Development of social cognition: the essential role of the cerebellum

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Abstract

Seminal work in the 1990's found alterations in the cerebellum of individuals with social disorders including autism spectrum disorder and schizophrenia. In healthy populations, distinct portions of the posterior cerebellum are consistently activated in fMRI studies of social cognition and it has been hypothesized that the cerebellum plays an essential role in social cognition, particularly in theory of mind. Here we review the lesion literature and find that the effect of cerebellar damage on social cognition is strongly linked to the age of insult, with dramatic impairments observed after prenatal insult, strong deficits observed after childhood damage, and mild and inconsistent deficits observed following damage to the adult cerebellum. To explain the developmental gradient, we propose that early in life, the *forward model* dominates cerebellar computations. The forward model learns and uses errors to help build schemas of our interpersonal worlds. Subsequently, we argue that once these schemas have been built up, the *inverse model*, which is the foundation of automatic processing, becomes dominant. We provide suggestions for how to test this, and also outline directions for future research.

1.INTRODUCTION

The human brain does not mature in a uniform manner and regional differences are thought to reflect something important about the maturation of thought and behavior. A great deal of attention has been paid to the late-developing prefrontal cortex and more recently, the uneven development of different white matter tracts (Hoffman et al., 2022; Reynolds, Grohs, Dewey, & Lebel, 2019). In this paper, we focus on the cerebellum, which has been relatively ignored by cognitive neuroscientists who believed its functions were limited to locomotion, playing an essential role in the calibration of smooth movements through the creation and ongoing modification of motor programs (Brodal & Bjaalie, 1997; Holmes, 1939). However, the pure-motor viewpoint was challenged over forty years ago and it is now widely acknowledged by cerebellum researchers that this brain region contributes to social, cognitive, and affective components of human behavior (Dow, 1974; Frick, 1982; Snider, 1950).

A new emerging view is that the cerebellum plays a key role in social cognition in general, and theory of mind (ToM) in particular (Heleven & Van Overwalle, 2019; Van Overwalle, Baetens, Marien, & Vandekerckhove, 2015; Van Overwalle & Vandekerckhove, 2013). ToM is also referred to as mentalizing, which is our ability to attribute mental states to others, and interpret their intentions, perspectives, and beliefs (Blakemore, 2008). Social cognition and its development critically underpins how we learn, think, communicate, and assimilate into our cultural context (Poulin-Dubois, 2020).

Beginning at birth with the emergence of socially-oriented perceptual capacities, such as face and emotion recognition, the antecedents to ToM unfold throughout infancy into early childhood (Korkmaz, 2011; Senju, 2012). Perceptual precursors of ToM include joint attention which arises at 3 months of age (reviewed in Korkmaz, 2011; see also Scaife & Bruner, 1975), while nonverbal forms of social communication such as pointing manifests between 9 to 14 months of age (reviewed in Korkmaz, 2011; see also Camaioni, Perucchini, Bellagamba, & Colonnesi, 2004). It is widely believed that nascent ToM schemes surface between the ages of 13 – 15 months (reviewed in Korkmaz, 2011; see also Frith & Frith, 2003; Onishi & Baillargeon, 2005; Surian, Caldi, & Sperber, 2007; Wiesmann & Southgate, 2021), coinciding with the emergence of pretend play at 18 months, which is thought to be critical for nurturing mature ToM abilities, and which is

notably impaired in ASD (Korkmaz, 2011; see also Connolly & Doyle, 1984; Lewis & Boucher, 1988). In addition, the sense of self (e.g. self recognition, sense of agency) is developed by approximately 1.5 years of age and is thought to be a prerequisite for ToM (e.g., self-other distinguish in the false belief task). However, it is not until the ages of 3 – 4 years that theory of mind occurs intuitively, without provocation (Korkmaz, 2011). This is also the ontological window in which children are able to correctly identify false beliefs in others, thereby correctly predicting the actions of non-self-agents who hold differing ideas about the world (Wiesmann & Southgate, 2021; see also Baron-Cohen, Leslie, & Frith, 1985).

As evinced by studies of mentalizing in adults with high-functioning ASD, the *spontaneous* evocation of ToM that normally arises around age 4 never develops in these individuals, despite their ability to solve benchmark false-belief tests by the mental age of 11 years (Senju 2012; see also Southgate, Senju, & Csibra, 2007). Over the last 30 years, several studies have reported alterations in the cerebellum in individuals with ASD (Wang, Kloth, & Badura, 2014).

Given that social cognition is an essential component of human development, and the cerebellum has been implicated in social cognition, the question of how cerebellar damage at different points in development affect the emergence of social cognition remains. In this paper we review the lesion literature as it provides stronger inferential power than neuroimaging for the involvement of particular brain regions in different cognitive and behavioral processes. We start by briefly discussing cerebellar anatomy before turning to our review of the effects of cerebellar damage on social cognition in infants, children, and adults.

