supervision and establish rehabilitation facilities.

To increase their degree of rehabilitation, it is advised to actively carry out health education on ASD and increase support for ASD families.

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2. SCHIZOPHRENIA AND THE FREQUENCY OF DAYTIME NAPPING: A BIDIRECTIONAL TWO-SAMPLE MENDELIAN RANDOMIZATION STUDY

Introduction:

A functional psychotic condition called schizophrenia is characterized by hallucinations, delusional beliefs, and changes in cognition, perception, and behavior. The clinical diagnosis of schizophrenia is only made when a thorough psychiatric history has been taken and all other potential causes of psychosis have been ruled out. Due to potential confounding factors that are challenging to demonstrate by observational epidemiological methods, such as sleep duration, insomnia, daytime sleepiness, and wake-up time, the bidirectional causality between more frequent daytime napping and schizophrenia may be biased. In order to give more reliable data, this study used two-sample Mendelian randomization (MR) to infer the bidirectional causality between more frequent daytime napping and schizophrenia.

Methods:

Summary statistics of the major genetic variants connected to the frequency of daytime napping and schizophrenia from genome-wide association studies were used in a bidirectional two-sample Mendelian randomization (MR) study. Daytime napping frequency GWAS single nucleotide polymorphisms (SNPs) data came from the UK Biobank (n = 452,633) and 23 and Me study cohort (n = 541,333), whereas schizophrenia GWAS data came from the Psychiatric Genomics Consortium. The major method used was the inverse variance weighted (IVW) analysis, with sensitivity analysis using the weighted median, MR-Robust Adjusted Profile Score (RAPS), Radial MR, and MR-Pleiotropy Residual Sum Outlier (PRESSO).

Results:

The results of the MR analysis demonstrated a bidirectional causal relationship between more frequent daytime napping and the likelihood of developing schizophrenia. The odds ratio (OR) for a one-unit increase in napping category (never, sometimes, usually) on schizophrenia was 3.38 (95% confidence interval [CI]: 2.02–5.65, P = 3.58 × 10– 6), and the beta for the relationship between the frequency of daytime naps and the likelihood of developing schizophrenia was 0.0112 (95%CI: 0.0060–0.0163, P = 2.04 × 10– 5). The same results were found by the sensitivity analysis.

Discussion:

Sleep abnormalities are a feature of schizophrenia and are linked to more severe psychotic symptoms and poor clinical outcomes.[2] Insomnia, nightmares, poor sleep hygiene, and other sleep abnormalities are common in schizophrenia patients and can exacerbate mental illness symptoms like hallucinations, delusions, and cognitive decline.[3] While daytime napping can effectively predict nocturnal sleep spindle density in schizophrenia patients, studies have shown that patients with schizophrenia have problems in the sleep spindle.[4,5] The current study findings might serve as a foundation for daytime napping in schizophrenia intervention and therapy.

Exposure	Outcome	SNPs (n)	Cochran's *Q* *P*-value	MR-Egger pleiotropy test *P*-value	Methods	OR (95% CI)	*P*-value
Daytime Napping Frequency	Schizophrenia	64	1.04E-19	0.27	IVW	3.48(1.54,7.85)	2.65E-03
					Weighted median	3.30(1.52,7.15)	2.56E-03
					MR-RAPS	4.50(2.92,6.93)	8.50E-12
					MR-PRESSO	3.68(1.65,8.20)	2.19E-03
					Radial MR	3.67(1.65,8.18)	1.48E-03
Daytime Napping Frequency (Without outliers)	Schizophrenia	47	0.154	0.68	IVW	3.38(2.02,5.65)	3.58E-06
					Weighted median	3.49(1.67,7.27)	8.57E-04
					MR-RAPS	3.55(2.18,5.78)	3.67E-07
					MR-PRESSO	3.38(2.02,5.65)	2.97E-05
					Radial MR	3.37(2.01,5.63)	3.81E-06

Causal estimates for daytime napping frequency on schizophrenia by Mendelian randomization. Abbreviations: SNP, single nucleotide polymorphism; IVW, inverse variance weighted; MR-RAPS, Mendelian randomization robust adjusted profile score; MR-PRESSO, Mendelian randomization pleiotropy residual sum and outlier

Conclusion:

The outcomes of this two-sample Mendelian randomization study with a bidirectional design suggested a bidirectional causal relationship between more frequent daytime napping and the development of schizophrenia, suggesting that increasing the frequency of daytime napping may be a potential intervention for the progression and treatment of schizophrenia.

