The Cerebellum
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REVIEW

# Cerebellar Roles in Motor and Social Functions and Implications for ASD

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## Abstract

The cerebellum, traditionally linked to voluntary motor coordination, is now recognized for its role in nonmotor functions, including cognitive and social behaviors. This expanded understanding is vital for identifying neurodevelopmental disorders such as autism spectrum disorder (ASD), where cerebellar abnormalities are common. Recent research has identified specific cerebellar circuits contributing to these diverse functions, revealing interconnected pathways that regulate both motor and social behaviors. The cerebellum communicates extensively with the cerebral cortex, thalamus, and limbic structures through converging and diverging pathways, integrating sensory and motor information to fine-tune outputs and influence higher-order functions. Mouse models have been instrumental in dissecting cerebellar functions, with studies using genetic and neuroanatomical techniques to manipulate specific circuits and observe behavioral outcomes. Disruptions in cerebellar pathways can lead to motor deficits and social impairments, mirroring human neurodevelopmental disorders. This review explores the anatomical and functional organization of cerebellar pathways in mice, their role in behavior, and the implications of cerebellar dysfunction in disorders such as ASD. Understanding these pathways enhances knowledge of cerebellar contributions to behavior and informs therapeutic strategies for cerebellar and neurodevelopmental disorders, emphasizing the integral role of the cerebellum in motor and social functions.

**Keywords** Cerebellum · Autism spectrum disorder (ASD) · Motor coordination · Cognitive functions · Neural circuits

## Introduction

The cerebellum, traditionally associated with the coordination of voluntary motor activity, has been increasingly recognized for its role in a range of nonmotor functions, including cognitive and social behaviors[1, 2]. This expanded understanding is particularly relevant in the context of neurodevelopmental disorders such as autism spectrum disorder (ASD), where cerebellar abnormalities are frequently reported [3]. Recent studies have begun to elucidate the specific neural circuits within the cerebellum that contribute to these diverse functions, highlighting the presence of distinct yet interconnected pathways that regulate both motor and social behaviors [4]. Understanding the contribution of the cerebellum to these functions involves dissecting the intricate network of cerebellar connections and their influence on broader neural circuits [5]. The cerebellum communicates extensively with various regions of the brain, including the cerebral cortex, thalamus, and limbic structures, through a series of well-orchestrated pathways [6]. These pathways can be broadly categorized into converging and diverging circuits. Converging pathways integrate sensory and motor information to fine-tune motor outputs, while diverging pathways project to regions involved in higher-order functions, influencing cognition and social interactions [7]. Mouse models have proven invaluable in dissecting the cellular and molecular mechanisms underlying cerebellar function. Through advanced genetic and neuroanatomical techniques, researchers have been able to manipulate specific cerebellar

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circuits and observe the resulting behavioral outcomes [8, 9]. These studies have provided significant insights into how disruptions in cerebellar pathways can lead to motor deficits and social impairments, mirroring aspects of human neurodevelopmental disorders [10]. In this review, we explore the current understanding of cerebellar pathways that contribute to motor and social behaviors in mice [11]. We will discuss the anatomical and functional organization of these pathways, their role in behavior, and the implications of cerebellar dysfunction in neurodevelopmental disorders [12]. By examining the converging and diverging pathways within the cerebellum, we aim to shed light on the complex neural mechanisms that underlie the cerebellum's multifaceted contributions to both motor and social functions.

