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1 Introduction

As an altricial species, humans face an intriguing challenge. We are endowed at birth with overabundant cortical connections, some of which must be eliminated while others are selectively stabilized as a consequence of sensorimotor experience (Hadders-Algra, 2004). Sensorimotor behaviors that occur with no obvious extrinsic stimulation, and that are often clustered together in time, are called "spontaneous" or "intrinsic" (Goldfield & Wolff, 2004). Spontaneous behaviors include arm waving, kicking, and babble vocalizations. Infants may actively contribute to the selective remodeling of initial cortical connections by transforming these spontaneous behaviors into functional relationships between the body and environment (Einspieler & Prechtl, 2011; Goldfield & Wolff, 2004; Hadders-Algra, 2000; Lee, Bhat, Scholz, & Galloway, 2008). Consider supine kicking: infants begin exploring their environment with their feet even before they learn to use their hands (Galloway & Thelen, 2004). Joint angle rotations at the hip, knee, and ankle during spontaneous kicking are initially synchronous, but

gradually become decoupled (Thelen, 1989). Is spontaneous kicking the exploratory foundation upon which infants decouple the timing of joint rotations so they can learn to use the leg for the locomotor functions of support, steering, and propulsion?

At the initiation of spontaneous kicking, the timing of joint rotations is relatively unstructured. But by the time infants become independently mobile, joint angle rotations are well coordinated for locomotion (Angulo-Kinzler, 2001; Goldfield, 1995, 2000). With typical development, coordination of hip, knee, and ankle joint angles becomes progressively more variable, indexed by progressively larger phase differences or, relatedly, progressively weaker cross correlations of hip, knee, and ankle joint rotations (Piek, 2002; Thelen, 1985; Vaal, van Soest, Hopkins, Sie, & van der Knaap, 2000). More complex relationships between the joints may signify the opportunity to re-organize motor degrees of freedom for locomotion.

The role of the hip in locomotion is related to initiation of phase transitions during alternating stepping (e.g., while supported upright on a treadmill; Thelen & Ulrich, 1991). Thus, the dominance of the hip contribution to spontaneous kicking may be an indication of the establishment of a functional role for the hip during gait (Jensen et al., 1994; Jensen et al., 1995). However, the postural support role of the foot in stepping during gait invites the hypothesis that there should also be measurable effects of the distal extremities on the more proximal parts of the leg. In short, if spontaneous kicking establishes new functional relationships between different parts of the leg in preparation for gait, then we should expect to find both proximo-distal (hip to ankle) and distal-proximal (ankle to hip) kinematic relationships between hip-knee-ankle joint rotations. Studies of animal running (Daley et al., 2007) and human exoskeleton-assisted gait (Lewis & Ferris, 2011) support this view of a proximo-distal gradient that distinguishes neural control strategies at the more proximal hip joint from the more distal knee and ankle joints.

The complex timing of spontaneous movements, such as kicking, may indicate a rich substrate of chaotic processes. From the newborn period onward, the temporal distribution of clusters of spontaneous behaviors fluctuates dramatically. These fluctuations in infant spontaneous behaviors may exhibit what dynamicists call "intermittency": the tendency for fluctuations to vary in size, and to do so in a

deterministically chaotic way (Mandelbrot, 1997; Ihlen & Vereijken, 2010). Intermittency in infant behavior may be an indication of the continuous nonlinear (i.e., multiplicative) interplay of stochastic and deterministic components across very many scales in the information flow between body and brain. For example, functional MRI (fMRI) studies have identified brain networks, called resting state networks (Raichle & Snyder, 2007; Durstewitz & Deco, 2008; Deco & Corbetta, 2011), which are active during periods without obvious external stimulation. One hypothesis is that these networks are in a constant state of exploration in order to generate predictions about likely network configurations that will be appropriate for future encounters with novel contexts (Deco, Jirsa, McIntosh, 2011). Incomplete resting state networks are already apparent in newborns (Fransson et al., 2007), and an increasing number of networks constitute the resting state during the first year (Gao et al., 2009). As brain development progresses during childhood, local connectivity weakens, and connectivity between more distant networks increase (Power et al., 2010).

Probing intermittency in a spontaneous behavior such as kicking could be a first step towards demonstrating a relationship between the internal exploratory activity of resting state networks and the process by which infants explore their own behavior to map body states onto brain network activity. However, there is little direct evidence for intermittency in infant kicking behavior. What properties of the leg might be a basis for information exchange between body systems and brain networks? The body consists of multifunctional mechanical components—bones, muscles, tendons, and other non-muscular tissue with elastic properties. These components may be used in different ways to perform many kinds of work: the leg may become a rigid strut for postural support, or a spring system for gait. The multifunctional leg has been modeled in many different animals as a mass-spring system (Holmes, Full, Koditschek, & Guckenheimer, 2006; Holt et al., 2006; Nishikawa et al., 2007). Active kicking may reflect a nonlinear coupling of brain network oscillations—themselves exhibiting intermittent, chaotic dynamics (Durstewitz & Deco, 2008)—to an excitable mechanical system, with the muscles re-exciting the intrinsic oscillations of the leg's pendular dynamics as energy is lost. If infant kicking exhibits intermittency due to nonlinear interactions, this may open a window to the process by which particular flexibly-assembled couplings of mechanical components enact internal exploratory processes.

The present work has two major goals. First, it investigates whether the complex, potentially intermittent temporal structure of spontaneous kicking movements reflects the presence of interactive chaotic processes. Interactivity in this chaotic sense may be most concretely understood in terms of multiplicativity. Whereas stochastic processes depending on strictly independent, strictly summative components reflect only additive factors, a stochastic process that depends also on the interactions among these components will reflect a nonzero influence of multiplicative factors. So, our first goal was to test probe the degree of multiplicativity in spontaneous kicking, at each of three major joints (i.e., hip, knee, and ankle). Second, we investigated whether interactivity evident in the kinematics of spontaneous kicking might also play a role in multijoint coordination. In more concrete terms, we sought to test whether multiplicativity at any of the joints predicted later changes multiplicativity at other joints. That is, should we find evidence of interactivity within the kinematics of any joint individually, it may be important to know whether this interactivity actually serves to underwrite interactivity among the joints of the leg as a whole.

We addressed these goals by measuring the fluctuations in joint rotations at the hip, the knee, and the ankle to probe for their relative influences on spontaneous kicking. Bodily fluctuations generally exhibit a temporal structure termed "fractal" (West, 2006; Bassingthwaighte, Liebovitch, & West, 1994), that is, fluctuations grow as a power-law of time scale, according to potentially noninteger (i.e., fractional or "fractal") exponents (Mandelbrot & Van Ness, 1968). Perceptual sensitivity to information depends on significant, structured variability in these fractal exponents, that is, on the multifractality of fluctuations in movement (Stephen, Arzamarski, & Michaels, 2010; Stephen & Anastas, 2011; Stephen & Dixon, 2011; Stephen & Hajnal, in press; Dixon, Stephen, Boncoddo, & Anastas, 2010; Dixon, Holden, Mirman, & Stephen, in press). Interestingly for present purposes, multifractality is also a feature of resting state networks (Ciuciu, Varoquaux, Abry, & Almog, 2011).We approached the problem of demonstrating exploration in leg kinematics by probing the multifractality of fluctuations at each joint and testing for relationships between multifractal fluctuations at different joints along the leg.