2.A BRIEF REVIEW OF CEREBELLAR DEVELOPMENT

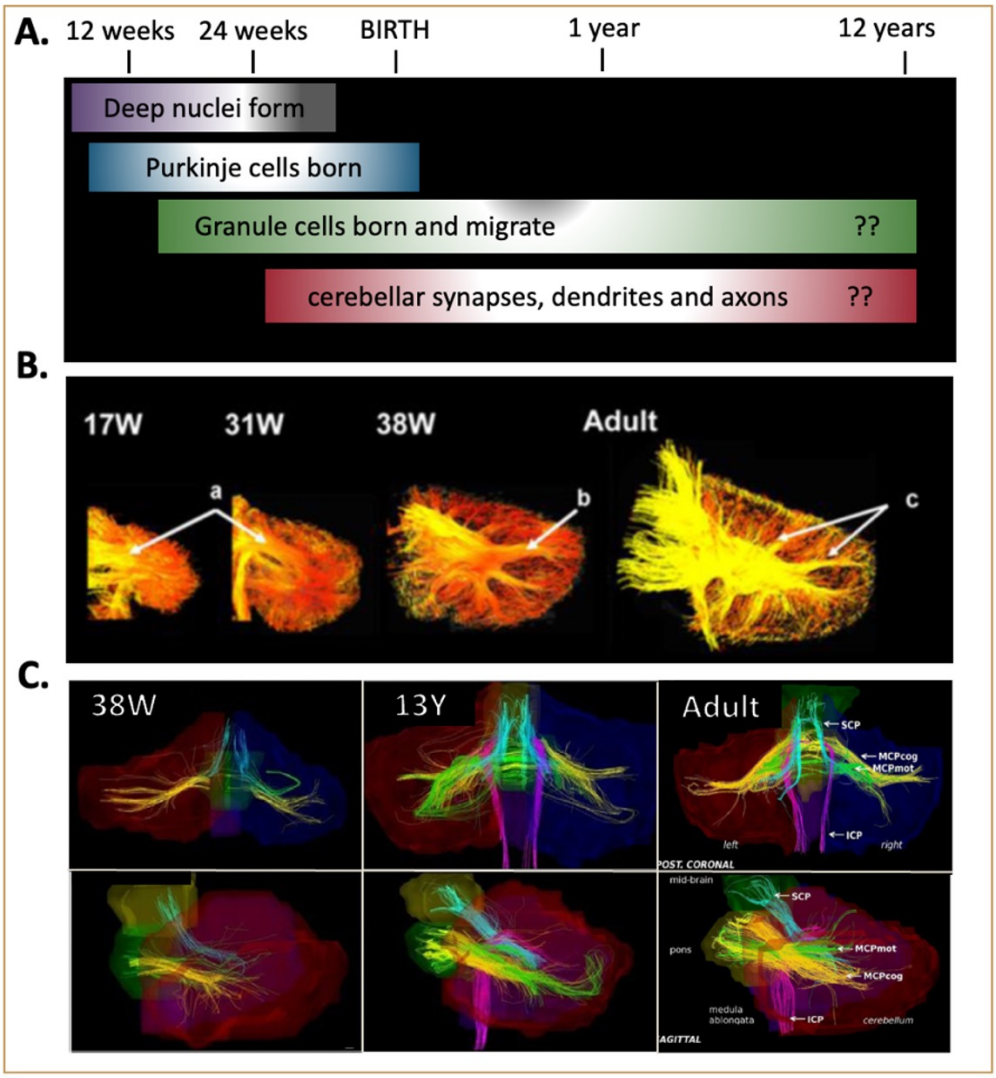
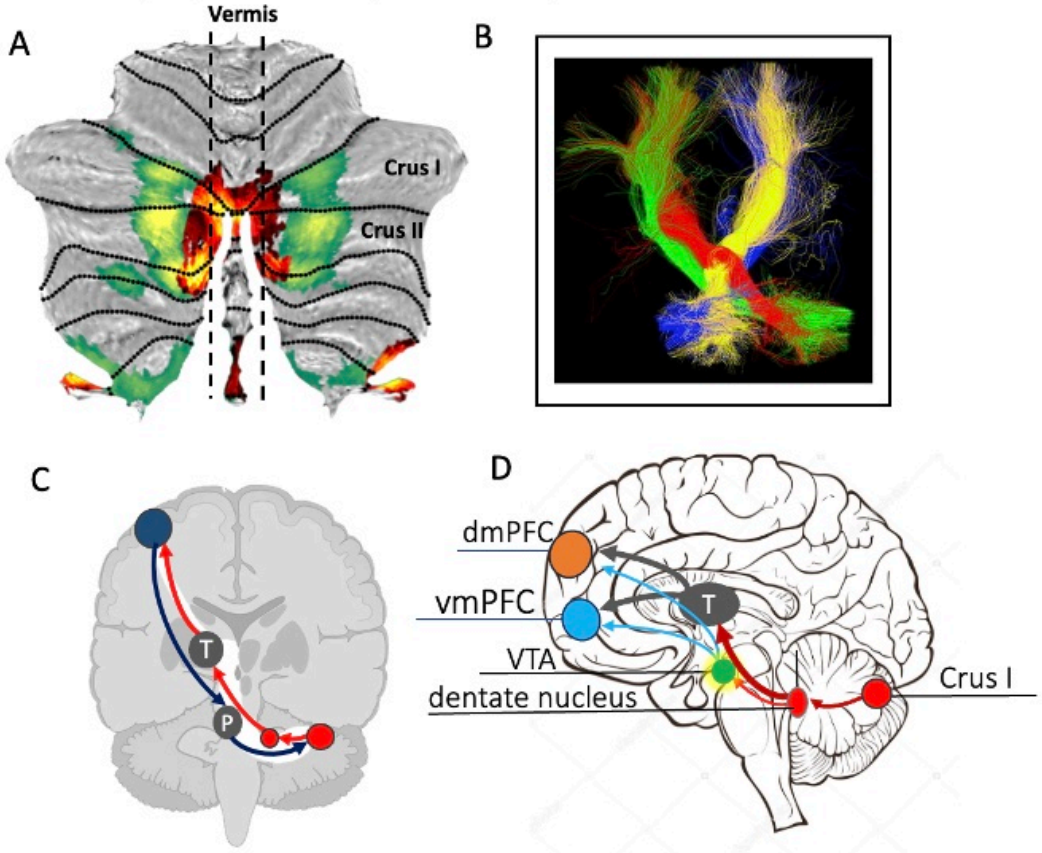


Figure 1. **A**. Schematic diagram, modified from Sathyanesan et al. (2019) depicting the timeline of major cellular events in the cerebellum. Note that beyond year 1, human cellular data are scarce. A few structural MRI studies indicate that the cerebellum increases in size through adolescence (Sussman, Leung, Chakravarty, Lerch, & Taylor, 2016), however, more data are needed to determine the source and meaning of this increase. **B**. Sagittal views of cerebellar pathways, from a post-mortem diffusion imaging study, imaged at 17 – 38 weeks (W) and in the adult (Takahashi et al., 2014). High fractional anisotropy (FA) values are indicated with arrows. Many pathways had low FA values until 38 gestational. **C**. HARDI tractography showing development of the cerebellar peduncles. The superior cerebellar peduncle pathway (SCP) is shown in light blue. The middle cerebellar peduncle (MCP) is in green and yellow. The inferior cerebellar peduncle pathways (ICP), shown in pink (Re et al., 2017). Images in B and C used with permission of authors.

The cerebellum begins to develop early in the first trimester with the deep cerebellar nuclei, the output channels of the cerebellum, forming at the end of the first trimester (Amore et al., 2021). During the second trimester, the granular layers (consisting of the most numerous cell type in the cerebellum – granule cells) and a midline region called the vermis form (Bromley, Nadel, Pauker, Estroff, & Benacerraf, 1994; Isumi, Mizuguchi, & Takashima, 1997). During the third trimester, cerebellum volume begins to increase exponentially and axonal connections are formed with the rest of the central nervous system (Pierson & Al Sufiani, 2016; Sathyanesan et al., 2019). This rapid cerebellar growth presents a five-fold increase in volume, indicating that the third trimester is a critical period in cerebellum development (Limperopoulos, Benson, et al., 2005; Limperopoulos, Soul, et al., 2005; Volpe, 2009b). The cerebellum continues to grow throughout childhood, possibly through an increase in intra-cerebral cellular processes and white matter proliferation (Takahashi, Hayashi, Schmahmann, & Grant, 2014), peaking in volume between the ages of 12-16 (Limperopoulos, Benson, et al., 2005; Limperopoulos, Soul, et al., 2005; Tiemeier et al., 2010). From birth to age 30, the volume and microstructure of the cerebellar peduncles also increases (Re et al., 2017). Once the cerebellum is fully formed, it has approximately five times more neurons than the cerebrum (Herculano-Houzel, 2009). These events are depicted in **Figure 1** and additional information about gross anatomy and connectivity can be found in **Box 1**.

Box 1. A Brief Guide to Cerebellar Anatomy

Just like the cerebrum, the cerebellum has a functional topography. **Figure A** depicts a labeled flatmap of the cerebellar cortex with BOLD activations in Crus I/II to a social mentalizing task (=green) and in the vermis and lobule IX to an emotion recognition task (=red) from the Human Connectome Project dataset (Metoki, Wang, & Olson, 2022). **Figure B** shows high resolution diffusion imaging data from a human infant brain (Pieterman et al., 2017). This image shows the *cortico-ponto-cerebellar tract (CPC; green and blue streamlines),* projecting from the cerebral cortex to the pons, which then projects to a region on the opposite side of the cerebellum. The *cerebello-thalamo-cortical* tract *(CTC; red and yellow streamlines)* is the output pathway, projecting from the cerebellum to the opposite hemisphere of the brain at the level of the inferior colliculus then synapsing onto the thalamus, and terminating in various regions of the cortex. Smaller, direct connections, that do not follow this scheme, exist between the cerebellum and subcortical regions such as the amygdala and ventral tegmental area (VTA; Carta, Chen, Schott, Dorizan, & Khodakhah, 2019; Middleton & Strick, 1997, 2001; Schmahmann & Pandya, 1997). **Figure C** is a schematic depiction of generic CTC and CTC tracts. **Figure D** is a schematic diagram of the social portion of the CTC tract, based on findings from Metoki et al (2022), who found that most structural connections were to the dorsomedial prefrontal cortex (dmPFC) and ventromedial prefrontal cortex (vmPFC), and from rodent work showing monosynaptic connections to the VTA. A similar circuit has been reported in rodents (Kelly et al., 2020). T=thalamus; P=pons. All images used with permission of author.



Early in life, cerebellar volume loss can result from direct damage, such as hemorrhages or tumors, or indirect damage. Indirect damage is a secondary effect related to damage in a remote but connected area of the brain and is called *diaschisis*. Crossed cerebellar diaschisis is a reduction in metabolism and blood flow in the cerebellar hemisphere contralateral to the affected cerebral hemisphere, usually in the frontal lobe (Hausen, Lachmann, & Nagler, 1997; Shamoto & Chugani, 1997). Although this was first documented in adults, it has also been documented in extremely premature infants and in fact, is more severe than what is observed in adults because it includes a striking decrease in cerebellar volume following cerebral damage (Limperopoulos, Benson, et al., 2005). The opposite also holds true – cerebellar damage can cause a striking volume reduction in the opposite hemisphere of the cerebrum (Limperopoulos, Benson, et al., 2005; Rousseaux & Steinling, 1992). These findings hint that early in development, the rapid growth of the cerebellum may be yoked to frontal lobe growth and activity.

