The Cerebellum (2022) 21:1123–1134
https://doi.org/10.1007/s12311-021-01330-y

COMMENTARY

# The Role of the Posterior Cerebellum in Dysfunctional Social Sequencing

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Accepted: 27 September 2021 / Published online: 12 October 2021


## Abstract

Recent advances in social neuroscience have highlighted the critical role of the cerebellum in social cognition, and especially the posterior cerebellum. Studies have supported the view that the posterior cerebellum builds internal action models of our social interactions to predict how other people's actions will be executed and what our most likely responses are to these actions. This mechanism allows to better anticipate action sequences during social interactions in an automatic and intuitive way and to fine-tune these anticipations, making it easier to understand other's social behaviors and mental states (e.g., beliefs, intentions, traits). In this paper, we argue that the central role of the posterior cerebellum in identifying and automatizing social action sequencing provides a fruitful starting point for investigating social dysfunctions in a variety of clinical pathologies, such as autism, obsessive–compulsive and bipolar disorder, depression, and addiction. Our key hypothesis is that dysfunctions of the posterior cerebellum lead to under- or overuse of inflexible social routines and lack of plasticity for learning new, more adaptive, social automatisms. We briefly review past research supporting this view and propose a program of research to test our hypothesis. This approach might alleviate a variety of mental problems of individuals who suffer from inflexible automatizations that stand in the way of adjustable and intuitive social behavior, by increasing posterior cerebellar plasticity using noninvasive neurostimulation or neuro-guided training programs.

**Keywords** Social mentalizing · Emotional mentalizing · Crus · Mental disorders · Non-invasive stimulation

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## What Is the role of the Cerebellum in Dysfunctional Social Thinking?

When making decisions or executing actions, it is crucial to understand the current situation and to predict upcoming events. In social interactions, we need to understand the "mind" of other persons as well as our own "mind" (i.e., intentions, preferences, beliefs, traits, and feelings). The capacity to read the "mind," including thoughts and emotions of self and others, is termed mentalizing and is postulated to be a key factor to predict, control, and anticipate upcoming behavioral and emotional responses. *Social mentalizing* or "theory of mind" is supported by a prefrontal-temporal circuitry (the *mentalizing* network which consists of a larger part of the default mode network; see meta-analyses by [1–3] while *emotional mentalizing* or "empathy" is supported by roughly the same network extending to areas in the *salience/ventral attention* and *somatosensory* networks (see [2]). Recent research also points to the involvement of another key brain region, the *posterior cerebellum*. The role of the posterior cerebellum in this social and emotional mentalizing network was for a long time ignored but has recently been uncovered with respect to social mentalizing [4, 5]. Early evidence on the role of the cerebellum in psychiatric pathologies demonstrating emotional or social dysfunctions has been reported by several labs [6, 7], and novel breakthroughs in uncovering the specific cerebellar dysfunctions in these pathologies is the focus of this paper.

## Goal and Outline

In this opinion paper, we want to highlight recent evidence demonstrating that the posterior cerebellum has a crucial predictive role in a variety of mentalizing functions [8–11] and propose research to extend our insight in this novel, but crucial role for social cognition. More importantly, we further suggest that the posterior cerebellum may play a central role in social dysfunctions observed in many clinical pathologies. Indeed, deficits in mentalizing at large, and in the posterior cerebellum in particular, may play a crucial role in the onset and maintenance of psychiatric disorders related to limited mentalizing and emotion recognition, such as *autism spectrum disorder* [12–14], *depression* [15, 16], *bipolar disorder* [17]**,** *obsessive compulsive disorder* [18–21]**,** *addiction* [22–25]**,** and *schizophrenia* [26–30].

Given the recent evidence that this posterior location is preferentially involved in social mentalizing (especially Crus 2, see [9]), we put forward the hypothesis that a number of dysfunctions in these psychiatric pathologies are related to deviances in social and emotional mentalizing, such as impaired understanding of others, social conduct, self-representation, and self-evaluation. These novel theoretical insights on the social role of the posterior cerebellum and its clinical implications may aid to develop several *neuro-guided tools and procedures* to alleviate suffering in these pathologies. Before introducing our hypothesis, we first provide a brief overview of the function of the cerebellum.

