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Locomotor efference copy signaling and gaze control: an evolutionary perspective

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Highlights

1. Locomotor EC-driven eye movements is an evolutionary conserved mechanism
2. EC signaling has adapted to the evolutionary increase in locomotor complexity
3. The role of ECs also changed with the evolution of vestibular sensory coding

§We dedicate this review to the memory of our colleague and dear friend Hans Straka who passed away early in the writing process. His contribution to the field of vestibular and ocular control research stands as an enduring example of scientific creativity and integrity.

Abstract

Neural replicas of the spinal motor commands that drive locomotion have become increasingly recognized as an intrinsic neural mechanism for producing gaze-stabilizing eye movements that counteract the perturbing effects of self-generated head/body motion. By pre-empting reactive signaling by motion-detecting vestibular sensors, such locomotor efference copies (ECs) provide estimates of the sensory consequences of behavioral action. Initially demonstrated in amphibian larvae during spontaneous fictive swimming in deafferented *in vitro* preparations, direct evidence for a contribution of locomotor ECs to gaze stabilization now extends to the ancestral lamprey and to tetrapod adult frogs and mice. Behavioral evidence also exists for other mammals, including humans, therefore further indicating the mechanism's conservation during vertebrate evolution. The relationship between feedforward ECs and vestibular sensory feedback in ocular movement control is variable, ranging from additive to the former supplanting the latter, depending on vestibular sensing ability, and the intensity and regularity of rhythmic locomotor movements.

Introduction

Maintaining visual acuity in spite of perturbing head/body movements is a challenge for all vertebrates during locomotion. The ability to generate eye movements that counteract head movement during self-motion can arise from multiple sources, including vestibulo-ocular reflexes (VORs) - considered the main mechanism for ensuring retinal image stability during head motion¹⁻³ - and optokinetic and proprioceptive reflexes⁴. In addition to these reactive sensory feedback processes, an intrinsic feedforward mechanism has been more recently found to contribute to gaze stability during locomotion (for recent review, see ⁵). In this scheme, efference copies (ECs; ^{6,7}) of the rhythmic motor commands from the spinal central pattern generators (CPGs) that drive actual locomotor movement, by providing predictive estimates of the resultant head motion, are employed to directly generate compensatory eye adjustments.

Here, we firstly briefly describe evidence for such EC control of ocular compensation as originally established in swimming larval frogs. We then propose that this intrinsic mechanism is not only restricted to the relatively simple undulatory swimming of amphibian larvae, like other aquatic animals, but also extends to terrestrial vertebrates, including mammals, with their more complex limb-based modes of locomotion. Finally, the functional relationship between spinal EC signaling and vestibular sensory encoding is considered in the context of maintaining visual stability in the face of the evolutionary increase in the complexity of locomotor strategies, accompanying biomechanical properties and changing sensory reference frames for head/body motion.

Locomotor EC-driven eye movements in larval frogs

The first demonstration of spinal EC control of gaze-stabilizing eye movements during locomotion derived from reduced brainstem-spinal cord preparations of larval frogs during episodes of spontaneous swimming motor activity^{8,9}. During so-called 'fictive' swimming in such preparations of *Xenopus* larvae, robust rhythmic burst discharge timed to spinal ventral root (Vr) activity is expressed in extraocular motor nerves innervating the lateral rectus (LR) and medial rectus (MR) eye muscles, despite the absence of any visuo-vestibular sensory feedback. Impulse bursts in each LR nerve occur in phase with Vr bursts on the opposite spinal cord side, whereas MR nerve bursts are in time with Vr bursts on the same cord side (Figure 1a, left). This strict coupling pattern is therefore appropriate for driving gaze-stabilizing eye movements during actual swimming *in vivo*: alternate contractions of synergistic pairs of LR/MR eye muscles produce conjugate eye movements that are oppositely directed to head undulations resulting from the tadpole's active tail motion, which occurs predominantly in the horizontal plane (Figure 1a, middle; ⁹⁻¹¹). The temporal relationship between ocular and spinal motor bursts observed *in vitro* also remains phase-constant, corresponding to eye movements that continuously compensate for horizontal undulations of the mid-caudal tail region where propulsive thrust is maximal¹². Moreover, the uniquely spinal origin of this EC signaling was evidenced by the lack of effect on the coupling following surgical removal in reduced preparations of supraspinal areas¹⁰ that elsewhere are known to be involved in locomotor control (midbrain¹³; cerebellum^{14,15}).

