

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

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26

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.  
62

## 63 Introduction

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

74 If, and how, the cerebellum contributes to feedback control is less clear. One clue comes  
75 from intentional tremor, a prominent feature of CD where low-frequency oscillations occur  
76 around the movement endpoint. Intentional tremor is reduced when movement is produced  
77 without visual feedback (Day et al., 1998). Such behavior is broadly consistent with a control  
78 system that relies on visual feedback (Beppu et al., 1987), and could occur if the gains on  
79 sensory feedback errors are larger in individuals with CD. Larger gains could lead to  
80 overcorrections for errors and the need for additional counter-corrections. This hypothesis is  
81 supported by evidence showing that, relative to controls, individuals with CD produce larger  
82 compensatory responses to auditory perturbations of speech (Parrell et al., 2017; Houde et al.,  
83 2019; Li et al., 2019).

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

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

102 The preceding hypotheses are based on inferences drawn from disparate studies that  
103 distinct methods and typically focus on a single motor domain. In the present study, we used a  
104 2 x 2 design to evaluate feedback and feedforward control in two motor tasks, one involving  
105 visually-guided reaching and the other speech production. To avoid potential ceiling effects in  
106 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 Participants

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

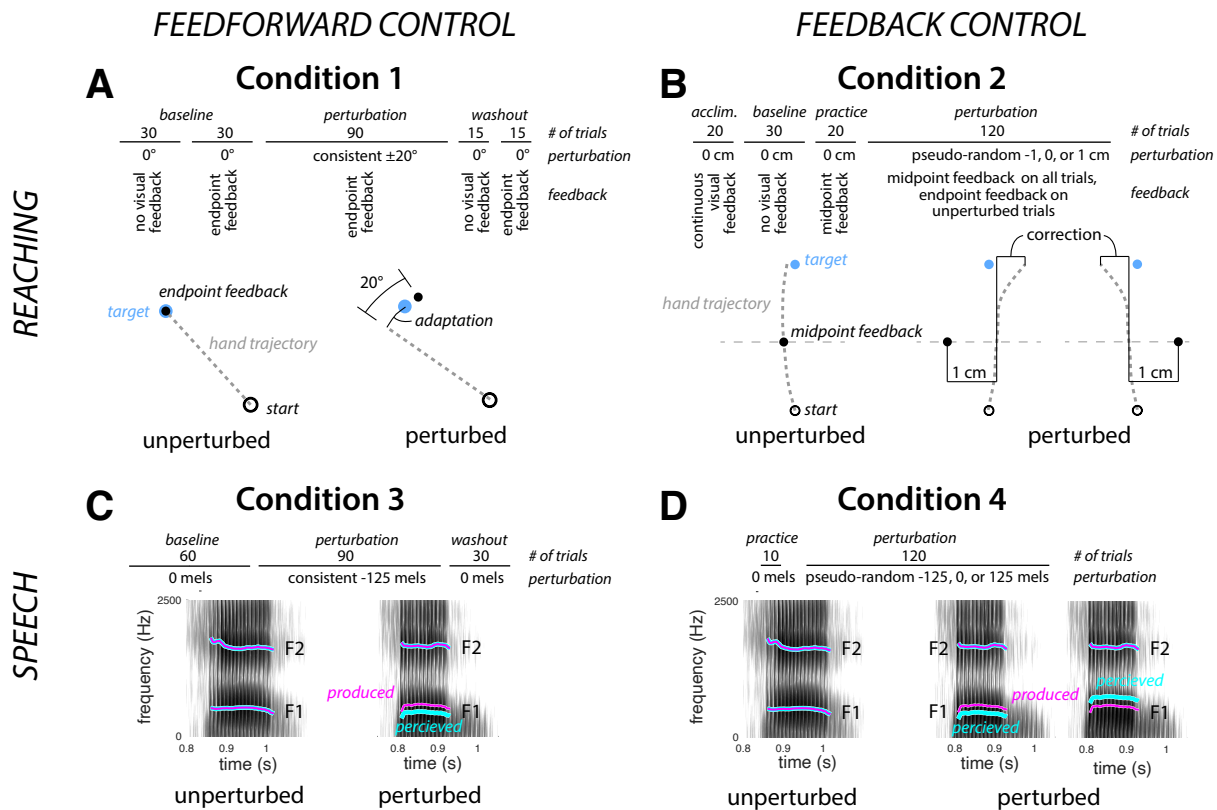
### 134 Experimental Design

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

145

146



147  
 148 **Figure 1: Experimental design.** The top row of each panel shows the structure of the experimental  
 149 session and the bottom part depicts the perturbation. **A:** Condition 1, feedforward control in reaching:  
 150 Participants made 8 cm center-out reaches to targets (blue dot) with endpoint feedback. During the  
 151 perturbation phase, the location of the feedback cursor was rotated by 20° from actual hand position  
 152 (direction cross-balanced across participants). The dashed line represents the unseen hand movement. **B:**  
 153 Condition 2, feedback control in reaching: Participants made 16 cm reaches to a target (blue dot).  
 154 During the perturbation phase, visual feedback about hand position (black dot) was given at reach  
 155 midpoint with the feedback shifted by -1, 0, or 1 cm, with the shift on each trial determined in a  