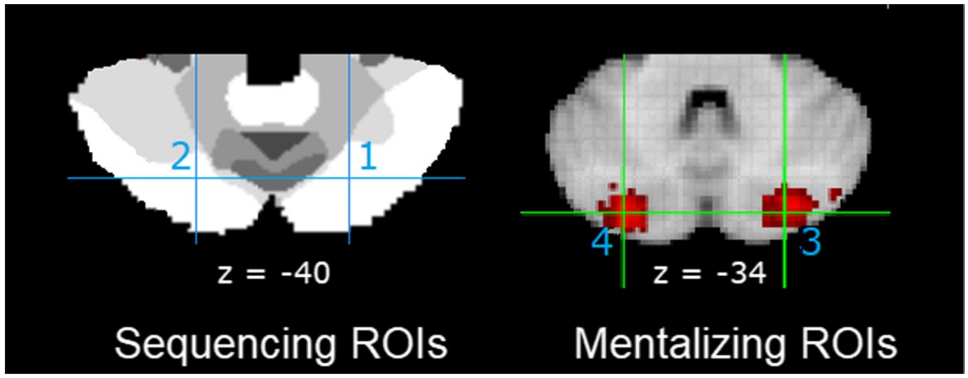
## The Social Function of the Posterior Cerebellum

A primary function of the cerebellum overall is to support *sequence learning and memories* supported by internal models that underpin not only motor acquisition, but also non-motor mental processes involving event sequences (e.g., [31]). According to this "sequence detection hypothesis," this allows the cerebellum to automatize and fine-tune a variety of motor and non-motor/mental (cognitive, social, and affective) action sequences and to send back prediction error signals when an anticipated event sequence does not fit with current behavior and its intended consequences [31].

When looking at complex social interactions or facial expressions, attention to a sequence of social and facial cues is required to infer the mental belief or emotion conveyed by the other person [32–34]. For example, it matters a lot to our impression of a person whether that person enters a room first or whether he or she opens the door and lets another pass first or when a person instigates an aggressive act or merely responds in self-defense. According to the sequence detection hypothesis, the cerebellum encodes typical social action sequences encountered in our interactions with other people and which are more or less rewarding [35]. This allows us to automatically predict and intuitively execute desirable behaviors in future interactions to attain successful outcomes and to automatically and rapidly adjust behavioral execution when the outcome is unexpected or undesirable.

Only recently, the contribution of the posterior cerebellum to social interaction processes and prediction has been recognized. A large-scale meta-analysis of fMRI studies on social cognition by Van Overwalle et al. [4] consistently found activation in the cerebellum. One of the most predominant activations during mentalizing was observed in the posterior cerebellar Crus 1 and 2. Although authoritative reviews on the functional anatomy of the cerebellum reported that these posterior areas are mainly involved in processing of language, words, and narratives, besides mentalizing [36, 37], a meta-analysis by [9] (see Fig. 1) suggests that the *posterior cerebellar Crus 2* appears to be a preferential site of social and emotional mentalizing. It was convincingly shown, across multiple studies, that social mentalizing recruits a circuitry of closed-loops between the posterior cerebellar Crus 2 and the temporo-parietal junction (TPJ),

**Fig. 1** A top view on the region of interest (ROIs) in the posterior cerebellar Crus 2 preferentially engaged during social sequencing tasks (ROIs 1–2) and in the social mentalizing automated meta-analysis of NeuroSynth (ROIs 3–4; adapted from[9])




and so to the medial prefrontal cortex (mPFC), two key cortical areas of the mentalizing network [9, 38, 39]. Animal research with mice [40] even demonstrated the existence of direct connections from the cerebellum to the brain's reward center (ventral tegmental area), and how this modulates preferences for social contact.

### Social Dysfunctions and the Posterior Cerebellum

With respect to clinical populations, recent studies have demonstrated impairments in social and emotional mentalizing in several pathologies. A clinical disorder entailing severe social disorders is *autism* [14] which includes dysfunctions in spontaneous social mentalizing (e.g., [41]), and social dysfunctions also exist in other clinical populations [42]. For example, *addicted* patients have long-lasting impairments in emotional recognition [43–46] and display reduced social mentalizing skills [25, 47–51]. Similar mentalizing dysfunctions have been observed in *depression* [15], *obsessive–compulsive disorder* [18], and *schizophrenia* [26, 27].

We will argue that the posterior cerebellum might play a critical role in many dysfunctions of social and emotional mentalizing and behavior in these clinical disorders. Evidence that these mentalizing deficits are related to the posterior cerebellum has been put forward for symptoms of impaired social interaction in autism [12], impaired emotion recognition [26], and for excessive addiction-related craving [52, 53]. However, mentalizing impairments related to the posterior cerebellum have not yet been systematically explored in many clinical pathologies.