An important reason for expecting joint fluctuations to provide an insight into the interactivity in spontaneous kicking is that multifractality is a key feature of

multiplicativity. Expressed most simply as the range of fractal exponents relating fluctuations to scale (Halsey et al., 1986; Mandelbrot, 2004), the degree of multifractality can index the strength of multiplicative factors in producing a stochastic process. For any given time series of fluctuations, a test for multiplicativity involves comparing the degree of multifractality of the original time series with the degree of multifractality for a distribution of surrogate time series. Surrogate time series mimic the linear structure (i.e., mean, variance, and autocorrelation) and so reflect approximations of the original time series built from strictly additive factors. Greater degrees of multifractality for the original time series than for surrogate time series suggests the role of multiplicative factors. That is, multiplicativity will produce greater degrees of multifractality for the original time series than what might be found in surrogate time series. Hence, multifractal analysis of fluctuations at the joints of the leg may provide insights into the multiplicativity.

We predicted that the multiplicativity apparent in multifractal analysis would shed light on the exploratory aspects of spontaneous kicking. To test this broad prediction, we sought to demonstrate the presence of multiplicativity at each joint and evidence that multiplicativity at each joint contributed to interaction amongst the joints of the leg. Specifically, our hypotheses were twofold. First, we hypothesized that fluctuations in joint rotation at the hip, the knee, and the ankle would exhibit multiplicativity (Hypothesis 1). The second hypothesis dealt with the interaction of multiplicative fluctuations across the different joints. Because of the dominant role of the hip during kicking, we expected fluctuations at the hip to generate later fluctuations at the knee and ankle; similarly, we expected that multiplicativity at the hip would promote later multiplicativity at the knee and ankle. However, because multifractal fluctuations may also reflect the developing functionality of the knee and ankle joints for locomotion, we also expected effects of multiplicativity at the knee and ankle for promoting later multiplicativity at the hip. Thus, we hypothesized that spontaneous kicking movements would exhibit both a top-down influence of multiplicativity (i.e., from hip to knee and ankle) and a bottom-up influence of multiplicativity (i.e., from knee and ankle to the hip) among the joints of the leg (Hypothesis 2).

2 Methods

2.1 Materials

2.1.1 Platform

A 36" x 48" x 6" mattress was secured with Velcro to the top of a 36" x 48" x 18" support structure built of four 18" x 24" x 18" double-wall cardboard boxes (Uline #S-4958) connected by Velcro. This central support structure was flanked by a peripheral support structure composed of a 24" x 24" x 12" double-walled cardboard box (Uline #S-4910) at each corner, a 48" x 24" x 12" double-walled cardboard box (Uline #S-15062) on each of the long sides of the central support structure, and a 36" x 24" x 12" double-wall cardboard box (Uline #S-14234) at one of the shorter sides. All of these exterior pieces were secured around the central support structure with Velcro. On top of the peripheral support structure, 14 foam wedges (with 24" x 24" base and 26.6-degree rise from one edge of the base to a height of 12" at the opposite edge of base) were placed with slope facing the central support structure. This foam-wedge perimeter served as a soft cushion preventing the infant from continuing to roll in the event that s/he rolled off the mattress. Eight of the foam wedges were cut diagonally from the 12" height to the opposite edge and were fit together so as to form the corners. One foam wedge was cut in half, parallel to the triangular face to form a wedge with a 24" x 12" base. This truncated wedge was attached to one of the other full size wedges to cover the top of the short side of the peripheral support structure. The trough 12" wide around the perimeter of the mattress was filled with blankets. A 24" x 36" x 12" foam pad was placed on the gap in the peripheral support structure to provide a place for the parent to sit by the infant's feet. Figure 1 shows the implementation of this design in the laboratory.

2.1.2 Mobile

A wooden mobile was suspended 2' above the center of the mattress with a wire secured to the ceiling so as to be central to the infant's visual field.

2.1.3 Vicon motion-capture system

Eight MX-T40 cameras (http://www.vicon.com/products/t40.html) with near-infrared light bulbs surrounding the lens were mounted on the walls (3 on the long walls and 2 on the short walls). Cameras were connected to a Dell desktop computer (Intel Xeon E5430 2.66 GHz, http://ark.intel.com/Product.aspx?id=33081) via an MX Giganet (http://www.vicon.com/products/mxgigagnet.html). Fourteen 14mm-diameter spherical infrared-reflective markers were placed with an electrode adhesive on the infant's hips, legs, and feet to record three-dimensional trajectories at a sampling rate of 120 Hz. The Vicon motion-capture system (Vicon Motion Systems, Oxford, UK, http://www.vicon.com) was operated using Vicon Nexus 1.5.1 (http://www.vicon.com/products/nexus.html) on a Windows XP operating system. For time-locked digital video recording, the Vicon motion-capture system was connected to a Canon Vixia HV30 1080i HDV digital video camera (http://www.usa.canon.com/cusa/support/consumer/camcorders/high_definition_camcorders/vixia_hv30).

2.1.4 Anthropometric measurement materials

A 2-foot paper measuring tape graduated in inches on one side and in centimeters on the other side and an electronic caliper were used to measure the infant's anthropometric parameters. A Scale-Tronix Pediatric Scale 4802 (http://www.scale-tronix.com/pages/productgroup/products/4802.html) was used to weigh the infant.

2.2 Participants

We collected motion-capture data for four infants (3 male and 1 female), mean age 5 months (SD = 1.47). For each infant, a parent provided written informed consent according to the guidelines of the institutional review board at Children's Hospital Boston.

2.3 Procedure

2.3.1 Vicon marker placement

Marker placements followed the configuration shown in Figure 2. Once the markers were attached, we placed the infant on his/her back on the mattress, with his/her feet pointed towards the parents.

2.3.2 Recording data

We recorded trials of approximately 30-second duration each. During each trial, we manipulated the mobile using a wire attached to the mobile to visually engage the infant and to encourage kicking. There was no purposeful contingency between infant kicking and movement of the mobile. In the intervals between the 30-second trials, we replaced fallen markers. If the infant was in the middle of kicking as the 30 seconds came to an end, we chose to extend the recording duration as long as the kicking bout continued. Recording 30-second trials continued until the infant became fussy.

Every third row of Table 1 shows the age of each infant as well as the corresponding number of individual joints recorded. During one trial for an infant aged 6 months, the motion-capture markers specifying the position of the left ankle fell off, and so there was one fewer recording of the left ankle than there were for all other joints. In all other cases, all six joints were successfully recorded during each trial, and with this sole exception, there were an equal number of recordings across joints.

2.3.3 Anthropometric parameters

We measured infant's anthropometric parameters to calculate their limb segments' mass M, center of mass CoM, and moment of inertia I according to the regression equations published by Schneider and Zernicke (1992). These parameters are infant's age, body weight, segment lengths, widths, and circumferences. Table 2 shows the anthropometric parameters and the regression equations for mass, center of mass, and moment of inertia calculation.

