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A Biologically Based Neural System Coordinates the Joints and Legs of a Tetrapod

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Abstract. A biologically inspired neural control system has been developed that coordinates a tetrapod trotting gait in the sagittal plane. The developed neuromechanical system is used to explore properties of connections in inter-leg and intra-leg coordination. The neural controller is built with biologically based neurons and synapses, and connections are based on data from literature where available. It is applied to a planar biomechanical model of a rat with 14 joints, each actuated by a pair of antagonistic Hill muscle models. The controller generates tension in the muscles through activation of simulated motoneurons. The hind leg and inter-leg control networks are based on pathways discovered in cat research tuned to the kinematic motions of a rat. The foreleg network was developed by extrapolating analogous pathways from the hind legs. The formulated intra-leg and inter-leg networks properly coordinate the joints and produce motions similar to those of a walking rat. Changing the strength of a single inter-leg connection is sufficient to account for differences in phase timing in different trotting rats.

Keywords: Neural Controller, Rat, Simulation, Central Pattern Generator, Inter-leg Coordination

1 Introduction

Animals dynamically adapt to varying terrain by changing gaits, adjusting footfall positions, and responding to perturbations. The neural circuits which accomplish these tasks reside in complex hierarchies, oscillating and interacting with each other at different time scales based on feedback from sensory information, making them difficult to understand. The prevailing theory in neurobiology is that hierarchies in the central nervous system sub-divide complex tasks into sub-tasks [1]. With the brain at the top and motoneurons at the bottom, each level relies on level-appropriate sensory information to predict outcomes at different time-scales and act on the levels below [2]. Neurological experimentation suggests that a large set of neurons involved in steady state walking are located in the thoracic ganglia for arthropods [3], and the spinal cord for vertebrates [4].

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Many of these neurological measurements suggest hypothetical circuits, and modeling is a useful tool to test that controllers based on these circuits are sufficient to produce the desired behavior [5]. Modeling typically starts at lower, basic levels and builds on findings to develop more complex models. Insect modeling has progressed significantly in this way. Early hypotheses concerning insect walking began with a set of leg coordination rules based on years of observing stick insects [6]. Implementation of these rules on a robot helped to test how they could produce reliable, coordinated walking over even ground [7]. Further models of insect walking have focused on coordination methods in individual legs, beginning with finite state rules [8], and how descending commands can be used to affect leg states and transitions in behavior [9, 10]. More detailed modeling of these systems has examined specific sensory pathways and how central pattern generators (CPGs) can be coordinated to produce different stepping behaviors with more detailed biological neuron models [11, 12].

Vertebrate systems are also being modeled. Knowledge of stance to swing transitions in stepping cats [13] has resulted in a model that demonstrates the necessity of both joint position feedback and limb loading feedback on replicating the behavior [14]. Further research on this model has shown that the replicated stepping patterns are inherently stable [15]. Research on salamanders has demonstrated how CPGs can be organized for the generation of different gaits and allow smooth transitions between swimming and walking [1].

Non-detailed evidence of CPGs in mammalian systems has resulted in many modeling theories. Early theories hypothesized the existence of a single CPG per leg, driving transitions between stance and swing. However, more recent models utilize multiple oscillating circuits at multiple hierarchical levels [16] which is supported by recent neurological data [17], indicating that mammalian CPG systems may look more similar to those in insects than previously hypothesized [18]. A model of a CPG coordinated through sensory feedback pathways has been shown to successfully replicate many behaviors seen in CPG coordination [19].

Our recent model of a rat walking in the sagittal plane used these CPG systems and coordination rules. We conducted deletion experiments on this model of proposed mammalian inter-leg pathways and developed hypotheses for the roles each performs in maintaining coordination between fore and hind legs [20]. We found that a mechanism in which elbow flexion encourages hind leg stance is sufficient and necessary to produce coordination in the proposed scheme. The mechanism in which the end of hip flexion initiates swing of the front limb is not sufficient for producing coordination because the hind legs are not coupled contraterally. However, its removal from the network reduces the likelihood of walking coordination. Having hip and knee flexion encouraged by elbow extension is also not capable of producing symmetrical coordination by itself. However, this work determined that this connection appears to be important in reducing deviations in walking coordination.