Together, these findings thus provided compelling evidence that in larval frogs at least, locomotor ECs deriving from spinal CPG circuitry generate an ocular motor command during swimming behavior that contributes to gaze stabilization⁹⁻¹¹. Significantly, the target-specificity, strength and timing of these feedforward signals produce ocular motor responses that are closely comparable to the motion-generated feedback responses arising from the horizontal angular VOR¹⁰. Specific lesions and neuroanatomical tracing also enabled the neural pathways that distribute the spinal EC signals in the brainstem to be identified (Figure 1a, right). After first crossing contralaterally in the spinal cord, the ascending EC from a given cord side is conveyed directly, bypassing the vestibular nuclei, to the contralateral Abducens nucleus from where LR motor neurons arise. The EC signal then re-crosses the midline via Abducens internuclear neurons to MR motor neurons on the opposite side. Thus, on arriving in the brainstem, the spinal EC command in swimming larval frogs also employs the same premotor pathways that mediate horizontal aVOR-driven eye movements, this circuitry being a common and highly conserved feature in vertebrates generally^{1,16,17}.

Locomotor EC gaze control in adult frogs and other vertebrate lineages

Locomotor EC-generated eye movements persist through frog metamorphosis when the animal's body is entirely remodeled and its locomotor strategy switches from tail-based to limb-based propulsion in the adult (Figure 1b; ^{18,19}). In contrast to the conjugate left-right eye oscillations that counteract tail/head motion in the larva, in juvenile adult *Xenopus*, the eyes now rotate inwardly (converge) during cyclic hindlimb-driven propulsion. This oculomotor behavior timed with limb movement is achieved by lumbar CPG ECs that co-activate the left/right MR motor nerves in time with bilaterally-synchronous hindlimb extensor motor nerve bursts during the forward accelerating phase of each limb kick cycle (Figure 1b, left, middle). Correspondingly, the ascending pathways mediating the coupling switch from crossed in larvae to uncrossed projections directly to oculomotor motoneurons innervating the MR muscles of each side (Figure 1b, right). During metamorphosis, as the adult appendicular system progressively emerges and replaces larval swimming²⁰, the spino-ocular coupling pathways associated with both axial CPG circuitry and the newly emerging hindlimb CPG co-exist within the same animal. As metamorphosis proceeds, the effective EC drive to the extraocular motor centers gradually shifts its influence from producing conjugate to convergent eye motion as the axial system declines and eventually disappears with tail resorption. Consequently, the EC control of eye movements remains continuously adapted to the changing visual needs associated with the switch in dominance of the animal's mode of locomotion¹⁹.

In an evolutionary context, anuran frogs like *Xenopus* lie at the interface between two of the major modes of locomotion found in the animal kingdom (Figure 2). Effectively, the metamorphic transition occurring in *Xenopus* over a period of weeks equates to the several 100 M years it took vertebrates to evolve from aquatic to terrestrial life. Since the origin of vertebrates nearly 500 M years ago, the extraocular motor and gaze-stabilizing VOR systems have been highly conserved through all lineages, despite a wide diversity in habitats and locomotor strategies^{21,22}. Therefore, the clear evidence for EC-mediated gaze control during aquatic locomotion in *Xenopus* frogs and the close functional relationship between this intrinsic feedforward mechanism and the VOR raises questions about the possible wider employment of spino-ocular motor signaling during vertebrate evolution.

Cyclostomes (hagfish and lamprey) constitute the present-day descendants of ancestral jawless craniates that possess reduced vertical semicircular canals considered homologous to their gnathostome counterparts, while lacking a horizontal semicircular canal system proper^{23,24}. Significantly, it has been recently reported that lamprey also expresses locomotor EC-evoked eye movements²⁵. Like *Xenopus* larvae, head fixed semi-intact lamprey preparations produce conjugate eye movements in phase opposition with undulatory tail oscillations in the horizontal plane. Moreover, these oculomotor responses persist after bilateral labyrinth removal and optic nerve

section, confirming the intrinsic spinal origin of the motor command. This locomotor EC control of eye movements discovered in lamprey might therefore represent an ancestral condition in early jawless chordates (see below).

