1 Differential effects of cerebellar degeneration on feedforward versus feedback control across
2 speech and reaching movements

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27 **Abstract**

28

29 Errors that result from a mismatch between predicted movement outcomes and sensory
30 afference are used to correct ongoing movements through feedback control and to adapt
31 feedforward control of future movements. The cerebellum has been identified as a critical part
32 of the neural circuit underlying implicit adaptation across a wide variety of movements
33 (reaching, gait, eye movements, and speech). The contribution of this structure to feedback
34 control is less well understood: although it has recently been shown in the speech domain that
35 individuals with cerebellar degeneration produce even larger online corrections for sensory
36 perturbations than control participants, similar behavior has not been observed in other motor
37 domains. Currently, comparisons across domains are limited by different population samples
38 and potential ceiling effects in existing tasks. To assess the relationship between changes in
39 feedforward and feedback control associated with cerebellar degeneration across motor
40 domains, we evaluated adaptive (feedforward) and compensatory (feedback) responses to
41 sensory perturbations in reaching and speech production in individuals with cerebellar
42 degeneration and neurobiologically healthy controls. As expected, the cerebellar group
43 demonstrated impaired adaptation in both reaching and speech. In contrast, the groups did not
44 differ in their compensatory response in either domain. Moreover, compensatory and adaptive
45 responses in the cerebellar group were not correlated within or across motor domains.
46 Together, these results point to a general impairment in feedforward control with spared
47 feedback control in cerebellar degeneration. However, the magnitude of feedforward
48 impairments and potential changes in feedback-based control manifest in a domain-specific
49 manner across individuals.

50

51 **Significance Statement**
52 The cerebellum contributes to feedforward updating of movement in response to sensory
53 errors, but its role in feedback control is less understood. Here, we tested individuals with
54 cerebellar degeneration (CD), using sensory perturbations to assess adaptation of feedforward
55 control and feedback gains during reaching and speech production tasks. The results confirmed
56 that CD leads to reduced adaption in both domains. However, feedback gains were unaffected
57 by CD in either domain. Interestingly, measures of feedforward and feedback control were not
58 correlated across individuals within or across motor domains. Together, these results indicate a
59 general impairment in feedforward control with spared feedback control in CD. However, the
60 magnitude of feedforward impairments manifests in a domain-specific manner across
61 individuals.
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Introduction

Coordinated movement relies on a combination of feedback control and anticipatory mechanisms. A mismatch between the predicted and actual feedback resulting from a motor command can lead to online corrections as well adaption of feedforward control for future movements (Shadmehr and Krakauer, 2008). The cerebellum plays a critical role in this latter process, helping ensure that the predictive system is optimally calibrated. One line of supportive evidence comes from the substantial literature showing markedly impaired performance of individuals with cerebellar degeneration (CD) during sensorimotor adaptation tasks involving upper limb movement (Martin et al., 1996; Smith and Shadmehr, 2005; Tseng et al., 2007; Donchin et al., 2012; Schlerf et al., 2013), gait (Morton and Bastian, 2006), eye movements (Xu-Wilson et al., 2009), and speech (Parrell et al., 2017).

If, and how, the cerebellum contributes to feedback control is less clear. One clue comes from intentional tremor, a prominent feature of CD where low-frequency oscillations occur around the movement endpoint. Intentional tremor is reduced when movement is produced without visual feedback (Day et al., 1998). Such behavior is broadly consistent with a control system that relies on visual feedback (Beppu et al., 1987), and could occur if the gains on sensory feedback errors are larger in individuals with CD. Larger gains could lead to overcorrections for errors and the need for additional counter-corrections. This hypothesis is supported by evidence showing that, relative to controls, individuals with CD produce larger compensatory responses to auditory perturbations of speech (Parrell et al., 2017; Houde et al., 2019; Li et al., 2019).

There is mixed evidence concerning feedback gains in other types of movement. Individuals with CD produce a smaller long-latency muscle response to mechanical perturbations (Kurtzer et al., 2013), consistent with a decreased gain in the response to proprioceptive feedback. However, their feedback-based corrections during split-belt treadmill walking are relatively normal (Morton and Bastian, 2006), suggesting proprioceptive gains are unaffected for at least some tasks. Moreover, individuals with CD have normal feedback gains in a continuous visual tracking task, but with a substantial phase lag (Zimmet et al., 2020). Computationally, this is consistent with an increased reliance on (delayed) feedback in the absence of a predictive function for state estimation (Wolpert et al., 1998; Miall et al., 2007).

It is possible that potential increases in feedback gains in non-speech tasks may be obscured by a ceiling effect. While the auditory feedback response in speech only partially compensates for the perturbation, the responses to perturbations in tasks such as visually-guided movements or walking typically provide nearly complete compensation to the perturbation (Morton and Bastian, 2006; Tseng et al., 2007). As such, reductions in feedback gains could be readily measured but increases in gain might be hard to detect. Alternatively (or additionally), modification of feedback gains in CD may be task-dependent, with variable changes across movement types. This would stand in contrast to feedforward control, where the cerebellum appears to play a similar role in implicit adaptation across domains.

The preceding hypotheses are based on inferences drawn from disparate studies that distinct methods and typically focus on a single motor domain. In the present study, we used a 2 x 2 design to evaluate feedback and feedforward control in two motor tasks, one involving visually-guided reaching and the other speech production. To avoid potential ceiling effects in the former, we used a task shown previously to induce only partial corrections to the visual

107   perturbation (Körding and Wolpert, 2004). Moreover, by testing the same individuals on all four
108   tasks, we can perform a correlational analysis, focusing on two key questions related to
109   individual differences associated with CD. First, are patterns of impairment similar across the
110   two motor domains and/or forms of control? Second, is the degree of impairment in
111   feedforward control (adaptation) predictive of feedback gains, a signature that would be
112   consistent with the hypothesis that enhanced feedback gains arise as a compensatory
113   mechanism.