## Traditional Role of the Cerebellum in Motor Control

Historically, the cerebellum has been predominantly studied in the context of motor control. Classical studies have demonstrated that the cerebellum is essential for the coordination, precision, and timing of new movements, and therapeutic possibilities may be revealed by discovering how the cerebellum is connected to pain. pain is considered more than sensation but also emotion and perception. This complexity is reflected in the network of cortical and subcortical systems that represent and process pain [13]. Lesions in the cerebellum lead to ataxia, characterized by uncoordinated and imprecise movements, underscoring its critical role in motor function [14]. Early cerebellar manipulations significantly impact behavior, as observed in adult mice with specific cerebellar mutations. The link between abnormal cerebellar improvement and autism-like behaviors in more genetically relevant models of early cerebellum involvement in nonmotor functions influences behavior during the preadult stages of cerebellar manipulation during development and can induce autism-like behaviors [15]. This review highlights recent advances and insights into the traditional understanding of cerebellar function in motor activities, integrating findings from the last decade that enhance our comprehension of its mechanisms and influence on motor behavior [16]. Traditionally recognized for its role in motor control, has been the focus of extensive research to elucidate its anatomical structures, functional integration, and contributions to motor learning and coordination [17, 18]. Anatomically, the cerebellum is divided into the vestibulocerebellum, spinocerebellum, and cerebrocerebellum, each contributing uniquely to balance, limb movement, and the planning of voluntary actions [19]. Recent studies have highlighted the cerebellum's ability to integrate sensory inputs with motor commands, ensuring precise and smooth movements [20]. This integration is crucial for motor learning, where synaptic plasticity within the cerebellar cortex allows for the adaptation and refinement of motor skills through practice [21, 22]. Additionally, the cerebellum's role in timing and predicting motor actions is essential for executing movements at the correct moment, as supported by evidence from neuroimaging studies [18]. The cerebellum's extensive connectivity with other motor regions, such as the motor cortex and basal ganglia, facilitates the coordination and modulation of motor commands [23]. Clinically, understanding cerebellar function has led to therapeutic strategies for cerebellar disorders, including neurostimulation techniques, intensive rehabilitation programs, and pharmacological interventions aimed at improving motor control [10]. These advances underscore the integral role of the cerebellum in motor function and its potential as a target for therapeutic interventions in motor disorders [24].

## Expanding the Role of the Cerebellum to Nonmotor Functions

Recent research has expanded the understanding of the cerebellum beyond motor control, implicating it in cognitive processes and social behaviors [25, 26]. Schmahmann's dysmetria of thought hypothesis proposed that the cerebellum contributes to cognitive functions through its extensive connections with the cerebral cortex [27, 28]. Functional imaging studies in humans have shown cerebellar activation during tasks that require working memory, language processing, and social cognition. The cerebellum is structurally divided into three main parts: the vestibulocerebellum, the spinocerebellum, and the cerebrocerebellum, each contributing differently to motor control [29]. The vestibulocerebellum, including the flocculonodular lobe, is involved in balance and eye movements. The spinocerebellum, comprising the vermis and intermediate zones, regulates body and limb movements [30]. The cerebrocerebellum, which consists of the lateral hemispheres, is associated with planning and initiating voluntary activity [31].

### Sensorimotor Integration

Recent studies have emphasized the role of the cerebellum in integrating sensory inputs with motor commands to fine-tune motor actions [32]. Functional imaging and electrophysiological studies have shown that the cerebellum receives proprioceptive feedback from muscles and joints, which it uses to adjust movements dynamically [4, 33]. This study demonstrated how proprioceptive signals are processed in the cerebellum to ensure smooth and coordinated movements [20, 34]. The cerebellum plays a crucial role in sensorimotor integration, seamlessly combining sensory inputs with motor commands to produce coordinated