2.4 Data analysis

2.4.1 Prefatory remarks about analysis strategy

Data analysis followed three main steps. In the first step, the motion-capture data for each trial was used to generate a time series of joint angles for each of six concurrently recorded joints (i.e., left hip, left knee, left ankle, right hip, right, knee, and right ankle). These joint angles reflected the 30-second recordings from the Vicon motion-capture system at 120 Hz. Hence, each trial generated six joint-angle time series, each consisting of 3600 successive samples equally spaced in time by 1/120 seconds. In the second step, we estimated the multifractal spectrum (according to an algorithm described below) for each joint-angle time series. Therefore, six multifractal spectra were estimated from each trial. As described below, the width of these multifractal spectra were compared to that of corresponding surrogates' multifractal spectra. The term "surrogates" here refers to artificial time series designed to mimic the strictly linear structure (i.e., mean, variance, and autocorrelation) of each original time series and, hence, to represent candidate additive cascade processes producing time series similar to the original. If the multifractal-spectrum width for an original time series exceeded the average of its corresponding surrogates' multifractal-spectra widths, this observation was taken as evidence of multiplicative cascade dynamics (Ihlen and Vereijken, 2010). The third step of analysis dealt with a relative multiplicativity ratio, defined for each joint in each trial as its multifractal-spectrum width divided by the average width of the corresponding surrogates' multifractal spectra. In the third step, the sequence of those relative multiplicativity ratios served as a time series at a relatively coarser grain than the raw joint-angle time series. Specifically, each value of the relative multiplicativity ratio in this third step carried summary information about a single time series of 30 seconds (i.e., 3600 samples) in the first step; hence, the sequence at the third step represented spontaneous kicking at a grain 3600 times more coarse than the time series at the first step. Whereas the time series of joint angles had equal number of evenly spaced samples recorded by the Vicon motion-capture system, the time series of relative multiplicatiivity ratios were dramatically shorter and heterogeneously spaced in time due to unavoidable short interruptions in the data capture. The 30-second trials were recorded with varying amounts of time between them, depending on the need for

replacing markers, removing occlusions to the view of Vicon cameras, or calming the infant. Nonetheless, we sought to describe structure within the sequence of relative multiplicative ratios across successive trials.

2.4.2 Data processing and inverse kinematics

The resulting Vicon Nexus files were converted to files containing trajectory information. We used the freely downloadable OpenSim software from SimTk (https://simtk.org/home/opensim-utils/) to make these conversions.

For each infant, we created an individualized OpenSim musculoskeletal model based on the measured anthropometric parameters. Then we used an inverse kinematics tool from OpenSim to acquire joint coordinates. The inverse kinematics tool is a computation that best matches the experimental data (marker positions) and model posture sample by sample, minimizing the discrepancy between real markers and model markers. The computation can be expressed in terms of weighted least-squares optimization:

$$\min \sum_{i=1}^{N_M} w_i \left(X_i^{participant} - \hat{X}_i \right)^2 \quad (1)$$

where N_M is the number of markers, w_i is the weight of marker i, $X_i^{participant}$ is the actual position of marker i on the participant, and $\hat{X}_i$ is the model's predicted position of the ith marker (Delp, Loan, Hoy, Zajac, Topp, and Rosen, 1990; Delp, Anderson, Arnold, Loan, Habib, John, Guendelman, and Thelen, 2007).

The OpenSim inverse kinematics tool computes joint angles in degrees, such that positive angles represent joint flexion and negative angles represent joint extension. However, the only exception is the ankle joint for which the neutral anatomical position is 90 degrees, as in standing. As demonstrated in the example joint-angle time series, ankle-joint angles are centered at a neutral position of zero, that is, with 90 degrees automatically subtracted. Hence, for ankle-joint angles, positive angles respresent dorsiflexion (i.e., originally, values greater than 90 degrees), and negative angles represent plantar flexion (i.e., originally, values less than 90 degrees).

2.4.3 Direct estimation of the multifractal spectrum

Multifractal analyses seek to specify the degree of heterogeneity in a given distribution. For any homogeneously distributed system, proportion P of total

fluctuations within any given observation window increases with window size n as

$$P(n) \sim n^{\alpha}, \quad (2)$$

where α is the singularity strength (Vicsek, 1992). As we dealt with continuous time series, the proportion measure described in Eq. 2, "fluctuations" were defined as "positive excursion from zero." "Total fluctuations" were treated as the total sum of an entire time series $x(t)$ (e.g., Yang, He, Zhou, and Ning, 2010). For our purposes, $x(t)$ can be thought of as a joint-angle time series. Because the algorithm defined below requires only non-negative numbers, for each joint-angle time series, we first subtracted the minimum of the time series from all other values in time series.[1] Proportion $P(n)$ was the ratio of average local sum for windows of size n (i.e., for time windows of n successive samples from the joint-angle time series) to total sum for the entire time series $x(t)$.

$$P(n) = \frac{\sum_{j=1}^{N_n} \sum_{i=1+n(j-1)}^{jn} x(i)}{\sum x(t)} \sim n^{\alpha}, \quad (3)$$

n is a scale factor that is incremented by one from an arbitrary minimum to encompass a variety of windows; we designated the number of nonoverlapping windows as N_n to distinguish it from N indicating the time-series length (as in Kantelhardt et al., 2002). In the present case, we incremented n by one across the interval $4 \le n \le N/4$. In Eq. 3, for a given values of n, the jth window of the time series $x(t)$ begins with $x(1 + n(j\text{-}1))$ and ends with $x(jn)$. Eq. 3 expresses $P(n)$ as the sum of each of these local window sums divided by the total sum or,

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[1] Two points suggest against any bias in this transform. First, multifractal analysis pertains to the amount of heterogeneity across different samples, and simply adding a constant to all values in a time series did not compromise the inter-sample heterogeneity. Second, results from multifractal analysis for any time series were only considered in light of the results from multifractal analysis of a distribution of its corresponding surrogates, whether explicitly in the case of comparison with average of the surrogates' multifractal spectrum or implicitly in the case of the calculation of the relative multiplicativity ratio.

equivalently, $P(L)$ might be thought of as a ratio of average window integral to total integral.

For heterogeneously distributed systems, fluctuation density can vary from window to window; in other words, a window can contain more or less fluctuation "mass." In this case, when fluctuation mass varies appreciably, proportion P of total fluctuations in different windows may increase according to diverse relationships with window size:

$$P_j(n) \sim n^{\alpha_j}. \quad (4)$$

That is, each jth window may exhibit its own singularity strength α_i. The frequency of each singularity strength is approximated by a Hausdorff measure f, a scaling relationship between Shannon entropy and window size (Halsey et al., 1986). Hence, it is possible to find a different value of f for windows with different singularity strength, that is, $f(\alpha_j)$. The term $f(\alpha_j)$ expresses a fractal dimension of the set of windows exhibiting the same singularity strength α_j. The relationship $f(\alpha)$—called the multifractal (or "singularity"; Mandelbrot [2004]) spectrum—serves as a frequency distribution of the singularity strengths needed to specify the various rates at which proportion grows with window size. The width of this frequency distribution represents the heterogeneity of system dynamics, "heterogeneity" referring to the variety of rates (i.e., singularity strengths) according to which proportion of fluctuation grows with window size. It is defined only for those values of f and α that can be reliably estimated from scaling relationships, defined below, of fluctuation-mass entropies and mass-weighted proportions, respectively, with window size n.

A number of multifractal methods derive the multifractal spectrum from generalized fractal dimensions (e.g., Kantelhardt et al., 2002; Halsey et al., 1986; Muzy, Bacry, and Arneodo, 1994), using a Legendre transformation (e.g., Callen, 1985) to calculate both f and α from different attributes of the same generalized fractal dimension. Although there may be strong theoretical motivation for transforming the generalized fractal dimension to derive the multifractal spectrum, these indirect methods may be prone to bias (Chhabra and Jensen, 1989). In this report, we use the direct estimation of the multifractal spectrum: calculating f and α independently and then admitting only those singularity strengths α for which

both α and its corresponding f are reliably defined (Chhabra and Jensen, 1989; Zamir, 2003).