There is a bidirectional causal relationship between more frequent daytime napping and schizophrenia, suggesting that increasing the frequency of daytime napping may be a therapy option for schizophrenia.

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4. A FOLLOW-UP STUDY ON SLEEP AND PSYCHIATRIC SYMPTOMS IN YOUNG CHILD PSYCHIATRIC OUTPATIENTS

Introduction:

Sleep plays an important role in healthy physical and mental development. It is especially crucial for young brains since it helps regulate emotions, regulate behavior, and build cognitive abilities.[1]Although studies imply a bidirectional relationship between sleep disorders and psychiatric symptoms as well as an obvious connection between children's sleep and emotional and behavioral development, investigations of child psychiatric patients' shorter sleep durations have not being published frequently. In the future, it would be helpful to have a clearer understanding of the frequency and relationship between sleep issues and the severity of psychiatric symptoms. This could lead to the development of more effective treatments for pediatric patients with psychiatric conditions.[2] The goal of this study was to learn more about how sleep at a young age predicts sleep problems and psychiatric symptoms at a later age in children with mental health issues.

Methods:

Participants (n = 68) were University Hospital outpatients in child psychiatry over a period of two years. Age 4 to 7 years, Finnish-speaking parents, and daycare attendants were requirements for inclusion of the children in the baseline sample. During baseline (ages 4–7 years) and again at follow-up, caregivers assessed sleep with the Sleep Disturbance Scale for Children (SDSC) and psychiatric symptoms with the Child Behavior Checklist (CBCL). Both times, background data on the family were collected

Results:

Sleep problems in early childhood predicted sleep issues in later life (R2Adjusted =.48, p <.001). Persistent sleep issues were significantly correlated with the severity of psychiatric symptoms (p =.001). Even after adjusting for age, sex, and psychiatric symptoms at preschool age, sleep issues were still associated with internalizing symptoms (p =.038).

Discussion:

The outcomes of this study imply that preschool sleep issues are related to psychiatric symptoms in children who received mental health care at a young age. After adjusting for age, sex, and T1 mental symptoms, preschool sleep issues were found to predict internalizing problems at school age. Even after further adjusting for parents' educational backgrounds, the finding was remained close to statistical significance. Only a small number of prior studies have investigated at potential connections between sleep-related issues and the severity of psychiatric symptoms in pediatric patients.[2] In the study by Bai et al., it was discovered that among children treated for pediatric anxiety disorders, having higher levels of dysregulated sleep and more severe internalizing symptoms during the duration of a 4-year follow-up increased the likelihood of poor mental health. However, compared to the present study sample, theirs was older on average (11-26 years, median age). Despite receiving anxiety/depression medication, a bidirectional relationship was seen in adolescence and young adulthood.[3]

		Mean	SD	Subclinical range, n(%)	Clinical range, n(%)
T1 (4–7 years)	Total problems	61.7	12.8	6 (8.8)	32 (47.1)
	Internalizing problems	60.0	13.0	11(16.2)	28 (41.2)
	Externalizing problems	61.9	12.8	6 (8.8)	31 (45.6)
T2 (8–13 years)	Total problems	62.4	12.1	3 (4.4)	38 (55.9)
	Internalizing problems	62.2	11.4	6 (8.8)	37 (54.4)
	Externalizing problems	59.8	12.4	7 (10.3)	30 (44.1)

Descriptive statistics of T-scores of Child Behavior Checklist, (total, internalizing, and externalizing scale scores) in child psychiatric outpatients (n = 68) at time 1 and 2.

Conclusion:

This study emphasizes the need of identifying and treating sleep issues in young child psychiatric patients since these issues may exacerbate or contribute to psychiatric symptoms. Severe psychiatric issues are linked to sleep issues, especially those that are persistent. As their treatment needs may

differ from those of their normative peers, it is critical to develop sleep problem treatments particularly for pediatric patients.