**Table 1** Cerebellar pathways and neuroanatomical connections

| Pathway | Origin | Destination | Function |
|---|---|---|---|
| Corticopontocerebellar | Cerebral Cortex | Cerebellar Cortex via Pontine Nuclei | Involved in planning and initiation of movement; conveys information from the cerebral cortex to the cerebellum [47] |
| Spinocerebellar | Spinal Cord | Cerebellar Cortex | Conveys proprioceptive information from the body and limbs to the cerebellum; crucial for coordination and adjustment of limb movements [18] |
| Vestibulocerebellar | Vestibular Nuclei | Flocculonodular Lobe | Involved in balance and eye movements; integrates vestibular input for postural control and ocular stability [48] |
| Olivocerebellar | Inferior Olive | Cerebellar Cortex | Important for motor learning and timing of movements; conveys signals from the inferior olive to the cerebellum [17] |
| Reticulocerebellar | Reticular Formation | Cerebellar Cortex | Contributes to motor coordination and posture; integrates information from the reticular formation to the cerebellum [21] |
| Cerebellothalamic | Deep Cerebellar Nuclei (especially Dentate Nucleus) | Thalamus | Modulates and coordinates voluntary movements; sends processed motor information from the cerebellum to the thalamus [49] |
| Cerebellorubral | Deep Cerebellar Nuclei (especially Interposed and Dentate Nuclei) | Red Nucleus | Involved in the coordination of limb movements; transmits information from the cerebellum to the red nucleus [29] |
| Cerebellovestibular | Fastigial Nucleus | Vestibular Nuclei | Contributes to balance and eye movements; sends signals from the cerebellum to the vestibular nuclei [50] |
| Cerebelloreticular | Fastigial Nucleus | Reticular Formation | Regulates muscle tone and posture; sends outputs from the cerebellum to the reticular formation [51] |

and precise movements [35]. It receives proprioceptive feedback, vestibular information, and visual and auditory inputs, integrating these signals to adjust and refine motor actions in real time [32]. Key mechanisms include intricate neural circuits involving Purkinje cells, synaptic plasticity, and predictive coding, which allow the cerebellum to anticipate and correct movement errors [5]. Advances in functional imaging and electrophysiological studies have deepened our understanding of these processes, highlighting the importance of the cerebellum in maintaining balance and coordination. Clinically, this knowledge informs therapeutic approaches such as neurostimulation and targeted rehabilitation to improve motor performance in individuals with cerebellar disorders.

## Motor Learning and Adaptation

The cerebellum plays a crucial role in motor learning by integrating sensory feedback with motor commands to refine and optimize movements through practice [36]. Recent research has highlighted the cerebellum's involvement in error-based learning, where it detects discrepancies between intended and actual movements and adjusts future actions accordingly [37]. Key mechanisms include synaptic plasticity at parallel fiber-Purkinje cell synapses, where long-term depression (LTD) and long-term potentiation (LTP) enable the adaptation of motor commands [22, 38]. Additionally, the cerebellum acts as an internal clock, ensuring precise timing for coordinated movements, and employs predictive coding to anticipate and correct errors [39, 40]. Advances in neuroimaging and electrophysiological studies have provided deeper insights into these processes, demonstrating the essential function of the cerebellum in motor learning. Clinically, this understanding has informed the development of targeted therapies, such as neurostimulation and specialized rehabilitation programs, aimed at enhancing motor learning and recovery in individuals with cerebellar disorders [23]. The cerebellum is crucial for motor learning, particularly in the adaptation and refinement of motor skills through practice [22]. This function is mediated by long-term synaptic plasticity within the cerebellar cortex, especially in parallel fiber-Purkinje cell synapses [41]. Studies have shown that synaptic modifications in the cerebellum are essential for acquiring new motor skills and adapting to changing environments. Notably, motor learning involves changes in the

**Table 2** Various mouse models used in cerebellar research, including genetic modifications, observed phenotypes, and relevance to human cerebellar disorders