114   **Materials and Methods**
115   <u>Participants</u>
116   23 individuals with cerebellar degeneration (CD: 19 female, 37-89 years, mean age 62 years)
117   and 15 age-matched neurobiologically healthy controls (CO: 8 female, 43-85 years, mean age 61
118   years) were recruited for the study. The participants had normal or corrected-to-normal vision,
119   and none reported any history of speech or hearing impairments, or significant neurological
120   issues (other than ataxia in the CD group). Participants provided informed consent and received
121   financial compensation for their time. The protocols were approved by the institutional review
122   boards at the University of Wisconsin–Madison and the University of California, Berkeley.
123   All of the participants were administered the Montreal Cognitive Assessment (MoCA) as
124   a gross measure of cognitive impairment. The CD group was additionally administered the
125   Standard Ataxia Rating Scale (SARA) to assess the severity of their ataxia. SARA subscores were
126   calculated for both upper limb control and speech. One individual with CD was excluded due a
127   MOCA score < 18, indicative of moderate cognitive impairment. See the Appendix for a full
128   characterization of the CD group. Data from one control participant was excluded from Exp 4
129   due to equipment failure during data collection. The final sample size was based on typical
130   sample sizes in previous studies and, with sample sizes of 22 and 15 for the CD and control
131   groups, gives a power of 0.75 to detect a large between group effect (d = 0.8) as well as a
132   power of 0.75 detect a medium effect (d = 0.5) for correlations within the CD group.
133
134   <u>Experimental Design</u>
135   Each participant completed four conditions in a single experimental session that lasted
136   approximately one hour, including breaks of approximately 5 min between each condition.
137   Feedforward and feedback control during reaching were assessed in Conditions 1 and 2,
138   respectively; similarly, feedforward and feedback control during speech were assessed in
139   Conditions 3 and 4, respectively. A schematic of each of the four conditions is shown in **Figure**
140   **2**. The same order of the four conditions was used for each participant. While this approach
141   introduces order confounds, we opted to keep the order fixed given that a) this is preferable for
142   correlational analyses and b) the sample size was insufficient to properly assess order effects.
143   Stimulus presentation and data collection was controlled with Matlab for all conditions.
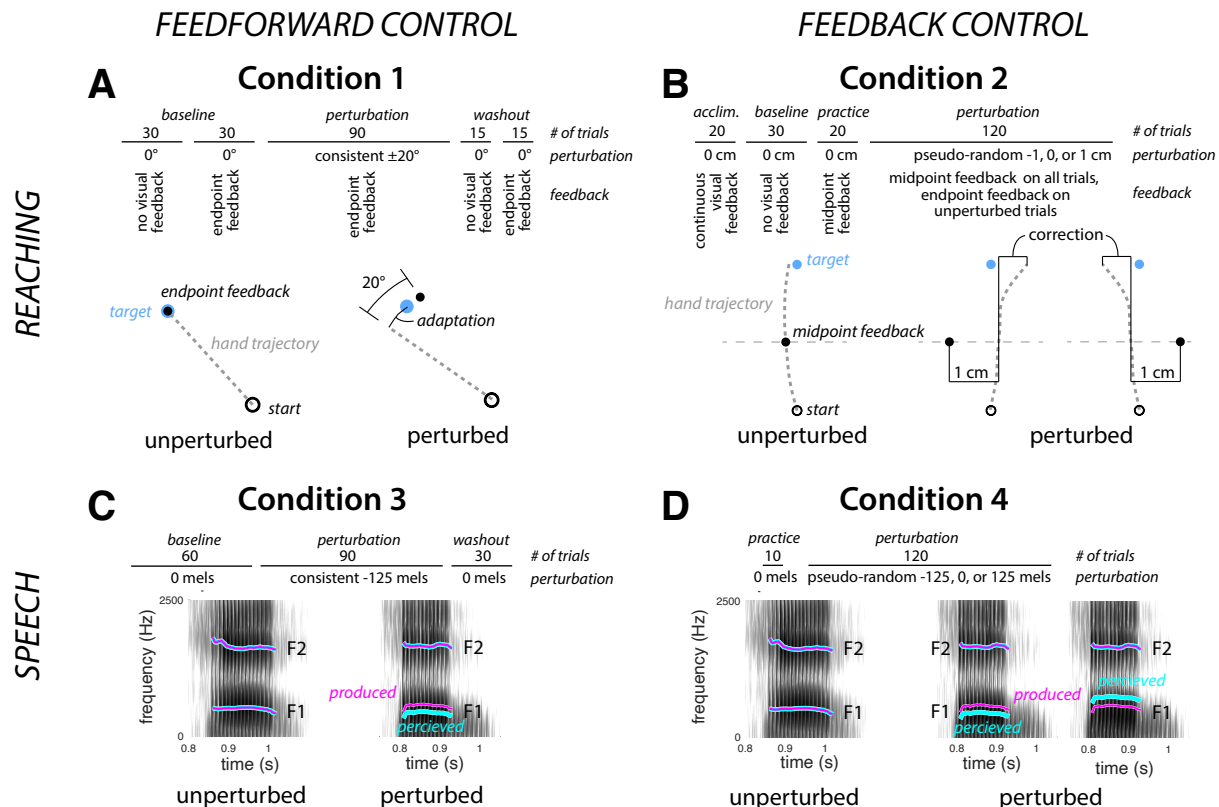144
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Figure 1: Experimental design. *The top row of each panel shows the structure of the experimental session and the bottom part depicts the perturbation.* ***A:*** *Condition 1, feedforward control in reaching: Participants made 8 cm center-out reaches to targets (blue dot) with endpoint feedback. During the perturbation phase, the location of the feedback cursor was rotated by 20° from actual hand position (direction cross-balanced across participants). The dashed line represents the unseen hand movement.* ***B:*** *Condition 2, feedback control in reaching: Participants made 16 cm reaches to a target (blue dot). During the perturbation phase, visual feedback about hand position (black dot) was given at reach midpoint with the feedback shifted by -1, 0, or 1 cm, with the shift on each trial determined in a pseudo-random manner. The thin gray line depicts location of reach midpoint, but was not visible to the participant.* ***C:*** *Condition 3, feedforward control in speech: Participants spoke a single word on each trial, hearing a playback of their speech over headphones with minimal delay. During the perturbation phase, feedback of the first formant during the vocalic portion of the utterance was perturbed by imposition of a − 125 mel shift (cyan trace superimposed on the spectrogram).* ***D:*** *Condition 4, feedback control in speech: Participants spoke a single word on each trial. During the perturbation phase, auditory perturbations (cyan trace) of -125, 0, or 125 mels were pseudo-randomly applied to the first formant during the vocalic portion of the utterance.*

Condition 1: Feedforward control in reaching.

Participants were seated in front of a 53.2 x 20 cm LCD screen (ASUS) that was horizontally encased in a table frame mounted 27 cm above a 49.3 x 32.7 cm digitizing tablet (Intuos 4XL, Wacom, Vancouver, WA). Participants held a modified air hockey paddle and made reaching movements by sliding the paddle across the table. The position of a stylus embedded in the paddle was recorded by the tablet at 200 Hz. Feedback, when available, was presented in the form of a cursor on the LCD screen. Participants' view of their hand was blocked by the LCD

171   screen. To further limit vision of the upper arm, the experiment was conducted in a darkened
172   room. The experiment was controlled with Psychtoolbox (Brainard, 1997; Pelli, 1997; Kleiner et
173   al., 2007).
174   Participants made center-out reaches, moving to targets located at a radial distance of 8
175   cm from the center of the workspace. The start location was marked by a white ring (6 mm
176   diameter). On each trial, the participant moved the digitizing stylus to position the hand within
177   the start location. Visual feedback about the stylus position was provided by a small (3.5 mm
178   diameter) white circle. After the participant had maintained the stylus position within the start
179   location for 500 ms, one of 3 equally-spaced targets (6 mm diameter) appeared (0°, 120°, or
180   240°). The participant was instructed to reach, attempting to "slice through" the target. The
181   instructions emphasized that the movements should be made quickly and, to minimize
182   demands on endpoint (radial) accuracy, should terminate beyond the target location. RT was
183   not emphasized; the movement could be initiated at any time after the presentation of the
184   target.
185   Visual feedback about the movement was limited to endpoint feedback, eliminating the
186   opportunity for visually guided corrections. The feedback cursor disappeared at movement
187   onset and reappeared when the radial distance of the hand movement reached 8 cm. The
188   cursor remained visible for 50 ms and then was blanked. If participants reached the 8 cm
189   threshold in ≤ 500 ms, a knocking sound was played, indicating that the movement was "fast
190   enough and far enough." If the movement duration was > 500 ms, participants heard a
191   recording of the words "too slow". During the return movement to the start location, visual
192   feedback was withheld until the stylus was within 3 cm of the start position.
193   The condition consisted of five phases. 1) A *no feedback baseline* phase of 30 trials to
194   measure movement variability in the absence of visual feedback. 2) A *baseline phase* of 30 trials
195   with veridical endpoint feedback of the cursor. 3) A *perturbation phase* of 90 trials in which the
196   location of the cursor at movement endpoint was rotated 20° from the true position. The 20°
197   rotation was chosen to limit awareness of the perturbation and minimize the use of explicit re-
198   aiming (Bond and Taylor, 2015; Morehead et al., 2015; Werner et al., 2015). The perturbation
199   was either clockwise or counter-clockwise, counterbalanced across participants. 4) An
200   *aftereffect phase* of 15 trials in which there was no visual feedback. 5) A *washout* phase of 15
201   trials with veridical endpoint feedback.
202   The hand angle for each trial was measured as the angle between a line connecting the
203   start location and the hand position at the time of peak radial velocity, relative to the line
204   connecting the start and target locations. To remove intrinsic biases in reaching, the mean hand
205   angle during the baseline phase was subtracted from the heading angle for each trial. We focus
206   on two measures of adaptation, the mean of the last 15 trials of the perturbation phase
207   (*asymptote*) and the mean of the 15 trials in the aftereffect phase. We additionally measured
208   reaction time (time from target appearance to movement onset) and movement time (time
209   from movement onset to target), as well as the percentage of trials where any part of the
210   cursor representing hand position overlapped with the target (*hits*).
211
212   *Condition 2: Feedback control in reaching.*
213   The experimental apparatus was identical to Exp 1. On each trial, the white ring
214   indicating the start location appeared near the bottom edge of the screen at the horizontal