For an observable time series $x(t)$, direct estimation of the multifractal spectrum begins with calculation of individual proportions $P_j(n)$ for each jth window of size n:

$$P_j(n) = \frac{\sum_{i=1+n(j-1)}^{jn} x(i)}{\sum x(t)}. \qquad (5)$$

The next step is to express the fluctuation density for each jth window of size n in terms of a partition function $\mu_j(q,n)$:

$$\mu_j(q,n) = \frac{(P_j(n))^q}{\sum_{j=1}^{N_n} (P_j(n))^q}, \qquad (6)$$

where q is a statistical moment used to selectively emphasize gradually different proportions. For the present report, the q parameter was incremented and decremented from $q = 1$ by .05 until the correlations decreased below a criterion. When $q = 1$, $\mu(q,n)$ reflects the mass of fluctuations based on raw proportions $P(n)$ as originally calculated in Eq. 3, but when $q > 1$ or $q < 1$, $\mu(q,n)$ reflects the mass of fluctuations for windows with higher proportions or lower proportions, respectively. For each value of q, the partition function $\mu(q,n)$ allows calculation of the Hausdorff measure f for different values of q, that is, $f(q)$, defined as

$$\begin{aligned} f(q) &= -\lim_{N_n \to \infty} \frac{1}{\ln N_n} \sum_{j=1}^{N_n} \mu_j(q,n) \ln[\mu_j(q,n)] \\ &= \lim_{n \to 0} \frac{1}{\ln n} \sum_{j=1}^{N_n} \mu_j(q,n) \ln[\mu_j(q,n)]. \end{aligned} \qquad (7)$$

In the foregoing paragraphs, we had seen that the Hausdorff measure might differ for windows with different singularity strength α, but now we see that it is also possible to express differences in the Hausdorff measure with different values of q. The generalized Hausdorff measure $f(q)$ is estimated as the scaling relationship of Shannon entropy of fluctuation masses $\mu_j(q,n)$ with window size n. Thus it has a meaning independent of the singularity strengths of individual windows.

Similarly, it is possible to generalize singularity strengths over different values of q as $\alpha(q)$, defined as

$$\begin{aligned}\alpha(q) &= -\lim_{N_n \to \infty} \frac{1}{\ln N_n} \sum_{j=1}^{N_n} \mu_j(q,n) \ln[P_j(n)] \\ &= \lim_{n \to 0} \frac{1}{\ln n} \sum_{j=1}^{N_n} \mu_j(q,n) \ln[P_j(n)].\end{aligned} \tag{8}$$

The singularity strength $\alpha(q)$ is estimated as the scaling relationship between mass-weighted proportion $P_j(n)$ with window size n; it represents the average of singularity strengths $\alpha_j(q)$ for each jth window for a given q. Provided that $f(q)$ and $\alpha(q)$ form a single-peaked curve $f(\alpha(q))$ for those values of q producing linear scaling relationships in Eqs. 7 and 8, the observable time series $x(t)$ is multifractal (Zamir, 2003). This $f(\alpha(q))$ is the singularity (or "multifractal") spectrum, and its width serves as an index of multifractality (Ivanov et al., 2001; Ihlen and Vereijken, 2010). For the present report, we included only those values of q for which the scaling relationships $f(q)$ and $\alpha(q)$ in Eqs. 7 and 8 reflected correlation at $r \geq .95$.

The singularity spectrum estimated from a time series should not be judged in isolation. Non-zero width of the singularity spectrum is evidence for multifractality (i.e., significant heterogeneity in the relationship of proportion $P_j(n)$ with window size as in Eq. 4), but it does not address the source of the multifractal fluctuations. Width of the singularity spectrum may reflect non-Gaussian distributions or autoregressive temporal structure in the original time series (Ihlen and Vereijken, 2010; Veneziano, Moglen, and Bras, 1995). So, it is important to compare the singularity-spectrum width estimated for the original time series against the distribution of singularity-spectrum widths for surrogate time series that mimic that distribution and autocorrelation. A commonly used algorithm for producing such surrogate time series is iterative amplitude-adjusted Fourier-transformation (IAAFT; Schreiber & Schmitz, 1996) method; it randomly reshuffles the values of the original time series while preserving the probability density and autocorrelation functions of the original time series. When the singularity spectrum of the original time series is significantly wider than the distribution of singularity spectra for the surrogate time series, the original time series can be said to exhibit multiplicative cascade dynamics (Figure 3).

Significant difference in width may be tested as a one-sample *t*-test, with critical value 1.96 for significance at $p < .05$. For the present report, we compared the singularity-spectrum width for each original time series with the singularity-spectrum widths for 50 of its IAAFT surrogates.

Because direct estimation of the singularity spectrum according to the foregoing algorithm does not produce real-numbered for time series containing negative numbers, we subtracted the minimum value from each time series before submitting it both to multifractal analysis and to surrogate generation. We generated 50 IAAFT surrogate time series for each original time series to test for multiplicative cascade dynamics.

2.4.4 Vector autoregressive (VAR) modeling

Essentially, VAR modeling tests the mutual effects among endogenous variables, controlling for effects from exogenous variables. Vector autoregressive (VAR) modeling tested mutual effects of multiplicativity among the hip, knee, and ankle joints on each side. That is, there were two models which included all infants, one for the right legs and the other for the left legs. The dependent measure from each joint was, for each 30-second recording, a relative multiplicative ratio, i.e., the ratio of original singularity-spectrum width to the average surrogate singularity-spectrum width. Because the singularity-spectrum width can be biased by non-stationarities of the corresponding time series, this ratio served as a general continuous measure of relative multiplicativity. The dichotomous result of the typical surrogate comparison (i.e., in which either the singularity spectrum is wider than the average surrogate singularity spectra or it is not) omits information about the finer-grained variability in the comparison between original singularity spectrum and surrogate singularity spectra. There may be considerable variability in the width of the original spectra on either side of the arbitrary cut-off entailed by a 95% confidence interval. The continuous aspect of the relative multiplicativity ratio may allow a more subtle test of how multi-joint interactions might influence multiplicative cascade dynamics. Multiplicativity may not always be sufficiently strong to push spectra widths beyond a 95% confidence interval on each individual trial (i.e., to generate a *t*-statistic greater than 1.96), but there may be statistically significant relationships in the relative multiplicativity across the joints that may predict reliable changes in spectra widths (relative to surrogate

spectra widths) over several trials. We sought to test relationships among the joints in terms of relative multiplicativity, whether or not absolute singularity-spectrum width suggested significantly (i.e., at 95% confidence levels) strong multiplicative factors.

For the present research, VAR modeling tested mutual effects among the endogenous variables represented by relative multiplicativity ratios for each of the three joints. It included relative multiplicativity ratios only for those 30-second recordings when all joint-angle time series were available from all six joints. We only generated a VAR model for one side at a time, but for the one occasion when markers were occluded or detached during motion capture, there was no joint-angle time series for one or more joints on only one side. However, it would be misleading to include all available data if it meant that an infant might contribute different numbers of relative multiplicativity ratios to the two models. The exogenous variables that we incorporated in VAR modeling comprised i) dummy-coded variables for each infant and ii) a trial variable expressing the number of successive trials with the complete six-joint set. Further explanation and details of VAR modeling, both in general and in specific application to the present dataset, may be found in the Appendix.

