Computation of linear acceleration through an internal model in the macaque cerebellum

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A combination of theory and behavioral findings support a role for internal models in the resolution of sensory ambiguities and sensorimotor processing. Although the cerebellum has been proposed as a candidate for implementation of internal models, concrete evidence from neural responses is lacking. Using unnatural motion stimuli, which induce incorrect self-motion perception and eye movements, we explored the neural correlates of an internal model that has been proposed to compensate for Einstein’s equivalence principle and generate neural estimates of linear acceleration and gravity. We found that caudal cerebellar vermis Purkinje cells and cerebellar nuclei neurons selective for actual linear acceleration also encoded erroneous linear acceleration, as would be expected from the internal model hypothesis, even when no actual linear acceleration occurred. These findings provide strong evidence that the cerebellum might be involved in the implementation of internal models that mimic physical principles to interpret sensory signals, as previously hypothesized.

The brain maintains internal models of the environment to interpret sensory inputs and prepare actions3–7. One function of internal models is to resolve sensory ambiguities3. In the vestibular system, sensing one’s movement during passive motion is complicated by an ambiguity related to Einstein’s equivalence principle: linear (inertial) accelerations experienced as one translates in the world are physically equivalent to the gravitational acceleration present whenever one changes orientation (that is, tilts) relative to earth-vertical. Consequently, otolith afferents in the inner ear encode the net gravito-inertial acceleration (GIA)9,10. Theoretical11–19 and behavioral5,20 studies have suggested that the brain resolves this ambiguity by using physical principles to implement an internal model of gravity. This model integrates rotation signals from the semicircular canals or vision to track an internal estimate of head orientation relative to gravity. This estimate is then used to extract linear acceleration through subtraction from the otolith-driven GIA signal, thereby resolving the gravito-inertial ambiguity.

The fact that the brain uses canal-driven signals to compute linear acceleration has been verified using lesion studies20–22. Translation-selective neurons have been found in the vestibular and cerebellar nuclei9, cerebral cortex23,24, thalamus25,26 and in Purkinje cells in the nodulus/uvula of the caudal cerebellar vermis (lobules X/IX)22,27. The cerebellum is often conceptually associated with the implementation of internal models2,28–31, suggesting that the previously identified translation-selective Purkinje cells9,22,27 represent the output of the internal model postulated to resolve the gravito-inertial ambiguity11–19. However, given that previous studies used stimuli that consisted of actual tilt and translation movements, this hypothesis could not be tested. Thus, it is presently unknown whether cerebellar responses reflect computations through an internal model or via simpler, but computationally inappropriate, filtering.

We addressed this question using an experimental protocol that induces an erroneous linear acceleration signal.

Tilt while rotating (TWR, also known as vestibular cross-coupling or vestibular Coriolis effect) is a disorienting and nauseating stimulus in humans that is commonly used in motion sickness training32–36. It consists of tilting the head while rotating continuously around an earth-vertical axis (Fig. 1a and Supplementary Movies 1a and 2a), similar to tilting the head while riding a merry-go-round (Fig. 1; also see Fig. 2 and Supplementary Modeling). During continuous rotation, the output of the semicircular canals is attenuated over time. As a result, when the head tilts in pitch during steady state, an erroneous roll rotation signal is generated in the canals (Fig. 1b and Supplementary Movie 1b,c). According to theory, this roll canal signal is integrated by the internal model to create an erroneous roll tilt estimate (as if the head were tilted ear-down; Fig. 1c). Given that the head is in fact upright, the output of the internal model of gravity is in conflict with the otolith cues, which signal that GIA is aligned with the head-vertical axis (Fig. 1b–d). The only inference consistent with these signals is that the head is simultaneously translating (Fig. 1d and Supplementary Movie 2b).

Thus, the internal model hypothesis predicts that TWR should induce an erroneous translation signal. Indeed, such induced roll tilt and erroneous translation have both been observed in reflexive eye movements5,37. We reasoned that, if cell responses in the macaque cerebellum reflect these internal model computations, translation-selective neuron activity should also correlate with this erroneous translation signal. We tested this hypothesis by recording from nodulus/uvula Purkinje cells and deep cerebellar nuclei neurons during TWR.

RESULTS

The disorienting effects of TWR are the consequence of two phenomena. First, as a result of their physical properties, the semicircular...
canals emit incorrect rotation signals during steady state (Supplementary Movie 1). Second, because of these erroneous canal signals, the hypothesized internal model generates an erroneous estimate of orientation relative to gravity, thereby causing an illusory translation (Supplementary Movie 2).

