REVIEW ARTICLE

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Autism spectrum disorder in patients with Dravet syndrome

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Abstract

Introduction and Objective. Dravet syndrome (DS) is a condition associated with a genetic mutation that causes epileptic seizures resistant to classical treatment. Symptoms may also co-occur that overlap with features of the neurodevelopmental disorder Autism Spectrum Disorder (ASD). The aim of the study is to demonstrate the relationship between the occurrence of Dravet syndrome and autism, and to provide suggestions relating to the cause of the co-occurrence of these disorders.
Review Methods. An evaluation was conducted of the literature available in the PubMed and Scopus databases Articles published between 2018 –2024 were searched using the phrases 'epilepsy', 'autism spectrum disorder', 'autism', 'pathogenesis', 'case', 'Dravet syndrome', 'child', 'prevalence', 'epidemiology', 'therapy', 'treatment', 'symptoms', 'causes', and 'genetics'.
Brief description of the state of knowledge. Epilepsy is a neurological disorder characterized by recurrent seizures and is correlated with ASD. The relationship between these disorders is incompletely understood. Similarly, it has been shown that Dravet syndrome can have symptoms that overlap with or even co-occur with features of autism. There are several hypotheses explaining this finding. A *de novo* mutation in the *SCN1A* gene is most often responsible for the onset of DS, which could indicate behavioural disorders and autistic behaviour, among other problems associated with this disease entity.
Summary. Dravet syndrome not infrequently manifests with complaints that may resemble ASD. There are a number of assumptions that explain the reason for the correlation between DS and ASD, based primarily on the pathophysiology of Dravet syndrome.

Key words

Dravet syndrome, epilepsy, autism spectrum disorder

INTRODUCTION AND OBJECTIVE

Dravet syndrome (DS) is a genetically determined, severe childhood epileptic encephalopathy characterized by drug-resistant, recurrent seizures [1, 2]. Patients with this syndrome often also struggle with other motor and cognitive dysfunctions, including behavioural problems [3]. Behavioural problems also occur in patients with Autism Spectrum Disorder (ASD), which is a neurodevelopmental disorder. It is worth noting that autism spectrum disorders are more common in Dravet syndrome than in the general population [4].

Epilepsy co-occurs in some patients with ASD, and the specific risk values depend on its type. This may be related to autism risk factors, among which epilepsy, duration, and frequency of seizures are mentioned [5].

The aim of this study is to draw attention to the co-occurrence of autism spectrum disorders among patients with Dravet syndrome.

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REVIEW METHODS

Between December 2024 – April 2025, the online databases PubMed and Scopus were searched. In the first stage, free full texts published in English in the years 2018–2024 were analyzed. Articles were searched using various combinations of the following key words: 'epilepsy', 'autism spectrum disorder', 'autism', 'pathogenesis', 'case', 'Dravet syndrome', 'child', 'prevalence', 'epidemiology', 'therapy', 'treatment', 'symptoms', 'causes', and 'genetics'. Articles that did not fit the subject of our work topic being investigated were excluded. In the second stage, the remaining papers were studied, and 39 that contained key information on DS, ASD, epilepsy, and correlations related to them were selected.

DESCRIPTION OF THE STATES OF KNOWLEDGE

Dravet syndrome (DS). A rare genetic disorder that affects 1 in 16,000 – 40,000 live births [6]. It is a severe childhood epileptic encephalopathy, characterized by drug-resistant, recurrent seizures. This epilepsy is incurable, and its onset may already occur before the first year of life [1, 2]. Triggers of seizures include infections, fever, ambient temperature, sunlight, and exercise [7, 8].

A characteristic feature of epileptic seizures in Dravet syndrome is their multiplicity. In infancy, alternating

unilateral or bilateral tonic-clonic febrile or afebrile seizures are observed. Between the first and fourth years of life, atypical absence of seizures and myoclonic seizures additionally develop [4]. In the first 5 years, status epilepticus is common. In the following years of life, the frequency of seizures decreases, and the older the child, the more often nocturnal seizures are observed. They are the main type of seizures in adults [9].