3.EFFECTS OF CEREBELLAR DAMAGE

3.1. FETAL STAGE: CHANGES IN SOCIAL BEHAVIOR ARE DEVASTING

The United States has the highest rate of premature births in the world. About 1.6% of all births in the United States occur before the gestational age of 32 weeks, which is considered severely premature (Purisch & Gyamfi-Bannerman, 2017). To illustrate the magnitude of this, in 2018 alone, 3,788,235 babies were born in the United States (Martin, Hamilton, & Osterman, 2019); 1.6% of this is over 60,000 infants. Cerebellar injury and volume loss is observed in about 20% of infants born before 32 weeks, with incidence increasing in those born extremely prematurely (e.g. less than 28 weeks; Steggerda et al., 2009). Cerebellar underdevelopment at this point in life is associated with a spectrum of severity, with the most severely affected children exhibiting cerebral palsy, epilepsy, mental retardation, and ASD (Volpe, 2009a).

Another way in which the cerebellum can be damaged early in life is by cerebellar hemorrhaging or ischemic events which tend to occur in conjunction with prematurity. Limperopoulos, Benson, et al. (2005) studied 35 children who had been born prematurely with cerebellar hemorrhage (mean

gestational age, 26 weeks), and 35 age-matched premature infants without such hemorrhaging. The neurological sequelae of the cerebellar damage were profound: motor abnormalities (66%) receptive and expressive language disabilities (37% and 42%, respectively), cognitive deficits (40%), social deficits (34%), and abnormal results on an autism screener measure (37%). Notably, children with damage to a part of the cerebellum called the *vermis* exclusively accounted for those with social deficits (Limperopoulos et al., 2007).

The cerebellum can also be damaged early in life by congenital malformation, underdevelopment, or absence (e.g. agenesis) of the cerebellum (reviewed in Millen & Gleeson, 2008), conditions which fortunately, are quite rare. A study of 27 individuals (Tavano et al., 2007) with a range of cerebellar congenital malformations found that the loss of midline regions, which includes portions of the vermis (which part is never defined), was associated with severe social and affective problems. The most severely affected individuals were socially withdrawn, had limited language abilities, and had obsessive interests. All of the children and young adults with absent vermal areas were classified as having pervasive developmental disorder or autism spectrum disorder. The authors noted: “It is difficult to interpret the neurological evaluation of these patients as they were poorly cooperative or uncooperative” suggesting that there were gross changes in affect regulation and social interactions (Tavano et al., 2007, p. 2653).

In a study translated from the original Russian, M. Bobylova, Petrukhin, Dunaevskaia, Piliia, and Il'ina (2006) studied children who had cerebellar dysgenesis (e.g. abnormal development) and discussed how parents began noticing that their children’s behavior was odd at around age 3. “These odd behaviors primarily affected responses to mothers: lack of attention to mothers or a symbiotic type of contact. With other people – adults and especially children – social behavior showed indifference or negativism,” (M. Y. Bobylova, Petrukhin, Dunaevskaya, Piliya, & Il'ina, 2007, p. 757). They also noted the variable outcomes in children, with some of them fulfilling all criteria for ASD, while other children had “…normal or high levels of intellect but with degraded development, delayed development of scholarly skills, attention deficit hyperactivity disorder, marked behavioral impairments and motor automatisms, and high levels of aggression,” (M. Y. Bobylova et al., 2007, p. 758).

Table 1. Effects of cerebellar damage on socio-emotional behavior during infancy and childhood. Papers are listed by age group, then alphabetically. Abbreviations: Patient (P); autism spectrum disorder (ASD); cerebellum (CB) generalized anxiety disorder (GAD).

Author	Etiology	N	Description
A. Fetal			
Akanli, Cohen-Addad, Malabanan, Margono, and Krilov (1997)	hemorrhage	1	Developmental delays when tested at 9 months
Bolduc et al. (2011)	malformation	49	71% had some sort of abnormal development. Global developmental delays in 39%; 41% screened positive for early signs of ASD, 24% had internalizing behavioral problems, 31% had impaired functional communication, 29% had impaired daily living skills, 20% had socialization problems
M. Bobylova et al. (2006)	agenesis/dysgenesis	20	Socially indifferent; inappropriate emotionally responses (exaggerated or absent); no eye contact; some aggression; described as "autistic"
Bulgheroni et al. (2016)	agenesis/dysgenesis	54	Global developmental delay in 81%; 39% showed emotional fragility, withdrawal, inattention, irritability, poor tolerance to frustration, and oppositional behavior linked to environmental stress (two oppositional defiant and two bipolar); more internalizing than externalizing problems
Hayashi et al. (2015)	hemorrhage	1	15 months showed continued gross motor and speech delay with significant right eye esotropia, trouble with balance, did not follow simple commands, 3-word vocab.
Hickey et al. (2018)	malformation (Joubert)	3	All 3 history of behavioral and emotional dysregulation (hallucinations, mood dysregulation, depression, general anxiety disorder, unprovoked aggression, suicidality, delusions of grandeur). Developmental delays, below age-based expectations across cognitive, academic, and adaptive skills domains.
Limperopoulos et al. (2007)	hemorrhage	35	Significantly lower scores on all tested measures on motor, language, and cognition. Also, 37% did not pass autism screeners and 34% had internalizing behavioral problems. Global development, functional, and social-behavioral deficits more common and profound with vermal injury
Parker et al. (2008)	malformation (preterm)	65	Worse mental well-being in those w/ decreased cerebellar volume in late adolescence; volume correlates positively with full scale, verbal and performance IQ; improved verbal fluency associated w/ increased cerebellar size

Steggerda et al. (2009)	hemorrhage (preterm)	108	57% of 14 infants with small cerebellum had mildly or severely abnormal neurodevelopmental outcome at 2 years, but 48% of 70 infants without cerebellums had the same.
Steinlin, Zangger, and Boltshauser (1998)	non-progressive congenital ataxia	34	Significant delays in developmental milestones; 90% needed special schooling, 66% unmotivated; 35% hyperactive and 35% shy; only 55% of subjects 18+ integrated into normal working life, and most were either extremely introverted or inappropriate extroverted
Tavano et al. (2007)	malformation	27	Disorders of affect and social interaction (33% with pervasive developmental disorder, others showing emotional fragility, rigidity, and anxiety), particularly in vermis; disorders of attention; delay in all developmental milestones
B. Childhood			
Aarsen, Van Dongen, Paquier, Van Mourik, and Catsman-Berrevoets (2004)	tumor	23	65% had behavioral disturbances (anxious, disinhibited, nightmares, posttraumatic stress, hyperactive); most had flattened affect (long term disturbances)
Baillieux et al. (2006)	tumor	1	Pre-surgery was hyperactive, impulsive, and rebellious. 1 yr post-op was emotional indifference, withdrawn, lacked initiation and planning.
Beebe et al. (2005)	tumor	103	Poor scores on adaptive domains of social functioning and communication. Significant impairment on Achenback Internalizing subscale.
Courchesne et al. (1994)	tumor	6	Autistic symptoms; problems shifting attention
Hopyan, Laughlin, and Dennis (2010)	tumor	74	P's normal on all emotion identification except for sadness; also problem with cognitive control of emotions
Karatekin, Lazareff, and Asarnow (2000)	tumor	4	Impaired executive functions
Kingma, Mooij, Metzemaekers, and Leeuw (1994)	tumor	4	1 year post-op: 1 had serious behavior and attention-deficit disorders, 1 had poor sustained attention, 1 had cognitive deficits, and 1 only had slight change in personality (more severe effects in younger kids)
Kupeli et al. (2011)	tumor	9	55% were irritable and emotionally labile following tumor resection
LeBaron, Zeltzer, Zeltzer, Scott, and Marlin (1988)	tumor	15	87% had problems with social relationships and school functioning
Levisohn, Cronin-Golomb, and Schmahmann (2000)	tumor	19	32% were irritable and emotionally labile following tumor resection