| Mouse Model | Genetic Modification | Observed Phenotypes | Relevance to Human Disorders |
|---|---|---|---|
| L7-Pcp2-Cre | Purkinje cell-specific Cre expression | Selective manipulation in Purkinje cells | Studying Purkinje cell function and cerebellar circuitry of offering insights into potential therapeutic strategies [51, 62] |
| Grid2 | Mutation in Grid2 gene | Ataxia, impaired motor coordination | Models for Grid2-related ataxias of Grid2-related ataxias, offering insights into their genetic and physiological mechanisms in humans [63] |
| Nna1 (Purkinje Cell Degeneration; pcd) | Mutation in Nna1 gene | Purkinje cell degeneration, ataxia | Model for neurodegeneration and cerebellar ataxias of model studies Nna1 gene mutations, shedding light on neurodegeneration and cerebellar ataxias in humans [64] |
| Atxn1 | Expanded CAG repeats in Atxn1 gene | Progressive ataxia, Purkinje cell loss | Spinocerebellar ataxia type 1 (SCA1) is model studies SCA1, exploring how Atxn1 gene mutations contribute to neurodegeneration and ataxia progression in humans [65] |
| Lurcher (Lc) | Gain-of-function mutation in Grid2 gene | Purkinje cell death, ataxia, cerebellar degeneration | Model for cerebellar degeneration and motor function research of lurcher mouse model, with a Grid2 gene mutation, shows Purkinje cell loss and severe motor deficits, modeling olivocerebellar degeneration." [66] |
| Staggerer (Rora) | Mutation in Rora gene | Ataxia, cerebellar hypoplasia | Insights into RORα-related ataxias and cerebellar development of model explores RORα-related ataxias and Rora gene mutations in cerebellar development [67] |
| Weaver (Girk2) | Mutation in Girk2 gene | Ataxia, granule cell loss, cerebellar hypoplasia | Models for granule cell development and Girk2-related cerebellar disorders of weaver mouse model, characterized by a mutation in the Pcnt gene, exhibits cerebellar granule cell degeneration and motor impairment, serving as a model for Dandy-Walker syndrome [68] |
| Reeler (Reln) | Mutation in Reln gene | Ataxia, cerebellar malformation | Models for understanding Reelin signaling in brain development of Reelin signaling in brain development, focusing on ataxias and cerebellar abnormalities linked to Reln gene mutations [69] |
| Scn8a | Mutation in Scn8a gene | Ataxia, seizures, motor deficits | Epilepsy and cerebellar ataxia of mouse model, carrying a mutation in the Scn8a gene, shows cerebellar atrophy, motor incoordination, and seizures, serving as a model for epilepsy and cerebellar ataxia [67] |
| Hotfoot (Grid2) | Mutation in Grid2 gene | Ataxia, impaired motor learning | Model for studying Grid2 function and associated ataxias in Grid2 function and ataxias, offering insights into their genetic and physiological mechanisms in humans [70] |

**Table 2** (continued)

| Mouse Model | Genetic Modification | Observed Phenotypes | Relevance to Human Disorders |
|---|---|---|---|
| Tottering (Cacna1a) | Mutation in Cacna1a gene | Ataxia, epilepsy, dystonia | Model for episodic ataxia type 2 (EA2) and familial hemiplegic migraine of EA2 and familial hemiplegic migraine, exploring the role of Cacna1a gene mutations in these neurological conditions [71] |
| Tsc1 KO | Knockout of Tsc1 gene in cerebellar neurons | Abnormal cerebellar development, ataxia | Model for Tuberous Sclerosis Complex of Tsc1 knockout mouse, with Tsc1 gene deletion in cerebellar neurons, shows abnormal cerebellar development and ataxia, relevant [72, 73] |
| PKCγ KO | Knockout of Prkcg gene | Motor deficits, impaired synaptic function | Model for cerebellar ataxia and dystonia of PKCγ knockout mouse model, lacking the Prkcg gene, shows motor deficits and impaired synaptic function, relevant for cerebellar ataxia and dystonia [74] |
| SCA3 YAC | Human Ataxin-3 transgene | Progressive ataxia, Purkinje cell loss | Spinocerebellar ataxia type 3 model mimics SCA3, offering a platform to study Ataxin-3 gene mutation mechanisms [75] |