 156 pseudo-random manner. The thin gray line depicts location of reach midpoint, but was not visible to  
 157 the participant. **C:** Condition 3, feedforward control in speech: Participants spoke a single word on  
 158 each trial, hearing a playback of their speech over headphones with minimal delay. During the  
 159 perturbation phase, feedback of the first formant during the vocalic portion of the utterance was  
 160 perturbed by imposition of a -125 mel shift (cyan trace superimposed on the spectrogram). **D:**  
 161 Condition 4, feedback control in speech: Participants spoke a single word on each trial. During  
 162 the perturbation phase, auditory perturbations (cyan trace) of -125, 0, or 125 mels were  
 163 pseudo-randomly applied to the first formant during the vocalic portion of the utterance.

164 **Condition 1: Feedforward control in reaching.**

165 Participants were seated in front of a 53.2 x 20 cm LCD screen (ASUS) that was horizontally  
 166 encased in a table frame mounted 27 cm above a 49.3 x 32.7 cm digitizing tablet (Intuos 4XL,  
 167 Wacom, Vancouver, WA). Participants held a modified air hockey paddle and made reaching  
 168 movements by sliding the paddle across the table. The position of a stylus embedded in the  
 169 paddle was recorded by the tablet at 200 Hz. Feedback, when available, was presented in the  
 170 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 ( $0^\circ$ ,  $120^\circ$ , or  
180  $240^\circ$ ). 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  $\leq 500$  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  $20^\circ$  from the true position. The  $20^\circ$   
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:

$$254$$
$$255 x_{\text{target}} \sim \beta_0 + \beta_1 x_{\text{midpoint}} + \beta_2 \text{perturbation(L)} + \beta_3 \text{perturbation(R)} + \beta_4 x_{\text{midpoint}}:\text{perturbation(L)} +$$
$$256 \beta_5 x_{\text{midpoint}}:\text{perturbation(R)}$$

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,  $\beta_2$ ,  $\beta_3$ ) and 2)  
260 corrections for self-produced variability (the midpoint position estimate,  $\beta_1$ , and its adjustment  
261 in the presence of perturbations,  $\beta_4$ ,  $\beta_5$ ). For statistical analysis, the sign of  $\beta_2$  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 ( $\beta_0$ ), 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 values 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 /ε/. 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 Statistical Analysis

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 ( $\beta_2, \beta_3$ ). 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 ( $\beta_1$ ),  
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**