Children treated in child psychiatry clinics frequently and persistently experience sleep issues, which are linked to psychiatric symptoms.

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4.A CASE REPORT OF OBSTRUCTIVE SLEEP APNEA AND MAJOR DEPRESSIVE DISORDER CO-OCCURRING

Introduction:

The World Health Organization (WHO) estimated that by 2030, major depressive disorder (MDD) will be the leading cause of disease burden worldwide.[1] Obstructive sleep apnoea (OSA) and major depressive disorder (MDD) are common in the general population. Early research also revealed a bidirectional relationship between the two diseases and negative health outcomes. People who also have concomitant MDD have a greater incidence of OSA. As many as two thirds of OSA patients may also have concomitant MDD. In the age of globalization, a sedentary lifestyle and psychological stress may raise the risk of MDD and OSA.[2] In this case presentation, the assessment and treatment of a patient with concomitant OSA and MDD are the main topics.

Case Report:

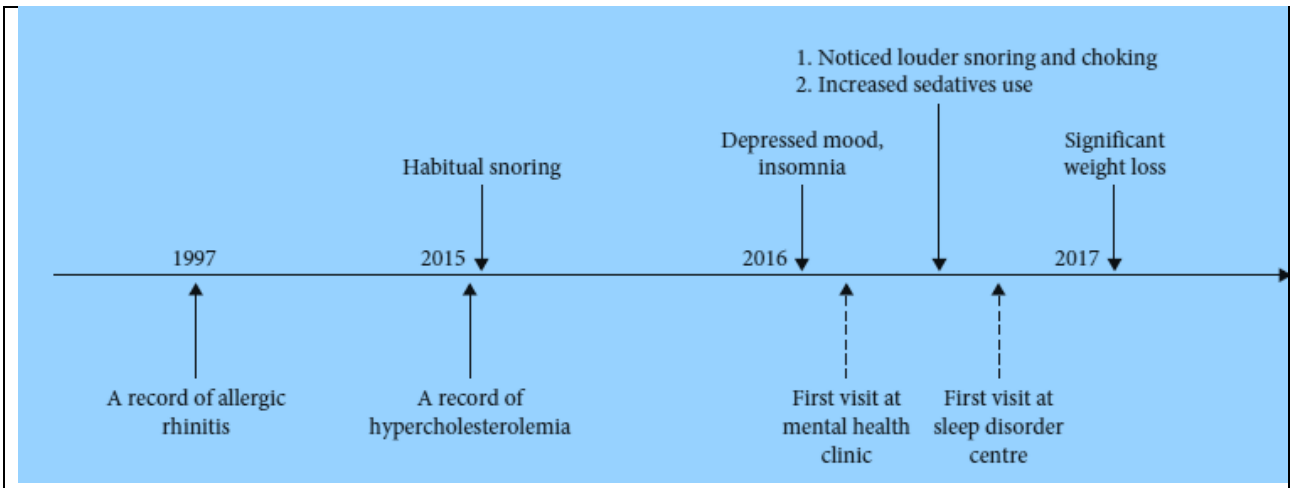
The patient, a 30-year-old single man, had a history of hypercholesterolemia and allergic rhinitis (AR). His mother became aware of his loud snoring two years prior to the initial visit to the psychiatry health facility. Although he had nasal congestion, there was no choking, grasping. After waking up in the morning, the patient had a dry throat, a clear nasal drip, and a headache. Despite getting 8 to 9 hours of sleep, he felt tired during the day because he didn't sleep during the day. He did not consume any tonics or beverages containing caffeine. But almost every day, these symptoms were present. He did not experience hypnagogic or hypnopompic hallucinations, periodic limb movement during sleep, parasomnia, sleep paralysis, or cataplexy. The patient had severe stress six