**Table 3** Cerebellum in autism spectrum disorder (ASD): key findings, affected regions, proposed mechanisms, and implications

| Aspect | Details |
|---|---|
| **Key Findings** | - Reduced Purkinje cell numbers |
| | - Abnormalities in cerebellar structure (e.g., hypoplasia) |
| | - Altered cerebellar connectivity |
| | - Impaired motor coordination and timing |
| | - Cerebellar role in cognitive and affective processing [79, 80] |
| **Affected Regions** | - Vermis |
| | - Hemispheres |
| | - Lobules VI and VII |
| | - Crus I and Crus II [65, 81] |
| **Proposed Mechanisms** | - Disruption in cerebellar-thalamic-cortical circuits |
| | - Altered synaptic plasticity |
| | - Impaired sensory integration and processing |
| | - Developmental disruptions affecting cerebellar growth and connectivity |
| | - Dysregulation of cerebellar output affecting social, cognitive, and motor functions [82] |
| **Implications** | - Understanding cerebellar contributions to ASD symptoms |
| | - Potential targets for therapeutic interventions |
| | - Insights into early developmental markers and early diagnosis |
| | - Influence on designing interventions for motor and cognitive deficits in ASD [83] |

strength of synaptic connections, which are critical for the accurate execution of learned movements [32].

## Timing and Prediction

One of the key contributions of the cerebellum to motor control is the precise timing and prediction of motor actions [42]. The cerebellum is thought to act as a predictive model that anticipates the sensory consequences of motor commands, allowing timely adjustments [43]. Studies using neuroimaging techniques have shown cerebellar activation during tasks that require precise timing and sequencing of movements, such as playing a musical instrument or typing. provided evidence that the cerebellum's predictive

**Table 4** Specific pathways implicated in motor and social behaviors, including the pathway name, involved brain regions, associated functions, and relevance to disorders

| Pathway | Involved Brain Regions | Associated Functions | Relevance to Disorders |
|---|---|---|---|
| Corticospinal Tract | Motor Cortex, Spinal Cord | Voluntary motor control | Impairments in ALS, cerebral palsy [86] |
| Corticobulbar Tract | Motor Cortex, Brainstem (Cranial Nerve Nuclei) | Facial and head movements | Dysfunctions in stroke, ALS [87] |
| Cerebellothalamic | Cerebellum, Thalamus | Coordination and timing of movements | Ataxias, coordination disorders [88] |
| Cerebellocerebral | Cerebellum, Cerebral Cortex | Fine-tuning motor activities, cognitive processing | Autism Spectrum Disorder, ADHD, ataxias [89] |
| Dorsal Column-Medial Lemniscus | Dorsal Column of Spinal Cord, Medulla, Thalamus | Proprioception, fine touch, vibration sense | Sensory processing disorders, proprioceptive deficits [90] |
| Spinocerebellar | Spinal Cord, Cerebellum | Proprioception, coordination of limb movements | Ataxia, proprioceptive disorders [91] |
| Hypothalamic–Pituitary–Adrenal (HPA) Axis | Hypothalamus, Pituitary Gland, Adrenal Glands | Stress response, regulation of cortisol | Anxiety disorders, depression, PTSD [92, 93] |
| Amygdala-Prefrontal Cortex | Amygdala, Prefrontal Cortex | Emotional regulation, social behavior | Anxiety, depression, social behavior disorders [94] |
| Default Mode Network (DMN) | Medial Prefrontal Cortex, Posterior Cingulate Cortex, Angular Gyrus | Self-referential thought, social cognition | Autism Spectrum Disorder, depression, ADHD [95, 96] |
| Salience Network | Anterior Insula, Anterior Cingulate Cortex | Detecting and filtering salient stimuli | Schizophrenia, ASD, anxiety disorders [97] |

capabilities are crucial for coordinating complex motor sequences [44, 45].

### Cerebellar pathways and neuroanatomical connections

The cerebellum interacts with various brain regions through a network of converging and diverging pathways. Converging pathways primarily involve the integration of sensory and motor information [19, 46]. These pathways include connections from the spinal cord, vestibular nuclei, and cerebral cortex to the cerebellar cortex and deep cerebellar nuclei [19]. Diverging pathways project from the cerebellum to multiple brain regions, including the prefrontal cortex, which is involved in higher-order cognitive functions, and the limbic system, which regulates emotions and social behaviors (Table 1).