During TWR, the subject tilts back and forth (pitch tilt; Fig. 2a–c and Supplementary Movie 1a) relative to a fixed, earth-vertical axis of rotation (EVAR; Fig. 2a and Supplementary Movie 1b). In an egocentric frame of reference, the projection of the EVAR velocity vector (Fig. 2a) on the head’s yaw (head-vertical) axis (Fig. 2a) is approximately constant (Fig. 2d). In contrast, the projection of the EVAR vector on the head’s roll (forward-pointing) axis (Fig. 2a) is negative when the head tilts forward and positive when the head tilts backward (Supplementary Movie 1b). Consequently, the roll component of head velocity reverses ($\pm 7.8^\circ$ s$^{-1}$) with each tilt movement (Fig. 2e). The brain continuously reconstructs the net rotation of the head by summing the yaw and roll velocity signals (Supplementary Movie 1c).

The semicircular canals, however, emit incorrect rotation signals during steady state because they are sensitive to angular acceleration (that is, changes in head velocity). During rotation at constant velocity (Fig. 2d), canal responses fade away with a time constant of $\sim 4$ s (ref. 38) (Fig. 2d,e and Supplementary Movie 1c). The brain has the ability to somewhat increase the duration of the rotation signal (Fig. 2d,e) via a process known as velocity storage$^{17,39}$. Nevertheless, after central rotation signals decrease to zero, the resultant rotation signal following each tilt movement is erroneously aligned with the roll axis, that is, it erroneously indicates a sideward rotation.

The key assumption of the internal model hypothesis (Fig. 3a, Supplementary Modeling and Supplementary Fig. 1) is that the brain tracks an internal model of gravity through the integration of central rotation signals$^{13,13,16-19}$. A general formula for tracking the gravity vector, $G(t)$, by integrating rotational velocity, $\Omega(t)$, in three dimensions is

$$G(t)=\int G(t)\times\Omega(t)dt$$  \hspace{1cm} (1)$$

where $\times$ denotes the vector cross-product. Translational acceleration can then be extracted as the difference between this gravity estimate and GIA, the signal carried by primary otolith afferents

$$A(t)=G(t)-GIA(t)$$  \hspace{1cm} (2)$$

Ideally, equation (1) would provide an exact estimate of gravity. In practice, however, limitations exist because the semicircular canals do not always provide a reliable rotation signal (Fig. 2d,e). Any error in the rotation velocity, $\Omega$, would cause an error in the tilt estimate (which is exactly what happens during steady-state TWR). In the absence of a correction system, errors would remain indefinitely and accumulate over time$^{17}$. To avoid severe disorientation, the brain corrects such errors by a feedback loop (somatogravic feedback), which slowly, but continuously, aligns the internal model output with the GIA$^{16,17}$. Incorporating this loop into the model results in equation (1) being replaced by

$$G(t)=\int G(t)\times\Omega(t)dt - \frac{1}{\tau_s}(G(t)-GIA(t))$$  \hspace{1cm} (3)$$

where $\tau_s$ is the time constant (one of five model parameters; Supplementary Modeling). The rationale behind this process is...
as follows: in general, the GIA (otolith response) may differ from the output of the gravity estimator (G) because of linear accelerations. However, long-duration accelerations are infrequent in everyday life. Thus, long-duration mismatch between G and GIA should be corrected by aligning G toward the GIA. The somatogravic feedback reflects the expectation that it is more likely that we are stationary (but tilted) than accelerating in the world. Accordingly, it has also been modeled as a Bayesian prior centered on zero linear acceleration\textsuperscript{16,17}.

Based on the model shown in Figure 3a, the induced roll signal is integrated by the gravity estimator (equation (3)) to generate an erroneous roll tilt and, given equation (2), an erroneous translation signal. Without somatogravic feedback, the induced roll velocity signal would result in a large erroneous roll tilt (\(-50^\circ\)), thereby creating a huge translation estimate (\(>7.5 \text{ m s}^{-2}\)), both of which would remain indefinitely (Fig. 3b). The somatogravic feedback substantially reduces both the magnitude and duration of the induced tilt and erroneous linear acceleration (Fig. 3b).

The simulations shown in Figure 2f are based on the model shown in Figure 3 (see Supplementary Modeling for model parameters). Note that tilt movements delivered at the onset of EVAR (initial TWR) should not create an erroneous translation (Fig. 2f) because the yaw and roll canal-driven signals decay together, thereby suggesting a vertical rotation direction (Supplementary Movie 1c). Thus, by examining differences in the magnitude of responses to initial versus steady-state TWR, we can test the hypothesis that the postulated internal model (Fig. 3a) uses three-dimensional rotation information, as predicted by the implementation of the vector differential equation (3).