In patients, the increased risk of death (nocturnal seizures, status epilepticus – SE), and the most common cause, Sudden Unexpected Death in Epilepsy (SUDEP)) must be taken into account. Mortality in patients with DS ranges from 3.7% – 20.8% [9–11].

The most common cause of the syndrome is a *de novo* mutation in the voltage-gated sodium channel gene *SCN1A*, which causes haploinsufficiency of Nav1.1, the alpha-1 subunit of the sodium channel [2, 6]. In some cases, the *SCN1A* mutation can also be detected in parents [3]. In the case of familial occurrence, it is worth considering the presence of mosaicism of a given variant in the parent, which is found in up to 10% of patients suspected of *de novo* mutations, and may affect the severity of disease symptoms [12]. Mutations in the *SCN1A* gene cause a decrease in sodium current in inhibitory GABAergic interneurons which, in turn, leads to neuronal hyperexcitability and seizures. Purkinje cells are also sensitive to a decrease in sodium current, and as a result, motor and cognitive dysfunctions are created, including behavioural problems [3]. Therefore, Dravet syndrome may be accompanied by behavioural disorders, autistic behaviour, cognitive deficits, developmental delay, sleep and gait problems (crouch gait) [2–4].

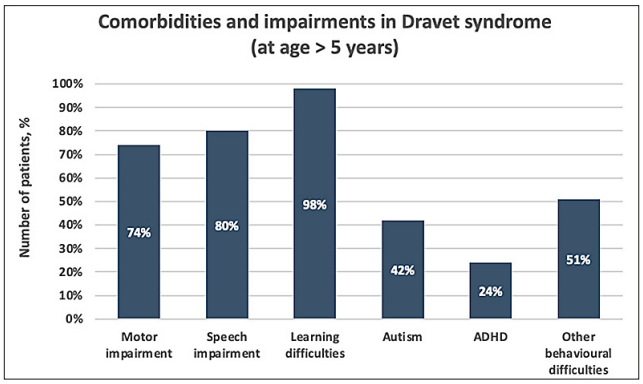

Figure 1. Distribution of comorbidities and additional impairments present in Dravet syndrome in patients older than 5 years [13].
ADHD – Attention Deficit Hyperactivity Disorder

The majority of people with ADHD meet the criteria for intellectual disability (ID), the pooled prevalence of which is estimated at 86% based on the analysed studies. The degree of disability can be varied, vary from mild to profound [14]. Children with significant ID present a greater number of autistic features [15]. ID, ASD, and epilepsy are correlated, and it is estimated that the occurrence of ID may be associated with a more than 7-fold increase in the risk of developing ASD [5 There are also indications that there is a significant frequency of co-occurrence of adaptive disorders in Dravet syndrome, which are also observed more often in children with ASD [14].

Considering the relatively frequent occurrence of behavioural disorders in Dravet syndrome, the role of screening tests in children, as well as appropriate diagnostics at an early stage, i.e., in the case of ASD, from the age of 2 years, should be emphasized [15]. Early diagnosis gives a chance to avoid the administration of contraindicated drugs, e.g., sodium channel blockers, while implementing treatment with an effective regimen of antiseizure medication (ASM) [10].

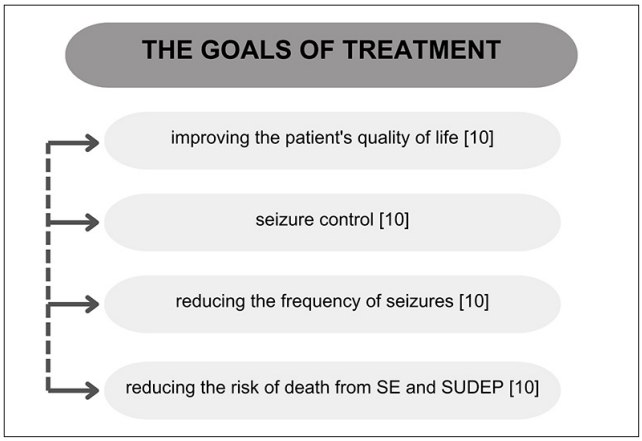

Figure 2. Th aims of DS treatment.
SE – status epilepticus, SUDEP – Sudden Unexpected Death in Epilepsy