215   meridian. After this positioned was maintained for 500 ms, the target appeared at a fixed
216   position, 16 cm in front of the start location. The longer distance was used so that feedback
217   could be presented at the midpoint of the movement, providing sufficient distance (and time)
218   for an online correction.
219   There were five phases to Condition 2. 1) An *acclimation phase* of 20 trials with
220   continuous veridical feedback. 2) A *baseline phase* of 30 trials in which there was no visual
221   feedback, either during the movement or at the target distance. 3) A *practice* phase of 20 trials
222   to introduce the method for providing limited visual feedback. The cursor appeared twice on
223   these trials, first when the radial amplitude of the stylus was 8 cm from the start location
224   (midpoint, duration = 100 ms), and second when the radial amplitude reach 16 cm (endpoint,
225   duration = 50 ms). Participants were instructed to "use what you see midway through the
226   reach to get as close as possible to the target". 4) A *perturbation phase* of 120 trials with
227   midpoint feedback. On 50% of the trials, the midpoint feedback was given at the true location
228   of the stylus, and on the remaining 50% of the trials, the midpoint feedback was shifted 1 cm to
229   the left of the stylus position or 1 cm to the right (25% each). Each cluster of 4 reaches
230   consisted of 2 unperturbed trials, and 1 trial with the leftward and rightward perturbation, in a
231   randomized order. Endpoint feedback was not provided on perturbed trials to minimize
232   learning effects (i.e., anticipatory effects based on prior responses to the perturbation).
233   Endpoint feedback was provided on the unperturbed trials to help participants remain
234   calibrated to the target reaching location. 5) A *final phase* of 20 trials with no visual feedback.
235   Data from this last phase was not analyzed further.
236   As in Condition 1, participants were instructed to slice through the target. The
237   movement time criterion was increased to 1200 ms. The extra time was employed to
238   compensate for the larger amplitude movements and to encourage participants to adopt a
239   movement speed that allowed them time to use the midpoint feedback to make an online
240   correction (if warranted). The knocking sound was played if the target amplitude was reached
241   within 1200 ms, and if the movement duration was between 400 and 1200 ms, the target circle
242   turned green. If the movement was < 400 ms, the words "too fast" appeared on the screen. If
243   the movement duration was longer than 1200 ms, the target circle turned red and a recording
244   of the words "too slow" was played.
245   The focus here was on the online corrections made in response to the horizontally
246   displaced midpoint feedback; that is, the horizontal position of the hand relative to the target
247   at the target distance. However, the raw horizontal displacement would also reflect the
248   horizontal position of the hand at reach midpoint, which may vary between trials. To account
249   for this, we fit the trial-by-trial data of each participant with a linear model that predicted the
250   final horizontal position of the hand on each trial from the horizontal hand position at reach
251   midpoint, the visual perturbation (treated as a categorical variable and coded using separate
252   dummy predictors for each direction), and the interaction between midpoint hand position and
253   the perturbation:
254
255    $x_{target} \sim \beta_0 + \beta_1 x_{midpoint} + \beta_2 \text{perturbation(L)} + \beta_3 \text{perturbation(R)} + \beta_4 x_{midpoint}{:}\text{perturbation(L)} +$
256    $\beta_5 x_{midpoint}{:}\text{perturbation(R)}$
257

258 This model allows us to estimate two dependent measures of feedback-based corrections: 1)
259 the magnitude of the correction for the perturbation (direction effect estimates, β_2, β_3) and 2)
260 corrections for self-produced variability (the midpoint position estimate, β_1, and its adjustment
261 in the presence of perturbations, β_4, β_5). For statistical analysis, the sign of β_2 values were
262 flipped such that negative values always reflected a compensatory response to the
263 perturbation, regardless of perturbation direction. Additionally, this method allows us to
264 estimate the effect of the correction for the visual perturbation while accounting for any
265 individual differences in the bias of the overall reach trajectory, an effect captured by the
266 intercept term (β_0), which reflects the horizontal displacement relative to the target position for
267 unperturbed reaches. We measured movement time, reaction time, and proportion of trials
268 where the cursor hit the target as for Condition 1. Hit proportion was calculated only for
269 unperturbed trials.
270
271 *Condition 3: Feedforward control in speech.*
272 Participants were again seated in front of the horizontally-aligned monitor. Participants
273 wore a head-mounted microphone (AKG C520) and close-backed, over-the-ear headphones
274 (Beyerdynamic DT 770). On each trial, participants spoke the word "head" after it appeared on
275 the monitor. The utterance was digitized through a Scarlett 2i2 sound card and processed with
276 Audapter (Cai et al., 2008; Tourville et al., 2013) to synthesize a sound for playback over the
277 headphones. The recording, processing, and playback occurred in near real time (~20 ms delay).
278 The intensity of the synthesized feedback was set to roughly 80 dB based on each participants
279 comfortable speaking volume, mixed with speech-shaped noise at ~60 dB to mask air- or bone-
280 conducted direct feedback of the utterance. The actual intensity level of the feedback varied
281 with changes in the intensity of participants' speech.
282 There were four phases in Condition 3. For each phase, the word "head" remained
283 visible for 2.5 s on each trial, and the trials were separated by 1.1-1.3 s (randomly jittered). 1) A
284 *baseline phase* of 60 trials with speech feedback resynthesized through Audapter with no
285 auditory perturbation applied. 2) A *perturbation phase* of 100 trials during which a constant
286 shift of -125 mels was applied to the first vowel formant (F1) throughout the utterance, shifting
287 the F1 value of "head" towards that of "hid". 3) A *washout phase* of 30 trials in which there was
288 no auditory perturbation.
289 Vowel onset and offset was labelled for each trial using a semi-automated procedure.
290 First, automatic labels were generated by identifying on the waveforms where the speech
291 amplitude first crossed (onset) or fell below (offset) a participant-specific amplitude value. The
292 automatic labels were then visually inspected and corrected when the waveform and
293 spectrogram indicated that the automatic markings were inappropriate. A semi-automated
294 procedure was used to track the formants during the vocalic phase of the utterance using
295 participant-specific vales for LPC order and pre-emphasis. Formant tracking was performed
296 with Praat (Boersma and Weenink, 2019). Using the Wave Runner software package (Niziolek
297 and Houde, 2015), the tracks were visually inspected and, when they did not align with visible
298 formants on the speech spectrogram, the formant tracking parameters (pre-emphasis, LPC
299 order) were modified.
300 The primary outcome measure was adaptation of the vocalic portion of the utterances
301 across trials. For each trial, we calculated the average F1 value from 50-100 ms after vowel

302   onset. This window avoids the transitional phase of the formant during the word-initial
303   consonant (initial 50 ms) as well as any online compensatory response to the perturbation,
304   which typically begins later than 100 ms after vowel onset (Tourville et al., 2008; Cai et al.,
305   2012; Parrell et al., 2017). To facilitate comparisons across participants, the F1 values were
306   normalized with respect to the average F1 value taken over the 50-100 ms window during the
307   second half of the baseline phase (trials 31-60). We chose to use the second half of the baseline
308   phase to allow participants time to acclimate to the processed auditory feedback.
309
310   *Condition 4: Feedback control in speech.*
311   The stimulus set consisted of four words, "dead", "fed", "said", and "shed", selected
312   because they share the same vowel /ɛ/. The condition began with a calibration phase designed
313   to shape the participant's speaking rate such that the produced vowel duration would be
314   between 300 and 500 ms. This criterion was important to ensure that there would be sufficient
315   time for feedback-based corrections, shown in previous work to have a latency of ~150 ms
316   (Tourville et al., 2008; Cai et al., 2012; Parrell et al., 2017). On each trial, one of the four words
317   was displayed. After each utterance, the automated estimate of the vowel duration in
318   milliseconds was displayed on the monitor. This procedure was repeated for 10 trials. If the
319   duration fell outside the 300-500 ms window on more than 2 of these 10 trials, the procedure
320   was repeated for 10 more trials. When the utterances fell within the criterion window for at
321   least 8 of the 10 trials, the main experiment began.
322   Condition 4 had two phases. 1) A *practice phase* of 10 trials with veridical auditory
323   feedback. 2) A *perturbation phase* of 120 trials. During the perturbation phase, participants
324   heard veridical feedback on 50% of trials, a + 125 mel shift of F1 (moving the vowel towards
325   that in "had") on 25% of trials, and a -125 mel shift of F1 (moving the vowel towards that in
326   "hid") on the remaining 25% of trials. Each group of 4 trials consisted of 2 unperturbed trials,
327   and 1 trial each of positive and negative F1 perturbations, randomly ordered. Stimuli were
328   presented in a random order (selection with replacement), with the constraint that the total
329   number of each stimulus word be as equal as possible across the experimental condition.
330   Vowels were tracked as in Exp 3. The primary outcome measure was the online
331   correction to the perturbation, calculated following standard approaches (Cai et al., 2012;
332   Niziolek and Guenther, 2013; Parrell et al., 2017; Daliri et al., 2020). First, an average baseline
333   F1 trajectory was calculated for each stimulus word from the unperturbed productions of that
334   word. Second, the F1 trajectory from each perturbed trial was normalized by subtracting the
335   appropriate word-specific average baseline F1 trajectory, giving a normalized F1 response for
336   each perturbed trial. Trajectories from trials with upward and downward F1 perturbations were
337   separately averaged to generate an average F1 response in each direction. To generate a
338   composite feedback response, the sign of the average response to the upward perturbation
339   was flipped, such that positive values always reflected a compensatory response to the
340   perturbation, regardless of perturbation direction. Note that because of the normalization
341   process (and lack of an explicit target), this differs slightly from the approach in Exp 2, where
342   midpoint variability had to be accounted for. To quantify the magnitude of the compensatory
343   response, the mean value of each average F1 response trajectory between 200-300 ms after
344   vowel onset was calculated. This window begins well after the expected 150 ms latency of the
345   compensatory response is ~150 ms (Tourville et al., 2008; Cai et al., 2012; Parrell et al., 2017).