The work presented in this paper expands upon our previous inter-leg model of tetrapod coordination by simplifying the structure in the hind leg intra-leg
controller and refining the foreleg controller to more closely mimic the pathways of the hind leg. The neural system controls a dynamic model of a rat so that its joint movements closely match kinematic data. This model is then used to test the hypothesis that changes in strengths of connections may be sufficient to account for the difference in phase timing in different trotting rats. Changes of synapse strength in the connection between foreleg elbow extension and hind leg flexion produce changes in phase timing by up to 17%.

2 Biomechanical Model

The simulation is conducted in the Vortex physics engine (CM Labs, Montreal, Quebec) implemented by Animatlab [21]. Rigid body dynamics, ground contact, body collisions, and friction are all simulated. Both the physics and neural system are simulated every 0.1 ms using single step forward Euler integration. The inertia of a solid body is determined by the shape of a triangulated mesh and a given density. The biomechanical model was constructed with scans of rat bones which were digitally matched to a rat walking up a rope using 3D x-ray high speed video. The model reconstruction was limited to motion in the sagittal plane, resulting in a total of 14 degrees of freedom, four for each front leg (scapula, shoulder, elbow, and wrist) and three for each hind leg (hip, knee, and ankle). To reduce the control problem to systems which coordinate the joints and match the animal experimental setup in which the ipsilateral coordination rules were developed [22], body weight is supported at the hip, which is fixed 6.5 cm above the ground. Linear Hill muscle models generate forces about the joints within the rat model [21] and is illustrated in Figure 1.

\[
\frac{dT}{dt} = \frac{k_{sc}}{b} \left( k_{pe} x + b \dot{x} - \left( 1 + \frac{k_{pe}}{k_{sc}} \right) \cdot T + A \right)
\]  

Fig. 1. Hill Muscle Model

Tension, \( T \), is developed in the muscle according to:
where $x$ is the muscle length, $k_{se}$ and $k_{pe}$ are the series and parallel stiffness, $b$ is the damping element, and $A$ is the activation level of the muscle, which has a length-tension component. Muscles are chosen as the activation method for two main reasons. First, it is likely that some aspects of the control system are tuned directly to the mechanical properties inherent in muscles. For example, the length-tension relationship promotes stability of positive force feedback pathways [23]. Other properties which could prove important are self-limiting velocity-tension, and physiological delays in tension development. Second, actuator compliance is an important part of achieving robust motions that can reject perturbations passively. To reduce the control problem, each joint is actuated by two antagonistic muscles and biarticular muscles are omitted. Biarticular muscles act mainly to counterbalance gravity [24, 25] and serve to reduce energy consumption [26]. Their addition to the model would add complexity with little benefit to studying pathways that affect intra- and inter-limb timing but we may include them in future models. Muscle properties were set to produce the desired torques calculated by Witte et al. while maintaining stability of motion [27].

Sensory feedback in the model is similar to types Ia, Ib, and II, and although simplified representations of nature, they contain important elements.

\[ \text{Ia} = k_a x_{\text{series}} \quad \text{Ib} = k_b T \quad \text{II} = k_c x_{\text{parallel}} \]  

(2)

where $k_a$, $k_b$, and $k_c$ are gain parameters set by the user to act as injected current into a neuron. The series elastic spring, $x_2$, is sensitive to muscle velocity as well as muscle length because the dashpot in the parallel element causes the parallel element to change lengths at a slower rate than the series spring. The length of the series spring is used to stimulate Ia feedback, known to be dependent on both length and velocity in animals. Ib feedback is dependent on the tension developed in the muscle, and type II feedback is dependent on length through measurement of the parallel elastic element $x_1$. The gains were experimentally adjusted to produce an injected current which gives usable information through a sensory neuron across the full range of muscle lengths and contractions.