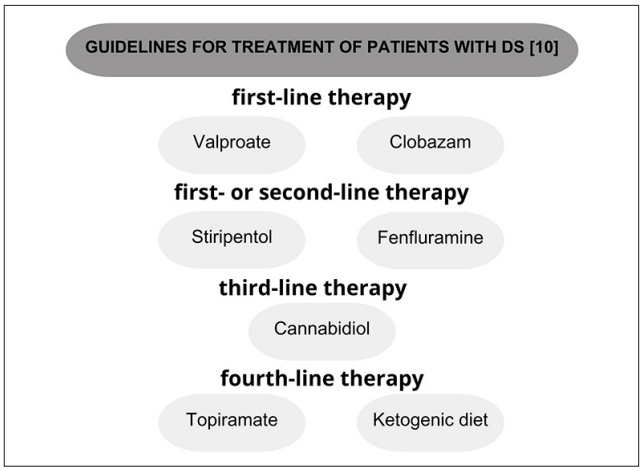

Figure 3. Guidelines for treatment of patients with DS (United States and European consensus recommendations).
DS – Dravet syndrome

Autism Spectrum Disorder (ASD). Belongs to the group of neurodevelopmental disorders. A person with this disorder shows characteristic features: interpersonal communication disorders, repetitive behaviours, a limited number of interests, as well as disorders in sensory integration [16, 17].

Factors causing ASD include genetic, prenatal, perinatal, and postnatal factors [16], with other factors such as infertility [18], genetically determined production of IL-6 [19], use of antibiotics in very early life in children, long-term use of antibiotics [20], or use of antibiotics by the mother during pregnancy [21], are also taken into account in the latest publications.

According to WHO data, it is estimated that 1 in 100 people have autism, although this number may be higher due to insufficient data from low- and middle-income countries [16]. A study by M. Solmi shows that the median age of diagnosis of ASD among children is 9 years [22], and in order to make

Table 1. Criteria of neurodevelopmental disorder based on DSM-5 [17]

Neurodevelopmental disorder	
Sub-criteria	Persistent deficits in social communication and interaction in various situations. Restricted and repetitive behaviour patterns, interests, or activities.
Needed to diagnose	Dyad: 2/2 diagnostic criteria must be met.
Diagnostic criteria	Persistent deficits in social communication and interaction across a variety of situations, manifested by: Deficits in social-emotional reciprocity (including abnormal social approaches, failure to participate in reciprocal conversations, reduced sharing of interests, feelings, or emotions, and inability to initiate or respond to social interactions). Deficits in non-verbal communication behaviours used in social interactions (poor verbal and non-verbal communication integration, abnormalities in eye contact, gestures, and body language). Deficits in developing, maintaining, and understanding relationships (including adapting behaviour to different social situations, difficulties sharing imaginative play or making friends, or a lack of interest in peers). Limited and repetitive behaviours, interests, or activities that are displayed by at least two of the following: Stereotyped or repetitive motor movements, use of objects, or language. Insistence on uniformity, adherence to routines, or ritualized verbal or non-verbal behaviour patterns. Markedly restricted or fixed interests of unusual intensity or focus. Hyper- or hyporeactivity to sensory stimuli or an uncommon hobby in sensory elements of the environment.
Age at development	Symptoms must be present early in development, but may only emerge when social demands exceed limited capabilities, or are masked by learned strategies.

DSM-5 – Diagnostic and Statistical Manual of Mental Disorders

a diagnosis of ASD, the patient must meet specific criteria (Tab. 1).

There are documented attempts to treat autism symptoms with a gluten-free diet [23], medical marijuana [24], or probiotics [25]. However, the research results are inconclusive, and further investigation is needed.