346
347   <u>Statistical Analysis</u>
348   Statistical analysis for all conditions was conducted in R (R Core Team, 2013) using
349   mixed ANOVAs or Welches' two-sample t-tests. When necessary, post-hoc tests were
350   conducted with a Tukey HSD correction. Where appropriate, one-sample t-tests with Holm-
351   Bonferroni corrections were used to determine whether the responses for each group differed
352   significantly from 0. Means are reported with standard deviations. Effect sizes are given as
353   Hedges *g* or partial eta-squared.
354   For conditions 1 and 3, a mixed ANOVA was used that included group (ataxic vs control)
355   and phase (end of perturbation and aftereffects), as well as the interaction between the two
356   factors. Similarly, for condition 4, the mixed ANOVA included group (ataxic vs control) and
357   perturbation direction (up and down), as well as the interaction between the two.
358   For condition 2, separate ANOVAs were used to evaluate differences between the ataxic
359   and control group in 1) the magnitude of the correction in response to the perturbation and 2)
360   the magnitude of correction in response to self-produced variability. These were conducted as
361   second-level analyses, on the beta coefficients estimated in the first-level analyses that were
362   conducted within each individual (see above). The dependent variable for the perturbation
363   correction model was the direction effect coefficient, estimated separately for each
364   perturbation direction (β_2, β_3). In addition to the perturbation direction factor (left vs right), this
365   model included a group factor as well as the interaction between perturbation direction and
366   group. For the variability correction, the dependent variable was the midpoint coefficient
367   representing the magnitude of correction for variability at reach midpoint in unperturbed (β_1),
368   left ($\beta_1+\beta_4$), and right ($\beta_1+\beta_5$) conditions. In addition to the main variable of interest, group, this
369   model also included perturbation condition (no perturbation, left, right). Welches' t-tests were
370   used to compare the ataxic and control groups on the reaction time, movement time, and hit
371   percentage in unperturbed trials.
372   In order to assess the relationship between feedback and feedforward control, we
373   conducted a set of planned correlational analyses using data from pairs of conditions. One set
374   of correlations compared the measures of feedback and feedforward control within each motor
375   domain (i.e., one correlation between the two reach tasks and a second between the two
376   speech tasks). We also performed a second set of correlations of similar forms of control across
377   the motor domains (i.e., one correlation of the feedforward measures from the reach and
378   speech tasks, and a second correlation of the feedback measures from the reach and speech
379   tasks). Because the primary aim of these analyses is to investigate the possibility that changes
380   in motor behavior due to cerebellar degeneration are correlated across sensory domains and
381   control systems, we limited these correlational analyses to the data from the CD group. We also
382   examined the extent to which our experimental measures of feedforward and feedback control
383   in the ataxic group were related to ataxia symptom severity, as measured by SARA speech and
384   upper limb subscores.
385

386   **Results**
387
388   <u>Condition 1: Feedforward control in reaching.</u>

389   In the baseline phase with visual feedback, the CD group was substantially more variable
390 than controls (standard deviation of 6.2 ± 2.0° vs 4.0 ± 0.8°, t(31.6) = 4.6 p < 0.0001, g = -0.89)
391 and less accurate (percentage of targets hit, 67 ± 25% vs 87 ± 15%, t(35.8) = -3.0, p = 0.005, g =
392 1.28). The CD group also produced slower movements than controls (347 ± 148 ms vs 264 ± 61
393 ms, t(31.6) = 2.4, p = 0.02, g = 0.67) and had longer reaction times (544 ± 116 ms vs 442 ± 58
394 ms, t(34.0) = 3.6, p = 0.001, g = 1.02).
395   Our principle outcome measure, adaptation, was operationalized as the change in
396 heading angle following the introduction of a 20° rotation of the feedback cursor. As can be
397 seen in Figure 2, the perturbation induced a gradual change in heading angle, with the
398 functions appearing to reach or approach asymptote by the end of the perturbation trials.
399 Adaptation in the control participants compensated for approximately 83% of the perturbation;
400 the comparable figure for the CD participants was only 45%. A decline in adaptation is visible
401 throughout the aftereffect phase in which there was no visual feedback and the following
402 washout phase in which veridical feedback was reintroduced.
403   Statistically, we first confirmed that the sign-dependent changes in heading angle were
404 significantly different than zero, the signature of adaptation. When measured during the final
405 trials of the perturbation block, the heading angle values were different than zero for both the
406 control (16.6 ± 2.7°, $t(14) = 24.1$, $p < 0.0001$, $g = 5.90$) and CD groups (9.2 ± 5.7°, $t(22) = 7.8$, $p <$
407 0.0001, $g = 1.56$). The adapted response persisted into the aftereffect block (controls: 15.1 ±
408 2.9°, $t(14) = 20.0$, $p < 0.0001$, $g = 4.87$; CD: 7.3 ± 7.5°, $t(22) = 4.7$, $p < 0.0001$, $g = 0.95$), although
409 the value was smaller than observed at the end of the perturbation block for both groups
410 ($F(1,36) = 7.4$, $p = 0.01$, $\eta^2 = 0.17$). Across both the perturbation and aftereffect phases,
411 adaptation was significantly greater in the control group ($F(1, 36) = 20.1$, $p < 0.0001$, $\eta^2 = 0.36$)
412 and the Group x Phase interaction was not significant ($F(1,36) = 0.05$, $p = 0.82$, $\eta^2 = 0.001$).
413 These results are in accord with previous results (for a review, see Krakauer et al., 2019) in
414 showing that adaptation of feedforward control for reaching is impaired in individuals with CD.
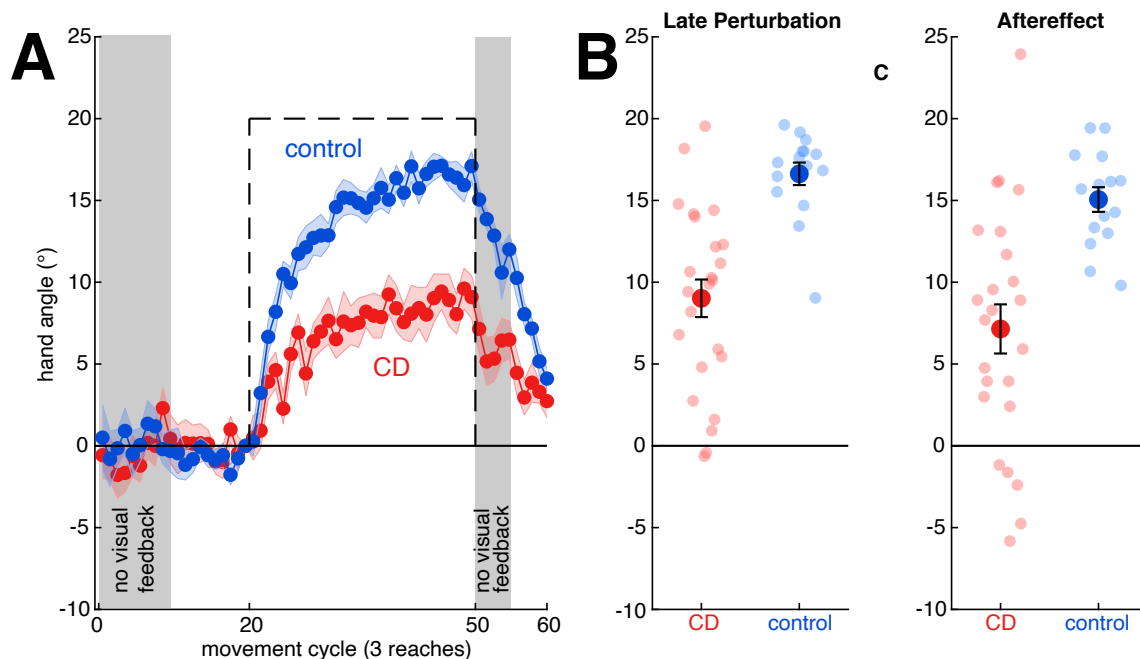
Figure 2: In the visuomotor rotation task, the CD group reached a lower level of adaptation and exhibited a smaller aftereffect relative to the control group. A: *Hand angle over the course of the experimental condition. Means and standard errors are shown for control participants (blue) and individuals with cerebellar degeneration (red). Visual feedback is withheld in the shaded phases. The perturbation is shown with a dashed black line.* **B,C:** *Individual data (semi-transparent dots) and group means ± standard error for the CD and control participants during the last 15 reaches of the perturbation phase (B) and 15-trial aftereffect phase in which the visual feedback was withheld (C).*