Moxon-Emre et al. (2019)	tumor	36	P's worse than controls on facial emotion recognition, but no difference in parent-reported social functioning. Radiation group had more difficulties.
Pollack, Polinko, Albright, Towbin, and Fitz (1995)	tumor	3	All developed post-op mutism and emotional changes (lability, depression) that disappeared by 6-mo. check
Rechtman et al. (2020)	arachnoid cyst	15	Eye tracking during visualization of social interaction scenes: some patients had reduced viewing time to the faces, not explained by general problems with eye movements
Richter et al. (2005)	tumor	12	Minor behavioral and affective changes in some (both positive and negative) but some had no changes at all.
Riva and Giorgi (2000)	tumor	26	Irritability, decreased sociability and eye contact, autism-like; 4/5 cases returned to normal in 3-4 weeks but 1 met criteria for autism for 1 month - behavior was associated with disinhibition-afterwards still had lack of empathy
Wolfe-Christensen, Mullins, Scott, and McNall-Knapp (2007)	tumor	21	24% were anxious, withdrawn, depressed, and had obsessive compulsive disorder

Across these different research findings that include individuals with a range of pathological processes that all co-localize to the cerebellum (see **Table 1**), there are some commonalities. First, the social and emotional deficits never occur in isolation. Instead, they occur as part of a larger spectrum of disordered motor, intellectual, and behavioral functions. Indeed the authors of a meta-analysis termed the behavioral pattern "developmental cerebellar cognitive affective syndrome" (M. Bobylova et al., 2006) in reference to a neurological disorder termed cerebellar cognitive affective syndrome (CCAS), observed in some adults following cerebellar damage (Manto & Mariën, 2015). Second, more diffuse damage that includes portions of the brainstem and the peduncles is associated with worse social outcomes compared to damage restricted to the cerebellum (Brossard-Racine, du Plessis, & Limperopoulos, 2015). Last, the social and affective deficits tend to be profound and of long duration. Indeed, authors of several studies commented that a high percentage of children in their studies met criteria for ASD (Limperopoulos et al., 2007; Limperopoulos et al., 2014), which is consistent with the view that gross cerebellar damage early in life is one of the highest known risk factors for the later diagnosis of ASD. Remarkably, as early as 1991, it was reported that individuals with Joubert Syndrome, a genetic disorder that has a

signature dysgenesis of the cerebellum, displayed symptoms of ASD (Holroyd, Reiss, & Bryan, 1991).

3.2. EARLY CHILDHOOD: CHANGES IN SOCIAL BEHAVIOR ARE STRIKING

Around 1200 children in the United States are diagnosed with brain tumors each year and of that number, around 70% are found in the intracranial cavity that contains the brainstem and cerebellum, and are thus termed *posterior fossa* tumors (Mortimer, 2011). Upon resection of these tumors, approximately one fourth of the cases develop posterior fossa syndrome which is defined as a temporary mutism lasting no more than 6 months, as well as extreme emotional lability and changes in social behavior (Kirk, Howard, & Scott, 1995). This disorder has been extensively researched and there are many high-quality review articles on the topic, with a primary focus on changes in language (Pitsika & Tsitouras, 2013). Changes in social and emotional behavior, which has received less attention, includes excessive irritability, social withdrawal, contextually inappropriate laughter and crying, akin to pseudobulbar syndrome (reviewed in Frazier, Hoffman, Popal, Sullivan-Toole, Olino, & Olson, 2022), depression, and obsessions (Pitsika & Tsitouras, 2013; see Table 1). For a depiction of the topographic location of these tumors, see **Figure 2**.

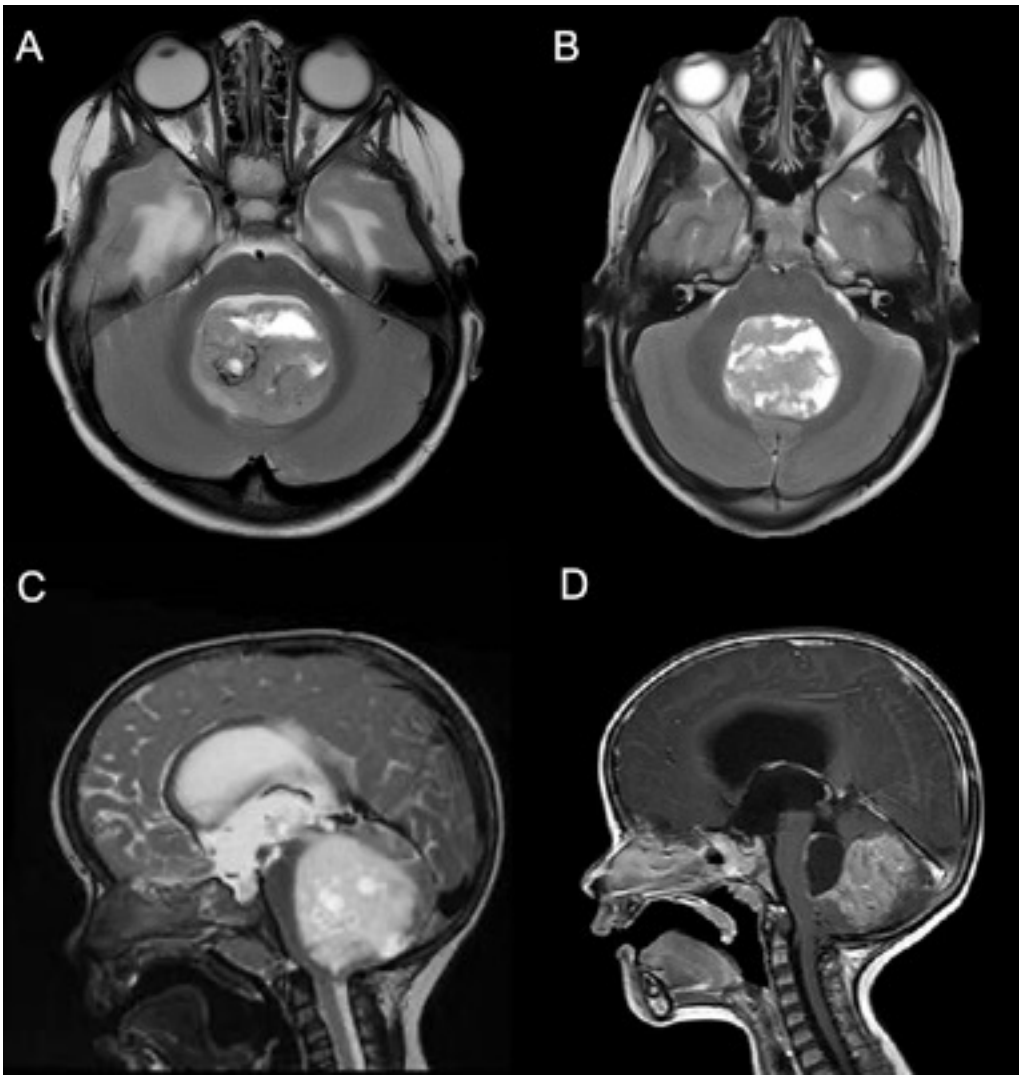


Figure 2. A series of FLAIR scans depicting different posterior fossa tumors in pediatric case studies. A. Axial view in a case that yielded cerebellar mutism following resection (reproduced with permissions from "The Posterior Fossa Society"); B. Axial view in a 16 year old female, presenting with binocularly blurred vision and emotional changes such as "giddiness" (Shah, 2012); C. Sagittal view in a 5-year-old male presenting with cerebellar symptoms (AlMatter). D. Sagittal view of medulloblastoma in a 2-year old female presenting with ataxia, lethargy, and post-operative cerebellar mutism (Gadgil, Hansen, Barry, Chang, & Lam, 2016). Images used with permission of authors. Note that images B-C are reproduced for publication in compliance with Creative Commons licenses.