Epilepsy. A neurological disease characterized by constant exposure to seizures that can lead to permanent damage to the central nervous system or body. The disease arises from an imbalance of excitation and inhibition in the brain, either through a breach in the continuity of neurons or dysfunction of neurotransmitters responsible for excitation and inhibition processes [26]. Epilepsy is a condition that often co-occurs with other neurodevelopmental disorders, such as autism. Despite different pathophysiology, individuals with ASD are more likely to have epileptic seizures than the general population [27].

The etiology of the co-occurrence of the two neurological disorders is not definitively clear. At the same time, there are some hypotheses suggesting that epilepsy, together with ASD, is due to defects in GABA-A neuron receptors or immune dysregulation [28]. The incidence of ASD in patients with epilepsy is estimated at 6.3%, with the risk varying by type of epilepsy [5] (Tab. 2).

It has now been shown that epilepsy with early onset may contribute to the onset of autism in children. Both of these neurological disorders may be linked to reduced brain plasticity through an impaired excitation-inhibition ratio.

Table 2. ASD risk in epilepsy (divided by type) [5]

Type of epilepsy	Risk of ASD
Generalized	4.7%
Infantile spasms (West Syndrome)	19.9%
Focal seizures	41.9%
Dravet Syndrome	47.4%

ASD – Autism Spectrum Disorder

Table 3. Key statistics characterizing Autism Spectrum Disorder and Dravet syndrome

Global prevalence of ASD	0.6%	[35]
	650/100,000	[36]
Male-to-female ratio in ASD	4.2	[36]
ASD prevalence in Dravet syndrome	22–46%	[14]
Number of patients with comorbidities or additional impairments in Dravet syndrome	91% of all patients older than 5 years	[13]

ASD – Autism Spectrum Disorder

Seizures in early infancy may disrupt neuronal plasticity and thus predispose to ASD [27].

Studies of brainwave activity in children with ASD have raised the possibility that this hypothesis is true. It is not uncommon for children with autism to have an abnormal EEG recording, even without a clinical picture of epilepsy [27]. It has been hypothesized that inadequate EEG results contribute to behavioural disorders. However, despite the hypotheses related to the pathogenesis of ASD and epilepsy, the emergence of cognitive impairment related to excitation/inhibition imbalance is more complicated due to the ineffectiveness of anticonvulsant treatment. However, positive effects of treatment with valproic acid or levetiracetam have been demonstrated in patients with ASD and co-occurring seizures, or abnormal electroencephalograph (EEG) results. Nevertheless, some therapies cover co-occurring disorders such as ASD and epilepsy. An example of such non-pharmacological therapy is the ketogenic diet used to treat epilepsy, among other disorders. The effects that are achieved in the nervous system with this diet suggest that the ketogenic diet can also be used in ASD [27]. In summary, it is still uncertain whether epileptic seizures or abnormal EEGs are the cause of or co-exist with ASD [29].

Moreover, subclinical electroencephalographic EEG abnormalities, often present in patients with ASD, not infrequently correlate with lower intellectual functioning in these patients. The mere presence of full-blown epilepsy in patients with autism is associated with more severe symptoms of ASD [30].

The publication suggests that adenosine, which is an endogenous anticonvulsant drug, affects both epilepsy and autism. By regulating neuronal excitability and inhibiting DNA methylation, adenosine inhibits the development of epilepsy. It is also an important neuromodulatory agent and has been shown to have an effect on symptoms seen in ASD [29].

Autism and epilepsy are closely correlated disorders. By gender and age, they are particularly linked in female epilepsy and early-onset epilepsy [31].