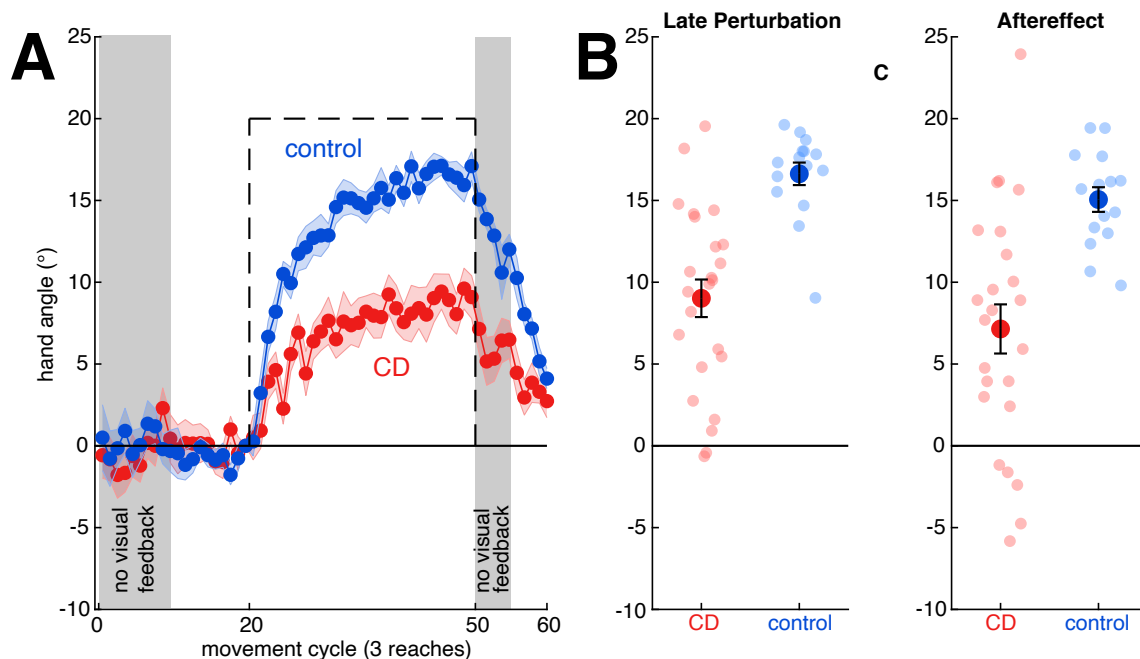
387

### 388 Condition 1: Feedforward control in reaching.

389 In the baseline phase with visual feedback, the CD group was substantially more variable  
390 than controls (standard deviation of  $6.2 \pm 2.0^\circ$  vs  $4.0 \pm 0.8^\circ$ ,  $t(31.6) = 4.6$ ,  $p < 0.0001$ ,  $g = -0.89$ )  
391 and less accurate (percentage of targets hit,  $67 \pm 25\%$  vs  $87 \pm 15\%$ ,  $t(35.8) = -3.0$ ,  $p = 0.005$ ,  $g =$   
392  $1.28$ ). The CD group also produced slower movements than controls ( $347 \pm 148$  ms vs  $264 \pm 61$   
393 ms,  $t(31.6) = 2.4$ ,  $p = 0.02$ ,  $g = 0.67$ ) and had longer reaction times ( $544 \pm 116$  ms vs  $442 \pm 58$   
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  $20^\circ$  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 ( $16.6 \pm 2.7^\circ$ ,  $t(14) = 24.1$ ,  $p < 0.0001$ ,  $g = 5.90$ ) and CD groups ( $9.2 \pm 5.7^\circ$ ,  $t(22) = 7.8$ ,  $p <$   
407  $0.0001$ ,  $g = 1.56$ ). The adapted response persisted into the aftereffect block (controls:  $15.1 \pm$   
408  $2.9^\circ$ ,  $t(14) = 20.0$ ,  $p < 0.0001$ ,  $g = 4.87$ ; CD:  $7.3 \pm 7.5^\circ$ ,  $t(22) = 4.7$ ,  $p < 0.0001$ ,  $g = 0.95$ ), 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.



415  
416 **Figure 2: In the visuomotor rotation task, the CD group reached a lower level of adaptation**  
417 **and exhibited a smaller aftereffect relative to the control group. A:** Hand angle over the course  
418 of the experimental condition. Means and standard errors are shown for control participants  
419 (blue) and individuals with cerebellar degeneration (red). Visual feedback is withheld in the  
420 shaded phases. The perturbation is shown with a dashed black line. **B,C:** Individual data (semi-  
421 transparent dots) and group means  $\pm$  standard error for the CD and control participants during  
422 the last 15 reaches of the perturbation phase (B) and 15-trial aftereffect phase in which the  
423 visual feedback was withheld (C).