For instance, Riva and Giorgi (2000) published an influential article that detailed six cases of children who underwent surgical ablation of the right vermis to treat posterior fossa tumors. Following the surgery, five of the children were strongly irritable and avoided all physical and eye contract. Language was poor and they were uncommunicative. After a few days, the social

aversion subsided and they began speaking. However, one 9-year-old girl in the study had, "…complete gaze aversion, severe intolerance of physical proximity to others, complex rhythmic rocking stereotypes of the trunk, and a high number of linguistic eccentricities," (Riva & Giorgi, 2000, p. 1056). She was additionally described as psychotic and her speech was perseverative, echolalic, and at times filled with obscenities. Other children were described who exhibited post-surgical decreases in intellect, had problems in executive tasks, and various language deficits.

Posterior fossa syndrome is fairly common, which has allowed researchers to delve deeply into factors that affect outcomes. First, outcomes are typically worse at younger ages (Dennis, Spiegler, Hetherington, & Greenberg, 1996). Second, the social and emotional deficits are part of a larger spectrum of behavioral changes, and each has its own trajectory of recovery. Last, though it was initially believed that posterior fossa syndrome was caused by damage to the vermis, this view has been supplanted by the idea that the pathophysiology is due to disruption of the CTC white matter tract that sends cerebellar signals to the thalamus then cortex. This view would predict that damage anywhere along this pathway will result in the core symptoms of posterior fossa syndrome. However, in practice, the constellation of deficits observed in posterior fossa syndrome are most common after damage to regions encompassing the start-point of the CTC tract - the dentate nucleus of the cerebellum or cerebellar peduncles - due to the high prevalence of posterior fossa tumors over other types of brain damage in children (Pitsika & Tsitouras, 2013).

One study (Schreiber et al., 2017) tested the 5-year cognitive outcomes of children who had had posterior fossa syndrome compared to children with similar tumor resections that did not result in the same disorder. They found that the posterior fossa group exhibited IQ, working memory, speed of processing, and general attention functions that were more than 1 standard deviation below the mean and that there was almost no recovery of function over the 5-years post-surgery. This suggests that social and emotional deficits may persist over time as well.

As shown in Table 1, evidence linking childhood-onset cerebellar damage to social and emotional changes is very strong. Several factors are not well understood, however. The average age for pediatric posterior fossa tumors is age 9 but they can occur from age 0 to age 18. Are social and

emotional deficits only observed when the tumors appear during a certain age window? Also, what is the longevity of the social and affective deficits?

3.3.ADULTHOOD: CHANGES IN SOCIAL BEHAVIOR ARE MILD

Cerebellar injury in adults typically occurs in the context of stroke or neurodegenerative disorder in individuals over the age of 60. In rare cases, it can give rise to a constellation of symptoms known as Cerebellar Cognitive Affective Syndrome (CCAS). CCAS is typified by deficits in four key areas of cognition, including spatial processing, executive functioning, linguistic abilities, and personality changes (Manto & Mariën, 2015). It has been noted that individuals with CCAS can have flattened affect, reduced emotional expressivity, disinhibition, and childish behavior as well as changes in linguistic output, mutism, and agrammatism. It is believed that the social consequences of CCAS arise following damage to the posterior lobe, especially when the vermis is damaged (Schmahmann & Sherman, 1998).

Jeremy Schmahmann has provided richly detailed case studies delineating the existence of CCAS. However, this disorder remains controversial because it is observed only rarely in the clinic. In addition, the effects of cerebellum damage on readily-observable social behavior in adults are described as rare (Branch Coslett, 2022), transient (Schmahmann & Sherman, 1998), or mild (Wang et al., 2014). This should be contrasted to the striking changes in social comportment observed after lesions to orbitofrontal cortex (e.g. pseudo-psychopathy; Schneider & Koenigs, 2017) or after bilateral amygdala-temporal polar damage (e.g. Kluver-Bucy disorder), or following degeneration of the temporal poles and orbitofrontal cortex (e.g. behavioral variant of frontotemporal dementia/primary progressive aphasia; Olson, Plotzker, & Ezzyat, 2007). The point of this comparison is that adult-onset cerebellar damage does not cause adult-onset autism spectrum disorder, Kluver-Bucy like symptoms, or pseudo-psychopathy.

Do individuals with CCAS have problems with ToM? We focus specially on ToM because it has been widely tested in different patient populations plus it is disrupted in ASD (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). Moreover, researchers have specifically linked ToM to cerebellar processing (Van Overwalle, Van de Steen, van Dun, & Heleven, 2020; Van Overwalle

& Vandekerckhove, 2013). In one case study, the authors studied a 52-year-old patient who had bilateral cerebellar infarcts that damaged both gray matter and white matter three months prior to testing. He exhibited marked deficits in theory of mind capacities as measured by the Reading the Mind in the Eyes task (RMET) and Faux Pas task (FPT). This can be conceptualized as difficulties in perceptual emotion attribution and in the externalization of social norm behavior. This finding bolsters the theory that the cerebellum indispensably subserves affective-cognitive processes even in adulthood, and hints at the presence of ToM deficits in CCAS (Roldan Gerschcovich, Cerquetti, Tenca, & Leiguarda, 2011).

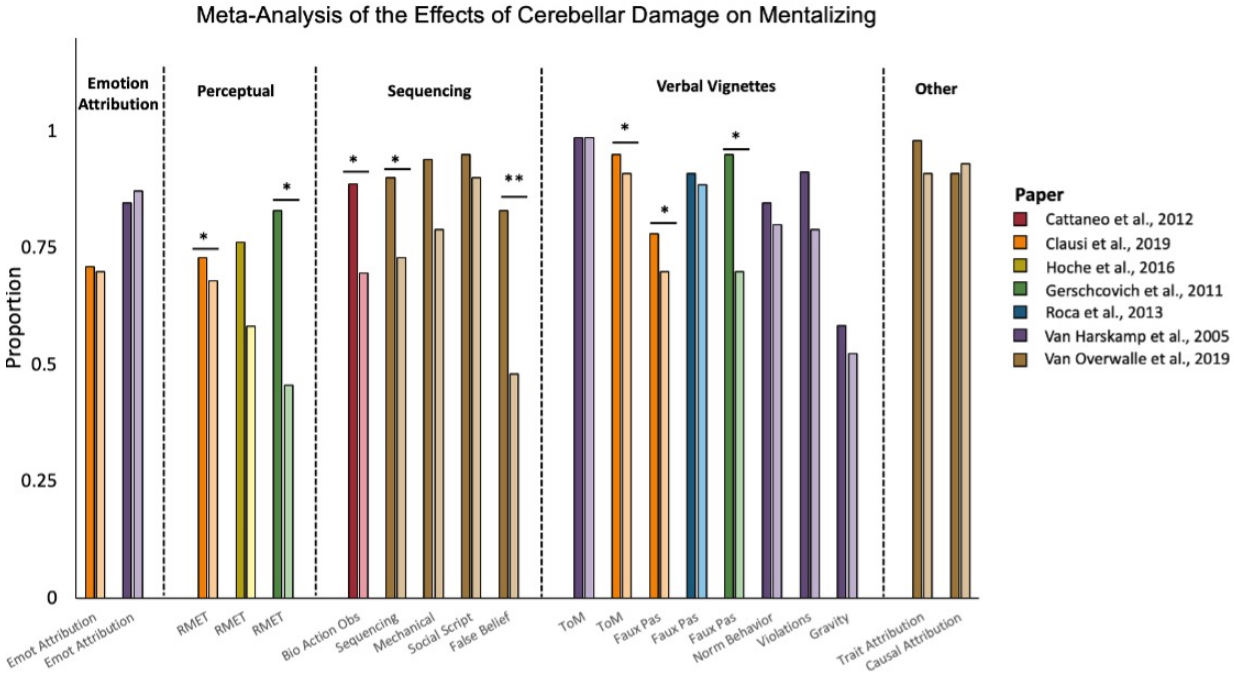


Figure 3. A compilation of studies in which adults with cerebellar damage had theory of mind formally tested. Studies were excluded if they did not test a control group (e.g., Lupo et al., 2015; Moriarty et al., 2016; Parente et al., 2013), failed to provide numerical values for the control group (e.g., Garrard et al., 2008), or did not report raw accuracy scores for each group (e.g., Beuriat, Cohen-Zimerman, et al., 2022). Developmental studies were excluded because pediatric populations are rarely administered ToM tests. Studies are grouped by task category, then by article.