## Mouse models for cerebellar research

Mouse models have been instrumental in elucidating the role of specific cerebellar circuits in behavior [52]. Genetic tools, such as Cre–loxP recombination, have allowed for the selective manipulation of cerebellar neurons [53]. Studies using these techniques have revealed that disrupting cerebellar Purkinje cells can lead to deficits in both motor coordination and social behavior, suggesting a shared neural substrate for these functions [54]. For instance, deletion of the Tsc1 (Tuberous Sclerosis Complex 1) gene in Purkinje cells results in autism-like behaviors in mice (Table 2). Tuberous sclerosis complex gene 1 (TSC1) plays a pivotal role in the insulin receptor pathway and is often disrupted in tumors. Encoded via the TSC1 gene, hamartin plays an essential role in regulating human cells. Understanding the role of phosphorylation requires identifying upstream kinases [55]. The TSC is a compelling model for examining how the cerebellum contributes to ASD pathogenesis. TSC patients exhibit cerebellar pathology and a strong association with ASD, while also revealing novel roles for Tsc1 in Purkinje cell function, shedding light on the role of the cerebellum in cognitive disorders such as autism [56]. Sufferers with familial TSC inherit a germline mutation in TSC1 or TSC2, leading to somatic loss and diverse renal epithelial tumors. TSC is an autosomal dominant genetic disease characterized by hamartomatous tumors across organs caused by mutations in TSC1 or TSC2. Chronic kidney disease is a critical risk factor for renal neoplasms, and TSC-associated renal tumors are often compared with sporadic cases [57]. Initially, thought to cause phenotypic variability in TSC, mtDNA variants were strongly correlated across tissues and tumors. Several key genotype–phenotype correlations in TSC have been identified [58, 59]. Considerably, traditional genetic testing fails to reveal mutations in 15% of patients. It is postulated that the majority of these cases are

**Table 5** Therapeutic Advances, Human and Mouse Models of Cerebellar Dysfunction and ASD

| Drug Name | Mechanism of Action | Therapeutic Target | Indications | Current Status and functions |
|---|---|---|---|---|
| Rapamycin | mTOR inhibitor | mTOR pathway | Tuberous Sclerosis Complex, cerebellar disorders | Approved for TSC, research for cerebellar applications [59] |
| Everolimus | mTOR inhibitor | mTOR pathway | Tuberous Sclerosis Complex, cerebellar tumors | Approved for TSC, research in cerebellar dysfunction [98] |
| Cannabidiol (CBD) | Modulates neurotransmission, anti-inflammatory | Endocannabinoid system | Seizures associated with cerebellar dysfunction | FDA approved for epilepsy, research ongoing for cerebellar conditions [99] |
| Baclofen | GABA_B receptor agonist | GABAergic system | Spasticity in cerebellar ataxia | Approved for spasticity, used off-label in cerebellar disorders [100] |
| Riluzole | Inhibits glutamate release | Glutamatergic system | Cerebellar ataxia, neuroprotection | Approved for ALS, research for cerebellar ataxia [101] |
| Cerebellar Activating Peptide | Enhances cerebellar neuron function | Cerebellar Purkinje cells | Cerebellar ataxia, motor dysfunction | Experimental stage [102] |
| Dalfampridine | Potassium channel blocker | Voltage-gated potassium channels | Improves motor function in cerebellar ataxia | Approved for MS, research for cerebellar ataxia [103] |
| N-Acetylcysteine (NAC) | Antioxidant, reduces oxidative stress | Cellular oxidative stress pathways | Neuroprotection in cerebellar dysfunction | Widely used antioxidant, clinical trials for cerebellar ataxia [104, 105] |
| Levodopa | Dopamine precursor | Dopaminergic pathways | Dopaminergic cerebellar dysfunction | Approved for Parkinson's, used off-label in cerebellar disorders |
| Gabapentin | Modulates GABA release | GABAergic system | Cerebellar ataxia, neuropathic pain | Approved for neuropathic pain, used off-label for cerebellar ataxia [106] |
| Lamotrigine | Stabilizes neuronal membranes, inhibits glutamate | Glutamatergic system | Seizures, cerebellar dysfunction | Approved for epilepsy, research for cerebellar dysfunction [107] |
| Acetazolamide | Carbonic anhydrase inhibitor | Alters ion balance in neurons | Episodic ataxia, cerebellar edema | Approved for episodic ataxia [108] |
| Topiramate | Inhibits glutamate receptors, enhances GABA | Glutamatergic and GABAergic systems | Seizures, cerebellar dysfunction | Approved for epilepsy, research for cerebellar disorders [107] |