In addition, the brain shows sexual dimorphism in terms of function and structure, as well as in various neuropsychiatric disorders. ASD in the female gender manifests itself with

Table 4. Selected studies on the comorbidity of Dravet Syndrome with other disorders (years 2017–2024) [13–15,37–39]

Author	Type of work	Year	Population	Age	*SCN1A* mutation prevalence	ASD	Supplementary results
Reilly C et al. [15]	Original work, population-based study	2024	41 children with DS Population: Sweden	<18 years	95%	ASD was diagnosed in 25 assessed children (61%) using DSM-5 criteria	Severe ID was strongly linked to increased severity of ASD features (p<0.001)
Jansson JS et al. [14]	Systematic review (29 studies)	2020	1,291 patients with DS Population: NA	4 months – 60 years	Pooled prevalence 93% (range 58–100%)	ASD comorbidity: – mean prevalence: 31% (range 0–67%) – prevalence in studies using ASD-specific instruments: 22–46% – prevalence in studies utilizing standardized 'gold standard' autism assessment tools, i.e. Autism Diagnostic Interview-Revised (ADI-R) and Autism Diagnostic Observation Schedule (ADOS-2): 22–39%	Prevalence of behavioural difficulties in individuals with DS (range: 43–100%) Pooled prevalence of ID was observed in 86% of patients (range 67–100%): – 8% within normal range (IQ >85); – 6% within borderline range (IQ 70–84); – 22% had mild ID (IQ 50–69) – 26% had moderate ID (IQ 35–49); – 38% had severe to profound ID (IQ <35).
Brown A et al. [37]	Original work, cohort study	2020	45 patients with DS and their primary caregivers Population: Australia	2–30 years	NA	ASD co-occurrence: – 22% of patients with DS (10 out of 45 participants) had a formal diagnosis of ASD – The Social Communication Questionnaire (SCQ) identified social communication deficits in 67% of participants (18 out of 27), with 10 patients at risk for ASD. A preserved ability to distinguish basic emotions in a social perception test indicated that certain social-cognitive skills remain intact.	Behavioual disorders: – An assessment of 36 caregivers was conducted using the BASC-2 scale. – The most common issues were attention problems, affecting 65% of the participants, and atypical behaviour resembling autism. observed in 70% of patients – Additionally, 58% of participants exhibited deficits in social skills. Co-occurrence of ID: – An assessment of 38 participants was carried out using the FSIQ scale. Reliable results were obtained for 28 participants. while 10 did not complete the full assessment. Among participants: – 8% (3 out of 38) were within the normal range (IQ >80); – 11% (4 out of 38) were within borderline range (IQ 70–79); – 29% (11 out of 38) had mild ID (IQ 50–69); – 26% (10 out of 38) had moderate ID (IQ 40–49); – 26% (10 out of 38) had severe to profound ID (IQ <40). This estimate was applied to participants who did not complete the full FSIQ assessment. – Intelligence tends to decrease with age.
Ouss L et al. [39]	Original work, cohort study	2018	35 patients with DS Population: NA	24 months – 7 years	77%	The following assessments were conducted: DSM-5 criteria, ADI-R tools, and ADOS-2. – 39% of patients (11 out of 30) were diagnosed with ASD, while 7% (2 out of 30) had Social Communication Disorder (SCD). A specific ASD profile characterized by relatively preserved social skills, was observed in children with DS. Co-occurrence of ASD and Dravet Syndrome was associated with greater impairments in several areas, including: – direct gaze (p=0.004); – range of facial expressions (p=0.04); – imaginative group play (p=0.01); – interest in interacting with other children (p=0.001); – showing attention (p<0.001).; – sharing enjoyment (p=0.02); – appropriate social response (p=0.01); – use of others' body in play (p=0.03);	– Abnormal development on the PEP-3 scale was found in 97% of children (29 out of 30 examined). – Adapted emotional expression on the PEP-3 was found in 57% of children (17 out of 30 examined). – Communication skills were partially preserved. although social behaviour was qualitatively unadapted. Co-occurrence of ID was observed in 77% of the children (23 out of 30 examined). – A higher level of cognitive impairment was observed in children with both DS and ASD.