2.1 Neural Model

This work is built on the dynamics of a leaky integrator, and is capable of modeling individual non-spiking interneurons, the firing rate of a population of neurons, or a single spiking neuron after the addition of a spiking threshold. We are currently not concerned with the specifics on how action potentials are generated and have left out Hodgkin-Huxley sodium and potassium currents. Instead, we are concerned with how signals propagate through the network, and how individual neurons and populations of neurons activate, deactivate, and contribute to network behavior. Each neuron is governed by the equation:

\[ C_m \frac{dV}{dt} = g_L \cdot (E_L - V) + I_{\text{ext}} + I_{\text{ions}} \]  

(3)

where $V$ is the membrane potential, $C_m$ is the membrane capacitance, $E_L$ is the leak potential, $g_L$ is the leak conductance, $I_{\text{ext}}$ are the external synaptic and
injected current inputs and \(I_{\text{ions}}\) are additional ionic currents that can be added to increase the dynamics of particular neurons (e.g. the calcium current in the rhythm generating neurons in equation 6). Constants such as \(C_m\), \(g_L\), and \(E_L\) are based on typical spinal cord interneuron values, but may be more directly implemented with known values when knowledge of them is made available. This limits the number of free parameters in the network and allows us to focus on the strength and types of connections between neurons. The synapse model is:

\[
I_{\text{syn}} = g_{\text{syn}} \cdot (E_{\text{syn}} - V_{\text{post}})
\]

where \(E_{\text{syn}}\) is the potential of the synapse, \(V_{\text{post}}\) is the postsynaptic membrane potential, and \(g_{\text{syn}}\) is piecewise linear activation function defined as

\[
g_{\text{syn}} = g_{\text{max}} \cdot \min \left( \max \left( \frac{V_{\text{pre}} - E_{\text{lo}}}{E_{\text{hi}} - E_{\text{lo}}}, 0 \right), 1 \right)
\]

where \(g_{\text{max}}\) is a user defined maximum conductance, \(V_{\text{pre}}\) is the presynaptic membrane potential, and \(E_{\text{lo}}\) and \(E_{\text{hi}}\) are the two user-defined voltage threshold values that define the piecewise-linear function where \(E_{\text{lo}} < E_{\text{hi}}\). The above model is also capable of representing the activation of a population of neurons with the same type of synapse where the population activity varies between 0 when \(V_{\text{pre}} < E_{\text{lo}}\) and \(g_{\text{max}}\) when \(V_{\text{pre}} = E_{\text{hi}}\). Most neurons used in this model are used in this way and represent average voltage values of a population, similar to those of other networks that model mammalian walking behaviors [19,11].

This neural model offers many advantages. Because the model is biologically based, it is expandable and not limited to the currently modeled system. Known neural pathways, ionic currents, and synapse plasticity which influence locomotor behavior can be implemented directly into the model, several of which are described in 4.1 and 4.2. These additional currents and properties that present a significant impact on the behavior of a particular area of the neural system may be added to individual neurons appropriately without further increasing the complexity of the rest of the system. One particular area of relevance to our study is how a set of neurons can produce a pattern generating behavior. The ion channels we have included in the rhythm generating (RG) neurons that contribute to active bursting phases are calcium channels, where the calcium current, \(I_{\text{Ca}}\), is defined as:

\[
I_{\text{Ca}} = g_{\text{Ca}} \cdot m(V) \cdot h(V) \cdot (E_{\text{Ca}} - V)
\]

\[
\frac{dm}{dt} = \frac{m_{\infty}(V) - m}{\tau_m(V)} \quad \frac{dh}{dt} = \frac{h_{\infty}(V) - h}{\tau_h(V)}
\]

where \(E_{\text{Ca}}\) is the equilibrium calcium channel voltage, \(g_{\text{Ca}}\) is the calcium conductance and \(\tau_m(V) = \phi_m \sqrt{\epsilon_m(V)}\) and \(\tau_h(V) = \phi_h \sqrt{\epsilon_h(V)}\). The functions \(m_{\infty}(V) = \frac{1}{1 + \epsilon_m(V)}\) and \(h_{\infty}(V) = \frac{1}{1 + \epsilon_h(V)}\) are sigmoids, where \(\epsilon_m(V) = \exp(-S_m \cdot (V_m - V))\) and \(\epsilon_h(V) = \exp(-S_h \cdot (V_h - V))\). \(\phi_m, \phi_h, S_m, S_h, V_m\) and \(V_h\) are constants. By starting with a relatively simple model, we are able to lay out the basic
structure of the network in a tractable manner and through the use of biologically based neurons we are able to increase complexity where necessary. Higher levels of complexity can lead to the emergence of more capable and dynamic behaviors [28].