Other researchers have investigated ToM in larger samples of adults with cerebellar damage who do not necessarily exhibit CCAS. In Table 2, we list all known studies and in Fig. 3, we plot the findings (Cattaneo et al., 2012; Clausi et al., 2018; Hoche, Guell, Sherman, Vangel, &

Schmahmann, 2016; Roca, Gleichgerrcht, Ibanez, Torralva, & Manes, 2013; Roldan Gerschcovich et al., 2011; van Harskamp, Rudge, & Cipolotti, 2005; Van Overwalle et al., 2019). Note that the pathophysiology is mixed, the time since damage is variable, and different tasks are used. To understand whether any deficits are more apparent in some tasks than others, we clustered findings by task modality.

Table 2. A comprehensive list of studies that measured changes in social behavior after cerebellar lesions in **adults**. Purely descriptive papers were not included. Studies in which cerebellar insult occurred early in life (e.g., agenesis) can be found in Table 2. Papers are grouped by category, then listed alphabetically. Abbreviations: International Affective Pictures (IAPs); Patient (P); Healthy controls (HC); Reading the Mind in the Eyes Task (RMET); spinocerebellar ataxia (SCA); Tubingen Affect Battery (TAB); penetrating traumatic brain injury (pTBI); Voxel-based lesion-symptom mapping (VLSM).

Author	Etiology	N	Description
Emotion Recognition			
Adamaszek et al. (2019)	Stroke	22	P's impaired in recognizing facial and speech-related emotions (task= TAB)
Adamaszek et al. (2014)	Stroke	15	P's impaired in recognizing facial and speech-related emotions (task= TAB)
D'Agata et al. (2011)	Neurodegenerative	20	P's normal for basic emotions but some deficits in recognizing negative emotions and social emotions
Turner et al. (2007)	Stroke	6	Normal emotional thoughts and reactions to IAPs pictures
Theory of Mind			
Beuriat, Cohen-Zimerman, et al. (2022)	pTBI	24	P's performed the same as HC on ToM and faux pas tasks. VLSM showed worse ToM performance was associated with lesions in left cerebellum.
Cattaneo et al. (2012)	Stroke	8	P's performed worse than controls when they had to find the odd-picture out of 4 that depicted different biological actions.
Clausi et al. (2019)	Neurodegenerative	27	P worse than controls on (1) RMET; (2) faux pas; (3) Happe's advanced ToM task
Garrard, Martin, Giunti, and Cipolotti (2008)	Neurodegenerative	9	P's worse than controls at ToM vingettes. P's normal on emotion attribution tests and Faux Pas
Hoche et al. (2016)	Mixed etiology	31	P's worse than controls at RMET
Lupo et al. (2015)	Spastic ataxia	6	P's worse than controls at (1) RMET; (2) Happe's ToM task. Normal on emotion attribution task (except embarrassment)
Moriarty et al. (2016)	Neurodegenerative	13	At follow up, all P's had impaired and worsened ToM on vignettes as well as executive function tasks

Parente et al. (2013)	Neurodegenerative	1	P's worse than controls on (1) ToM vignettes; (2) RMET; (3) faux pas. Also, mild executive dysfunction.
Roca et al. Roca et al. (2013)	Stroke	11	Normal ToM on Faux Pas Test
Roldan Gerschcovich et al. (2011)	Stroke	1	P impaired at (1) RMET; (2) Faces Test; (3) Faux Pas Test
Sokolovsky et al. (2010)	Neurodegenerative	8	P's had normal ToM on vignettes. Mild, mixed performance on emotion attribution task.
van Harskamp et al. (2005)	Superficial siderosis	6	P's worse than controls at ToM vignettes. P's=controls on emotion attribution tests and Faux Pas test.
Van Overwalle et al. (2019)	Neurodegenerative	10	P's tested on multiple ToM tests. P's only showed deficits on Picture sequencing task
Biological motion perception			
Jokisch et al. (2005)	Stroke	7	Point light walkers in noise: normal performance. Non-biological motion: 4/7 patients did not solve the task
Sokolov et al. (2014)	Stroke	1	Point-light walkers in noise. Year 1: lower threshold; Year 3: normal.

The results show that in two of the categories – "emotional attribution" and "other", no deficits were reported across four tests. Two out of three studies reported deficits on RMET, in which the task is to look at pictures of eyes and assess the feelings of the person behind the eyes. Three out of five experiments in two papers reported deficits on "sequencing" tasks. This group of tasks requires subjects to put a disordered sequence of pictures depicting a social scenario or mechanical series of events in the correct sequence. This group of tasks may also require subjects to choose the image that is not part of a logical sequence of events from a set of disarranged photos depicting biological motion (Cattaneo et al., 2012; Van Overwalle et al., 2019). Last, individuals with cerebellar damage appear to show mild to no deficits (three of eight) on ToM tasks that employ verbal vignettes in which ToM is employed to understand character's beliefs, feelings, or intentions while reading a brief narrative (see also: Beuriat, Cohen-Zimerman, et al., 2022) .

It is worth spending a moment to consider the effects of cerebellar damage on tasks that involve sequential processing, because it has been proposed that this is one of the fundamental processes of the social cerebellum (Leggio & Molinari, 2015). Van Overwalle et al. (2019) found that patients with cerebellar damage were impaired on a social picture sequencing task, but not the

other ToM tasks. Although the sequencing idea is intriguing, across the reviewed studies, this idea does not hold up since two out of three studies reported that cerebellar patients had deficits on a task that uses static images - Reading the Mind in the Eyes Task (RMET). Also, two other studies explicitly tested perceptual thresholds to biological motion after cerebellar damage – a task that requires sequence perception and trajectory prediction – but found only modest deficits that were no longer apparent 1-year post injury (Jokisch et al., 2005; Sokolov et al., 2014; Sokolov, Gharabaghi, Tatagiba, & Pavlova, 2010).

Emotion recognition is important for accurate ToM, especially in tasks using facial stimuli, like RMET (Mitchell & Phillips, 2015). Using laboratory tasks, there are some reports of mild problems recognizing emotions from faces (Adamaszek et al., 2014; D'Agata et al., 2011; Hoche et al., 2016; Sokolovsky, Cook, Hunt, Giunti, & Cipolotti, 2010) and matching facial emotion with vocal prosody (Adamaszek et al., 2014; Adamaszek et al., 2019). As measured by these tasks, the effects are unreliable (see Table 2).

Our review of the lesion literature shows that changes in social comportment are only rarely reported after adult-onset cerebellar damage (for a similar view see (Beuriat, Cristofori, Gordon, & Grafman, 2022; Wang et al., 2014) and when found, tend to dissipate quickly. In the laboratory, effects of adult cerebellar damage on ToM exist but they are inconsistent across task and study, and when observed, are mild (Roldan Gerschcovich et al., 2011). In contrast, there are dramatic changes in social functioning following early life cerebellar damage.

There are several explanations for the developmental differences that cannot be ruled out by this review and thus must be investigated by future researchers. First, lesions in children could be relatively larger and more likely to impinge on Crus I/II and thereby affecting social behavior to a greater degree (in other words, dose and location variables may differ between children and adults). Second, lesions in children could affect deep nuclei and white matter tracts linking the cerebellum to the cerebrum to a larger degree. Last, it is possible that the tasks used to assess social cognition in adults do not have sufficient variance or do not sufficiently capture the nature of cerebellar social deficits.