The presence of spino-ocular motor coupling in the tetrapod adult frog^{18,19} also suggested that CPG EC signaling is not only relevant to the left-right tail/body motion of undulatory swimming animals like lamprey and larval amphibians, but might also contribute to stabilizing gaze in other limbed vertebrates during bi- or quadrupedal locomotion. Indeed, locomotor EC-driven ocular behavior has been recently established in mice²⁶. First, the Abducens motor nerve exhibited rhythmic burst discharge that was phase-coupled with fictive locomotion in ex-vivo brainstem spinal cord preparations of neonatal animals, comparable to the coupling found in *Xenopus*. This locomotion-related Abducens activity was elicited by an EC signal emanating from the forelimb CPG in the cervical cord region. Second, rabies virus injections into the LR muscle of juvenile mice revealed monosynaptic connectivity between bilateral cervical neurons and LR motoneurons, suggesting that the EC signals are conveyed directly by an ascending spinal cord-brainstem pathway, again similar to adult *Xenopus*¹⁸. Third, sustained treadmill-elicited locomotion in decerebrated adult animals evoked horizontal conjugate eye movements in phase with the forelimb gait pattern, despite the absence of vestibular and visual sensory inputs. Such intrinsically driven ocular behavior therefore complied with the spino-ocular motor coupling observed in ex-vivo neonatal preparations, indicating the mechanism's persistence throughout ontogeny.

There is also strong behavioral evidence for motor ECs contributing to gaze stabilization in other mammalian species. In walking cats, gaze shifts are tightly coordinated with the stride cycle, even in the dark, and coupling is stronger at faster speeds when rhythmic movements are more stereotyped²⁷. Since this eye movement-stride coordination during walking was not determined by vision, it was proposed that intrinsic ECs from the spinal CPG mechanism are mainly responsible for driving the gaze-stabilizing behavior. Similarly, spinal ECs related to active limb movement may reinforce gaze compensating horizontal eye movements in monkeys running in darkness²⁸. Also, in guinea pigs, compensatory eye movements during self-head motion were found to be anticipatory, occurred independently of vestibular sensing and were therefore also likely produced by an internal feedforward command that was predictive of the active movement²⁹.

A role for locomotor EC signaling in gaze control has also been reported in humans, in both normal and pathological contexts. For example, when performing a gaze fixation task during treadmill walking, subjects with a bilateral vestibular deficiency displayed reduced velocities of ocular compensation in response to head movement, but the phase of movement remained little different

from normal³⁰. This finding suggested that, whereas the gain component of compensatory eye movement is dependent on fully functional vestibular sensing, the timing parameter is probably governed by locomotor EC signals. Furthermore, in normal human subjects, angular head and compensatory eye movements in the vertical plane become increasingly phase-coupled as treadmill locomotor speed transitions from walking to running^{31,32}, again implicating spinal EC influences that become stronger and more effective during faster, more stereotyped running.

Altogether these findings strongly support the conclusion that spinal CPG-ocular motor coupling via ECs, initially found in *Xenopus* larvae and its tetrapod adult, is a conserved mechanism for stabilizing eye movements during locomotion, from an extant agnathan and anuran amphibians to mice and other mammals, including humans (Figure 2).

Interactions between locomotor EC signaling and vestibular sensory processing

Since spinal ECs provide a reliable estimate, both in timing and amplitude, of the sensory consequences of rhythmic propulsive movements³³, the question arises as to how these converging signals from different sources interact in producing ocular compensation. In the *Xenopus* tadpole, where tail-based swimming is mainly in the horizontal plane, the matching spatio-temporal and target specificities of spinal ECs and horizontal aVORs, and their use of common vestibulo-ocular circuitry, could reasonably predict that the two signals summate to ensure conjugate eye movement (Figure 3a). Such an interaction was originally tested in *Xenopus* tadpoles by monitoring locomotor and ocular motor activity without, or with, concurrent imposed head rotations using semi-intact *in vitro* preparations with intact vestibular endorgans¹⁰. During high frequency fictive swimming in conjunction with head rotations in the horizontal plane, oculomotor activity remained exclusively coordinated to, and modulated by, axial CPG activity. This therefore indicated that rather than being additive, horizontal aVORs are in fact suppressed during locomotion, presumably by the EC signals themselves^{10,34}. On the other hand, other VORs, such as roll-activated reflexes, remained unaffected. This selective horizontal aVOR cancellation is likely due to a temporal mismatch in feedforward/feedback signaling: the sensory-motor transformations underlying the VOR would otherwise produce cyclic ocular motor output that is too slow for preemptive EC-driven output, especially during rapid swimming, which attains cycle frequencies of >10 Hz²⁰.

However, when larval tail undulations occur at slower cycle frequencies, ocular motor output was recently found to arise from both spinal EC and horizontal semi-circular canal influences now acting in combination, with a clear contribution of the latter to compensatory eye movements³⁴. This summing interaction therefore indicated that during less intense or slower swimming, the spinal

ECs were no longer capable of gating out horizontal angular vestibular inputs. Furthermore, as swimming frequency decreases through larval life as the animal grows³⁵ and propulsive movement becomes less stereotyped¹¹, the horizontal VOR's contribution to gaze stability during self-motion becomes increasingly predominant³⁴. Significantly, this transition proceeds concomitantly with a developmental increase in sensing capability of the horizontal semi-circular canals themselves⁹.