With these predictions in hand, we next examined how translation-selective cells in the nodulus/uvula and cerebellar nuclei respond during TWR. For simplicity, we will refer to each of the components of Figure 2 as actual tilt (Fig. 2b,c), induced tilt (Fig. 2e) and induced linear acceleration (Fig. 2f). If the cerebellum indeed reflects the output of the postulated internal model to disambiguate gravito- inertial acceleration, we expect translation-selective Purkinje cells to respond during steady-state TWR in a manner consistent with the simulations shown in Figures 2f and 3b (hypothesis 1). Alternatively, if no internal model is used, or if cerebellar neuron activity does not reflect these internal model computations, translation-selective cells should not modulate during TWR, as there is no actual linear acceleration stimulus. In addition, responses to initial TWR would reveal whether the internal model uses three-dimensional canal signals to compute a veridical estimate of angular velocity (hypothesis 2). If so, these erroneous translation responses should build up gradually, as EVAR velocity decays after the initial TWR response.

We recorded simple spike activity from nodulus/uvula Purkinje cells\textsuperscript{22,27} and from cerebellar nuclei neurons\textsuperscript{23} (located mostly in the rostral fastigial nucleus\textsuperscript{10,41}) that responded selectively to translation, rather than GIA (Supplementary Fig. 2). We recorded from vestibular-only cells that do not respond to eye movements. We examined TWR responses from a representative translation-selective nodulus/uvula Purkinje cell\textsuperscript{22} (Fig. 4), which responded vigorously during sinusoidal translation (Fig. 4a), but only weakly during an equivalent roll tilt stimulus (Fig. 4b). The cell also responded vigorously during TWR, with large increases and decreases in firing rate following every tilt movement (Fig. 4c,d).

Whether the firing rate increases or decreases depended on both the direction of tilt (nose down versus nose up; Fig. 4f) and the direction of EVAR (Fig. 4c,d). During leftward rotation (Fig. 4c), this particular example cell was activated in response to forward (negative) tilt and inhibited in response to backward (positive) tilt. This pattern was reversed during rightward rotation (Fig. 4d). The observed dependence on the direction of EVAR indicates that these responses do not merely represent the tilt movement itself. Accordingly, the cell did not respond to an identical sequence of tilts performed in the absence of EVAR (Fig. 4e).

The characteristic reflexive horizontal eye movements (vestibulo-ocular reflex, VOR) generated during TWR are also relevant to this analysis (Supplementary Fig. 3). At the onset of constant velocity, a horizontal VOR was elicited that slowly decayed to zero (Fig. 4c,d). The direction of this eye velocity response was rightward (negative; Fig. 4c) during leftward EVAR and leftward (positive; Fig. 4d) during rightward EVAR, and this was not dependent on pitch tilt direction (Supplementary Fig. 3). Thus, the initial horizontal eye velocity is an angular VOR (aVOR) that is a response to the EVAR. Each subsequent pitch movement generated a vertical aVOR (Fig. 4e) and resulted in a short-lasting horizontal eye velocity whose direction depended on both the direction of tilt and the direction of EVAR (Fig. 4c,d). In contrast, no horizontal eye velocity was generated without EVAR (Fig. 4e). Indeed, as shown previously\textsuperscript{6}, these horizontal eye movements reflect a translational VOR (tVOR) in response to the induced translation signal generated according to the internal model (Fig. 3a). Notably, neither the horizontal eye movements nor the cell’s responses were a result of a yaw aVOR signal, as the horizontal canals were minimally activated during these tilts (Supplementary Movie 1c and Supplementary Fig. 4) and responses depended on both the direction of tilt and direction of rotation, whereas the yaw aVOR only depended on the latter (see Supplementary Fig. 4).

We sought to test whether the cells’ responses correlate qualitatively and quantitatively with this erroneous translation signal. We first analyzed steady-state responses (more than 30 s after the onset of TWR).
Steady-state responses during TWR

If cell modulation during TWR reflects responses to the erroneous translation signal, we would expect to find a correlation between the magnitude of the TWR response (Fig. 5a) and the cell’s gain during actual translation. Indeed, there was a significant correlation for TWR in both the preferred direction ($R^2 = 0.59$, $F$ test, $P < 10^{-4}$; Fig. 5b) and the anti-preferred direction ($R^2 = 0.35$, $F$ test, $P < 10^{-5}$; Fig. 5c) responses. Here, preferred direction is defined as the tilt and EVAR combinations that would theoretically elicit an erroneous translation in the preferred direction of the cell (expected to increase firing rate). Similarly, tilts in the anti-preferred direction refer to movements that would elicit an erroneous translation in the opposite direction (expected to decrease firing rate). In contrast, we found no significant response and no correlation during tilts without EVAR ($R^2 = 0.003$, $F$ test, $P = 0.7$; Fig. 5d). Nodulus/uvula Purkinje cells and cerebellar nuclei neurons responded similarly (similar regression slopes; Supplementary Fig. 5). Thus, nodulus/uvula and cerebellar nuclei cell responses are pooled hereafter.