Noise was added to the neuron models to encourage variation and perturb the simulation to loosely approximate noise in the animal’s nervous system. All neurons had a minimum of random, uniformly distributed noise of $\pm 0.1$ mV. CPG half centers and motor neurons have a maximum of $\pm 0.5$ mV, and motor neuron inputs have a maximum of $\pm 0.2$ mV. This noise is the source of the variability seen in the simulation data presented below, and the removal of the noise eliminates the variability.

3 Analysis of biological data: leg kinematics during walking in rats

Forelimb and hindlimb kinematics are the visible and measurable output of the nervous system in biological systems and reflect the role of muscles as mobilizers or stabilizers at a certain instant of a stride cycle. In order to test the validity of our model we analyzed the movements of leg elements and joints by means of X-ray-based motion analysis on four adult rats. Animal care was in accordance with German animal welfare regulations, and experimental procedures were registered with the Thuringian Committee for Animal Research.

The animals were filmed while they walked along a horizontal track. High-resolution videoradiography was used to collect data in lateral projection. The X-ray system (Neurostar, Siemens AG, Erlangen, Germany) operates with high-speed cameras (SpeedCam Visario G2, Weinberger GmbH, Erlangen, Germany) with a maximum temporal resolution of 2000 Hz and a maximum spatial resolution of 1536 dpi x 1024 dpi. For our purposes a frame frequency of 500 Hz and the maximum spatial resolution were used. The movement of the leg elements was quantified by manually tracking previously defined landmarks using SimiMotion 3D (Simi Reality Motion Systems GmbH, Unterschleissheim). Scapula angle is defined relative to the horizontal plane, leg joint angles are measured on the flexion side of each joint. Temporal gait parameters and inter-leg coordination were determined with the aid of additional normal light cameras helping to distinguish between left and right legs. The normal light cameras recorded the locomotion synchronously to the x-ray system. Further details of the method are published elsewhere [29]. Data illustrated in 7 were obtained from six complete cycles. The cycles were adjusted to the same duration by means of linear interpolation before mean and standard deviation were computed.

4 Control Network

4.1 Intra-Leg Network

The exact nature of how the nervous system controls muscle activation is not known [30]. For simplicity, the muscle control system in our network is tuned for
position based control, and leaves out the effect of Renshaw cells, Ia feedback, and other known motoneuron influences. Tension is developed in the muscle based on type II afferent feedback (stretch receptors) modulated by a constant force inhibiting neuron and is illustrated in Fig. 2. As the muscle gets closer to its desired length, excitation of the motor neuron decreases. This acts similar to low gain position control. We use kinematic data [31] and recently collected unpublished data to determine these desired joint angles and muscle lengths and tuned the low level controllers to reach these angles.

![Diagram](image)

Fig. 2. Left: Single leg joint controller. Circular ends represent inhibitory synapses while triangular ends represent excitatory synapses. CPGs are composed of two rhythm generating (RG) neurons with calcium channel dynamics which mutually inhibit each other via less dynamic interneurons. Each motoneuron is excited by type II (red) feedback unless inhibited by a CPG half-center (blue). Each motoneuron is also inhibited through an adjustable tonic inhibition (green). Right: Animatlab model of rat. Model reconstruction was done using bone matching of high speed, 3D x-ray video.