Indirect evidence for an attenuating interaction between spinal EC and vestibular sensory signaling has also been reported in mammals under pathological conditions. In canine³⁶ and human^{36,37} subjects with unilateral vestibular sensing deficits, resultant postural instabilities were found to be significantly diminished during running than during standing or walking. In both cases, this increased stability was proposed to arise from rhythmic spinal CPG output that reduces destabilizing vestibular influences during faster, more stereotyped rhythmic self-motion. Similarly, in normal human subjects, perturbing vestibular sensing by galvanic labyrinth stimulation causes smaller trajectory deviations during running than walking³⁸, again compatible with activity-dependent EC gating of vestibular sensory cues. Such EC signaling has also been proposed to attenuate the effects of fixation nystagmus intensity in vestibulo-cerebellar impaired patients during fast walking by selectively suppressing ocular motor control disturbances that otherwise occur during standing³⁹.

A selective negative interaction between VORs and locomotor ECs has also been reported in normal human subjects. During treadmill locomotion, gaze stabilization in the horizontal plane is predominantly horizontal aVOR-driven, independently of locomotor speed or gait^{31,32}. During faster walking or running, where head motion predictability increases and the head and eyes now move in phase, the vertical aVOR that drives compensatory eye movement during slower walking is suppressed and gaze stabilization now switches to an automated feedforward mode that again is ascribed to the involvement of spinal ECs^{31,32,40}.

The neural site(s) at which spinal ECs exert suppressive or permissive actions on vestibular sensory-motor processing during locomotion remain largely unknown. In *Xenopus* larvae, at least one gating process has been found to occur peripherally, at the level of the mechanosensory hair cells in the peripheral vestibular endorgans themselves⁴¹. In addition to accessing the ocular motor nuclei, locomotor EC signals are also conveyed by vestibular efferent pathways to the inner ear where, during swimming activity, they cause an overall reduction of mechanosensory transmission from hair cells onto first-order vestibular afferent neurons. However, it was recently reported that vestibular afferent encoding of head motion is unaltered when rhesus monkeys perform voluntary locomotion compared to passive head movements, thus indicating an absence of a spinal EC influence at the primate's vestibular sensory periphery⁴².

Despite this context-independent sensing, the central vestibular neurons targeted by the monkey's endorgan afferents respond differentially during passive versus active head turning^{43–45}. The responsiveness of neurons implicated in vestibulo-spinal postural reflexes and the vestibulo-thalamic pathway for self-motion perception are strongly attenuated by cancellation signals originating from neck muscle premotor/motor centers during voluntary head rotations^{46,47}. Experimental evidence (reviewed in ⁶) further suggested that by comparing internal predictions and actual proprioceptive inputs, cerebellar circuits can rapidly adapt and modulate vestibular inputs to disentangle self-generated from externally-imposed head motion⁴⁹. In contrast, vestibulo-ocular neurons encode similarly for stabilizing gaze in the two behavioral situations, but do not respond during voluntary gaze redirection^{44,50,51}. In light of this context-dependent, vestibular circuit-specific integration of ECs, experiments are now required to test whether a spinal CPG influence emerges in eye-controlling vestibular neurons when monitored with and without the rhythmic stereotyped expression of locomotor behavior.

Further evolutionary considerations

Early in vertebrate evolution, spinal CPG-ocular motor coupling could have been the initial strategy for retinal image stabilization during the tail-based locomotion of aquatic ancestral chordates, as occurs in present-day protochordates²⁵, before vestibular system endorgans had appeared^{24,52,53}. This notion is particularly relevant to the horizontal semi-circular canals, whose relatively late arrival in jaw-bearing vertebrates occurred long after undulatory swimming had emerged²³. In early tadpole-like chordate ancestors, therefore, a spino-ocular command that conveyed a predictive correlate of horizontal angular head motion in the absence of motion-encoding semicircular canals could likely have been the sole mechanism available for stabilizing gaze during locomotion. This mechanism's important role can be seen in present day lamprey that lack a lateral horizontal semicircular canal, and in early developing *Xenopus* tadpoles with their rudimentary vestibular endorgans and highly stereotyped swimming behavior (Figure 3a). Interestingly, although not possessing a horizontal semi-circular canal proper, present-day lamprey has a unique horizontal duct structure⁵⁴ that provides head movement sensing in the horizontal plane²⁵, which seems to have evolved in parallel with the gnathostome horizontal canal^{24,54}.