These results suggest that increases and decreases in firing rate that were time-locked to each tilt movement during steady-state TWR qualitatively follow the sign predicted for the induced translation signal and that the response magnitude correlates with the respective translation gain of each cell. Both of these findings are consistent with the hypothesis that translation-selective cerebellar cells carry a signal proportional to the erroneous translation during TWR. The slope of the preferred direction responses was $1.4 \pm 0.1° \text{s}^{-1}$, $95\%$ confidence interval $1.1, 1.7$ m s$^{-2}$. For TWR responses in the anti-preferred direction, the slope was $0.8 \pm 0.05$ m s$^{-2}$. These population response slopes can be interpreted as a way to decode the magnitude of the induced translation during steady-state TWR. The decoded value is close to that predicted by the internal model ($-1 \text{ m s}^{-2}$; Fig. 3a,b).

We analyzed the time course of the population response to steady-state TWR (Fig. 6a). The population activity decayed with a time constant of 4 s (monkey V), 2.2 s (monkey T) and 1.3 s (monkey K). The anti-preferred direction population responses were weaker, as is often the case for cerebellar neurons, and were negligible in monkeys T and K.

The tVOR responses lasted about 20 s (Fig. 6b), consistent with previous measurements (see Figure 3e–h in ref. 5). The dynamics of tVOR responses involved additional temporal filtering that is not included in the simplified model shown in Figure 3a (refs. 43, 44; Supplementary Fig. 6). The vertical aVOR that compensates for the actual tilt movement matched the imposed tilt velocity in both magnitude ($18 \pm 0.1° \text{s}^{-1}$, mean ± s.e.; that is, a gain of 0.87) and duration (1.4 s). The induced aVOR, which is a direct measure of the induced activation of the canals, reached a similar peak velocity ($12 \pm 1.3 \text{ m s}^{-2}$) and $10 \pm 0.5° \text{s}^{-1}$ in monkeys V, T and K, respectively; Fig. 6c and Supplementary Table 1), corresponding to a gain of $-0.8$ relative to the simulated induced signal. However, its time constant varied across monkeys (monkey V, 8.3 s; monkey T, 3.5 s; monkey K, 1.8 s). Model simulations followed the basic characteristics of both neural responses and induced aVOR (Fig. 6a,c). Finally, both the neuronal response and the behavioral output (tVOR) showed a sluggish rise, as the stimulus (that is, the induced tilt and translation signal) rose slowly over a period of 1.4 s (duration of pitch tilt). This slow build-up, predicted by the differential filtering of each variable in the internal model (Fig. 3), is highly consistent with experimental observations (Supplementary Fig. 6).

The internal model (Fig. 3a) is based on physical and geometrical rules and has only five parameters (Supplementary Modeling). Four parameters are used to describe the canals and the central processing of rotation information. These parameters were only used to compute the central rotation signal, $\Omega(t)$, which could be observed in the aVOR of each monkey. Thus, these four model parameters were chosen such that they simulated the evoked eye movement. In the steady state, $\Omega(t)$ is identical to the induced signal, which was reproduced accurately in all monkeys (Fig. 6c). Given $\Omega(t)$ and the otolith-driven GIA(t), the model computes the acceleration...
signal, $A(t)$, using equations (3) and (2), with only a single additional free parameter, $\tau_n$, which was set to $\tau_n = 0.5$ s. Notably, the simulated acceleration signal closely matched the neuronal population response along the preferred direction in all monkeys (Fig. 6a). This suggests that the variations in the duration of $\Omega(t)$ were sufficient to explain the quantitative differences in neural responses across monkeys. Model simulations also predicted larger induced linear acceleration for $120^\circ$ s$^{-1}$ versus $45^\circ$ s$^{-1}$ rotations ($2.1$ versus $0.9$ m s$^{-2}$, respectively), although increases in neural activity were smaller ($1.8 \pm 0.7$ versus $1.2 \pm 0.6$ m s$^{-2}$; Fig. 6a), possibly suggesting some saturation effect.