## Open Questions

Given the lack of research on the social (sequential) mentalizing function of the posterior cerebellum, especially for clinical populations, there is a need for a novel theoretical perspective on the role of the posterior cerebellum in social circuitries in the brain, to provide compelling functional as well as anatomical evidence for it, such as spelling out the functional pathways between cerebellar-cortical areas, and to make progress in the causal or modulatory role of the posterior cerebellum using posterior cerebellar neurostimulation in healthy and clinical populations [54].

Although social (sequential) mentalizing tasks or posterior cerebellar stimulation techniques on healthy participants show promising results, these methods have not been frequently tested in clinical populations with impaired social cognition, such as autistic spectrum, depression, bipolar, obsessive–compulsive, addictive, or schizophrenic disorders. The role of social impairments related to dysfunctional posterior cerebellar processes in healthy participants and in these pathological populations is still unclear. Moreover, the posterior cerebellum could be a potential new treatment target, given that classical neurostimulation techniques — though beneficial to some extent — do not yet result in large response and remission rates in clinical samples [55–57]. Providing empirical answers on these and related questions might have an important impact on the regular diagnosis, therapy, and clinical management of these pathologies.

## Our Hypothesis and Initial Evidence

In an extension of the *sequence detection hypothesis* (e.g., [31]), we claim that the cerebellar contribution to mentalizing sequencing about thoughts and emotions of others and self has gone awry in many clinical pathologies. Our key hypothesis is that the learning, automatization, and prediction of thought-emotion-action sequences in the posterior cerebellum has become inflexible and rigidly automatized, revealing lack of plasticity to learn new sequences, resulting in their chronical under- or overuse not only in cerebellar patients [58] but also in many other clinical pathologies. We hypothesize that a major characteristic of social cerebellar pathologies is that, as soon as a situational trigger is present, the posterior cerebellum automatically runs the associated thoughts and feeling as mental routines either shallowly or repeatedly, resulting in blunted or exaggerated thoughts and feelings. This leads to inflexibility in previously rewarded social and emotional behaviors with little tolerance and plasticity for learning more appropriate social conventions and responses in the given context, leaving little room for situational adjustments and more adequate social conduct.

To break this continuous cycle of maladaptive thought-feeling-action sequences, fundamental knowledge on posterior cerebellar functions and connectivity with cortical areas is crucial for a complete understanding of social processes, as well as the development of alternative ground-breaking approaches to neurostimulation and neuro-guided behavioral treatment. Behavioral programs focusing on the cerebellum's role in learning and automatizing social action sequences (and unlearning inappropriate sequences) as well as noninvasive brain stimulation focusing on the posterior cerebellum (e.g., *transcranial magnetic stimulation* [TMS], *transcranial direct current stimulation* [tDCS], *deep brain stimulation* [DBS]; [54] can profit from new insights gained from research on the sequencing role of the posterior cerebellum in social reasoning. Table 1 shows a selection of clinical pathologies that may be affected by a dysfunctional posterior cerebellum derived from this hypothesis.

Using the NeuroSynth database [59], Table 2 (left panel) shows evidence that these pathologies reveal deviant brain functionality or anatomy in the posterior cerebellum, more in particular in Crus 1 or 2 and Lobule IX, located in the default/mentalizing network [60].

Locations in the mentalizing network were also found in conventional meta-analyses exploring the differences between participants with and without specific pathologies, with respect to posterior cerebellar volume or activation during relevant social tasks (see Table 2; middle panel). Given that many studies tend to ignore the cerebellum, it is encouraging that some evidence of posterior cerebellar anomalies in mentalizing areas can be identified at a meta-analytic level for these pathologies, providing firmer ground for our theoretical hypothesis. Taken together, we suggest that a mentalizing deficit in the posterior cerebellum is a core transdiagnostic dysfunction of many clinical pathologies, in line with the "social system" approach of the Research Domain Criteria (RDoC) framework of psychiatric disorders [48, 61, 62].