#### 424 Condition 2: Feedback control during reaching

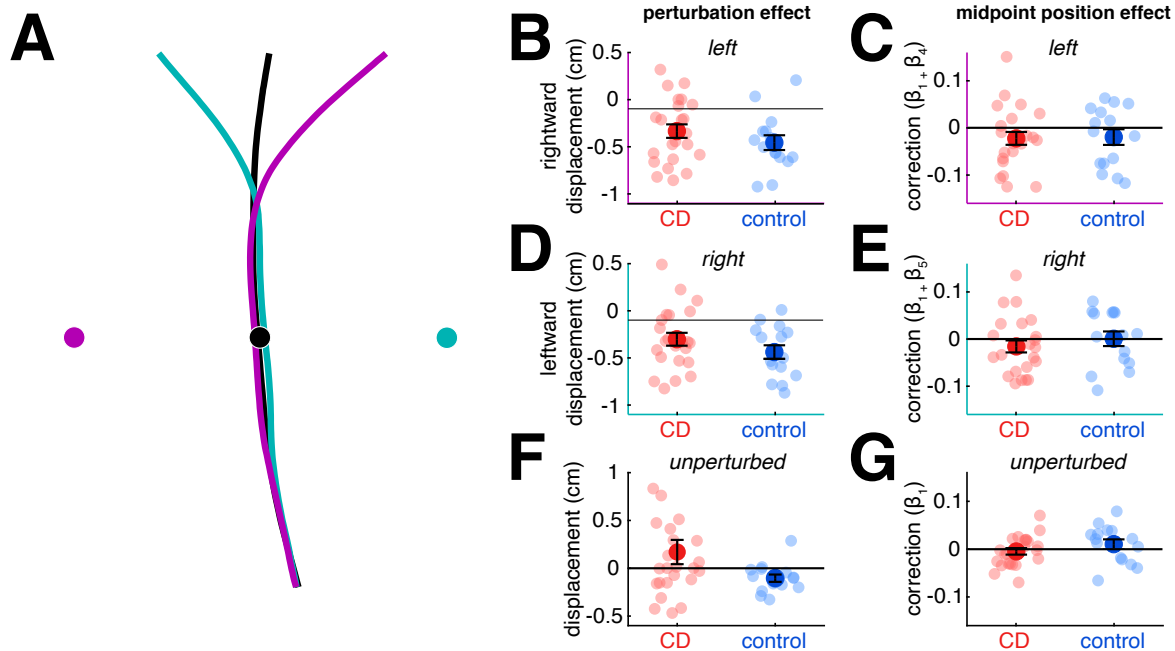
425 As in Condition 1, there were some kinematic differences between the two groups. The  
426 most salient group difference was in movement accuracy: control participants hit the target  
427 more often than the CD participants on the unperturbed trials (control:  $85 \pm 10\%$ , CD:  $70 \pm 24\%$ ,  
428  $t(31.9) = 2.7$ ,  $p = 0.01$ ,  $g = 0.75$ ). There were small differences between the groups in reach  
429 curvature, with controls showing a slight leftward shift from midpoint to target on unperturbed  
430 trials and the CD group showing a slight rightward shift on these trials (control:  $-0.17 \pm 0.193$   
431 cm, CD:  $0.13 \pm 0.52$  cm,  $t(30.2) = 2.5$ ,  $p = 0.02$ ,  $g = 0.69$ , **Figure 3F**). Reaction times were also  
432 slower in the CD group (control:  $452 \pm 79$  ms, CD:  $525 \pm 138$  ms,  $t(35.5) = 2.08$ ,  $p = 0.045$ ,  $g =$   
433  $0.61$ ) whereas movement time was similar between groups (control:  $717 \pm 114$  ms, CD:  $722 \pm$   
434  $114$  ms,  $t(30.1) = 0.14$ ,  $p = 0.89$ ,  $g = 0.05$ ).