months prior to his first appointment at the mental health facility due to relocating to a new location for employment. He was disheartened. He didn't want to leave the house or go to work. The patient used to walk for 40–60 minutes every day in the evening as exercise, but he no longer does it. He was unable to concentrate, was anxious about going to work, and kept blaming himself for changing jobs. Additionally, he ate more frequently and put on 4 kg in a month when under stress. Suicidal thoughts and manic or hypomanic episodes were denied by him. He was experiencing both acute and chronic insomnia. The more he slept, the more nervous and restless he felt. The signs of insomnia increased in frequency until they were eventually experienced virtually daily. So he made an appointment with a psychiatrist. He was prescribed 10 mg of escitalopram and 2 mg of clonazepam after being diagnosed with major depressive disorder and anxious discomfort. His mood slightly improved after taking the drugs consistently for two months. At first, he was able to sleep, but he continued to struggle with fragmented sleep, frequently waking up during the night. The patient felt like though he had been choking all night. He also noted that his snoring had increased in volume. It would take him longer than an hour to fall back asleep if he woke up in the middle of the night. This increased the patient's concern that if he didn't get enough sleep, he would become sleepy, lose attention, and be unable to perform at work. The patient continued to take an additional 2- 4 mg of clonazepam daily on his own, but noticed drowsiness the following day and a need for a nap between 5 and 6 pm as a side effect. His psychiatrist suggested him visit a sleep disorder clinic to assess any additional sleep issues. The patient often went to bed at 11:00 pm and woke up at 7:00 am on workdays, but on vacations, he went to bed at 12:00 am and woke up at 10:00 am. The patient typically played on his or her phone and watched television before bed, with a 20-minute sleep latency. He always remains in bed when he is unable to sleep. The sleep specialist evaluated the patient. A score of 3 on the STOP-BANG Sleep Apnea Questionnaire indicated an intermediate risk for OSA. His upper airway evaluation revealed that he had overbite malocclusion, Friedman tongue position III, tonsillar grade 1, swelling in both inferior turbines, and clear nasal discharge. The sleep specialists offered to perform an overnight polysomnography because they suspected OSA, but he never followed up with the sleep disorder centre. He continued taking 4-6mg of clonazepam and 10mg of escitalopram every day. The patient experienced polyphagia, unintentional weight loss (5 kg/month), polydipsia, polyuria, and nocturia six months later. He went to see an internist as a result. The HbA1C mg/dL and fasting blood sugar were 190 and 12.7%, respectively. He had glucosuria as well. He was confirmed to have type 2 diabetes. He eventually received a liraglutide injection along with oral

antihyperglycemic drugs, and he was also instructed to go to the sleep disorder centre and continue to visit the psychiatrist. Finally, he was assessed to have severe OSA with mild oxygen desaturation due to an AHI of 45.7 occurrences per hour and a nadir oxygen desaturation of 93%. The continuous positive airway pressure (CPAP) setting of 10cmH2O was proposed by the positive airway pressure (PAP) titration to normalise the respiratory disturbance and arousal indices. Additionally, snoring was eliminated, and the oxygen saturation level was maintained at or above 95%.

Discussion:

The case exemplifies the co-morbidity of type 2 DM, OSA, and MDD. There may be clinical overlap between MDD and OSA. Both illnesses, which were left untreated, contributed to or were the cause of a newly appearing medical condition, in this case type 2 DM. The patient's symptoms were unquestionably consistent with MDD with apprehensive anxiety. However, in this scenario, a doctor should be suspicious of concomitant OSA due to the patient's severe snoring, moderate insomnia, nocturia, morning headache with a dry throat, and his family history.[2] Additionally, a comprehensive physical examination is necessary to find OSA risk factors like obesity, hypertension, diabetes, respiratory, cardiovascular, and neurological abnormalities as well as signs of upper airway narrowing.[3] Finally, to confirm the OSA diagnosis, a confirmatory test is needed. According to recommendations from the American Academy of Sleep Medicine (AASM), the test comprises of either a PSG or a home sleep apnea test (HAST).[4,5]



Timeline of essential medical history

Conclusion:

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It's important to recognize that depression and OSA frequently coexist. Additionally, a bidirectional association exists between the two conditions. For effective patient results, a thorough assessment is necessary to ascertain the diagnoses and concurrent treatments for both illnesses.

Early diagnosis and treatment of major depressive disorder and obstructive sleep apnoea are essential to obtain positive patient results.

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