Presumably, the subsequent evolution of more complex body morphologies and locomotor strategies, particularly with the transition to appendicular locomotion and terrestrial life (Figure 2), required an accompanying elaboration of the semi-circular canal system to encode the increasing spectra of head motion. Moreover, the appearance of a flexible neck resulted in head movements that were much more independent of body motion during locomotion, with a gravity sensory

perception different from aquatic life (Figure 3b). These morphological and locomotor innovations would have in turn required the vestibular sensory system to encode more efficiently the larger modalities of head motion in aerial vertebrates like mammals and birds⁵⁵. Coupled with this increased complexity was the evolutionary innovation of other supraspinal structures in spino-vestibulo-ocular processing, notably the cerebellum (for recent review, see ⁵⁶), which interestingly, is absent in lamprey and is still non-functional in early larval *Xenopus*⁵⁷. Consistent with this scheme, for example, in head-fixed mice during treadmill running, locomotor EC-driven ocular movements are only apparent after decerebration²⁶, indicating that major cortical and subcortical circuit influences normally mask spinal EC signals in the intact animal.

Such considerations thus point to a changing contribution of locomotor ECs to gaze control during vertebrate evolution. In parallel with the evolutionary increase in locomotor repertoires, the spino-ocular control mechanism appears globally to have transitioned from a dominant role in aquatic undulatory animals (Figure 3a), to a more restricted role in limbed vertebrates (Figure 3b) with their necessarily more elaborate sensing and central processing of 3D head motion.

Conclusions and perspectives

Considerable evidence has now accumulated for an important role played by CPG EC signaling in maintaining visual acuity during locomotion throughout vertebrate lineages, providing a feedforward command to the ocular motor system that supplants or supplements reactive control by sensory feedback-mediated VORs. The contribution of locomotor ECs to eye/visual stability appears to have changed during the course of evolution, adapting from a dominant (Figure 3a) to a subordinate role (Figure 3b) in accordance with the evolutionary increase in propulsive and biomechanical complexity, and the associated functional impact of vestibular sensory signaling.

The central neural sites at which locomotor ECs and VOR-related signals interact, and the underlying cellular/synaptic mechanisms, remain to be established. The future use of cellular level patch clamp recordings, molecular genetic approaches and refined in vivo recording techniques during actual locomotor behavior will enable such issues to be addressed. Neuronal population calcium imaging will also aid in locating relevant populations and determining their real-time activity profiles with cellular resolution. Moreover, comparative studies with other tractable animal models should further advance our understanding of contribution that locomotor EC signaling make to gaze control during vertebrate evolution.

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** outstanding interest

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**This article reports on the role of optokinetic and VORs in stabilizing gaze in lamprey, the oldest extant vertebrate. Using an eye-brain-labyrinth preparation, they found that both reflex types are present and that visuo and vestibular inputs are integrated at several sub-cortical levels. Importantly also, this study demonstrates for the first time that after removal of visuo-vestibular inputs, a spinal CPG-extraocular motor coupling leading to gaze-stabilizing eye movements appears during fictive swimming. This therefore constitutes a further example of locomotor ECs contributing to gaze stabilization, and indicates the mechanism's presence at the dawn of vertebrate evolution.

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**This article demonstrates for the first time in a mammal (mouse) the existence of a spino-extraocular motor coupling via direct ascending neural pathways. In both isolated brainstem-spinal cord preparations during fictive locomotion and decerebrated adult mice during treadmill walking, locomotor feedforward signals were found to evoke rhythmic ocular motor bursting coordinated with cervical CPG activity or actual eye movements synchronized to rhythmic forelimb movements,

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**This study took advantage of the cat model where the mechanics of the body and limbs, head-neck and oculomotor systems are known. The cat's gaze behavior was investigated in relation with limb stride when walking on different surfaces and with changes in the visual environment. Importantly, it was found that gaze behavior remained coordinated with stride, even in complete darkness, demonstrating that the phase-coupling between limb and gaze-compensating oculomotor movements are independent of vision. The authors propose that while locomotor ECs are likely to contribute to the cat's gaze behavior during walking, particularly at faster speeds, their influence on the oculomotor system is not as strong as, for example, in *Xenopus*.