435 Our principle dependent measure, online corrective responses, was operationalized as a  
436 lateral shift in the trajectory in response to perturbed feedback that was presented at the  
437 midpoint of the movement, with the direction of the perturbation randomized across trials. As  
438 can be seen in the data from a representative control participant (Fig 3A), the hand trajectory  
439 deviated in the opposite direction of the perturbation, a signature of a feedback-based  
440 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  $-0.21 \pm 0.34$  cm,  $t(22) = 3.3$ ,  $p = 0.003$ ,  $g = 0.67$ ; control:  $-0.35 \pm 0.30$  cm,  $t(14) = 4.6$ ,  $p = 0.0005$ ,  
444  $g = 1.12$ ) and rightward perturbation (CD:  $-0.20 \pm 0.32$  cm,  $t(22) = 2.9$ ,  $p = 0.007$   $g = 0.60$ ;  
445 control:  $-0.34 \pm 0.28$  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  $p = 0.1$ ,  $\eta^2 = 0.07$ ). There was no difference between the responses to the two perturbation  
448 directions ( $F(1,36) = 0.16$ ,  $p = 0.69$ ,  $\eta^2 = 0.004$ ) and the Group x Direction interaction was not  
449 significant ( $F(1,36) = 0.009$ ,  $p = 0.93$ ,  $\eta^2 = 0.0002$ ). 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  $\beta_1$ ,  $\beta_1+\beta_4$ ,  
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 3C,E,G**), this shift  
457 was not significantly different from 0 after correction for multiple comparisons (CD:  $-0.010 \pm$   
458  $0.035$ ,  $p = 0.05$ ,  $g = -0.30$  ; control:  $-0.009 \pm 0.042$ ,  $p = 0.24$ ,  $g = -0.21$ ). There was no difference  
459 between the two groups ( $F(1,36) = 0.01$ ,  $p = 0.91$ ,  $\eta^2 = 0.0004$ ), nor any interaction between  
460 group and direction ( $F(2,72) = 2.4$ ,  $p = 0.10$ ,  $\eta^2 = 0.06$ ). However, there was a significant effect  
461 of perturbation direction ( $F(2,72) = 7.3$ ,  $p = 0.001$ ,  $\eta^2 = 0.17$ ) such that the shift in trajectory  
462 was greater in the presence of leftward perturbations ( $-0.021 \pm 0.0332$ ) compared to trials with  
463 no perturbation ( $0.001 \pm 0.038$ ;  $p = 0.008$ ,  $g = 0.62$ ). There was a similar trend in for trials with  
464 rightward perturbations ( $-0.010 \pm 0.036$ ), 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

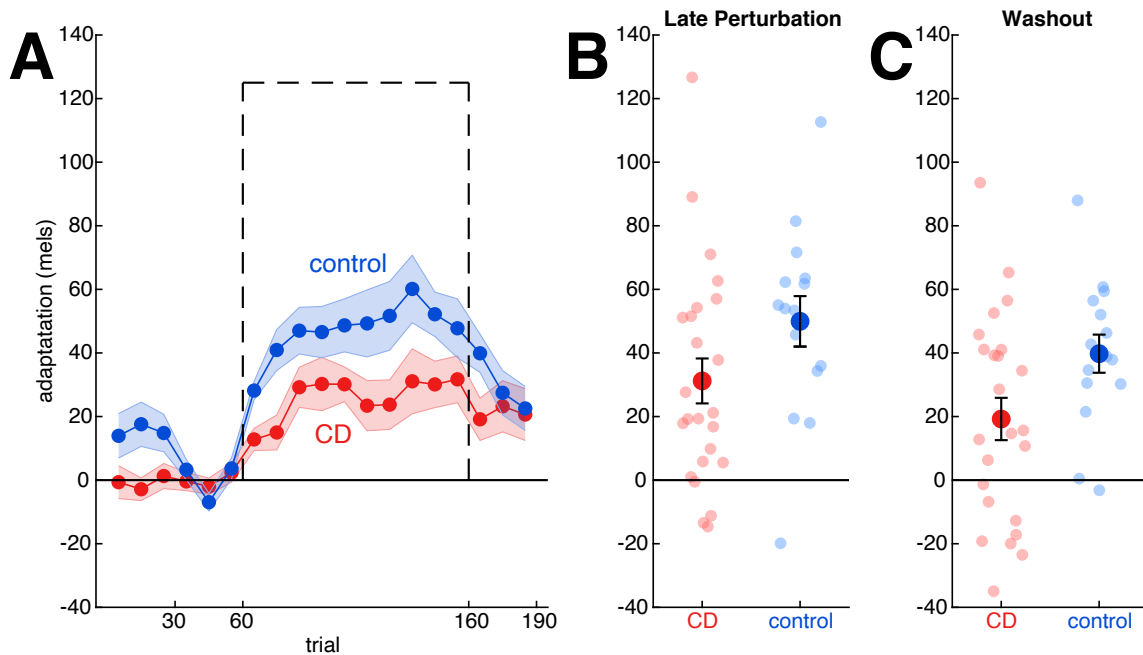


468  
 469 **Figure 3: In the reaching feedback control task, both the CD and control groups showed a**  
 470 **robust on-line correction to perturbed visual feedback presented at the midpoint of the reach,**  
 471 **with no group differences.** **A:** Example data from one control participant showing average  
 472 reach trajectories for unperturbed reaches (black), reaches with a 1 cm rightward visual  
 473 perturbation (teal), and reaches with a 1 cm leftward visual perturbation (purple). Dots in  
 474 corresponding colors show the location of the visual feedback. The corrective response only  
 475 partially corrects for the perturbation. **B, D:** Compensatory response to the feedback  
 476 perturbation. **F:** Estimate of the change in horizontal hand position from reach midpoint on  
 477 unperturbed trials. **C, E, G:** Estimates of the effect of true hand position at reach midpoint on the  
 478 compensatory response for leftward (C), rightward (E), and unperturbed (G) trials. Across all  
 479 plots, compensatory responses have negative values. Semi-transparent dots represent  
 480 individuals.