**Table 1** Proposed key dysfunctions in social and emotional mentalizing processes in clinical pathologies related to the posterior cerebellum

| Inflexibility in thought-feeling-action sequences |
|---|
| Underactive social attention and learning |
| Underactive social and emotional mentalizing |
| Overactive ruminative catastrophizing and negative self-evaluation |
| Overactive addiction-related ruminative craving |
| Overactive/underactive feelings of social responsibility |

## A Research Proposal and Expected outcomes

To address the pressing questions on the role of the posterior cerebellum in dysfunctional mentalizing in clinical pathologies, we believe that future research should address the following research questions and methodologies, leading to the following expected outcomes and impact:

### Mentalizing About Others and Self

Future research should explore in depth the functionality of the posterior cerebellum in mentalizing about others and the self in action sequences, using fMRI in healthy participants as main methodology. Cerebellar activity should be elicited and measured by tests on action sequences that require mentalizing about others. Figure 2 depicts a number of these tasks involving mentalizing about others from prior research. For example, in some tasks, events were portrayed using

**Table 2** Several pathologies showing a role in the default/mentalizing network of the cerebellum [60] as listed by coordinates in NeuroSynth (left) and in meta-analyses (middle)

| Pathology | NeuroSynth term and # studies | MNI Coordinates | | | Meta-analyses on volume, connectivity or activation | MNI Coordinates | | | Cerebellar lobule |
|---|---|---|---|---|---|---|---|---|---|
| Autism | ASD; 146 | 50 | − 58 | − 40 | [63] (Volume) | 31 | − 76 | − 27 | Crus 1 |
| Depression | MDD; 185 | 46 | − 52 | − 46 | [64] (Resting state) | 16 | − 84 | − 16 | Crus 2/1 |
| Bipolar | Bipolar; 157 | -5 | − 54 | − 54 | [27] (Connectivity) | − 12 | − 90 | − 30 | Lob IX/Crus 2 |
| Obsession | OCD; 81 | 20 | − 82 | − 44 | [65] (Volume) | − 5 | − 51 | − 44 | Crus 2/Lob IX |
| Addiction | Alcohol*; 130 | 46 | − 62 | − 40 | [66] (Reward anticipation) | − 20 | − 86 | − 20 | Crus 1 |
| Schizophrenia | Schizophrenic; 84 | − 48 | − 62 | − 40 | [26] (Emotional faces) | − 30 | − 77 | − 38 | Crus 1/2 |

Left: NeuroSynth meta-analysis key term and number of studies involved (*substance use disorder [SUD] is not available as term in NeuroSynth). Middle: Meta-analyses refer to either differences in volume or activation of relevant social processes in comparison with neurotypical participants. Right: Cerebellar lobules refer to NeuroSynth and meta-analyses, respectively, or both. Coordinates refer to MNI; Talairach coordinates were converted to MNI coordinates.