Most intra-leg network pathways in our model are developed directly from proposed mechanisms in mammalian literature, which is focused almost exclusively on the hind leg. Stance-to-swing transition is the most studied phenomenon, and it occurs from both reduced firing in Ib Golgi tendon afferents, and increased firing from hip flexor stretching [13]. This integration of signals is shown in Fig. 3 and can be visually followed with the purple dashed synapses; the ‘Load Release’ neuron is inhibited by ankle extensor Ib feedback and excited by hip flexor Ia feedback via the ‘Hip Angle Back’ neuron. When the ‘Hip Angle Back’ and ‘Load Release’ neurons become sufficiently active, they inhibit hip, knee, and ankle extensor activations causing a switch from stance to swing. Positive force feedback [23] also plays a role in the network. This load feedback (blue pathway) can initiate stance if the leg is not already in this state, as well as increase muscle activation (not shown). Experiments have shown that stance is initiated by reduced firing of the hip flexor type II afferents [32, 33]. This indicates that the hip is forward and causes contraction of the hip and ankle extensors and can be followed by the brown pathways. Using only these theories has proven difficult in developing a walking model that is able to step stably, and one notably absent piece to the puzzle is the initiation of contraction in the knee.
extensor muscles. We hypothesize that knee extensor contraction is initiated by stretching of the hip extensor muscle during the swing phase (black lines in Fig. 3). This causes the knee extensor to start contracting part way through swing, as can be determined through analysis of the kinetic data [27].

![Diagram of intra-leg network for the rear rat leg model](image)

**Fig. 3.** Intra-leg network for the rear rat leg model. Three joints are controlled in this diagram: hip, knee, and ankle. All blocks are a single integrator neuron as described in Eq. 3. Sensory information is transduced to current and injected into the sensory neurons (top). Feedback is filtered by a layer of interneurons (middle) and is used to coordinate the CPGs (bottom). Coordinating pathways are inhibitory (circle end) or excitatory (triangle end). Pathways inspired through previous biological research can be followed as dashed purple [13], dot-dashed blue [23], and dotted brown [32, 33] lines. The pathways in black are hypothesized as a result of this work.

Though the foreleg has an extra joint, analysis of several of the joints indicates a close match of stance and swing movements of the hind-leg. Scapula movement is similar to that of the femur for stance and swing while shoulder and elbow movements match closely with the knee, and ankle, respectively. Therefore, the extra joint in the front leg which requires additional control is the wrist. The front leg network is built on the hypothesis that these joints are controlled with analog sensory pathways (Fig. 4). Elbow extensor Ib feedback is used in parallel with ankle extensor Ib feedback for load detection. Continued elbow extensor Ib feedback inhibits swing for the highest and lowest joints, and when load is detected, it ensures those joints are in stance. Scapula flexor Ia and II feedback are used to determine when the leg is at the end of stance, and causes the shoulder and elbow joints to enter flexion. Scapula extensor II excitatory feedback is used to indicate when the leg is in the middle of swing similar to hip extensor II excitatory feedback used for the hind leg. This feedback causes the elbow and shoulder to begin extending. Elbow flexor II feedback initiates stance of the scapula and wrist similar to how hip flexor II feedback is used to initiate stance in the hip and ankle.
4.2 Inter-Leg Network

Inter-leg connections are based on behavioral mammalian experiments. They are an order of magnitude weaker than intra-leg connections [6]. Akay et al. [22] postulate three main coordinating influences between fore and hind legs. Influence 1) Foreleg extension reduces onset of the hind limb hip flexor. Influence 2) The end of activation of the hind limb hip flexor advances activation of forelimb swing. Influence 3) Activation of the forelimb elbow flexors contributes to inhibiting hind limb hip flexors. These influences are based on behavioral experiments in cats and have been implemented as network connections in the following ways: C1) Activation of the elbow extensor motor neuron applies inhibition to the hip flexor motor neuron. C2) Reduced activation of the hip flexor motor neuron allows the ‘Side Interneuron’ to escape and excite the ‘Init Swing’ neuron of the front leg for a short period. C3) Activation of the elbow flexor motor neuron depolarizes the "Hip Angle Forward" neuron on the hind legs, helping to initiate stance. These can be followed with the green pathways and labels in Fig. 5.