3 Results and Discussion

3.1 Overview

We tested two hypotheses. Hypothesis 1 was that fluctuations in joint angles at the hip, knee, and ankle during infants' spontaneous kicking would exhibit multiplicativity. Hypothesis 2 was that, independent of any proximal-to-distal effects of relative multiplicativity from the hip on later relative multiplicativity at the knee and ankle, the relative multiplicativity of fluctuations at the knee and ankle would promote later relative multiplicativity at the hip. As described belo, results were consistent with both of these hypotheses.

3.2 Multifractal analysis of joint-angle time series and comparison with surrogate time series

Figure 4 illustrates a portion of the joint-angle trajectories for each of three leg joints from an example trial. Because motion-capture data was recorded at 120 Hz for 30 seconds at a time, each recording included 3600 samples.

Direct estimation of the multifractal (or singularity) spectrum revealed that all 257 recorded joint-angle time series were multifractal, that is, had singularity spectra with nonzero width. Figure 5 shows singularity spectra for example joint-angle time series for the hip, knee, and ankle on each leg from an example trial with an example infant. Table 2 shows the numbers of joint-angle time series whose singularity spectra were significantly wider than their corresponding distribution of 50 surrogate singularity spectra. In all, there were 102 joint-angle time series (i.e., 36.69% of all joint-angle time series) whose spectra were wider than those of their surrogates. This result meant that these 102 joint-angle time series exhibited significantly more heterogeneity (i.e., exhibited significantly wider variety of growth rates of proportion $P_j(n)$ with window size n) than what would be expected from a distribution of artificial time series with the same linear structure. The failure of linear structure to exhibit comparable heterogeneity indicates that strictly additive cascade dynamics are not sufficient to generate the observed time series; there must therefore be a significantly multiplicative factor generating the observed time series.

3.3 Distribution of time series exhibiting significant evidence of multiplicative cascade dynamics

The distribution of joint-angle time series exhibiting significant evidence of multiplicative cascade dynamics revealed an interesting structure. That is, a 2 (side) x 3 (joint) analysis of variance (ANOVA) demonstrated that time series of the hip angle exhibited multiplicative cascade dynamics significantly more often than either the knee angle or the ankle angle, $F(2, 18) = 7.65$, $p < .01$. Side and the interaction of side with joint showed no other significant differences in the frequency of joint-angle time series exhibiting multiplicative cascade dynamics, Fs < 1. As Table 2 shows, joint-angle time series exhibited multiplicative cascade dynamics for roughly half of the hip joint-angle time series and for much less than half of the other two joints.

3.4 Preliminary implications and hypotheses for consideration in subsequent VAR modeling

This disproportionate frequency of multiplicative cascade dynamics in the hip joint warrants some discussion and the articulation of hypotheses guiding an understanding of subsequent VAR modeling. This section sketches out possible relationships among the joints that may account for the distribution of multiplicative cascade dynamics across the joints.

The disproportionate evidence of multiplicative cascade dynamics at the hip raises questions about the coordination among the different joints during spontaneous kicking. As discussed in the introduction, multifractal, multiplicative fluctuations can index the transmission of sensory and neural information among the environment and different parts of the motor system (Stephen and Dixon, 2011; Stephen and Hajnal, in press). What might this distribution of multiplicativity tell us about the flow of information through the leg? VAR modeling of relative multiplicativity may provide some insight into how the joints along a single leg might share multiplicative fluctuations, that is, how the fluctuations—and so perhaps information—in joint angles might propagate across the leg.

Generally speaking, there are three possible ways in which the joints might share multiplicative fluctuations. First, and most simply, there could be no relationship amongst the joints. Second, it is possible to envision a proximo-distal differentiation process according to which the multiplicativity at the hip feeds downward to the lower extremities (i.e., knee and ankle). Wide singularity spectra in heartbeat fluctuations (Ivanov et al., 2001) and postural fluctuations (Morales and Kolaczyk, 2002) for healthy, typically-developing individuals may point to a more general structure of multiplicativity through the torso for healthy development. Multiplicativity may simply taper off toward the more distal parts of the body, and what multiplicativity can be found in the extremities may simply be downstream effects of events in the more proximal regions of the body. To the degree that fluctuations may reflect the transmission of neural information, this second option alone would be most compatible with a view of motor coordination that is driven entirely by outwardly tending commands from a central executive. Third, is it possible that multiplicativity at the lower extremities feeds upwards. That is, just in the same way that multifractal fluctuations at the periphery of the body can reflect the flow of sensory information to the rest of the body (Stephen

and Hajnal, in press), perhaps the multifractal fluctuations in the knee and the ankle joints serve to promote multifractal fluctuations at the hip. Perhaps what multiplicativity the torso may exhibit in healthy, typical development is fostered by relatively weaker multifractal fluctuations distributed across the more distal regions of the body that somehow aggregate and produce stronger multifractal fluctuations in the more proximal regions of the body. This third option alone would be more compatible with a view of motor coordination entirely organized by bottom-up interactions, similar to what Van Orden, Holden, and Turvey (2003) suggested by implicating self-organized criticality (SOC; Bak, 1996). The second and third possibilities are not mutually exclusive, however, and together they may support a view of developing motor coordination as the product of nonlinear interactions at many different spatial and temporal scaling, including contributions from both top-down and bottom-up effects in the nervous and motor systems. Presently, the empirical results speak only to the time scales ranging from the smallest window size *n* within each 30-second trial to the coarser-grain series of trials considered in VAR modeling, but it may have further applications over longer developmental time scales (e.g., Plotnick and Spekoski, 2001).

3.5 VAR modeling of relative multiplicativity ratios to test for interactions among joints on each side

We ran lag-1 VAR modeling of relative multiplicativity ratios (*M*; i.e., original singularity-spectrum width divided by the corresponding average surrogate singularity-spectrum width) for each side, including exogenous variables for trial and for dummy-coding of individual infants. The Appendix describes the details of this VAR modeling.

The results of the VAR modeling are most simply presented in terms of the resulting impulse response functions (IRFs). As described in more detail in the Appendix, IRFs depict the later changes in (i.e. the response of) any endogenous variable due to a current increase in the current value of (i.e., due to an impulse from) another endogenous variable. Figures 7 and 8 depict the IRFs for the relatively multiplicativity ratio *M* for each of three joints along the leg on the right side and the left side, respectively. Solid curves represent the IRF, and the dashed lines represents the 95% confidence intervals for each value in the IRF. On the right side, there appeared to be no significant effect of relative multiplicativity at

the hip joint on later relative multiplicativity at both the knee and ankle joints. That said, the relative multiplicativity at both the knee and ankle significantly promotes the relative multipliciativity at the hip, knee, and ankle on later trials. On the left side, the relative multiplicativity at all joints significantly promotes the relative multiplicativity at all joints on later trials. Across the board, significant effects on later relative multiplicativity were positive and extended over many subsequent trials. Though not all infants contributed 10 trials, Figures 6 and 7 illustrate the predicted response of each variable to unique contributions of each variable based on the coefficients from VAR modeling, and the choice to present predicted responses over 10 trials is arbitrary. IRFs illustrate the average response predicted from the modeled lag-1 VAR relationships over the distribution of all 10-trial periods available from the observed sequence of relative multiplicativity ratios. For a VAR model with i.i.d. Gaussian residuals, the response (i.e., value of the IRF curve on the vertical axis) due to an impulse from any one variable will die out over sufficiently many trials.