Table 4. Selected studies on the comorbidity of Dravet Syndrome with other disorders (years 2017–2024) [13–15,37–39] (continuation)

He N et al. [38]	Original work, cohort study	2018	95 patients. including 45 with DS. Population: China	2–16 years (average 8,2 years)	NA	ASD was diagnosed in 22% of children with DS (10 out of 45 examined). using DSM-5 and ICD-10 criteria.	Co-occurrence of ID: – Assessment was performed using the Chinese Wechsler Intelligence Scale for Children (C-WISC) – scale for children from 6 years of age, and the Gesell developmental scale for children up to 6 years of age – ID was identified in 91.1% of patients with DS. – Mild ID (IQ 55–70) was observed in 11.1% of patients with DS. – Moderate to severe ID (IQ 25–54) was observed in 66.7% of patients with DS. – Profound ID (IQ <25) was observed in 13.3% of patients with DS. – Risk factors for ID include an early age of onset and a symptomatic etiology
Lagae L. et al. [13]	Original work, multi-national cohort study	2017	584 caregivers of DS patients Population: – Italy (14%). – UK (12%). – Germany (12%). – France (11%). – Spain (10%). – Netherlands (10%). – Poland (7%). – other European (16%). – rest of the world (8%)	From <1 year – 48 years. (average 10,6 years) – 6% Infants (I): < 2 years – 24% Pre-school (PS): 2–5 years – 35% Middle childhood (MC): 6–11 years – 18% adolescent (A): 12–17 years – 17% Adult (Ad): ≥18 years	NA	Co-occurrence of ASD in 33.6% of patients with DS: – I - 0%. – PS - 17.0%. – MC - 39.1%. – A - 51.4%. – Ad - 38%	Almost all (99.6%) patients aged ≥5 years had at least one or more motor-learning, speech and behavioural disorders. Behavioural disorders were observed in 45.5% of patients with DS: – I - 0%. PS - 17.0%. MC - 39.1%. A - 51.4%. Ad - 38% Speech disorders were found in 64% of patients with DS, and 14.9% were non-speaking: – I - 32.4% (23.5% with no speech). PS - 63.1% (17% with no speech). MC - 71.8% (11.9% with no speech). A - 67.3% (11.2% with no speech). Ad - 57% (19% with no speech) Motor disorders were present in 71.7% of patients with DS: – I - 55.9%. PS - 68.1%. MC - 77.2%. A - 68.2%. Ad - 75% Only 7.5% of patients with DS had no other comorbidities or impairments: – I - 2.9%. PS -3.5%. MC - 8.9%. A - 9.3%. Ad - 10%

IQ – Intelligence Quotient; **ID** – Intellectual Disability; **DS** – Dravet Syndrome; **ASD** – Autism Spectrum Disorder; **NA** – Not Available; **DSM-5** – Diagnostic and Statistical Manual of Mental Disorders; **ICD-10** – International Classification of Diseases Version 10; **ADI-R** – Autism Diagnostic Interview-Revised; **FSIQ** – Full-Scale Intelligence Quotient; **SCQ** – Social Communication Questionnaire; **BASC-2** – Behaviour Assessment System for Children (2nd. edn.); **PEP-3** – Psychoeducational Profile (3rd. edn.); **SCD** – Social Communication Disorder; **ADOS-2** – Autism Diagnostic Observation Schedule (2nd. edn.); **C-WISC** – Chinese Wechsler Intelligence Scale for Children, **I** – Infant (<2y); **PS** – Pre-school (2–5y), **MC** – Middle childhood (6–11y); **A** – Adolescent (12–17y), **Ad** – Adult (≥18y).

greater difficulties in social communication than in the male gender; however, females with ASD usually do not have as many restricted interests and activities as males. More severe implications characterize autism in the male gender. Epilepsy, on the other hand, varies by gender in terms of prevalence, with a higher incidence occurring in men [26].

Correlation between Dravet syndrome and autism spectrum disorder. Patients suffering from Dravet syndrome also often suffer from other conditions in addition to the syndrome's symptoms. It has been noted that attention deficit disorder, behavioural problems, gait and speech problems, and sleep disorders co-occur with Dravet syndrome [2]. Many of the disorders presented by the patients, such as failure to maintain eye contact, sticking to a rigid routine, a very narrow range of interests, delayed speech development, and poor ability to express emotions, overlap with symptoms that are characteristic of autism spectrum disorders [2]. Patients also show traits different from classic autistic behaviour, such as a relatively preserved ability to socialize and an excessive desire to interact with strangers [32]. This may be due to mutations in the sodium channel genes *SCN1A* and *SCN2A*, which are responsible for the occurrence of Dravet syndrome. There are reports that patients whose disease is caused by inheritance of these genes, have symptoms that can be attributed to symptoms of autism spectrum disorders [2, 32].