Coordination between the front legs is tested in two ways. The first is based on observations of the animal, in which it is seen that rats primarily move in a symmetrical gait with the front legs 50% out of phase with each other. These connections can be seen in Fig. 5. If one leg is in swing, the contralateral leg is encouraged to either enter stance if it is in swing, or continue in stance. Additionally, we have implemented the connection that if a leg is in stance then the contralateral leg is encouraged to enter swing. Kinematic and coordination data are shown from these connections. In addition to behavioral based connections, we have run some preliminary experiments with direct connections using commissural interneurons as have been found in mice [34] and cats [35], and further described with neural modeling [36]. With these connections, because we do not have a single CPG for each leg, the scapula joint CPGs are connected with CINi and CINE interneurons, and the rest of the CPGs remain unconnected. These pathways are set such that the CINi pathways provide three times as much inhibition as the CINE provides excitation, similar to [36]. These connections can
be seen in Fig. 6. Parameters were tuned to encourage coordination within a few steps of model initiation, but not impose coordination immediately.

Fig. 5. Inter-leg behavioral network. These connections are weaker than connections coordinating intra-limb movement. Contralateral connections encourage only one front foot to be on the ground at a time and can be followed with the orange dotted lines. Ipsilateral legs are coordinated based on research performed on cats, can be followed with the green dashed lines, and are numbered according to the influences determined by Akay et al. [22].

Fig. 6. Front leg commissural neural network. Scapula flexor activation both inhibits opposite leg scapula flexion (CINi neurons) and excites opposite leg flexion (CINe neurons), but at one third the strength of inhibition.

5 Results

5.1 Kinematic Results

The network produces stepping motions for both front and hind legs. Most joint kinematics match well, and many trends which are observed in the animal data
Fig. 7. Comparison of averaged biological recordings with results of the simulation for a single stride over multiple trials. Mean data for each is shown with the dashed and solid lines and one standard deviation is shown shaded or hashed around it. Stride is broken into stance from 0%-50% and swing 50%-100%.
are also captured in the simulation. The mean and one standard deviation for animal and simulation data are plotted in Fig. 7. The hip joint of the model moves more slowly at the beginning of stance but catches up at the end of stance, while swing is well matched. The knee joint movement closely matches the animal for both stance and swing including the double extension and flexion phases visible in the animal. The ankle joint also matches the double extension and flexion phases of the animal, though flexion during stance is less pronounced in the simulation due to external support of the body. Ankle flexion and extension of the model during swing follows closely to that of the animal.

Using network connections derived from the hind limb, the front limb is capable of producing steps which also share many of the same characteristics with the animal. The scapula extends and flexes in very similar timing to that of the animal for stance and swing with only slight differences in speed during relative periods. The shoulder shows the double extension and flexion phases visible in the animal, though flexion during stance is more limited than in the animal. The elbow does not undergo flexion during stance, which is likely due to the support of the body, similar to its analogous joint, the hind leg ankle. Elbow swing in the model matches the animal data closely. The wrist does not achieve the same range of motion as the animal, though it does undergo extension and flexion in the same phases.

5.2 Inter-leg Coordination Results

![Footfall diagram of simulation with different strengths in C1 compared with animal data taken at FSU Jena. Solid bars indicate stance of a particular foot. A single step cycle is approximately 450ms long for both the animal and simulation. For the symmetric gait, front legs alternate stance and swing and are approximately 50% out of phase. A C1 strength of 1.5 uS closely matches the data from Jena, while a decrease in strength shifts the phase of the hind legs forward until it closely matches data taken by Görsk et al. [37] (not pictured).]
The timing of hind leg touchdown to foreleg liftoff and between the forelegs which develops from the implemented control network is very close to that of the animal. Comparison of biological data collected in the Fischer lab at FSU Jena, data collected by Górskas et al.\cite{37}, and the simulation experiments can be seen in Table 1 with a visual representation of select data in Fig. 8. In the table, phase timing of a limb touchdown is described as a fraction of the period of another limb. For example a value of .5 in the column LF→RF indicates that the Right Front limb touches down on average half way through a full stride period of the Left Front leg. Rats primarily locomote in a trot where homologous and homolateral limbs are approximately 50% out of phase with each other and diagonal limbs are approximately directly in phase with each other. For all animals and simulation, homologous limbs were 50% out of phase with each other. Discrepancies between diagonal and homolateral limbs were observed when comparing animal data between FSU Jena and Górskas et al. Data collected by Górskas indicates that hind limbs touch down before fore limbs lift off (0.40-0.41), while data collected by FSU Jena shows liftoff of the forelimbs occurs before hind limb touchdown (0.60-0.65).