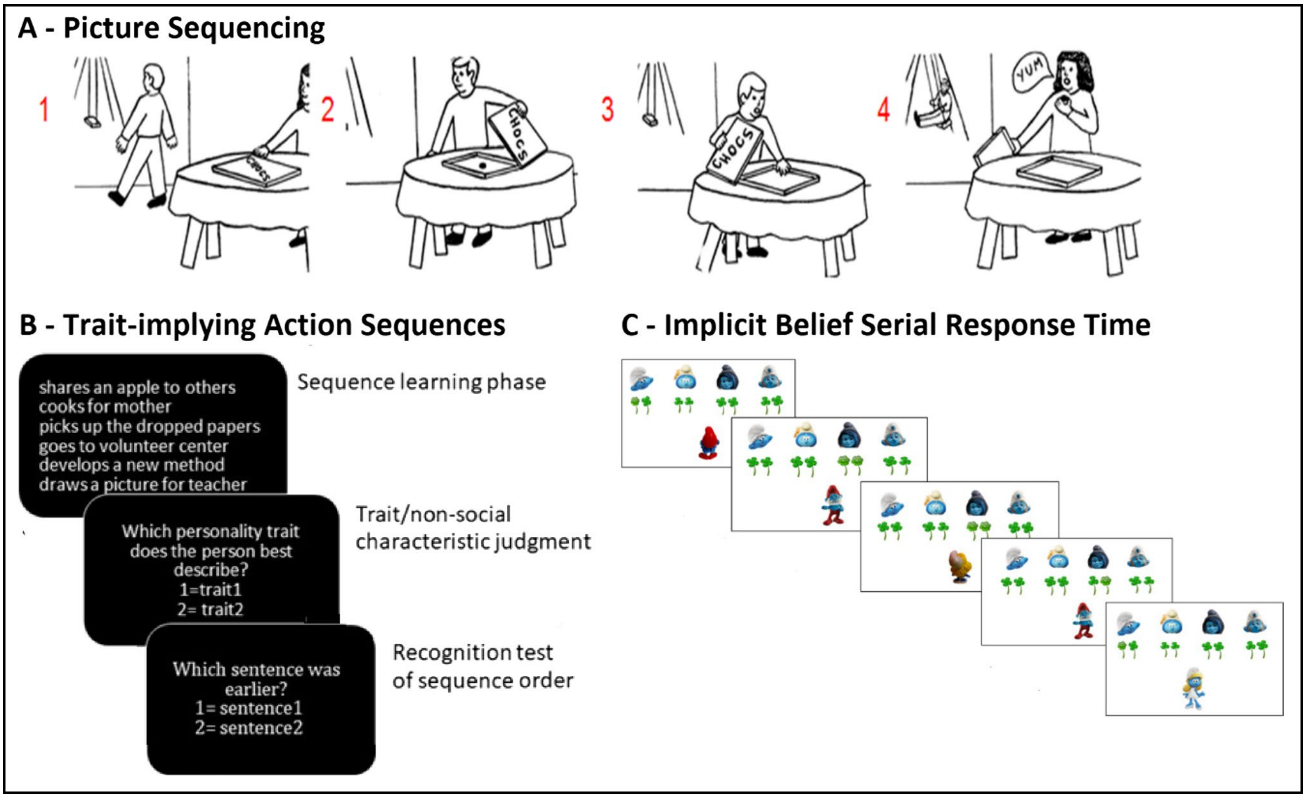



**Fig. 2** Several sequencing tasks used in prior research; trials go from left to right or from top to bottom. **A** Picture Sequencing: an example of a false belief sequence (the correct order is 2 – 1 – 4 – 3; [33]). **B** Trait-Implying Action Sequences: an example of a trial with six sentences implying a pro-social trait of which the order had to be memorized [34]. **C** Implicit Belief Serial Response Time: participants had to answer as fast as possible how many flowers the protagonist could see when oriented to the flowers; and when not, how many flowers he or she had seen previously. Unbeknownst to them, there was a fixed order of true and false beliefs held by the protagonists [68]

cartoon-like pictures (cf. Figure 2A) or brief sentences (cf. Figure 2B) in a randomized order. In all tasks, participants had to generate or memorize the correct sequence of non-motor actions that required *mentalizing on beliefs* [33, 38]; Fig. 2A), *goal-directed intentions* [67], and *traits of others* [34],Fig. 2B) not only at an explicit level, but also at an *implicit level* [68],Fig. 2C). These social sequencing tasks were typically compared against control conditions involving either social non-sequencing (i.e., no specific order had to be learned or generated) or non-social sequencing (i.e., goal-directed movements or trait-like characteristics of objects instead of persons). It was consistently found that the posterior cerebellum was recruited during generating or memorizing social sequences, and significantly more so than during both non-sequential and non-social control conditions. The control conditions sometimes elicited weaker posterior cerebellar activation, for instance, when a sequence was shown, but was not to be memorized. In the domain of emotion attributions of the self and others, tasks requiring to identify *dynamic emotional faces* (e.g., sequential transitions from neutral to angry) recruited the posterior cerebellum as compared to static emotional faces [69].

This is an area of active and promising investigation, and there are still numerous key questions for future research. Perhaps the main issues are: Is the posterior cerebellum:

- responsive to novel sequencing tasks on social action which have not been investigated yet (e.g., preferences, attributions of affective experiences)?
- responsive to higher-level social meaning, for instance, when actions contradict implied traits, emotions, and intentions of individuals or social groups (e.g., counter-stereotypical behaviors)? — this would establish the role of the posterior cerebellum as a truly high-level social processor.
- responsive to predictions of novel social actions of other people based on prior social judgements about them, such as their traits, preferences, or emotional responses? — this question turns around the predictive logic of previous tasks in which mental states and traits were inferred based on social behaviors.
- equally responsive to implicit and explicit learning of regularities in a social or emotional context? — how do

both learning mechanisms interact with each other in the cerebellum or brain in general?

- involved in closed-loops with canonical cortical areas in social processing (i.e., TPJ and mPFC) during these tasks, which seem to differ from well-known closed-loops in motor behaviors?
- also occasionally responsive when social non-sequencing information is provided, perhaps due to spontaneous processing of implicitly assumed sequencing?
- also equally responsive to language processing devoid of social cues, and what is the relationship with social processing (e.g., narratives are often stories about social protagonists)?