4.CELLULAR MECHANISM OF DYSFUNCTION

Studies in non-human animals have convincingly shown that social behavior can be altered by experimentally perturbing cells and circuits in the cerebellum. Purkinje cells are the primary output cell of the cerebellum and they overwhelmingly inhibitory. Purkinje cells in Crus I/II of the cerebellum (see Box 1 for neuroanatomical details) can have dopamine receptors. One study showed that when dopamine receptors on mouse Purkinje cells were inactivated, mice became less social and lost their preference for social novelty (Cutando et al., 2022). This extends prior work showing that inhibition of Purkinje cells in the rodent cerebellum can cause abnormal sociability and increased repetitive grooming behaviors (Tsai et al., 2012). Related to this, a recent study tested genetically altered mice using a social task in which the mice could choose to spend time in a chamber with another mouse, an empty chamber, or a chamber with a non-social object. At baseline, mice preferred the social chamber. However, after a pathway between the deep cerebellar nuclei and ventral tegmental area (VTA) was opto-genetically inactivated, mice lost this preference. Continuous inactivation of this pathway completely prevented the expression of social interest. Because the VTA is the origin of the dopaminergic cell bodies that form the reward circuitry of the brain, the authors interpret this finding as showing that portions of the cerebellum modulate the VTA which in turns, alters social motivation and social preferences (Carta et al., 2019). All of these studies were in adult rodents.

Is there any evidence for a developmental gradient? Badura et al. (2018) chemo-genetically perturbed cerebellar output (Crus I/II) in juvenile mice, let them grow up, then measured social preference in adulthood, and found that it was diminished. In contrast, when the same chemo-genetic perturbation occurred in adulthood, it did not alter social preference, once again suggesting that there is a shift in the computations performed by the social cerebellum as we mature.

5. THE STEEP AGE GRADIENT MAY BE CAUSED BY SHIFTS IN DOMINANT COMPUTATIONAL MODELS

The reviewed findings raise the question: how can we explain this age gradient? We attempt to do so here. A well-accepted model of cerebellar processing in the motor domain is the *forward internal model*. The goal of the forward model is to make prediction on how a given action will reach a desired outcome. This model describes how the cerebellum receives a command signal

from the cerebrum, compares that command to sensory feedback, and then creates a prediction error signal that fine tunes the command based on what the desired outcome is and sends it back to the cerebrum (Ito, 2008). This is contrasted with the *inverse internal model,* which seeks to produce the best actions to achieve a desired outcome. Here, the cerebellum would receive a desired outcome from the cerebrum and produce the action that reaches that outcome. While both models are forms of supervised learning which relies on a large amount of data where the action to outcome pairings are known, the inverse model in particular is optimal when extensive training has occurred.

Let us consider an example of how these models would be used to understand social knowledge. As we navigate through our social world, we meet new individuals and are quickly able to interact and carry a narrative with them based on solely knowing the relationship between individuals. For example, if a bartender wants to make small talk to their patrons, they may observe two people sitting together, talking, learning closely together, and infer that they are a couple. The bartender can then ask about their relationship, how long they have been dating, etc. This is done with the forward model of the cerebellum considering the features of the observed patrons (e.g., leaning in close to each other) and making a guess that they are a couple in order to achieve the desired goal of starting a conversation. If this prediction is inaccurate, for example if the patrons are actually siblings, then the cerebellum's forward model adjusts the bartender's mental model of their relationship and she can try talking about different things. The inverse model in this example would be if the patrons are known to the bartender and are known to be a couple. The inverse model then would readily provide information on appropriate subject matters that the bartender could use to initiate small talk.

We propose that the cerebellum builds and deploys mental models that shift from one dominant model-type to another model-type, over the course of development. As a child, the cerebellum's forward internal model mechanism would help create social mental models of the world that offer predictions of outcomes that are constantly being tested and retested to refine the model. These models would be stored in portions of the cerebral cortex referred to as the "social brain". If for some reason the cerebellum was not able to build social mental models, or was not able to access social mental models (because structural connections between the cerebellum and cortex were

damaged), individuals would find it difficult to predict how others might act or how their words or actions might be received, which would cause them to find social interactions frightening and unpredictable. This is at the heart of Wang et al. (2014) proposal that early damage to the cerebellum causes a *developmental diaschisis*. This view suggests that the cerebellum drives the maturation of functional circuits in the evolutionarily newer, and more functionally plastic, association cortex of the cerebrum to which it is connected. As such, cerebellar damage early in life causes downstream dysfunctions in the cerebrum because the cerebellum cannot create forward models that functionally tune the frontal lobe.

As we mature, models of the world are built up by the forward model to the degree that adjustments of those models by the cerebellum are needed less and less frequently. Because the forward model has already built up a repertoire of highly predictive social mental models, cerebellar damage in adulthood does not lead to dramatic changes in social thoughts or behavior and instead, is associated with mostly normal theory of mind and social comportment. We propose that at this point, the forward model ceases to be important for social behavior, and there is a shift in the dominant model executed by cerebellar computations. The dominant mode of activity is now the inverse internal model of the cerebellum, which executes automated thoughts and behaviors. Thus, social deficits following cerebellar damage in adulthood would likely take the form of slowness or difficulty initiating an otherwise automated response. Slowing has been observed in normal aging with cerebellum correlates (Bernard, 2022; Bernard & Seidler, 2014).

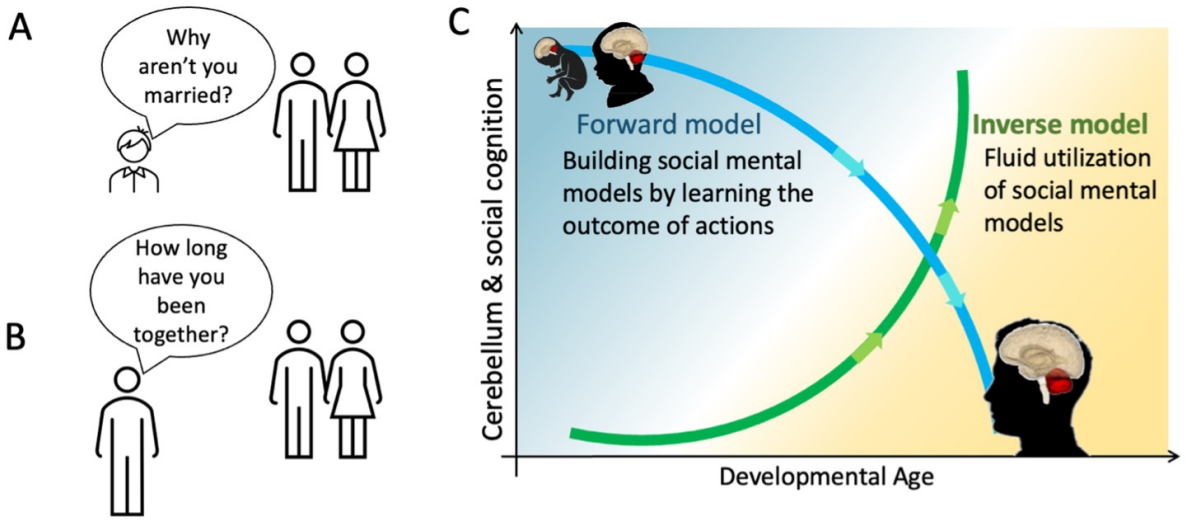


Figure 4. Cerebellar internal models and social development. A. The forward model is critical for the early development of social mental models which are used for learning about and predicting other people's attention, how they move and gesture, their motives, internal thoughts and emotions, and acquiring social knowledge, as illustrated here. B) The inverse model is important for the fluid utilization of social mental models which by adulthood, are mature. This is important for automatic mentalizing, deploying social norms, language pragmatics, and the quick processing of emotions. C) We hypothesize that earlier in life, the forward model will be critical to build social models and map accurate actions to desired outcomes. Later in life, the inverse model will be more widely used so that individuals can seamlessly interact with others. By this time, an individual will have extensive experience with the social world, new types of social situations are infrequent, and thus the forward model is not as crucial as it once was.