Adjustments to the strength of one connection in the model (C1, Fig. 5), is able to effectively alter the timing between fore and hind limbs such that both sets of animal data can be matched. Higher connection strengths cause the phase timing of the hind leg touchdown compared to foreleg liftoff to shift backwards. A strength of 1.5 uS in C1 creates an average phase timing of 0.62 for hind limb touchdown when compared to forelimb touchdown, which is similar to the phase timing of the data collected at FSU Jena. When this connection is weakened to a value of 0.2 uS, the phase is reduced to an average of 0.475, with the hind foot touching down before forelimb liftoff, and matching the data collected by Górskas et al. more closely.

Using the proposed front leg inter-leg connectivity in which CPGs are directly coupled also produces coordination in the front legs. Using strengths with similar proportionality as that for wild type mice in Rybak et al.\cite{36}, we produced alternating stepping patterns which match that of the walking animal. When the strength of the inhibitory connection is adjusted to 10% or lower of the original strength (CINi to Inhibit Scapula Extensor, Fig. 6), the model begins bounding in a similar manner to Netrin-1 Knock Out (KO) mice\cite{36}.

6 Discussion and Conclusions

6.1 Kinematics

The neural system introduced in this paper is capable of producing forward walking motions of the rat simulation with several kinematic movements similar to that of the rat. Hind leg movements closely mimic those made by the animal, including matching flexion during the beginning of stance while the extensors are activated. A reliance on purely kinematic data would not capture the eccentric muscle contractions, but estimates of the joint torques and knowledge of the neural systems involved informed the formulation of a simplified neural controller
Table 1. Comparison of biological data collected in the Fischer lab at FSU Jena, data collected by Górska et al.\cite{37}, and the simulation experiments. Phase timing of limbs are correlated as a fraction of the period of another limb. Standard deviation for all data sets is between 4-8%. For all animals and simulation, homologous limbs were 50% ± 3% out of phase with each other. Discrepancies between diagonal and homolateral limbs of the different rat data were mostly accounted for by a modification of the strength of C1.

<table>
<thead>
<tr>
<th>Test</th>
<th>Diagonal</th>
<th>Homolateral</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RH→LF</td>
<td>LH→RF</td>
</tr>
<tr>
<td>Górska</td>
<td>0.11</td>
<td>0.08</td>
</tr>
<tr>
<td>Jena Data</td>
<td>-0.10</td>
<td>-0.13</td>
</tr>
<tr>
<td>C1 = 0.2 uS</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>C1 = 1.5 uS</td>
<td>-0.14</td>
<td>-0.10</td>
</tr>
</tbody>
</table>

Table 2. Coordination of forelimbs in the developed model using behavioral rules, connections based on neural studies of wild type animals, and connections based on neural studies of Netrin-1 Knock Out (KO) animals. Phase timing of limbs are correlated as a fraction of the period of another limb. Behavioral rules and wild type neural connections both produce symmetric walking with front legs, while Netrin-1 KO rules produce in phase bounding. In all scenarios, the ipsilateral coordination is minimally affected.