Condition 2: Feedback control during reaching

  As in Condition 1, there were some kinematic differences between the two groups. The most salient group difference was in movement accuracy: control participants hit the target more often than the CD participants on the unperturbed trials (control: 85 ± 10 %, CD: 70± 24%, $t(31.9)$ = 2.7, p = 0.01, g = 0.75). There were small differences between the groups in reach curvature, with controls showing a slight leftward shift from midpoint to target on unperturbed trials and the CD group showing a slight rightward shift on these trials (control: -0.17 ± 0.193 cm, CD: 0.13 ± 0.52 cm, $t(30.2)$ = 2.5, p = 0.02, g = 0.69, **Figure 3**F). Reaction times were also slower in the CD group (control: 452 ± 79 ms, CD: 525 ± 138 ms, $t(35.5)$ = 2.08, p = 0.045, g = 0.61) whereas movement time was similar between groups (control: 717 ± 114 ms, CD: 722 ± 114 ms, $t(30.1)$ = 0.14, p = 0.89, g =0.05).

  Our principle dependent measure, online corrective responses, was operationalized as a lateral shift in the trajectory in response to perturbed feedback that was presented at the midpoint of the movement, with the direction of the perturbation randomized across trials. As can be seen in the data from a representative control participant (Fig 3A), the hand trajectory deviated in the opposite direction of the perturbation, a signature of a feedback-based response. The lateral shift accounted for roughly 33% of the perturbation magnitude in the

441   control group and 21% in the ataxic group (Fig 3B, D). These corrective feedback responses
442   were significantly different from 0 in both groups in response to the leftward perturbation (CD:
443   -0.21 ± 0.34 cm, $t(22)$ = 3.3, p = 0.003, g = 0.67; control: -0.35 ± 0.30 cm, $t(14)$ = 4.6, p = 0.0005,
444   g = 1.12) and rightward perturbation (CD: -0.20 ± 0.32 cm, $t(22)$ = 2.9, p = 0.007 g = 0.60;
445   control: -0.34 ± 0.28 cm, $t(14)$ = 4.7, p = 0.0003, g = 1.15). While the magnitude of the feedback-
446   based response was smaller for the CD group, this difference was not significant ($F(1,36)$ = 2.8,
447   p = 0.1, η^2 = 0.07). There was no difference between the responses to the two perturbation
448   directions ($F(1,36)$ = 0.16, p = 0.69, η^2 = 0.004) and the Group x Direction interaction was not
449   significant ($F(1,36)$ = 0.009, p = 0.93, η^2 = 0.0002). These results indicate that feedback-based
450   corrective responses to visual perturbations are not enhanced in individuals with CD; indeed, as
451   a group the trend was for attenuated feedback responses.
452   In a second measure of feedback control, we looked at adjustments in the reach
453   trajectory in response to motor variability (estimated as the change in horizontal position from
454   reach midpoint to endpoint unrelated to the visual perturbation; model parameters β_1, $\beta_1+\beta_4$,
455   and $\beta_1+\beta_5$). Although both groups showed a slight shift in trajectory in the compensatory
456   direction in response to the position of the cursor at reach midpoint (**Figure 3**C,E,G), this shift
457   was not significantly different from 0 after correction for multiple comparisons (CD: -0.010 ±
458   0.035, p = 0.05, g = -0.30 ; control: -0.009 ± 0.042, p = 0.24, g = -0.21). There was no difference
459   between the two groups ($F(1,36)$ = 0.01, p = 0.91, η^2 = 0.0004), nor any interaction between
460   group and direction ($F(2,72)$ = 2.4, p = 0.10, , η^2 = 0.06). However, there was a significant effect
461   of perturbation direction ($F(2,72)$ = 7.3, p = 0.001, , η^2 = 0.17) such that the shift in trajectory
462   was greater in the presence of leftward perturbations (-0.021 ± 0.0332) compared to trials with
463   no perturbation (0.001 ± 0.038; p = 0.008, g = 0.62). There was a similar trend in for trials with
464   rightward perturbations (-0.010 ± 0.036), but this did not reach significance (p = 0.15, g = 0.33).
465   In sum, there is little evidence that either group corrected for self-produced variability at reach
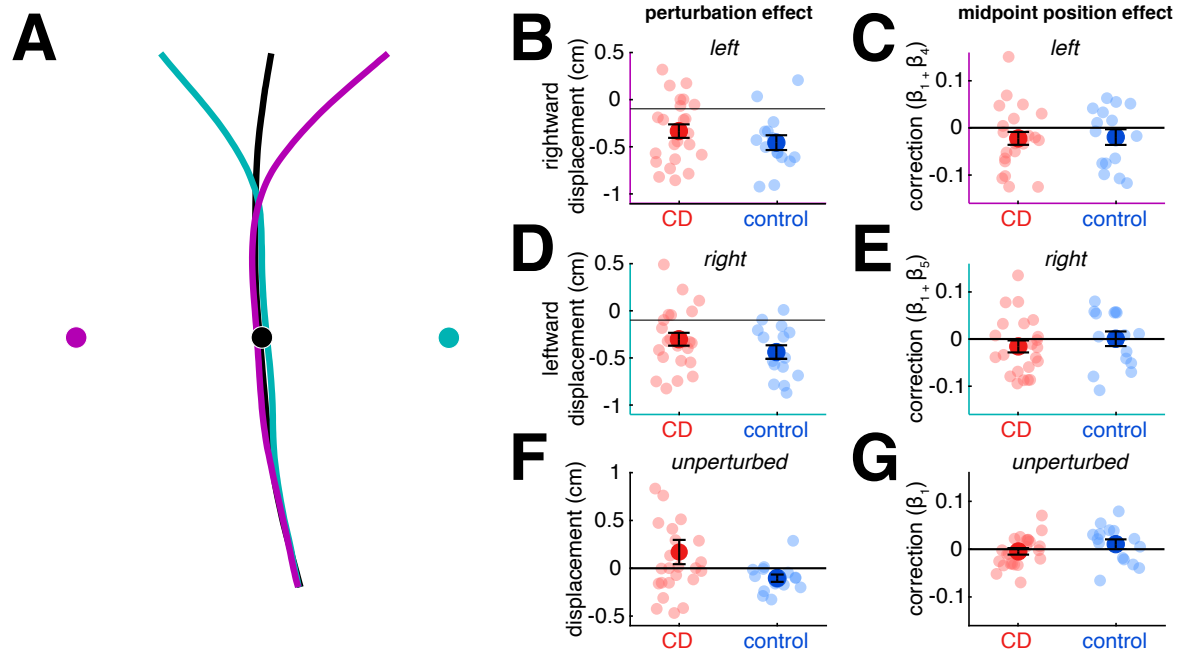466   midpoint.
467

468

Figure 3: In the reaching feedback control task, both the CD and control groups showed a robust on-line correction to perturbed visual feedback presented at the midpoint of the reach, with no group differences. A: Example data from one control participant showing average reach trajectories for unperturbed reaches (black), reaches with a 1 cm rightward visual perturbation (teal), and reaches with a 1 cm leftward visual perturbation (purple). Dots in corresponding colors show the location of the visual feedback. The corrective response only partially corrects for the perturbation. B, D: Compensatory response to the feedback perturbation. F: Estimate of the change in horizontal hand position from reach midpoint on unperturbed trials. C, E, G: Estimates of the effect of true hand position at reach midpoint on the compensatory response for leftward (C), rightward (E), and unperturbed (G) trials. Across all plots, compensatory responses have negative values. Semi-transparent dots represent individuals.