Interpreting existing fMRI studies in adults (for reviews see Van Overwalle, Baetens, Marien, & Vandekerckhove, 2014; Van Overwalle, D'Aes, & Marien, 2015) through the lens of this model can be difficult. If the social situation used in the study is easy and familiar, like many ToM tasks, activations likely reflect deployment of the inverse model. If the task requires learning new social rules or behaviors with error-driven feedback, the activations likely reflect deployment of the forward model. It is also possible that the cerebellum receives the input and concludes via low prediction errors, that no adjustments need to be made to what the cerebrum is computing.

5.1.TESTING THE MODEL

The most basic prediction of the model is that the forward model will be relatively more active early in life, when young children are forming models of the social world, as compared to in

adulthood. One way to test this is to first map the monosynaptic pathway between the cerebellum and VTA using tractography. Assuming this pathway exist, we predict there will be a positive relationship between tract microstructure and developmental age that will plateau in late adolescence. Microstructure may also correlate with social interest metrics and autism scores.

Second, associating a rewarding, pleasurable feeling to a social entity is a building block of all social cognition; there is evidence that low levels of social interest stunt the development of adult-levels of social cognition (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012). Rodent studies have linked Crus I/II to social preference in young, but not older mice. To test whether this developmental difference extends to humans, young children and adults could be asked to simply look at social and non-social scenes in the scanner while their gaze is being tracked. We predict relatively greater activations in children's Crus I/II, as compared to adults, while they gaze at social scenes and we would predict that activations would remain high across repetitions of the same social stimulus while adult activations would drop off steeply across repetitions as a sign that the forward model is already built. In addition, there should be strong functional connectivity between socially-sensitive regions of Crus I/II and the VTA during the social task but not the non-social task, which should correlate with developmental age.

Third, to test whether the adult cerebellum is involved in social learning using the forward model, a novel fMRI task could be designed in which participants engage in learning through trial and error with feedback, to acquire knowledge about the social norms and etiquette of space aliens. Social norms could be embedded in the space aliens body language. Based on our prior findings (Metoki et al., 2022), we predict that Crus I/II will be activated when learning the new body language and that activations will decrease more quickly in individuals who learn more quickly. We also predict that the social-activated region of Crus I/II will be functionally synchronized with socially-sensitive regions of the cerebrum. We additionally predict that adults with damage to Crus I/II will be able to learn alien social norms, but they'll do so extremely slowly and using explicit, language-based learning mechanisms.

6. CONCLUSIONS

A dominant belief in neuroscience is that the developing brain is more plastic than the adult brain (Johnston, 2009). This can be extremely beneficial because it allows children to rapidly acquire new skills while also making them more resilient to brain injury as compared to adults. However, the cerebellum does not abide by this rule: early life injury causes far more profound effects than later-life injury. In fact, the negative consequences of cerebellar damage show an inverted age gradient with strikingly worse outcomes from damage at the earliest periods of life, and milder and rapidly-recoverable effects on social behavior from damage later in life.

The reviewed findings indicate that early life cerebellar damage can serve as a model system for understanding the development of normal social behavior as well as understanding developmental disorders defined by social impairments, such as ASD. We are certainly not the first to observe this. As noted by D'Mello & Stoodley (2015), "*... damage to the cerebellum can directly lead to an ASD diagnosis in a way that damage to other regions commonly implicated in ASD cannot, including the prefrontal cortex, basal ganglia, and parietal cortex,*" (D'Mello & Stoodley, 2015, p. 13; Fatemi et al., 2012; Rice & Stoodley, 2021). This view is not without controversy; a recent large-scale study found no differences in cerebellar gross anatomy in individuals with ASD (Laidi et al., 2022) however it could be argued that examination of gross morphometric differences is too crude of an analysis. Obviously, most individuals with ASD do not have cerebellar lesions, but there are many other ways in which the cerebellum can become dysfunctional. For instance, cerebellar Purkinje cells are particularly sensitive to toxic exposure (Fatemi et al., 2012). Prenatal alcohol exposure and childhood lead exposure damage the developing cerebellum (Kumar, LaVoie, DiPette, & Singh, 2013; Sanders, Liu, Buchner, & Tchounwou, 2009) and newer findings show that perfluorinated carboxylates accumulate in the hippocampus and cerebellum (Eggers Pedersen et al., 2015). Whether any of these toxins are part of the pathogenic process leading to dysfunction in cells and circuits and ultimately a diagnosis of ASD is currently not known, but is mechanistically plausible should it occur early in life. Some of these toxins could trigger the proliferation of microglia, causing Purkinje cells to become over-excited (Yamamoto, Kim, Imai, Itakura, & Ohtsuki, 2019) and hence, too much inhibitory output from the cerebellum. This could decrease dopamine release from the VTA, and ultimately, decrease social behaviors. None of these cellular effects would necessarily produce macroscopic changes in cerebellar volume.

Our review is fairly dense so here we summarize our main points:

(1) A region of the posterior cerebellum, Crus I/II, is linked to social cognition. This region is structurally connected via the CTC and CPC tracts to social regions in the frontal lobe; (2) the cerebellum modulates functional networks in cerebral association cortices involved in social behavior (Adolphs, 2009), and potentially is a driver of functional specialization in these regions, as described by Wang et al., 2014; (3) disturbed cerebellar-VTA communication can alter social behavior in rodents; whether this circuit is involved in human social behavior is not currently known because histology is lacking. Last and most importantly, (4) the effects of cerebellar damage depend on the age of insult with much worse outcomes on social behavior from early life damage, and mild deficits from damage in adulthood. We propose that this is because early in life, the *forward model* dominates cerebellar computations, which helps build predictive models of the social world, while in adulthood, the *inverse model* dominates cerebellar computations (see **Figure 4**).

As compelling as these findings are, research on social cognition and the cerebellum has progressed in fits and starts. This review has identified several problems that have hindered progress. At a broad level, many developmental cognitive neuroscientists may be unaware of the cerebellum's early essential role in laying down the substrates of adult social behavior. At a practical level, the cerebellum is often an afterthought in fMRI studies so full coverage of this structure is rare and direct hypothesis testing is unusual. The large number of strong and consistent findings showing that early perturbations of the cerebellum give rise to a dramatic increase in ASD as well as the maturational gradient described in this paper should give all developmental neuroscientists pause. Outstanding questions are described in Box 2.

Box 2. Outstanding Questions

(1) Does the infant cerebellum have the same functional topography as the adult cerebellum? The rapid development the cerebellum during infancy makes the answer to this question uncertain (Garfinkle et al., 2020).
(2) One difference between the cerebellum and cerebrum is that the cerebellum's functions are largely unconscious such that the negative effects of cerebellar lesions can be partially compensated for by bringing the issue at hand to conscious awareness. Is the cerebellum more

relevant for implicit social learning as compared to explicit (Siciliano & Clausi, 2020)? Implicit ToM emerges very early in life, is crucial for later explicit ToM development. It is closely linked to eye-movements – which are calibrated by ancient structures in the cerebellum (Kheradmand & Zee, 2011) - since infants learn to use other's gaze to predict social outcomes (Slaughter, 2015).

(3) Self-recognition and a sense of agency develops in late infancy and is thought to be a prerequisite for ToM. The cerebellum creates a prediction (e.g., the forward model) of the sensory consequences of our actions which fundamentally allows us to distinguish self from other. Does early life cerebellar malfunction to Crus I/II disrupt a sense of self leading to a cascade of social deficits including problems with ToM? Problems in this mechanism could also cause problems distinguishing self-produced from other-produced thoughts and voices, such as that experienced in psychosis (Montgomery & Bodznick, 2016).

(4) The developmental window of the social cerebellum's role in adolescence is still uncharted. Do the preteen and teenage years constitute a significant sensitive period for cerebellum-dependent social maturation?

(5) How does neural plasticity manifest across development in terms of the cerebellum's afferent connections to the cerebrum and the efferent connections back to it? Is there a developmentally-informed gradient in relation to the strength of the structural and functional connections in the forward and inverse directions as we age? That is, do socially-relevant forward connections attenuate as inverse connections strengthen as we age?

(6) Is the developmental shift from forward to inverse models occur universal or limited to certain functions? How does advanced age affect the deployment of forward models? Do forward and inverse computations employ the same circuits or different circuits within the cerebellum?

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Competing Interests

None

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