**Influence of yaw rotation signals**

Quantification of the responses obtained during steady-state TWR provides support for hypothesis 1. It is known, however, that TWR does not have a disorienting effect if the tilt movement is performed shortly after the beginning of EVAR (initial TWR). This is fortunate, as such a movement is experienced very commonly, for example, when people nod their heads while walking around a corner. We next explored whether initial TWR responses are indeed reduced compared with steady-state responses (hypothesis 2). In theory, initial TWR differs from steady-state TWR in two respects.
First, the yaw component of the rotation signal provided by the canals is veridical, as it has not yet decreased (Fig. 2d). Second, the induced rotation signal is weaker (62%; Fig. 2e and Supplementary Modeling) during initial TWR than during steady state. If the erroneous acceleration during TWR is a consequence of the induced rotation signal alone, then the initial TWR should induce a substantial linear acceleration (62% as strong as during steady state). In contrast, the theoretical framework shown in Figure 3a predicts that initial TWR should not induce any translation signal (as the direction of $\Omega(t)$ is veridical; Supplementary Movie 1a).

Similar to steady state, the induced aVOR precisely followed the dependence predicted by model simulations, although the latter was half as large as the former (Figs. 2e and 7a). A large difference between initial and steady-state responses was also seen for the induced tVOR. Although small and short-lasting during the first tilt movement, horizontal eye velocity (tVOR) was strong during steady state (Figs. 6b and 7b).

We then compared the preferred direction neuronal population responses to initial and steady-state TWR (Fig. 7c). Like the tVOR (Fig. 7b), but unlike the induced aVOR (Fig. 7a), the initial TWR elicited only a small response, consistent with model simulations (Fig. 7c). We measured the increase in firing rate in a 1-s interval following the actual tilt movement on a cell-by-cell basis. If this response was driven by the induced rotation signal alone (without an internal model), we would expect the cells’ firing rates to be 62% as large during initial TWR as during steady-state TWR. In contrast, we found that the neuronal responses to initial TWR were, at the population level, only 25% as large as the responses to steady-state TWR (linear regression, slope = 0.25 ± 0.05, mean ± s.e.; Fig. 8a). The slopes were identical for nodulus/uvula ($n = 27$, slope = 0.24 ± 0.14, 95% confidence interval) and cerebellar nuclei ($n = 31$, slope = 0.16 ± 0.15) neurons (Supplementary Fig. 5).

That the initial TWR responses are suppressed provides further support for the hypothesis that these cerebellar responses result from processing through an internal model that uses three-dimensional signals from all semicircular canals, as necessary to implement equation (3). This finding was further quantified by measuring how TWR responses increase as a function of time from EVAR onset (Fig. 8b). The experimental data fell close to simulations based on the internal model and did not match simulations that assumed that the response is proportional to the induced aVOR (and no internal model). Altogether, our data indicate that cerebellar translocation-selective neurons encode a neural estimate of the erroneous linear acceleration whose magnitude increases as the yaw rotation signal decreases.

**DISCUSSION**

Using TWR stimuli, we found that translation-selective cerebellar cells also encode an erroneous linear acceleration, even when no actual translation occurs. Neural response properties followed theoretical predictions in which the brain generates an internal estimate of translation as the difference between the net otolith signal and the output of an internal model of gravity. In fact, simulations of a
simple model that captures these ideas result in predicted erroneous linear acceleration signals that are quantitatively similar to responses of translation-selective cerebellar neurons.

Using a similar experimental protocol, a previous study found that erroneous translation estimates can be measured in human tVOR responses. The illusion of induced tilt during TWR has also been measured in humans and primates. In contrast, direct evaluation of translation perception during TWR-like stimuli is difficult given the disorienting, confusing and nauseogenic nature of these movements in humans. It is unclear whether the confusion and disorientation experienced by humans during TWR masks the perception of linear acceleration or whether the induced translation signals are the cause of confusion and disorientation. It is for this reason that we, like others, focused on eye movements rather than perception as a behavioral correlate of the postulated linear acceleration signal.

The conclusion that responses of these cerebellar neurons during TWR reflect their sensitivity to a centrally generated translation signal is based on the following observations. First, steady-state TWR responses were significantly correlated with the cell's gain during actual translation (Fig. 5b,c) and there were no tilt-evoked responses in the absence of EVAR, a stimulus that did not elicit an erroneous translation signal (Fig. 5d). Second, steady-state TWR responses matched the translation signal predicted by the internal model hypothesis in both magnitude (Fig. 6a) and dynamics (Fig. 6a). Third, variations among monkeys were satisfactorily predicted by differences in the dynamics of the central processing of rotation signals, which are well understood and quantified from reflexive eye movements (induced aVOR; Fig. 6c). Finally, the magnitude of this tilt-evoked TWR activity was negligible in the early part of each trial (initial TWR), but gradually increased with time, with a time constant of ~14 s (Fig. 8).