481

Condition 3: Feedforward control in speech

Adaptation was operationalized as the change in F1 in response to a -125 mel F1 shift in the feedback heard by participants, introduced via the real-time resynthesis of their utterances. As can be seen in Figure 4A, this auditory perturbation caused a gradual change in F1 that opposed the perturbation. At asymptote, the adaptive response had reached approximately 40% of the perturbation in the control group and 25% in the CD group. The lower degree of compensation relative to reaching is consistent with previous studies (Houde and Jordan, 1998; Purcell and Munhall, 2006; Parrell et al., 2017). Note that, unlike Condition 1, post-perturbation trials (washout phase) always included veridical feedback; we did not include a no-feedback phase as masking auditory feedback with noise leads to substantial changes in speech (Lombard, 1911; Summers et al., 1988).

493   We first confirmed that both groups adapted during the perturbation phase (CD: 31 ± 39
494   mels, $t(1,22)$ = 3.9, p = 0.0007, g = 0.79; control: 53 ± 31 mels, $t(1,14)$ = 6.6, p < 0.0001, g = 1.61;
495   Figure 4B) and maintained their adapted state during the initial part of the washout phase (CD:
496   21 ± 32 mels, $t(1,22)$ = 3.1, p = 0.005, g = 0.64; control: 40 ± 23 mels, $t(1,14)$ = 6.6, p < 0.0001, g
497   = 1.62; Figure 4C). The CD group exhibited less adaptation than the control participants ($F(1,36)$
498   = 4.7, p = 0.036, , η^2 = 0.12), and this difference was similar in both phases (Group x Phase
499   interaction: $F(1,36)$ = 0.06, p = 0.80, η^2 = 0.002). In sum, we find that sensorimotor adaptation
500   in speech is impaired in individuals with CD, similar to the impairment observed for reach
501   adaptation in Condition 1 and consistent with previous findings (Parrell et al., 2017),
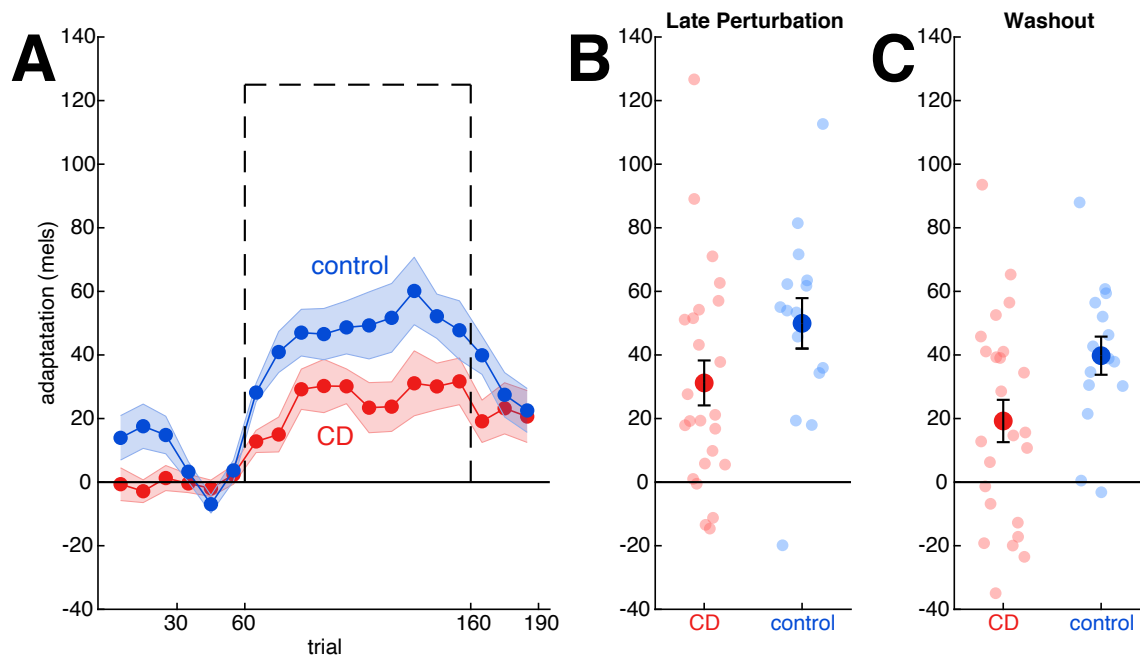

502
503   **Figure 4: In the speech adaptation task, the CD group adapted less than controls. A:** *Change in first*
504   *formant over the course of the experimental condition. Means and standard errors are shown for control*
505   *(blue) and CD participants (red). For each participant, the change was calculated relative to their mean*
506   *F1 value during the second half of the baseline phase, with positive values corresponding to changes in*
507   *the opposite direction of perturbation. The perturbation is shown with a dashed black line (sign flipped).*
508   **B,C:** *Group means ± standard error (solid dots) and individual data points (semi-transparent dots) of*
509   *adaptive response during the last 10 trials of the perturbation phase and first 10 trials of the washout*
510   *phase.*

511   Condition 4: : Feedback control in speech
512   Online corrective responses for speech were operationalized as the change in F1 during
513   the time window from 200-300 ms after vowel onset in response to an upward or downward
514   perturbation of F1, randomized across trials. The data are plotted relative to F1 values
515   measured on unperturbed trials. As can be seen in Figure 5A, the non-predictable auditory
516   perturbations resulted in compensatory responses that opposed the perturbation in both
517   groups. The magnitude of the corrective response (7.6% in controls, 9.2% in the ataxic group)
518   was similar to that typically observed in response to auditory perturbations of speech and much
519   lower than observed for the non-predictive perturbations during reaching used in Condition 2.