481  
 482 Condition 3: Feedforward control in speech

483 Adaptation was operationalized as the change in F1 in response to a -125 mel F1 shift in  
 484 the feedback heard by participants, introduced via the real-time resynthesis of their utterances.  
 485 As can be seen in Figure 4A, this auditory perturbation caused a gradual change in F1 that  
 486 opposed the perturbation. At asymptote, the adaptive response had reached approximately  
 487 40% of the perturbation in the control group and 25% in the CD group. The lower degree of  
 488 compensation relative to reaching is consistent with previous studies (Houde and Jordan, 1998;  
 489 Purcell and Munhall, 2006; Parrell et al., 2017). Note that, unlike Condition 1, post-perturbation  
 490 trials (washout phase) always included veridical feedback; we did not include a no-feedback  
 491 phase as masking auditory feedback with noise leads to substantial changes in speech  
 492 (Lombard, 1911; Summers et al., 1988).

493 We first confirmed that both groups adapted during the perturbation phase (CD:  $31 \pm 39$   
494 mels,  $t(1,22) = 3.9$ ,  $p = 0.0007$ ,  $g = 0.79$ ; control:  $53 \pm 31$  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  $21 \pm 32$  mels,  $t(1,22) = 3.1$ ,  $p = 0.005$ ,  $g = 0.64$ ; control:  $40 \pm 23$  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  $= 4.7$ ,  $p = 0.036$ ,  $\eta^2 = 0.12$ ), and this difference was similar in both phases (Group x Phase  
499 interaction:  $F(1,36) = 0.06$ ,  $p = 0.80$ ,  $\eta^2 = 0.002$ ). 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),



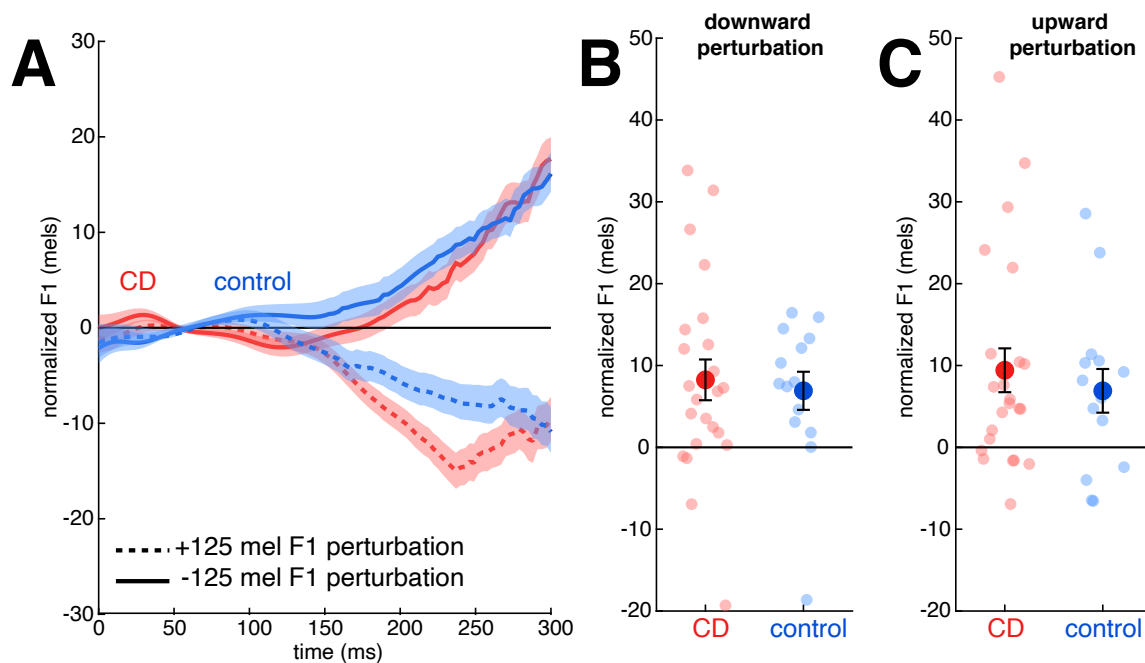
502 **Figure 4: In the speech adaptation task, the CD group adapted less than controls.** **A:** Change in first  
503 formant over the course of the experimental condition. Means and standard errors are shown for control  
504 (blue) and CD participants (red). For each participant, the change was calculated relative to their mean  
505 F1 value during the second half of the baseline phase, with positive values corresponding to changes in  
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  $\pm$  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:  $10.2 \pm 13.6$  mels,  $t(1,22) = 3.6$ ,  $p = 0.002$ ,  $g =$   
522  $0.73$ ; down:  $11.8 \pm 16.0$  mels,  $t(1,22) = 3.5$ ,  $p = 0.002$ ,  $g = 0.71$ ) and control group (up:  $11.0 \pm$   
523  $14.4$  mels,  $t(1,13) = 2.9$ ,  $p = 0.02$ ,  $g = 0.72$ ; down:  $8.2 \pm 12.4$  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  $\pm$  standard error are shown as solid dots; semi-transparent**  
543 **dots represent individuals.**