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*This study reports that spino-ocular coupling and EC signaling is likely to contribute to gaze stabilization during human locomotion. Eye and head movements were recorded during changing locomotor speeds in light and darkness to obtain the gain and phase of horizontal and vertical angular VORs. Whereas horizontal aVORs persisted with increasing speed, vertical eye-head coordination switches from a VOR-driven compensatory to a synergistic mode where the head and eye move in phase. This was taken to indicate that during faster and more stereotyped locomotion, as the predictability of resultant head movement increases, spinal EC commands suppress the vertical aVOR and assume the task of stabilizing gaze in the vertical plane.

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Figure legends

Figure 1. Spino-ocular motor coupling and efference copy-mediated gaze stabilization during undulatory swimming in *Xenopus* larvae **(a)** and during appendicular swimming in juvenile adults **(b)**. In the tadpole **(a, left panel)**, the axial CPG that drives alternating left/right ventral root (Vr) bursting underlying tail oscillations, also sends an ascending efference copy (EC) to produce bursting in the lateral (LR) and medial rectus (MR) motor nerves, which in turn drive conjugate eye movements that counteract resulting left/right head movement. The EC signals are conveyed by a crossed spinal-abducens nucleus (N) pathway, then are transmitted to the opposite oculomotor nucleus via circuitry employed by the horizontal VOR **(a, right panel)**. In the froglet, the lumbar CPG responsible for bilaterally-synchronous hindlimb kicking, sends an ascending EC signal that now generates convergent eye movements to compensate for head forward acceleration during each hindlimb extension **(b, left panel)**. This spino-ocular coupling is mediated by uncrossed pathways to the ipsilateral extraocular motor nuclei **(b, right panel)**. *Figure adapted from*^{10,18}.

Figure 2. Locomotor EC-induced ocular behavior exists in species representing key evolutionary steps in vertebrate lineages. This ranges from ancestral agnathans to latest emerged primates, despite changes in locomotor mode, increased biomechanical complexity and the accompanying increased impact of vestibular sensory signaling. Evidence for EC-mediated gaze control has so far been found in the primitive lamprey²⁵, anuran amphibians^{5,8} and mammals, including mice²⁶, guinea pigs²⁹, cats²⁷, monkeys²⁸ and humans^{31,32,39}. By compressing into several weeks events that spanned several hundred million years in evolution as vertebrates transitioned from undulatory to appendicular locomotion, metamorphosing *Xenopus* offers insights into the changing contributions of feedforward ECs and visuo-vestibular feedback to gaze stabilization through vertebrate evolution. *Figure adapted from*⁵.

Figure 3. Contrasting functional relationships between locomotor EC- and VOR-based signaling in larval *Xenopus* **(a)** and mouse **(b)**. The aquatic tadpole **(a)**, due to its compact body structure and stereotyped undulatory mode of locomotion in the horizontal plane, produces passive head motion almost identical to the tail kinematic profile. Also, with relatively weak VORs, this enables predictive CPG EC signals to be the dominant mechanism for generating compensatory eye movements during swimming. Conversely, in the mouse **(b)**, the presence of a flexible neck and complex patterns of terrestrial locomotor movement results in head motion that is highly different from the limb kinematic profiles. Consequently, gaze stabilization during locomotion can no longer be preferentially supported by CPG EC signals, but relies mainly on head motion sensing and now highly evolved VORs. *Figure adapted from*^{10,26}.