3.6 Implications of VAR modeling

We had highlighted three major alternative outcomes from VAR modeling of relative multiplicativity ratios across the three joints on each side. First, there could have been no evidence for effects of multiplicativity from one joint on any other. Second, the multiplicativity at the lower joints (i.e., knee and ankle) could have been the downstream effects of later multiplicativity at the hip. Third, the multiplicativity of the more distal joints could have been crucial for promoting multiplicativity at the hip. The IRFs suggested a combination of the second and third alternatives. That is, the IRFs provided evidence supporting the notion that multiplicativity at the knee and ankle promoted multiplicativity at the hip on both sides. The IRFs provided mixed evidence supporting the second alternative: only on the right side did multiplicativity at the hip have significant effects on later multiplicativity at the more distal joints (see Figure 8).

The positive effects illustrated by the IRFs served as new information independent from the raw frequencies of joint-angle time series exhibiting multiplicative cascade dynamics (Table 2). These raw frequencies of joint-angle time series exhibiting significantly strong multiplicative factors reflected only the number of joint-angle time series whose singularity spectrum with width greater than the

95% confidence interval around the average width of the corresponding surrogates' singularity spectra. Such joint-angle time series, as noted above, made up only about a third of the total joint-angle time series and were disproportionately more frequent at the hip than at the other joints. The findings from VAR modeling illustrated in the IRFs indicate positive effects for the relative multiplicativity of most joints on relative multiplicativity of all other joints. These positive effects might first seem to have suggested a runaway increase in singularity-spectrum widths across the board, and thus, they might seem to have run counter to the resounding evidence of relatively narrow singularity-spectrum widths the knee and ankle joint angles. However, the IRFs do not reflect the overall mean value of singularity-spectrum widths for the different joint angles. Instead, they addressed the effect of trial-to-trial changes in relative multiplicativity (i.e., singularity-spectrum width divided by surrogate singularity-spectrum width) above and beyond the mean differences across joints and how trial-to-trial changes in any one joint angle's relative multiplicativity influenced all joint angles' subsequent relative multiplicativity. Furthermore, IRFs only indicated effects consequent to positive change, and so positive values in the IRFs meant only that changes in one variable causes later changes in another variable in the same direction. Hence, positive effects in IRFs from a VAR model of the relative multiplicative ratio suggested only that increases and decreases in relative multiplicativity for one joint angle promote later increases and later decreases in relative multiplicativity for other joint angles.

The relationship among the joint angles demonstrated by the IRFs in Figures 6 and 7 may provide an explanation for the disproportionate distribution of relatively wide singularity spectra at the hip. The mutual positive effects between knee and ankle may serve to amplify later effects of each on the hip, but it remains unclear how the knee and the ankle joint-angle fluctuations maintain relatively narrow singularity spectra and, consequently, exhibit weak relative multiplicativity. There thus seemed to be negative effects on relative multiplicativity, whether from non-kinematic physiological factors or from external mechanical/gravitational factors, that served to narrow the singularity-spectrum width for knee and ankle joint-angle fluctuations. More longitudinal data, comparison with atypically developing groups, and experimental

manipulation of limb inertia might be assist future research in further exploring those factors narrowing the singularity spectrum.

4 General Discussion

Taken together, the evidence from multifractal fluctuations in the kinematics of the leg during infants' spontaneous kicking provides new insight into the flow of information coordinating movement. First, multifractality serves as evidence for multiplicative cascade dynamics, suggesting the presence of nonlinear interaction of various factors across temporal and spatial scales (e.g., Ihlen & Vereijken, 2010). The disproportionate distribution of multiplicative cascade dynamics across the leg during infants' spontaneous kicking suggests a structured relationship between joint-angle fluctuations at the hip and joint-angle fluctuations at the knee and ankle. VAR modeling indicated that changes in the relative multiplicativity of fluctuations at the knee and ankle serve to promote later changes in the relative multiplicativity at the hip (Figure 8). Because multifractal fluctuations are an important signature of information flow through the motor system (Stephen et al., 2010; Dixon, Stephen et al., 2010; Dixon, Holden et al, in press; Stephen and Anastas, 2011; Stephen and Hajnal, in press), these effects of relative multiplicativity from more distal joints to more proximal joints serve as new evidence for the exploratory function of spontaneous kicking. These results both corroborate previous findings concerning the multifractal fluctuations in physiology and suggest directions for further research in the dynamics of motor coordination. First, the relatively narrow singularity spectra from multifractal analysis of leg kinematics during infants' spontaneous kicking bears similarity to previous findings of narrow singularity spectra in gait (Muñoz-Diosdado, 2005; West & Scafetta, 2003). This similarity may suggest that infants' spontaneous kicking bears a close relationship with the development of processes underlying gait.

Further, the mutual effects amongst the relative multiplicativity of different joints across the leg serves as evidence that infants' spontaneous kicking reflects the interactions along the proximo-distal gradient of downstream effects of central nervous processes on the motor periphery and upstream effects of peripheral mechanics on more proximal control. This exchange of multiplicative fluctuations presents a complementary view of coordination along the proximo-distal gradient

to that presented by a biomechanical accounting of kinetics (e.g., Daley et al., 2007). Kinetics may provide information about the aggregate torques propelling motor components along their mean trajectories, but the dissipation of kinetic energy is a necessary aspect of movement that opens the motor system up to fluctuations (i.e., thermal energy; Alexander, 2000). Dissipation of kinetic energy and the resulting fluctuations represent a stochastic aspect of motor control that defies an accounting by torques. Dissipation is more often expressed in terms of multifractality and underpinning multiplicative noise processes (e.g., Frisch, 1996). Hence, differences in active control or passive reactivity of torques along the proximo-distal gradient of the leg (e.g., Jensen et al., 1994; Ulrich et al., 1994) reflects only one aspect of the developing motor system. The present finding serves to underscore a bidirectional flow of information across the leg that need not be underwritten explicitly by active and passive torques.

These results underscore the role of interactive, chaotic dynamics in coordinating movement. The bidirectional flow of multiplicative fluctuations along the proximo-distal gradient of the infant leg may provide a foundation for viewing infants' spontaneous kicking as the expression of resting state network (RSN) dynamics exhibited by the brain and nervous system (Deco et al., 2011; Deco & Corbetta, 2011). The potential extension of RSN dynamics across the motor system could have potentially dramatic entailments for motor development. First, it would provide a deeper framework for understanding the relationship between spontaneous movements and the refinement of neural pathways for motor control. That is, whereas previous accounts have understood changes in spontaneous kicking movements as a downstream effect of differences in neural development (e.g., the effect of white-matter brain injury as in Fetters et al. 2010), the present evidence opens up the possibility that fluctuations at the motor periphery may influence dynamics of neural mechanisms upstream. Crucially, in the framework of RSN dynamics run by nonlinear interactions, a distinction between "top-down" and "bottom-up" may not be so appropriate, and control of movements may originate from the motor periphery as well as the central nervous system.