*Current status:* This research has so far progressed quite well as can be observed from our brief review on social sequencing tasks above, is still going on, and has provided encouraging results. It appears that the theoretical hypothesis is healthy and that this approach may form the groundwork for more research on the mentalizing role of the posterior cerebellum in healthy and clinical populations.

*Expected outcome:* Given that the posterior cerebellum has seldom been implicated before in social neuroscience, this research will provide a breakthrough in theoretical models of (dysfunctional) social processes and stronger evidence for closed loops of activation between the posterior cerebellum and cortical key areas.

## Noninvasive Neurostimulation and Causal Impact

Although neuroimaging may demonstrate the role of the posterior cerebellum, this constitutes only correlational evidence and does not convincingly demonstrate the causal impact of the posterior cerebellum on social and emotional mentalizing. To investigate the causal impact of the posterior cerebellum on mentalizing, researchers can turn to noninvasive neurostimulation (e.g., tDCS, TMS; [54] targeting the posterior cerebellum with healthy participants, measuring its impact on mentalizing. Given that the 7-network structure of the cerebellum [60] shows a posterior-anterior axis going from mentalizing, executive control, and then roughly to limbic, salience/ventral attention, and somatomotor circuits, noninvasive stimulation may modulate the social mentalizing areas most directly. Modulation can be measured, for instance, on performance of aforementioned (adjusted) sequencing tasks, as well as on (sub)cortical processes using fMRI or EEG [70]. Although research on cerebellar patients can also be very informative [8, 38, 71], the causal role on social cognition is somewhat limited, because this may also result from compensatory alterations of neural wiring elsewhere during brain growth. Moreover, this approach cannot inform about potential noninvasive stimulation treatments to improve social functioning.

Only few studies have addressed this issue. To illustrate, cerebellar TMS applied online (i.e., at the onset or offset of stimulus presentation) pointed to cerebellar involvement in social cognition, such as understanding facial emotion expressions [32, 72], bodily motions [73], and group attitudes [74]. In addition, cerebellar tDCS modulated recognition of emotional facial expressions [75] and eye expressions at a rapid and automatic level [76]. More importantly, with respect to mentalizing during social sequencing, repetitive TMS targeting the posterior cerebellum subsequently increased the speed at which participants generated the correct sequence of true and false belief events as well as of social routines and non-social events on the Picture Sequencing task [77],Fig. 2A). Future research needs to investigate whether focal tDCS stimulation of the posterior cerebellar Crus (e.g., using high definition tDCS) may selectively facilitate social or emotional mentalizing, or both. Other questions for future research are whether noninvasive brain stimulation also facilitates processing of other mentalizing judgments, like intentions, traits, affect, and so on?

*Current status:* Research on noninvasive neurostimulations of the posterior cerebellum and its effect on social sequencing is just beginning to emerge. Fortunately, the posterior cerebellum is a relatively easy target for noninvasive stimulation because of its convenient location close to the skull at the back of the brain. However, a limitation is that cerebellar stimulation tends to result into significant activation of the occipital cortex [78] and that it also may stimulate the neck muscles, which is experienced as unpleasant. Given that many other questions still remain on noninvasive stimulation and its effectiveness (see below), we think that it is too early to experiment with invasive stimulation techniques, such as deep brain stimulation. In any case, due to the risks involved, the applicability of such interventions is restricted to a limited subset of patients where their use is warranted due to severe medical problems.

While past reviews paint several technical and methodological possibilities [54, 79], several questions need empirical answers:

- The key question is which stimulation protocol will have the strongest effect on social and emotional sequencing processes? TMS (which may also reach deeper subcortical areas), tDCS (with a more wide-spread cortical impact), or HD-tDCS (with more focal tDCS effects)?
- Which electrode placement is most effective in bringing about (causal) changes in brain areas related to social and emotional sequencing processes? Placement of the cerebellar and other electrodes directs the electric currents through different brain areas, potentially with crucial differences in effects.
- To what extent will neurostimulation affect some types of mentalizing sequences differently compared to others?

Will it modulate social mentalizing more strongly than emotional mentalizing? Will it facilitate posterior cerebellar flexibility, so that uncommon false beliefs (Picture Sequencing task), unexpected and trait-inconsistent action orders (Trait-Implying task), or implicit random orders (Belief Serial Response Time task) are processed more easily? Or will it instead reinforce stereotypical actions?