Mutations in the *PCDH19* gene, located on chromosome Xp22.1, encoding protocadherin 19, however, are associated with a Dravet syndrome-like (DS-like) phenotype. Carriers of pathogenic variants in the *PCDH19* gene account for up to 16% of Dravet syndrome patients without mutations within *SCN1A* [12, 33]. Rampazzo et al., in a systematic review, pointed to a higher prevalence of autism in carriers of pathogenic *PCDH19* variants. ASD was found in 62.5% of patients, while the incidence was estimated at 37.5% in *SCN1A* carriers. However, there was no correlation between the type of variant and the co-occurrence of autism, cognitive disorders, or behavioural problems [33].

Currently, there are three hypotheses that explain the co-occurrence of Dravet syndrome with neurocognitive disorders. The first is the dorsal stream vulnerability theory (according to the cognitive dual-stream theory formulated by David Milner and Malvyn A. Goodale), the second is the sensorimotor-integration deficit theory, and the third is the cerebellar-like pattern theory, being part of cerebellar cognitive-affective syndrome. In favour of all three theories are greater disabilities in executive intelligence than in verbal intelligence, abnormal eye movements, stereopsis, fixation shift, impaired motor skills, visuomotor integration deficits, impaired working memory, executive function impairments, planning, set-shifting, and verbal fluency. Behind the theories of sensorimotor-integration deficit and cerebellar-like pattern are abnormalities such as attention and hyperactivity disorders, comorbidity with ASD or autistic-like traits like poor eye contact, speech delay, adherence to routine, and poor ability to express emotions, abnormal gross motor abilities like delayed independent sitting and walking, and coordination problems during locomotion. The dorsal-stream vulnerability theory and sensorimotor-integration deficit theory are supported by pervasive visual perceptual impairments and visual attention impairments. There are also symptoms that speak only for one of the three above-mentioned theories. The sensorimotor-integration deficit is supported by abnormal phonological perception and detection, auditory-motor integration deficits leading to expressive language deficits in repetition, articulation, phonological accuracy, morphosyntactic accuracy, omission errors, agrammatism, and naming.

On the other hand, only cerebellar-like pattern theory is supported by dysarthric speech, imprecise articulation, abnormal nasal resonance, voice and pitch, and prosody errors. These arguments also include the dysrhythmic tapping of a finger. The interweaving of symptoms that can be subordinated to given hypotheses of the occurrence of disorders indicates that it is probably impossible to identify one leading hypothesis that causes the appearance of disorders in all patients equally. It is possible that different patients experience the appearance of changes based on different mechanisms, or the mentioned mechanisms co-occur in a given patient, and cause the appearance of different symptoms [34].

CONCLUSION

Autistic behaviour can often be seen in Dravet syndrome, the reasons for which are not fully explained, although suspicions fall on the *SCN1A*, *SCN2A*, or *PCDH19* gene mutations present in Dravet syndrome. Patients struggling with DS should receive early diagnosis for ASD and timely implementation of optimal therapy. Genetic mutations present in DS can also cause epileptic symptoms, which are characterized by drug resistance to classic drugs used in epilepsy therapy. The picture of DS very often also includes Intellectual Disability, which is associated with an increased risk of developing ASD.

Currently, there are three hypotheses for the pathophysiology of neurodevelopmental disorders in the course of DS. However, none of them has been singled out as the main cause, and the mechanisms probably correlate with each other. The clinical picture in both ASD and DS often overlaps in terms of autistic behaviour. However, there are some variations in the ASD picture, such as a relatively preserved ability to socialize and an excessive desire to interact with strangers.

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