caused by somatic TSC1/TSC2 variants, creating complicated diagnostic challenges [59]. Half of those with TSC1/TSC2 variants have a second hit in affected tissues, such as facial angiofibromas, renal AMLs, and LAM. Missense variants make up 6% of TSC1 pathogenic variants, while small indels make up more than 57% of TSC1 pathogenic variants [60]. Using a 3D CNN model with multicontrast (multicontrast magnetic resonance imaging) MRI, we were able to identify TSC in children early. Fluid-attenuated inversion recovery (FLAIR3), a novel modality, enhances the visibility of TSC lesions and improves classification accuracy. The technique utilizes 3D-EfficientNet and 3D-ResNet as feature extractors. FLAIR3 was previously used most effectively in MS [61]. Genetic disorder (TSC) is a caused by mutations inside the TSC1 and TSC2 genes cell growth and benign tumor formation in multiple organs of affect the brain, skin, and other organs.

## Cerebellar involvement in autism spectrum disorder (ASD)

There is substantial evidence linking cerebellar dysfunction to ASD. Neuroimaging studies have identified structural and functional abnormalities in the cerebellum of individuals with ASD [76, 77]. Animal studies have further supported this link, showing that mutations in genes associated with ASD often lead to cerebellar abnormalities [78]. For example, knockout models for genes such as Shank3 and Cntnap2 exhibit both motor and social deficits along with cerebellar anomalies (Table 3).

### Specific Pathways Implicated in Motor and Social Behaviors

Research has identified specific cerebellar pathways involved in distinct aspects of motor and social behaviors

[17, 25]. For motor control, the spinocerebellar and cerebrocerebellar pathways are crucial because they integrate sensory feedback and fine-tune motor outputs [84]. For social behaviors, cerebellar connections to the prefrontal cortex and limbic system are particularly important [85]. These pathways are thought to modulate social cognition and emotional regulation through their influence on cortical and subcortical circuits (Table 4).

Human and mouse models play a critical role in understanding the genetic and molecular underpinnings of those conditions. These models new therapies effectively, paving the way for targeted treatments (Table 5). Current studies focuses on various interventions, including pharmacological approaches, gene therapy, and behavioral strategies to develop effective treatments that can significantly improve the quality of lifestyles for people with cerebellar dysfunction and ASD, with ongoing studies continually refining these therapeutic approaches.

### Clinical Implications

Understanding the role of the cerebellum in motor control has significant clinical implications, particularly for individuals with cerebellar disorders [32]. Conditions such as cerebellar ataxia, dystonia, and tremor are characterized by impaired motor coordination and balance. Recent therapeutic approaches, including cerebellar stimulation and targeted rehabilitation strategies, aim to mitigate these motor deficits by enhancing cerebellar function [91]. Studies have shown that cerebellar transcranial magnetic stimulation (TMS) can improve motor performance in patients with cerebellar damage, highlighting the potential for neuromodulation in treating cerebellar disorders.

## Conclusion

The literature underscores the dual role of the cerebellum in motor and social functions, mediated through its converging and diverging pathways. Understanding these pathways in detail not only enhances our knowledge of cerebellar function but also provides insights into the neural mechanisms underlying neurodevelopmental disorders such as ASD. Continued research using advanced genetic and neuroanatomical techniques in mouse models will be crucial in unraveling the complex contributions of cerebellar circuits to behavior.

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