This latter finding is particularly intriguing when compared with model predictions. According to the internal model hypothesis, conceptualized by equation (3), the brain has access to an accurate estimate of three-dimensional angular velocity, $\Omega(t)$. The suppressed neural response to initial TWR suggests that the brain indeed uses signals from both the horizontal and vertical semicircular canals to compute a reliable three-dimensional estimate of $\Omega(t)$ and, consequently, G(t). Thus, even though nodulus/uvula Purkinje cells do not modulate during yaw rotations, their responses carry an explicit and functionally important yaw rotation signal. Our findings provide an explanation for the puzzling fact that, although nodulus/uvula Purkinje cells lose their ability to distinguish tilt from translation following local gabazine injections (T.A. Yakusheva, D.A. and P.M. Blazquez, unpublished observations), suggesting that it is possible that these internal model computations utilize GABAergic circuitry in the cerebellar cortex.

**METHODS**

Methods and any associated references are available in the online version of the paper.

Note: Any Supplementary Information and Source Data files are available in the online version of the paper.

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**AUTHOR CONTRIBUTIONS**

J.L. designed and performed the experiments, analyzed the data, and prepared the manuscript. H.M. performed experiments. D.E.A. designed the experiments and prepared the manuscript.

**COMPETING FINANCIAL INTERESTS**

The authors declare no competing financial interests.

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ONLINE METHODS

Animals and experimental setup. Three male rhesus monkeys (V, T and K, 6, 4 and 8 years old, respectively; Macaca mulatta) were chronically implanted with a single saccular search coil to measure horizontal and vertical eye movements\(^5\text{3-52}\) and were used for neural recordings. Two additional monkeys (P and N; both 5 years old) were implanted with a dual eye coil and used for three-dimensional (horizontal, vertical and torsional) eye movement recordings. All monkeys were also implanted with a circular delrin ring to immobilize the head and a delrin platform with staggered rows of holes for neural recordings. To provide better access to the midline of the cerebellum, the platform was tilted leftward 10° in two of the monkeys (T and K). In the third monkey (V), the platform had a double angle: it was tilted both rightward 10° and forward 10°. All experimental procedures were carried out in accordance with US National Institutes of Health guidelines and were approved by the Animal Studies Committee of Washington University in St. Louis. Monkeys were pair-housed under natural dark-light cycle. Monkeys T and K had no prior history of testing, monkeys P and N were used for cortical recording studies, and monkey V was used for other neurophysiology studies, including injections of a reversible GABA antagonist (gabazine).

During experiments, the monkeys were comfortably seated in a primate chair secured inside the inner gimbal of a vestibular stimulator composed of a three-axis rotator mounted on a 2-m linear sled (Acutorphins). The first two axes allowed the monkey to be tilted in any orientation and the third axis rotated the setup around an earth-vertical axis. The monkeys were positioned such that all three rotation axes were aligned with the center of the head and the stereotaxic-horizontal plane was earth-horizontal. The eye coil signals and the stimuli were filtered (200 Hz, 6-pole Bessel) and digitized at a rate of 833.33 Hz (model 1401, CED, 16-bit resolution, Cambridge Electronics Design).

Neural recordings. Cerebellar nuclei neurons and Purkinje cells in the nodulus (lobule 10) and uvula (lobule 9) (collectively referred to here as nodulus/uvula) were recorded extracellularly using epoxy-coated tungsten microelectrodes (9–12-MΩ impedance, FHC) through 26-gauge guide tubes. Neuronal data was acquired using an analog channel of the 1401 (33 kHz) and analyzed offline using a custom Matlab (MathWorks) script to extract spike timing from the raw neuronal data. We sorted spikes manually on the basis of spike statistics (amplitude, impedance, FHC) through 26-gauge guide tubes. Neuronal data was then used inrefs. 9, 20–22, 27. These stimuli consisted of either pure tilt, pure translation, or combined translation and tilt (translation-tilt and tilt + translation). The tilt stimulus was a 0.5-Hz sinusoidal rotation around upright with peak amplitude of 11.5° (and peak velocity of 36° s\(^{-1}\)). Because this motion reorients the head relative to gravity, otolith afferents were stimulated by a 0.5-Hz gravitational acceleration component in the horizontal plane with a peak magnitude of 2 m s\(^{-2}\) (0.2g). The amplitude of the translation stimulus (20 cm) was adjusted so that the inertial acceleration of 0.2g matched the horizontal gravitational acceleration during the tilt stimulus. During combined rotation and translation stimuli, inertial and gravitational acceleration components combined in either an additive or subtractive fashion depending on the relative directions of the two stimuli. As a result, the net GIA activating the otolith receptors either doubled (tilt + translation) or was nearly zero (tilt – translation), even though the actual translation of the monkey remained the same\(^9\).