520   The change on perturbed trials was significantly larger than 0 in response to upward and
521   downward perturbations in the CD group (up: 10.2 ± 13.6 mels, $t(1,22) = 3.6$, $p = 0.002$, $g =$
522   0.73; down: 11.8 ± 16.0 mels, $t(1,22) = 3.5$, $p = 0.002$, $g = 0.71$) and control group (up: 11.0 ±
523   14.4 mels, $t(1,13) = 2.9$, $p = 0.02$, $g = 0.72$; down: 8.2 ± 12.4 mels, $t(1,13) = 2.9$, $p = 0.02$, $g =$
524   0.72) (Figure 5B, C). While the mean values were larger in the CD group and individuals in this
525   group showed the largest compensatory response, the difference between the two groups was
526   not significant ($F(1,35) = 0.35$, $p = 0.56$, $\eta^2 = 0.009$). There were no differences between the
527   responses to the two perturbation directions ($F(1,35) = 0.1$, $p = 0.74$, , $\eta^2 = 0.003$) and the
528   Group x Direction interaction was not significant ($F(1,35) = 0.07$, $p = 0.80$, $\eta^2 = 0.002$). These
529   results suggest feedback gains for auditory perturbations in speech are similar in individuals
530   with CD and healthy controls, consistent with the reaching results in Exp 2. Of note, the null
531   effects here are inconsistent with the results from previous studies involving speech
532   articulation (Parrell et al., 2017) and vocal pitch production (Houde et al., 2019; Li et al., 2019)
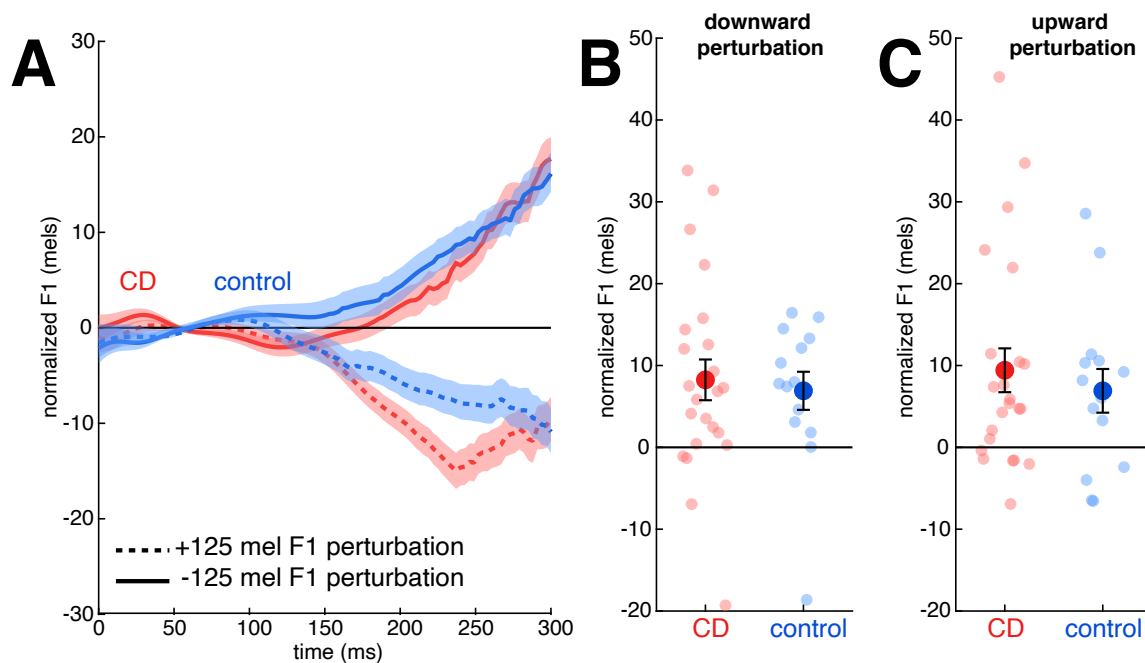533   in which individuals with CD were found to show an enhanced feedback response.

534
535   *Figure 5: In the speech feedback control task, both the CD group and controls corrected for formant*
536   *perturbations in both directions, with no difference between the two groups. A: Normalized formant*
537   *trajectories in response to a downward F1 perturbation (solid lines) or upward perturbation (dotted lines)*
538   *in the control (blue) and CD (red) groups. Shaded area indicates standard error. Data are normalized to*
539   *the formant trajectories produced in trials with no perturbation. B, C: Magnitude of F1 values in trials*
540   *with a downward (B) or upward (C) perturbation, averaged over the time window spanning from 150-*
541   *300 ms following onset of vocalic portion of the utterances. Compensatory responses are expressed as*
542   *positive values in both cases. Group means ± standard error are shown as solid dots; semi-transparent*
543   *dots represent individuals.*

544   Feedforward and feedback control within and across motor domains
545   By testing each participant in all four conditions, we can compare the measures of
546   feedback and feedforward control within and across task domains. Because our focus in this

547 analysis is how deficits in these domains may be correlated in individuals with cerebellar
548 degeneration, we limit this analysis to the 22 individuals with CD.
549   The between-domain comparisons assess the similarity of impairment (or lack thereof)
550 between reaching and speech. Although the ataxic group adapted less than the controls at the
551 group level, there was no significant correlation within the ataxic group between the magnitude
552 of adaptation between speech and reaching (Figure 6A). Similarly, the magnitude of feedback-
553 based compensation (Figure 6B) was unrelated across motor domains.
554   The within domain correlations provide a test of the hypothesis that feedback gains may
555 increase in response to impaired feedforward control: in this case, we would expect a negative
556 correlation between these measures such that individuals who are more impaired in
557 feedforward control should be more likely to exhibit a greater reliance on feedback control.
558 This relationship might hold even if there is no overall increase in compensatory responses
559 when comparing the CD and control groups. Contrary to this prediction, the relationship
560 between feedforward adaptation and feedback-based compensation was not significant in
561 either reaching (Figure 6C) or speech (Figure 6D).
562   We additionally tested whether any of our behavioral measures correlated with ataxia
563 severity as assessed with the SARA. Neither a summary measure of overall upper limit ataxia
564 nor intentional tremor severity were correlated with reach adaptation or compensation (all $p >$
565 0.2). Similarly, overall speech impairment was not correlated with speech compensation ($r = -$
566 0.24, $p = 0.26$). We did observe a positive correlation between the SARA measure of speech
567 impairment and speech adaptation, with lower levels of adaptation to the auditory
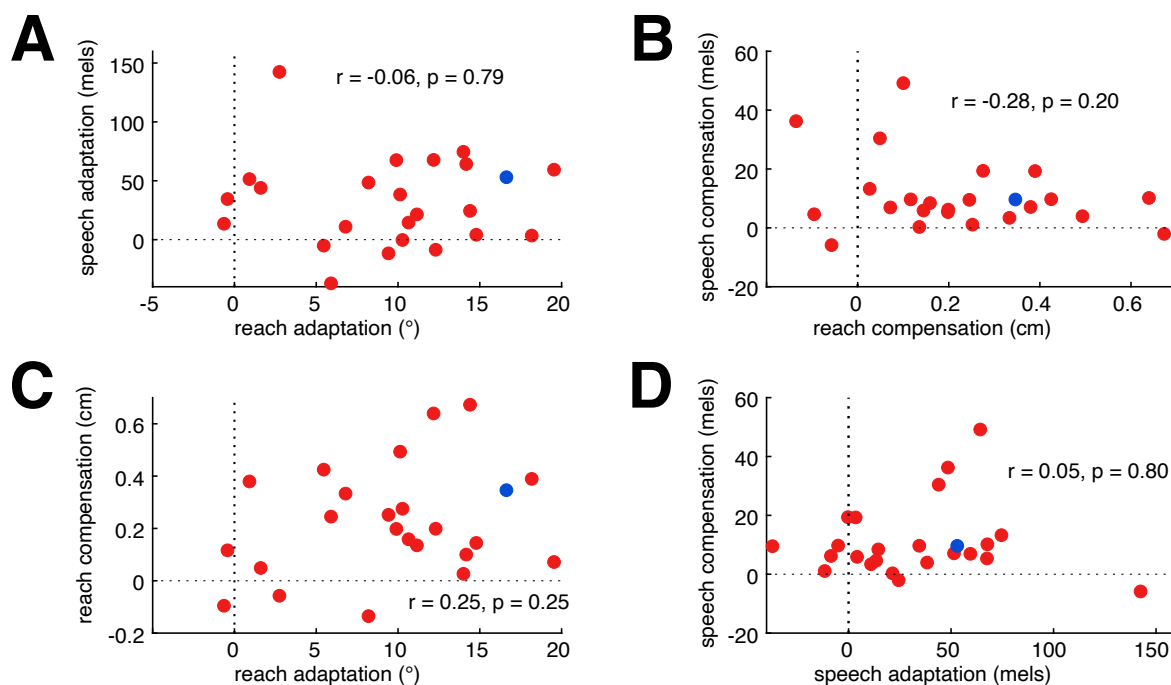568 perturbation associated with greater speech impairment ($r = 0.45$, $p = 0.03$).
569

Figure 6: Correlational analyses of feedforward adaptation and feedback-based compensation in individuals with cerebellar degeneration. *Within the CD group, there were no correlations in behavioral measures obtained across (**A-B**) or within (**C-D**) motor domain. Plots show individual CD participants as*

574 *red dots and the mean of the control participants as a blue dot. To simplify interpretation, the sign of*
575 *compensation in reaching has been flipped such that larger positive values reflect greater compensation,*
576 *as for all other measures. **A**: Adaptation in reaching and speech. **B**: Compensation in reaching and*
577 *speech. **C**: Adaptation and compensation in reaching. **D**: Adaptation and compensation in speech.*

578

579 **Discussion**
580 We conducted a set of experiments to evaluate the impact of cerebellar degeneration on
581 feedforward and feedback control in two motor domains, reaching and speech. Individuals with
582 cerebellar degeneration showed a marked impairment in feedback control relative to controls,
583 manifest as reduced adaptation in response to a sensory perturbation that remained constant
584 from trial to trial. Feedback control, measured in terms of the on-line response to a variable
585 perturbation, was intact in both motor domains.