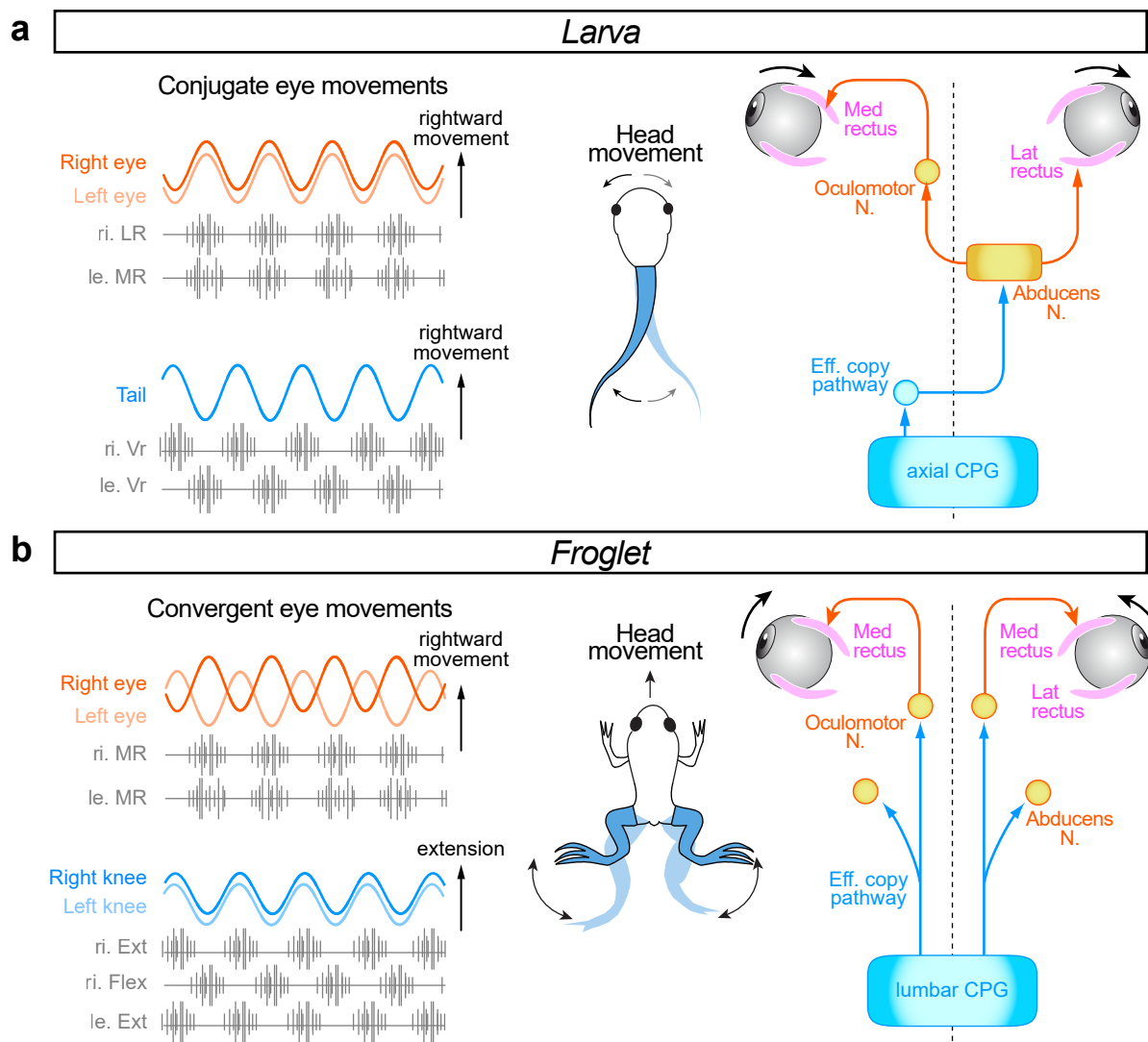


Figure 1

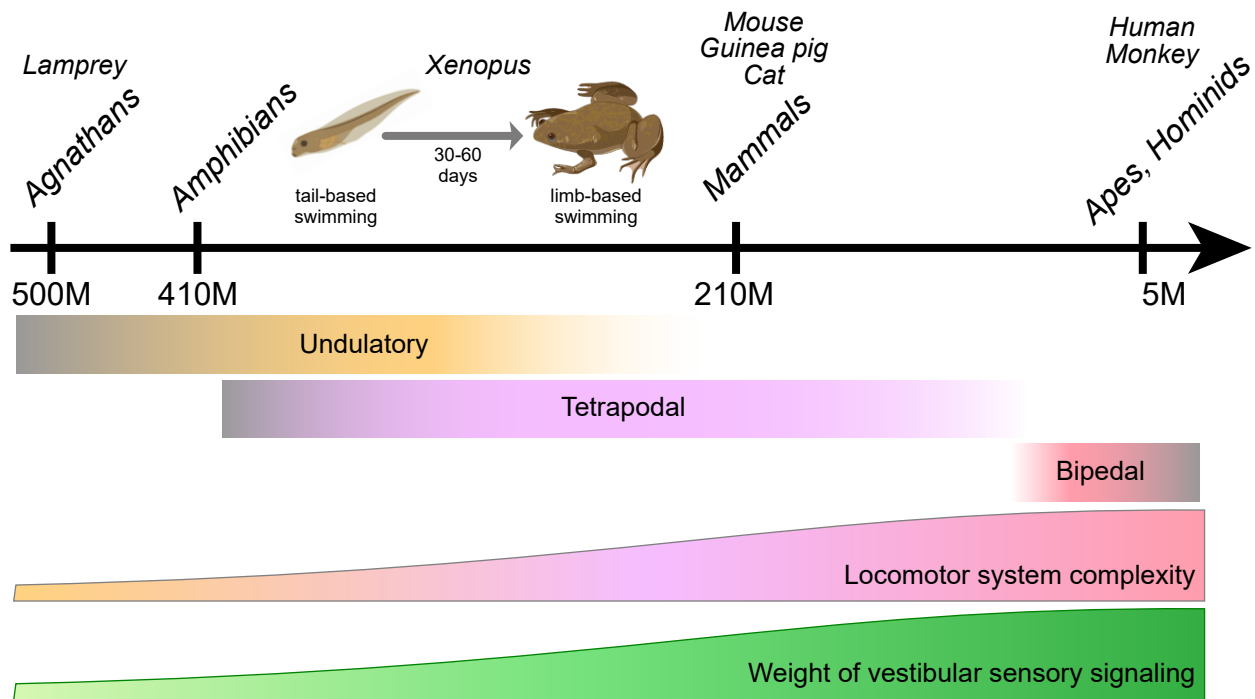