These stimuli were used to first identify translation-selective cells and then evaluate which of four directions (naso-occipital, interaural and 45° in-between axes) gave the largest response. TWR movements were then delivered along the direction with the largest translation response. Although cells were tested along any one of these four directions, for simplicity the model and stimuli (Figs. 1–3) are described as if TWR were always delivered along the naso-occipital axis (that is, using pitch tilts). Axes and sign conventions were as follows: positive directions for the three head axes are forward (naso-occipital), leftward (interaural) and upward (vertical axis). Yaw, pitch and roll rotations are defined around the vertical, interaural and naso-occipital axis (positive directions: leftward, nose down and right ear down, respectively).

Each TWR run was composed of a series of 10° tilt movements performed during a single period of constant-velocity EVAR (Fig. 1). The monkey was first statically tilted 10° in either one direction perpendicular to the axis with the largest translation response. 10 s later, and while the monkey remained in the tilted orientation, constant velocity EVAR (velocity, 45° and/or 120° s\(^{-1}\); acceleration, 45° s\(^{-2}\)) was subsequently delivered. As soon as the constant velocity was reached (1 s after EVAR onset for 45° s\(^{-1}\) and 2.6 s for 120° s\(^{-1}\)), the first tilt movement (toward the opposite 10° position) was initiated (Fig. 2a–c). A total of eight tilt movements were performed, separated by intervals of 30 s. Each movement reversed the direction of tilt and followed a trapezoidal velocity profile (peak acceleration 50° s\(^{-2}\), peak velocity 20° s\(^{-1}\), total duration 1.4 s; Fig. 2b, c and Supplementary Figs. 3–6). 30 s after the last tilt movement, the EVAR was stopped. After a resting period of at least 30 s, the monkey was brought back smoothly to a new initial position and another run was initiated. At least four TWR runs were performed with opposite initial tilt directions (as in Supplementary Fig. 3) and leftward/rightward EVAR (Fig. 4). In addition, one control run was performed during which tilt movements were applied without any EVAR (Fig. 4e). In the following, we refer to each discrete tilt movement as a trial.

Sample size. For these experiments, we specifically searched for translation-responsive cells. This was done by qualitatively comparing tilt and translation responses on-line. We then used the principles described in ref. 9 to quantitatively identify translation-selective cells offline (see below). In total, we recorded from 40 cerebellar nuclei and 38 nodulus/uvula cells, of which 31 cerebellar nuclei and 30 nodulus/uvula cells (16 were confirmed and 14 were putative Purkinje cells) were classified as translation-selective neurons offline. Only the latter group of cells was included in our analyses.

Responses to 45° s\(^{-1}\) were available for 58 cells (31 cerebellar nuclei, 27 nodulus/uvula). Responses to TWR at 120° s\(^{-1}\) were available in 11 cells (6 cerebellar nuclei, 5 Purkinje cells) from monkey T only (monkeys V and K were heavier (9.2 kg and 10 kg, respectively) than monkey T (5.3 kg)), and therefore our setup could not perform TWR at 120°/s in these animals). We compared (Supplementary Fig. 5) the responses of cerebellar nuclei and confirmed and putative Purkinje cells and found no difference. We therefore pooled all data for the main analyses. To investigate how responses depend on the time between the beginning of EVAR and the tilt movement, additional TWR runs (at 45° s\(^{-1}\)) were also delivered in a subset of cells (n = 11) for which the first tilt movement occurred 5, 10, 15 or 20 s after the beginning of EVAR.