<table>
<thead>
<tr>
<th>Test</th>
<th>Diagonal</th>
<th>Homolateral</th>
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<tbody>
<tr>
<td></td>
<td>LF→RF</td>
<td>RF→LF</td>
</tr>
<tr>
<td>Behavioral</td>
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<td>0.49</td>
</tr>
<tr>
<td>Wild Type</td>
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<td>0.50</td>
</tr>
<tr>
<td>Netrin-1 KO</td>
<td>-0.01</td>
<td>0.01</td>
</tr>
</tbody>
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Inter-leg Controller for Tetrapod Coordination

capable of producing these results. The front leg also produces steps similar to the animal, despite being developed mainly from hind leg connectivity properties.

There is room for improvement, however, as there are still some issues in kinematic matching. First, limits in the joint range of motion cause poor matching in the wrist. Methods for better designing muscle attachments to achieve greater ranges of motion could improve this, however without simulation capability which can actively wrap muscles around joints and update force directions, it is unlikely the same range of motion can be achieved in simulation as is seen in the animal. Second, external support of the body reduces the joint torques, especially at the beginning of stance. Development of local motor control networks which can produce the proper torque profiles required for walking may be able to eliminate the need for this external support. It is unclear how a change in low level muscle control would alter the coordination network, however the necessary torque profiles are smooth, and preliminary tests on individual legs indicate only moderate changes in synapse strength and thresholds may be required to maintain coordination. This is left as future work.

Though more detailed network construction may lead to better fitting of the biological data, most of phase transition and joint amplitudes are mimicked by the model. These results support the validity of the developed neural network and model and serve as a useful tool for testing the effect of network connectivity through means such as pathway deletions and strength modulation.

6.2 Inter-leg Coordination

Phase timing of ipsilateral and diagonal legs in the range tested is shifted by up to 17% by adjusting the strength of C1. Increasing the strength of C1 slows down the swing of the hind leg more when the front leg is in stance, causing it to reach its forward position later. Coordination as a result of higher connection strengths in C1 match that of the Jena data while coordination results with lower connection strengths match the data collected by Górska et al. With the data collected by Górska et al., rat hind legs touched down before liftoff of the front legs. With the Jena data, the hind legs touched down after the front legs lifted off. Both these outcomes are achievable with the implemented network, and these varied outcomes are produced by adjusting the strength of a single connection. In this study, changes of synapse strength in a single connection generated a continuum of coordination gaits. Such a mechanism may account for behavioral differences in gaits between animals.

Though rats primarily locomote with a trot, many other animals use different gaits and phase timing in these different gaits often require only slight changes in the timing between fore and hind legs, such as the difference between a trot and a gallop. Though not explored in this particular study, these changes may also be achievable by modulation of synapse strengths within the network. Alterations in phase timing may also be continuous with changes of speed within the same gait. Continuous alterations in phase timing is achievable with the network described.

When the direct neural connections found in mice and cats [34, 36, 35] are implemented, an alternating stable gait emerges. This occurs even though only
the scapula CPGs were influencing each other, and no other joints were involved in the coordination between the front legs. Additionally, coordination is maintained even though the scapula joints were receiving coordinating influences from within the leg to keep the joints coordinated in a walking motion, as well as coordinating influence from the hind legs to maintain ipsilateral coordination.

Ipsilateral coordination is also maintained when changes in the front leg coordination occurs. When the inhibiting synapses are weakened similar to what occurs in Netrin-1 KO mice, the front legs move together in a bound. Because the hind legs receive all their coordination cues from the ipsilateral foreleg, their coordination with the front legs is maintained and the hind legs fall into phase with each other as well, producing a well coordinated bound. It would be interesting to see if animals which regularly switch between trotting and bounding are able to modulate this connection via descending commands or some other means.

The developed controller can also be useful for robotic applications. By better understanding how animals coordinate their joints and limbs, we may be able to build better robot controllers which will approach the adaptability and flexibility that animal systems exhibit over a variety of terrains. This controller is able to coordinate four limbs in a trotting gait effectively. This gait is also adjustable, with smooth, continuous changes which affect limb coordination while maintaining individual joint coordination within the legs. The gait is also more drastically modifiable through the modulation of a single synapse coordinating the front legs, producing two different stable systems. Implementing a controller with these properties on a robotic system may enable more flexible changes in speed or even more drastic gait changes while maintaining control over individual leg functions.

References