- What are the functional neural mechanisms of effective cerebellar stimulation? To have more insight in this process, novel research is needed on changes in task-related activity and connectivity, besides behavioral changes, as measured by EEG markers [80] or fMRI.

*Expected outcome:* Modulation by posterior cerebellar stimulation will provide the needed evidence that the posterior cerebellum has causal impact on cortical processes, behavior, and performance, rather than being a mere by-product of cortical processes.

## Mentalizing in Clinical Populations

Applying the aforementioned sequencing tasks on social and emotional mentalizing in clinical populations will allow us to investigate the theoretical relevance and diagnostic value for pathologies like autism, depression, obsessions, addiction, schizophrenia, and perhaps many more. Is mentalizing in these populations distorted, or do other (perhaps more anterior?) cerebellar processes play a major role in these pathologies?

We hypothesize that clinical disorders in the posterior cerebellum may arise from at least three basic functional impairments, which are related to distortions in the posterior cerebellum or connections to and from the posterior cerebellum:

1. Ignoring or misinterpreting social environmental cues: A first dysfunction is failing to detect social information that is relatively uncommon, leading to biased recognition of, and an overreliance on, typical and expected social cues. This may initiate stereotypical and repetitive social action sequences that are inappropriate in a given situation (e.g., autism). This dysfunction most likely resides in anatomical and functional posterior cerebellar deficits due to this region's involvement in social sequential information processing. Anatomical and functional connectivity between the social cortical network and the posterior cerebellum may even distort normal functioning of the mentalizing cortical network.
2. Reduced social behavioral repertoire: The reduction of the posterior cerebellar volume in many clinical disorders may lead to a reduced social behavioral repertoire because of encoding limitations. Consequently, even if subtle social cues are recognized, limitations in the behavioral responses may often lead to less appropriate social behavior.
3. Inability to implicitly learn and automatize social behavioral repertoire: Another dysfunction is the lack of neurological resources to flexibly learn and automatize novel social action sequences that are appropriate in given circumstances. Consequently, even if the posterior cerebellar volume is sufficiently large, learning of appropriate behaviors in novel social environments is too slow to be adequate. Most likely, this dysfunction resides in the functionality of the posterior cerebellum and, in particular, the automatic learning of action sequences.

Each of the mentalizing tasks described earlier (see Fig. 2) may be more diagnostic for one dysfunction than the another:

1. Picture Sequencing Task

Generating sequences of false beliefs more slowly or incorrectly and/or lack of change in posterior cerebellar activation for false beliefs (in comparison with true beliefs and social routines) might be most diagnostic to reveal reductions in the social behavioral repertoire, because false beliefs are relatively complex and unfamiliar. However, these same differences might also be due to ignoring or misinterpreting unusual social environmental cues in sequencing tasks necessary to identify the correct order of actions. Tasks that exclusively manipulate environmental cues (e.g., [81]) might be more diagnostic for identifying distorted cue processing.

2. Trait-Implying Action Sequences Task

Failure to memorize the correct order of trait-implying behaviors that are inconsistent with initial behaviors as opposed to consistent, and/or no change in posterior cerebellar activation for inconsistent behaviors, might be most diagnostic to reveal slow learning of novel action sequences. In this task, environmental cues play little or no role at all, as these are not systematically manipulated.

3. Implicit Belief Serial Response Time Task

Slow learning of implicit environmental regularities during mentalizing and/or lack of posterior cerebellar activation might be most diagnostic to reveal an inability to learn and automatize novel social behavioral sequences. In this task, environmental cues are most likely not indicative of implicit social learning, as such cues are not systematically manipulated (apart from the protagonists' orientation).

*Current status:* Although research on social sequencing tasks on populations with clinical pathologies is just beginning, as detailed above, these tasks can diagnose with some specificity the functional or anatomical deficit that might be responsible for impaired performance, such as biased perception of environmental cues, limited capacity for extending one's behavioral repertoire, or slow implicit automatization of novel behaviors. A key concern for these tasks is whether they need some stimulus adaptation in function of specific clinical impairments under study. For example, in addictive patients, it might be more relevant and productive to investigate social action sequences related to their addiction (e.g., alcohol, drug use). However, for other patient populations, this might be less relevant as social mentalizing is a key diagnostic dysfunction (e.g., autism).