Data analyses. Responses to tilt and translation were analyzed offline using a variant of the partial correlation analysis previously employed to test whether neurons selectively encode translation, tilt or GIA\(^\text{3-22}\). Cells were classified as translation selective if the translation model fitted their activity significantly better (P < 0.05), and were excluded from our study otherwise. For cells that passed this test, the response to 0.5-Hz translation (in units of spikes s\(^{-1}\) per m s\(^{-2}\)) was used as a reference to characterize TWR responses quantitatively (see below). To quantify TWR responses, we first separated trials on the basis of whether model predictions should generate a linear acceleration in the cell’s preferred direction. Based on our sign conventions, model predictions for the direction of the induced linear acceleration depended on both the direction of tilt and the direction of EVAR. For example, a positive tilt movement around any axis (for example, pitch; Fig. 2) during positive (for example, leftward) EVAR was predicted to induce a positive acceleration along that axis (for example, leftward). Similarly, a negative tilt movement during negative EVAR should induce an identical (positive) acceleration signal. We examined whether, during sinusoidal translation, the cell increased its firing rate during acceleration...
in this direction (that is, if the peak response occurred while the acceleration was positive). If so, these trials were considered to be in the cell’s preferred direction; if not, then they were considered to be in the cell’s anti-preferred direction. Similarly, we determined whether the responses measured following negative tilt during positive EVAR and positive tilt during negative EVAR were in preferred direction or anti-preferred direction.

Simultaneously with the analyses of neural responses, we also quantified calibrated and de-saccaded eye position signals. Slow phase eye velocity was computed using digital differentiation, filtering and de-saccading\(^\text{15,53}\). The magnitude and dynamics of the tVOR depends strongly on the direction of linear acceleration and vergence angle\(^\text{54}\). Specifically, lateral linear accelerations evoke a horizontal tVOR in darkness, whereas conjugate horizontal slow phase eye velocity is negligible for fore-aft movements\(^\text{52}\). Thus, pitch TWR runs (Supplementary Fig. 3b), but not roll TWR runs (Supplementary Fig. 3e), produce an observable conjugate tVOR in darkness. Because of this property, it is possible to separate the tVOR from the yaw aVOR, using a similar method to that described in ref. 5 (Supplementary Fig. 3).

**Statistical analysis.** Data during steady-state TWR (that is, when tilt was delivered more than 30 s after the beginning of TWR) were analyzed first. For each cell, we averaged responses during all trials in preferred direction and anti-preferred direction. We measured the difference between the average firing rate over 1-s intervals immediately before the onset and after the offset of each tilt movement (Fig. 5a). This firing rate change was then plotted as a function of the cell’s sensitivity to sinusoidal translation and the Matlab function regress was used to search for correlation between the two response parameters. We confirmed that our sample is adequate for performing parametric statistics (that is, linear regression and Student statistics) by replicating all analyses after applying a method which identifies and removes outliers (Matlab function robustfit) and finding that the results were unaffected.

Correlations between the cell’s sensitivity to sinusoidal translation and its response to TWR were evaluated by performing linear regression and \(F\) tests. We computed the average firing rate following TWR in preferred direction, anti-preferred direction and control; 95% confidence intervals were computed using Student statistics (that is, as the s.d. of the firing rate across cells multiplied by \(t_{\alpha/2,n-1}/\sqrt{n-1}\)), where \(t_{0.05,n-1}\) is the critical value of the Student distribution with \(n-1\) degrees of freedom and \(n\) is the number of cells). All tests performed in the study were two-sided. All tests that yielded statistically significant results had very small \(P\) values (<10\(^{-4}\)), which compensated the effect of performing multiple comparisons.

The time course of the tilt-induced responses in translation-selective cells were also computed from TWR runs with tilt movements occurring 1–60 s after the onset of EVAR. This was done by computing correlation slopes with steady-state responses. These slope values were plotted as a function of time, and the time constant was quantified by an exponential fit using a gradient ascent procedure (function \texttt{lsqnonlin}, Matlab). The confidence interval of the exponential fit was computed by bootstrapping\(^\text{55}\). Note that the 95% confidence intervals computed for the time constant are large because exponential fits on a small number of samples can be quite variable.

**Modeling.** For comparisons with experimental data, we simulated a previously published model\(^\text{17}\) (Fig. 3a). This model can be seen as a synthesis of multiple studies based on the internal model hypothesis\(^\text{11–19}\). This model was also inspired by and consistent with another published model based on the Bayesian approach\(^\text{16}\).

Finally, simultaneously recorded eye movements were used to set the model parameters, under the assumption that they reflect signals proportional to the internal variables of induced tilt and translation signals (as previously shown\(^\text{5}\)). Note that, when the five model parameters were set such that they reproduced the behavioral responses satisfactorily (for example, Figs. 6c and 7a), the model also simulated the neuronal responses accurately. Note also that the tVOR slow-phase eye velocity likely reflects further filtering beyond an internal estimate of linear acceleration\(^\text{42,50}\) (Supplementary Fig. 6), which was not included in the five-parameter model (Fig. 3). Details about the model and the parameters used for simulations can be found in **Supplementary Modeling**.

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