Figure 2

Figure 3

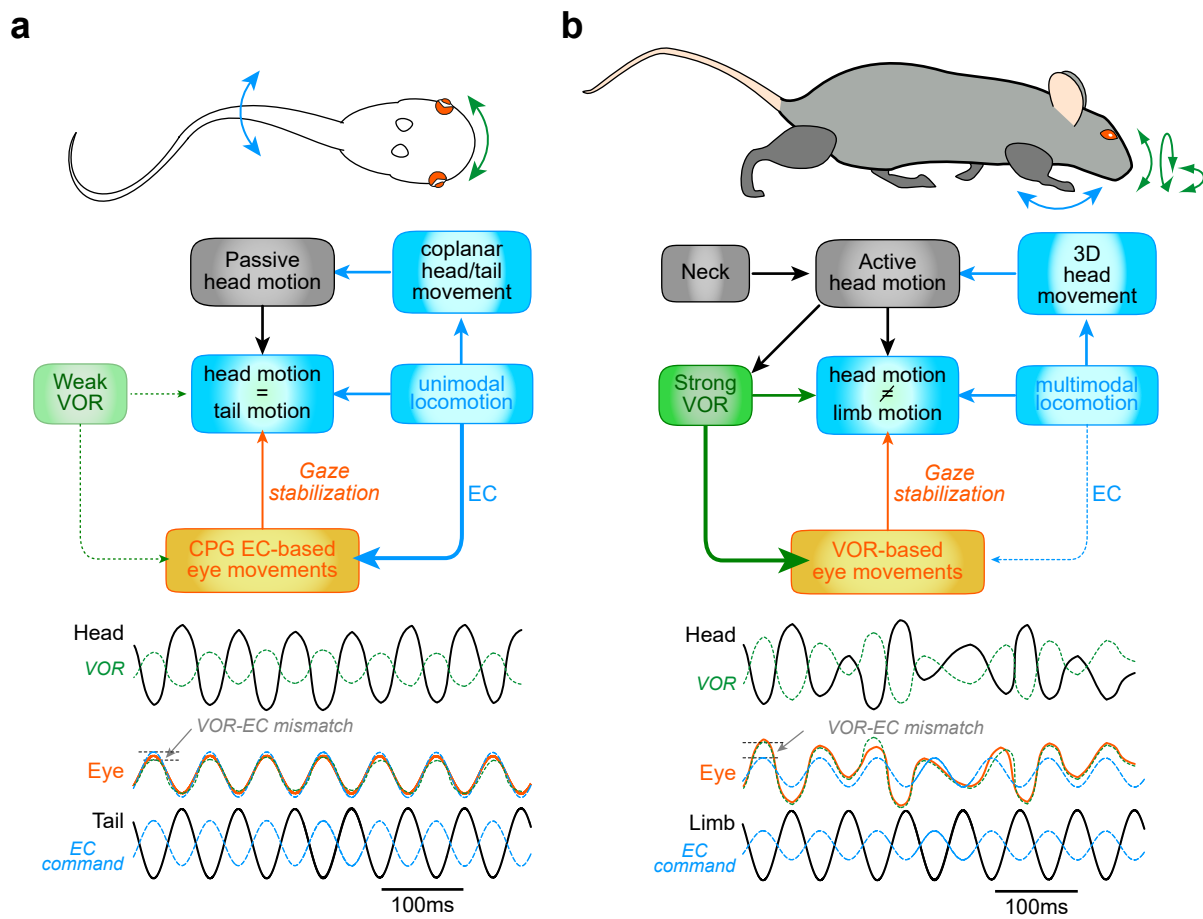


Figure 3