*Expected outcome:* This research will provide more solid ground for the proposed theoretical model on the posterior cerebellum, as it will provide compelling evidence for a modulatory role of the posterior cerebellum on impaired mentalizing in clinical pathologies.

### Neurostimulation and Neuro-guided Therapy in Clinical Populations

Applying posterior cerebellar neurostimulation using noninvasive (e.g., tDCS, TMS; [54]) and invasive techniques (e.g., DBS) might help to improve social performance in clinical populations (i.e., autism, depression, obsessive and bipolar disorder, and addiction). Cerebellar stimulation could help to replace habitual maladaptive patterns of impaired social, depressive, obsessive–compulsive, or addictive-related thought-feeling-action sequences, with more adaptive sequences. Previous clinical research has already shown that cerebellar tDCS improves memory and executive functions in bipolar depression [82, 83], decreases obsessive behaviors in obsessive–compulsive disorders (although evidence is mixed; see [84, 85]), and improves brain information processing in bipolar patients, as measured by electroencephalographic markers (EEG; [80]). It was further found that dysfunctional cerebellar metabolic activity was related not only to a longer duration of the depressive episode, but was also associated with lower response rates to classical prefrontal rTMS treatment in treatment resistant depressed patients [86].

As far as we are aware, invasive cerebellar stimulation (e.g., DBS) has been applied so far only for patients with severe motor deficits, such as tremor, Parkinson's, and epilepsy, and not for social deficits [87]. Given that research on social cognition in the cerebellum is still young, future possibilities for applying invasive stimulation for improving social thinking can certainly profit from research on the social consequences of other patient populations undergoing cerebellar DBS.

In addition to cerebellar stimulation, training programs may also improve flexibility and functionality of social sequences when applying the insights gained from posterior cerebellar research on social and emotional mentalizing. Too often, this focus is absent in existing therapies, and quite often, only attention to other's thoughts and sensations is practiced. The main crux in our approach is a novel focus on social temporal sequences (i.e., as exemplified in narratives) which are critical to understand the intentions and other mental states of other persons and the self. Attention to sequencing can be reinstated by paying more attention to (a) how other people narrate daily events that occurred to them, (b) what the essential narrative transitional cues are to structure their narrative in a temporal order, (c) how these narratives often involve thoughts or emotions, and (d) training patients in recounting these narratives including these transitional and mentalizing elements (see also [88]).

*Current status:* Noninvasive neurostimulation and sequence-guided therapy of clinical populations is in its infancy. However, preliminary experiments have been started. Additional questions on clinical work are: Is the plasticity of the posterior cerebellum large enough to allow for significant changes in clinical behavior after single or repeated noninvasive cerebellar stimulations? Or after short- or long-term training programs? When do we apply these noninvasive stimulation methods, just before training, or just after, or even during training? It has been argued that the application of noninvasive stimulation (rTMS and tDCS) concurrently with psychological tasks or with psychotherapeutic interventions will have the strongest behavioral and emotional outcomes in psychopathology by co-activating the same network involved [89, 90].

*Expected outcome:* We expect that posterior cerebellar noninvasive neurostimulation as well as behavioral training programs and/or psychotherapy with emphasis on narrative sequence elements, either alone or in combination, can substantially boost therapeutic effectiveness for autism, depression, obsession-compulsion, addiction, and perhaps many other psychiatric and neurological disorders manifesting similar symptoms.

## Conclusion

The sequencing hypothesis of social thinking and automatization in the posterior cerebellum sketched above has provided a fruitful ground for thinking about, and doing research on, the posterior cerebellum. If future research can pave the way for more detailed knowledge on the working of this brain area in social and emotional mentalizing, this would be decisive, not only by uplifting our scientific thinking about the posterior cerebellum, but also by taking a transdiagnostic approach to clinical pathologies. This might aid many individuals with distinct pathologies that were difficult to treat in the past. By increasing the plasticity of the posterior cerebellum using noninvasive neurostimulation, in combination with other proven behavioral and brain-related strategies, we can alleviate the mental problems of a large number of individuals who suffer from inflexible automatizations that stand in the way of adjustable and intuitive social behavior.

**Funding** This work has been supported by an SRP57 grant by the Vrije Universiteit Brussel to Frank Van Overwalle and Chris Baeken. There is no other funding source.

## Declarations

**Ethics approval** This article does not contain any studies with human participants or animals performed by any of the authors.

**Conflict of interest** The authors declare no competing interests.

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