Future research into the role of fluctuations in the chaotic dynamics of motor development might follow two broad currents: one investigating endogenous group differences in multiplicative fluctuations and the other pursuing the therapeutic applications of exogenously manipulating the flow of fluctuations for

atypically developing infants. For instance, future research in the former current could investigate whether infants with white-matter brain injury exhibit differences in the presence and flow of multiplicative fluctuations in spontaneous kicking; this work could be important for early diagnosis of cerebral palsy. Studying the patterns in multijoint coordination of multiplicative fluctuations longitudinally for both typically-developing and brain-injured infants might provide better insights into the chaotic dynamics underpinning motor development.

Future research on the design of a therapeutic device poses an even stronger, more provocative test of the role of chaotic dynamics in motor development. Although previously demonstrated differences in spontaneous kicking between typically-developing and brain-injured infants may reflect effects of brain injury (Fetters et al., 2010), it is possible that resulting differences in kicking serve to change the later flow of information back to the nervous system. A nervous system beginning at a baseline of brain injury might not be able to generate the kicking movements that would normally promote more typical development and more optimal outcomes. Orthotic devices designed to accentuate the flow of fluctuations from motor periphery to more central parts of the motor system could help support kicking movements that would stimulate more typical development, possibly preventing further adverse effects of brain injury. Research on enhancing sensorimotor function with noise (Priplata et al., 2006; Priplata et al., 2003; Priplata et al., 2002) and on the chaotic control of robotics (Steingrube, Timme, Wörgötter, and Manoonpong, 2010; Arena, De Fiore, and Patané, 2009) suggest that building peripheral fluctuations into the architecture of motor systems may foster the emergence of adaptive, flexible coordinations of motor degrees of freedom.

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Tables

Table 1. Regression equations for segment mass, center of mass, and moment of inertia calculations.

Segment	Parameters
Upper arm	$M = 1.2249 \times 10^{-2} BW + 1.3067L + 9.8645 \times 10^{-1} C - 1.9376 \times 10^{-1}$
	$CoM = 4.428 \times 10^{-1} L$
	$I = 2.753 \times 10^{-5} BW + 4.21 \times 10^{-3} L - 4.0818 \times 10^{-4}$
Forearm	$M = 5.2671 \times 10^{-3} BW + 9.7584 \times 10^{-1} L + 1.1492C - 1.6886 \times 10^{-1}$
	$CoM = 4.541 \times 10^{-1} L$
	$I = 1.07 \times 10^{-5} BW + 1.9878 \times 10^{-3} L - 8.7072 \times 10^{-4} C - 2.7489 \times 10^{-4}$
Hand	$M = 2.1345W - 4.6776 \times 10^{-2}$
	$CoM = 4.495 \times 10^{-1} L$
	$I = 8.2554 \times 10^{-4} W - 2.16 \times 10^{-5}$

Thigh	$M = 6.9126\times10^{-2}A + 2.9582L + 3.1541C - 6.7217\times10^{-1}$
	$CoM = 4.859\times10^{-1}L$
	$I = 1.7943\times10^{-2}L + 5.5699\times10^{-3}C - 2.7078\times10^{-3}$
Leg	$M = 6.5138\times10^{-3}BW + 1.8158L + 1.8743C - 3.546\times10^{-1}$
	$CoM = 4.377\times10^{-1}L$
	$I = 1.866\times10^{-5}BW + 8.5431\times10^{-3}L + 1.6127\times10^{-3}C - 1.1192\times10^{-3}$
Foot	$M = 2.9331\times10^{-3}BW + 1.2405L + 1.9337W - 1.025\times10^{-1}$
	$CoM = 3.469\times10^{-1}L$
	$I = 2.244\times10^{-5}A + 9.2595\times10^{-4}L + 2.6849\times10^{-3}W - 1.2675\times10^{-4}$

Table 2. Number of recorded joint-angle time series exhibiting significant multiplicative cascade dynamics, by infant, by joint, and by side.

		Hip		Knee		Ankle	
Age (mos.)	Multiplicative?	Left	Right	Left	Right	Left	Right
6	Yes	6	6	2	2	3	1
	No	7	7	11	11	9	12
	Total	13	13	13	13	12	13
4	Yes	5	4	1	2	3	4
	No	2	3	6	5	4	3
	Total	7	7	7	7	7	7
3.5	Yes	7	9	5	5	7	4
	No	6	4	8	8	6	9
	Total	13	13	13	13	13	13
6.5	Yes	7	5	4	3	3	4
	No	3	5	6	7	7	6
	Total	10	10	10	10	10	10

Figure Legends

Figure 1. Photograph depicting the position of the infant and the parent on the support structure.

Figure 2. Schematic of supine infant illustrating the location of Vicon motion-capture markers on the legs: left hip (A), right hip (B), left thigh (C), right thigh (D), left lateral knee (E), left medial knee (F), right medial knee (G), right lateral knee (H), left lateral ankle (I), left medial ankle (J), right medial ankle (K), right lateral ankle (L), left foot (M), and right foot (N).

Figure 3. Schematic comparisons of an original time series' singularity (or multifractal) spectrum (black curve) against surrogate time series' singularity (or multifractal) spectra (grey curves). The double-headed arrows indicate the width of the singularity spectra of matching color. The left panel shows the case in which the original time series' singularity spectrum falls within the distribution of surrogate time series' singularity spectra. The right panel shows the case in which the original time series' singularity spectrum is wider than the distribution of surrogate time series' singularity spectra. The right panel is consistent with multiplicative cascade dynamics, that is, significant complexity above and beyond additive cascade dynamics. The left panel is consistent with multiplicativity weak enough to leave original time series' singularity spectra indistinguishable from those of the surrogate time series.

Figure 4. Example joint-angle trajectories for each of three joints on each leg. The left and right panels depict joint-angle trajectories for the hip (black curve), knee (dark grey curve), and ankle (light grey curve) on the left leg and the right leg, respectively.

Figure 5. Example singularity spectra for joint-angle trajectories on each leg. The left and right panels depict singularity spectra for joint-angle trajectories for the hip (black curve), knee (dark grey curve), and ankle (light grey curve) on the left leg and the right leg, respectively.

Figure 6. Impulse response functions (IRFs) from vector autoregressive (VAR) modeling of relative multiplicativity ratios for the hip, knee, and ankle joint angles on right leg. The right, middle, and left columns contain responses due to impulses from relative multiplicativity ratios at the hip, knee, and ankle, respectively. The top, middle, and bottom rows contain the responses in relative multiplicative ratios at the hip, knee, and ankle, respectively. For each plot, the y-axis specifies the amount of later response to a current impulse (i.e., an increase by one standard error) from any of the endogenous variables (organized here by column), and the x-axis specifies the number of trials following the current impulse. The IRF (black curve) illustrates the later response to a current impulse as it decays over subsequent trials. Later response is significantly large for those subsequent trials when the 95% confidence intervals (dashed curve) does not cross the x-axis.

Figure 7. Impulse response functions (IRFs) from vector autoregressive (VAR) modeling of relative multiplicativity ratios for the hip, knee, and ankle joint angles on left leg. The left, middle, and right columns contain responses due to impulses from relative multiplicativity ratios at the hip, knee, and ankle, respectively. The top, middle, and bottom rows contain the responses in relative multiplicative ratios at the hip, knee, and ankle, respectively. For each plot, the y-axis specifies the

amount of later response to a current impulse (i.e., an increase by one standard error) from any of the endogenous variables (organized here by column), and the x-axis specifies the number of trials following the current impulse. The IRF (black curve) illustrates the later response to a current impulse as it decays over subsequent trials. Later response is significantly large for those subsequent trials when the 95% confidence intervals (dashed curve) do not cross the x-axis.