586

587 ***Multi-modal impairment in feedforward control in individuals with cerebellar degeneration***
588   Our principle positive result, that individuals with cerebellar degeneration are impaired
589 in adapting their motor behavior in the presence of sensory prediction errors, is consistent with
590 prior neuropsychological studies involving upper limb control and speech. These results are
591 consistent with the hypothesis that the cerebellum provides a domain-general mechanism for
592 generating sensory predictions and using error information to keep this predictive system well-
593 calibrated. In particular, these results agree with evidence that the cerebellum is critical for
594 implicitly updating motor behavior. Adaptation in speech is highly likely to be an implicit
595 process (Kim and Max, 2020; Lametti et al., 2020): the majority of participants are unaware of
596 the auditory perturbation (Parrell and Niziolek, 2020), and adaptation is similar whether
597 participants are unaware of the perturbation or made aware of the perturbation and explicitly
598 told to ignore the feedback (Munhall et al., 2009). The design for the reaching task in the
599 current study, one in which the perturbation was only 20°, was chosen to also primarily engage
600 implicit processes (Bond and Taylor, 2015; Morehead et al., 2015; Werner et al., 2015).
601   While adaptation was impaired in both reaching and speech at the group level, there
602 was no correlation between the magnitude of adaptation in reaching and speech in the CD
603 group. That is, individuals who were more impaired in reaching were not more likely to be
604 impaired in speech. It is possible that the failure to find such a relationship is due either to
605 unreliable estimates of adaptation and compensation in each domain (given that we only
606 tested each condition a single time, and no previous work, to our knowledge, has established
607 the reliability of these measures) or to a lack of power given our sample size of 22 individuals
608 with CD. However, previous work has identified a similar dissociation in individuals with CD
609 between adaptation to dynamic (force-field) and kinematic (visuomotor rotation) perturbations
610 during reaching, and deficits in adapting reach dynamics and reach kinematics have been
611 localized to different regions in the cerebellum in both individuals with CD (Rabe et al., 2009)
612 and acute cerebellar lesions (Donchin et al., 2012). Our results further specify this dissociation
613 by showing that cerebellar mechanisms of adapting kinematic perturbations to different motor
614 effectors do not overlap. Given somatotopic representations across subregions of the
615 cerebellar cortex (e.g., Marvel and Desmond, 2010; Mottolese et al., 2013), we would

616   anticipate that deficits in speech adaptation would be associated with more medial cerebellar
617   regions, relative to deficits in limb adaptation, though future work examining patterns of
618   cerebellar damage in patients will be needed to confirm this hypothesis.
619
620   ***Intact, but not enhanced feedback control in individuals with cerebellar degeneration***
621   Feedback-based corrections for errors were similar in magnitude between individuals
622   with cerebellar degeneration and controls for both speech and reaching. Although this result
623   agrees with estimates of feedback gains in a continuous visuomotor tracking task (Zimmet et
624   al., 2020), the absence of a difference between the CD and control groups on the speech task
625   fails to replicate a previous finding from our labs showing an enhanced feedback response to a
626   similar perturbation (Parrell et al., 2017). Moreover, a much larger increase in the feedback
627   response, relative to controls, has been observed in response to auditory perturbations of pitch
628   (Houde et al., 2019; Li et al., 2019). Our previous results had led to the hypothesis that the
629   enhanced feedback response reflected a compensatory mechanism, one to help offset the
630   disruptive effects of impairment in feedforward control. The failure to observe enhanced
631   feedback in the current study in both domains argues against this compensatory hypothesis.
632   The absence of a correlation between the feedforward and feedback measures within both
633   motor domains also argues against a compensatory hypothesis.
634   There are a few issues to consider in terms of the discrepancy between our results and
635   previous studies as well as the interpretation of the null results regarding the compensatory
636   hypothesis. First, we may have failed to find any changes in feedback control in individuals with
637   CD simply due to sampling issues. Our sample of this population (n = 23) is consistent with, or
638   larger than, many previous studies (Morton and Bastian, 2006; Parrell et al., 2017; e.g., Houde
639   et al., 2019; Li et al., 2019; Zimmet et al., 2020), and we were adequately powered to detect
640   relatively large between-group effects (.75 power to detect *d* of .8). However, it is possible the
641   effect size of the expected increase in feedback gains in certain domains, as in our previous
642   work on oral articulation, may be relatively small. Thus, we may simply have been under-
643   powered to detect any changes.
644   Second, it may be that increased feedback gains are a secondary, and somewhat
645   sporadic, effect of cerebellar degeneration. The consistent and striking deficit in feedforward
646   control points to inaccurate or attenuated predictive signals. In this case, some individuals may
647   learn to rely more on sensory feedback to help with movement accuracy as a compensatory
648   mechanism for impairments in feedforward control, though at some loss of movement speed
649   and fluidity. It may be that our sample did not include enough participants with altered
650   feedback gains to show an effect at the group level. If this is the case, we might still expect to
651   find some evidence that individuals with larger feedback gains have greater impairments in
652   feedforward control, even if there were no group differences. However, we found no evidence
653   of this negative correlation. While this does not eliminate this explanation, it may make it an
654   unlikely account.
655   A final potential explanation is that increases in feedback gains are domain-specific
656   while impairments in feedforward control are more general, at least at the group level. This
657   may have to do with domain-specific use of feedback for sensorimotor control. The strongest
658   example of higher feedback gains in individuals with cerebellar degeneration comes from work
659   on pitch control, where the on-line response to a pitch perturbation was roughly twice as large

660 in a CD group compared to a control group (Houde et al., 2019; Li et al., 2019). This increase
661 may result from an inherent reliance on auditory feedback for pitch control, compared to
662 higher reliance on feedforward control for speech and reaching. There is some evidence that
663 pitch control is indeed heavily weighted towards online feedback control even in healthy
664 individuals: pitch control rapidly degenerates after post-lingual hearing loss while oral
665 articulation is better maintained (Cowie and Douglas-Cowie, 1983; Lane and Webster, 1991).
666 Thus, cerebellar degeneration may cause increased feedback gains only in those motor domains
667 which are already primarily reliant on sensory-feedback-based control. The lack of any
668 correlation between the magnitude of feedback-based corrections across motor domains is
669 consistent with the idea that changes in feedback use may be domain-specific.
670
Conclusions
672 Adaptation of feedforward control based on sensory errors is impaired in individuals
673 with cerebellar degeneration in both reach and speech. Interestingly, these individuals
674 exhibited intact feedback control in both domains. Contrary to our initial hypothesis and data
675 from vocal pitch control, we found no evidence for increased feedback gains in either domain.
676 However, these results, together with those from a recent study of upper extremity control
677 (Zimmet et al., 2020), motivate further investigation into how feedback gains may be
678 differentially affected by the specific demands of different motor tasks, as well as to determine
679 the variability in feedback control associated with cerebellar dysfunction.
680

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Appendix

Table A1: Characteristics of individuals with cerebellar degeneration. One participant, shown in gray, was excluded due to a MOCA score indicative of moderate cognitive impairment.

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Diagnosis	Age	MOCA	SARA overall score	SARA upper limb subscore	SARA intention tremor subscore	SARA speech subscore
AOA2	42	27	22	6	2.5	2
SCA3	61	28	16	5	1.5	3
SCA3	75	26	16.5	5.5	1.5	0
SCA3	65	25	24	7	2	1
SCA6	77	22	8	2	1	1
SCA6	70	23	7.75	3.25	0.5	0
SCA6	60	26	3	0.5	0	0
SCA6	49	26	1	1	0	0
SCA6	65	24	9	3.5	0.5	0
SCA6	57	27	6	3	0	0
SCA8	57	27	11	3.5	1	0
SCA8	53	30	3.5	2	0	0
SCA, sporadic	57	22	10	4	1	2
SCA, unknown	65	17	25	6	2	3
SCA, unknown	89	26	9.5	2	0.5	4
SCA, unknown	68	27	23	5	2	1
SCA, unknown	64	25	8	1.5	0.5	1
SCA, unknown	37	27	14	3	1	2
SCA, unknown	72	27	18.5	4	1.5	1
SCA, unknown	64	28	11	3.5	0	1.5
SCA, unknown	54	28	8.5	4.5	0.5	3
SCA, unknown	65	25	9.5	2.5	1	0
SCA, unknown	62	30	12	5.5	1	1

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