#### 544 Feedforward and feedback control within and across motor domains

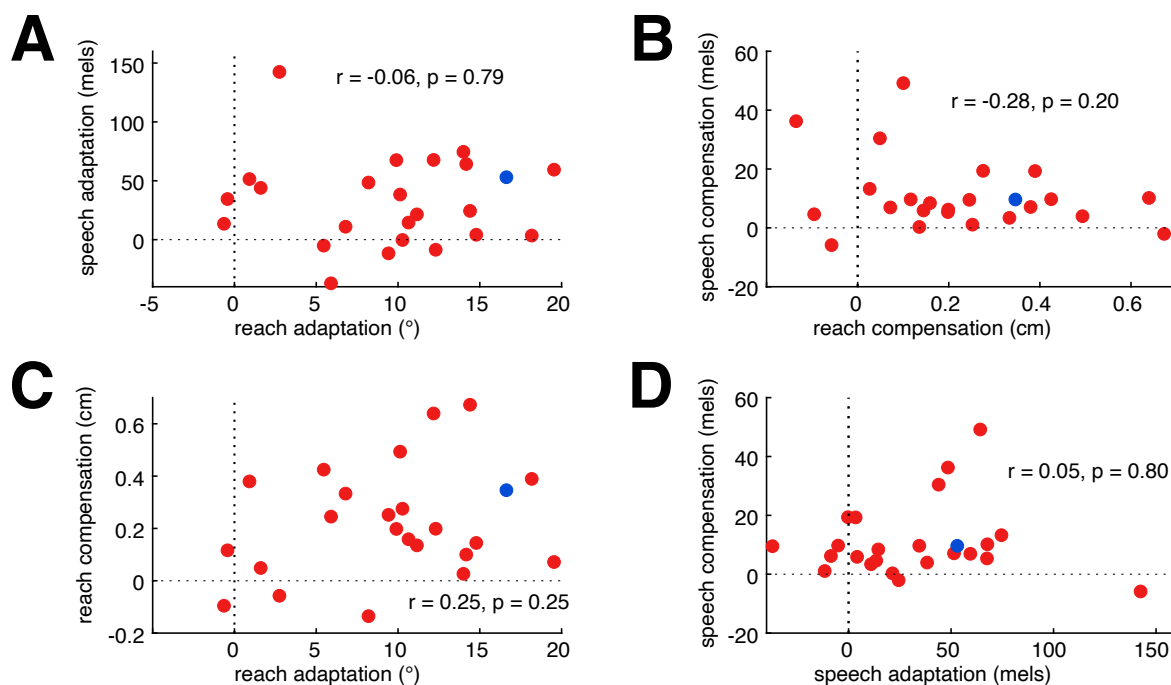
545 By testing each participant in all four conditions, we can compare the measures of  
546 feedback and feedforward control within and across task domains. Because our focus in this

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



570  
571 **Figure 6: Correlational analyses of feedforward adaptation and feedback-based compensation in**  
572 **individuals with cerebellar degeneration.** Within the CD group, there were no correlations in behavioral  
573 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

### 671 **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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800

801 **Appendix**

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

804

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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