Figure 8. Abstract schematic of the relationship between the joints found using VAR modeling of relative multiplicativity ratios (i.e. a schematic compression of information illustrated more completely in Figures 6 and 7). VAR modeling allows test of mutual effects among endogenous variables over time: the vertical axis pointing downwards suggests the forward passage of time, and columns represent relative multiplicativity ratios (i.e., the endogenous variables) for the hip, knee, and ankle (left, middle and right, respectively). Solid arrows represent positive relationships found among joints on both legs. Dashed arrows represent positive relationships found among joints on one leg. This schematic does not contain information about multiple subsequent trials (see Figures 6 and 7) but serves to illustrate the case that relative multiplicativity at most joints (all with the exception of the right hip) served to promote later relative multiplicativity.

Appendix

Vector autoregressive (VAR) modeling is a multivariate analysis that tests for interaction among cotemporaneous dependent variables. Because our VAR modeling tested mutual effects among relative multiplicative ratios at three different joints (i.e., hip, knee, and ankle), we plan to use VAR modeling with three dependent measures. So, the following examples describe VAR relationships among three variables r, s, and v. VAR modeling proceeds from the assumption that each individual variable r, s, and v is lag-m autoregressive, that is,

$$\begin{aligned} r(t) &= A_1 r(t-1) + \ldots + A_m r(t-m) + \varepsilon_r \\ s(t) &= B_1 s(t-1) + \ldots + B_m s(t-m) + \varepsilon_s \\ v(t) &= C_1 v(t-1) + \ldots + C_m v(t-m) + \varepsilon_v, \end{aligned} \qquad (9)$$

where each ε indicates independently and identically (i.i.d.) Gaussian residuals, and where A, B, and C are coefficients indicating the contribution of previous values to current values. Coefficients with subscript k, varying from 1 to m, indicates the contribution of previous values at k lags to the current value. VAR modeling tests mutual effects between each pair of variables above and beyond

autoregressive effects within each variable. The first typical step in VAR modeling is a lag-1 test of mutual effects, as in

$$\begin{aligned} r(t) &= A_{r1}r(t-1) + B_{r1}s(t-1) + C_{r1}v(t-1) + \varepsilon_r \\ s(t) &= B_{s1}s(t-1) + A_{s1}r(t-1) + C_{s1}v(t-1) + \varepsilon_s \\ v(t) &= C_{v1}v(t-1) + A_{v1}r(t-1) + B_{v1}s(t-1) + \varepsilon_v, \end{aligned} \qquad (10)$$

and lags may be increased until the residual terms meet the i.i.d. assumptions. As in Eq. 9, coefficients A, B, and C describe contribution of past values to current values of r, s, and v. However, the subscripts differ from Eq. 9: subscripts now index both the variable for which they indicate an autoregressive contribution to current values and the number of lags at which these contributions occur. That is, coefficients with subscript xk indicate the coefficient of a given previous variable at lag-k to current values of x. Although Eq. 10 only describes endogenous effects among r, s, and v, it is possible also to control for effects exogenous to the original endogenous variables. For example, r, s, and v might represent such interrelated econometric variables as production, employment, and consumer deman. Above and beyond endogenous effects among these interrelated variables, effects exogenous to the economy (e.g., a hurricane h or a war w impacting supply or operation of production plants) might contribute independent effects. The resulting model would expand Eq. 10 as follows:

$$\begin{aligned} r(t) &= A_{r1}r(t-1) + B_{r1}s(t-1) + C_{r1}v(t-1) + D_r h(t) + G_r w(t) + \varepsilon_r \\ s(t) &= B_{s1}s(t-1) + A_{s1}r(t-1) + C_{s1}v(t-1) + D_s h(t) + G_s w(t) + \varepsilon_s \\ v(t) &= C_{v1}v(t-1) + A_{v1}r(t-1) + B_{v1}s(t-1) + D_v h(t) + G_v w(t) + \varepsilon_v, \end{aligned} \qquad (11)$$

where D and G are coefficients indicating the contribution of exogenous variables h and w, respectively. Exogenous variables are typically expected to have concurrent rather than autoregressive effects on endogenous variables, and so their coefficients only need on subscript x denoting the endogenous variable to which they indicate a contribution from the exogenous variable.

Provided that the i.i.d. assumptions are met, it is possible to model the independent contributions of each endogenous variable to the forecasted values of each other endogenous variable. Following orthogonalization of the individual equations by Cholesky decomposition, these independent contributions find articulation in impulse response functions (IRFs) that illustrate the change in predicted value for each jth endogenous variable when an ith endogenous variable

increase by one standard error ε_i. That is, IRFs can express each endogenous variable's response to an impulse from specifically one or another of the endogenous variables. The VAR relationship presumes that each endogenous variable is contingent on a combination of effects from the endogenous variables. IRFs help to tease apart the unique contributions of each endogenous variable in change each other endogenous variable (or itself). Orthogonalizing the equations in Eq. 11 emphasizes their independent contributions to overall variance. Adding 1 to any one of the endogenous variables may produce an effect that spreads to any of the endogenous variables, producing significant change in the later predicted values (Lütkepohl, 2005; Sims, 1980).

Endogenous variables in the present report included the relative multiplicativity ratios for each of three joints. As noted in the main text, exogenous variables included dummy-coded variables for each infant and a trial variable. The dummy codes may be described as follows: each infant was assigned his/her own dummy-coded variable, equaling zero for all trials that the infant did not contribute and equaling 1 for all trials that the infant did contribute. These dummy-coded variables controlled for mean differences by infant on otherwise generic VAR relationships; the trial variable simply controlled for any effects of each infant's continued experience under the conditions of measurement (e.g., fatigue).

We ran VAR models to predict current relative multiplicativity ratios $M_{Hip}(t)$, $M_{Knee}(t)$, and $M_{Ankle}(t)$ each in terms of previous (i.e., lag-1) values of all three relative multiplicativity ratios $M_{Hip}(t\text{-}1)$, $M_{Knee}(t\text{-}1)$, and $M_{Ankle}(t\text{-}1)$ and also in terms of current values of trial T and the dummy-coded variables for individual infant I. Hence, to compare this modeling to Eq. 11, the models for either the left side or the right side could be expressed as follows:

$$\begin{aligned} M_{Hip}(t) &= a_{H1}M_{Hip}(t-1) + b_{H1}M_{Knee}(t-1) + c_{H1}M_{Ankle}(t-1) + d_H T(t) + g_H I(t) + \varepsilon_{M_{Hip}} \\ M_{Knee}(t) &= b_{K1}M_{Knee}(t-1) + a_{K1}M_{Hip}(t-1) + c_{K1}M_{Ankle}(t-1) + d_K T(t) + g_K I(t) + \varepsilon_{M_{Knee}} \\ M_{Ankle}(t) &= c_{A1}M_{Ankle}(t-1) + a_{A1}M_{Hip}(t-1) + b_{A1}M_{Knee}(t-1) + d_A T(t) + g_A I(t) + \varepsilon_{M_{Ankle}}. \end{aligned} \quad (12)$$

Tests for serial dependence, heteroscedasticity, and non-normality of the residuals of the lag-1 VAR models for the left side and the right side returned nonsignificant ($p > .10$) test statistics, indicating that the lag-1 VAR models met the i.i.d. assumptions.