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When human walking becomes random walking: fractal analysis and modeling of gait rhythm fluctuations

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Abstract

We present a random walk, fractal analysis of the stride-to-stride fluctuations in the human gait rhythm. The gait of healthy young adults is scale-free with long-range correlations extending over hundreds of strides. This fractal scaling changes characteristically with maturation in children and older adults and becomes almost completely uncorrelated with certain neurologic diseases. Stochastic modeling of the gait rhythm dynamics, based on transitions between different "neural centers", reproduces distinctive statistical properties of the gait pattern. By tuning one model parameter, the hopping (transition) range, the model can describe alterations in gait dynamics from childhood to adulthood—including a decrease in the correlation and volatility exponents with maturation. © 2001 Published by Elsevier Science B.V.

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1. Fractal scaling of healthy gait

What does human walking have to do with fractals? During gait, the locomotor system moves the body, one stride after the next, in an apparently regular fashion. Statistical physics typically deals with phase transitions, fluctuations, and interactions that occur at the microscopic level. Here we briefly describe our investigations of the subtle stride-to-stride fluctuations in gait and demonstrate the strong connection between human walking and random walks.[1] These investigations provide insight into the neural control of locomotion as well as its changes with aging and disease.

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[1] The present description is based upon Refs. [3–6,15,25]. For more details, see those references.

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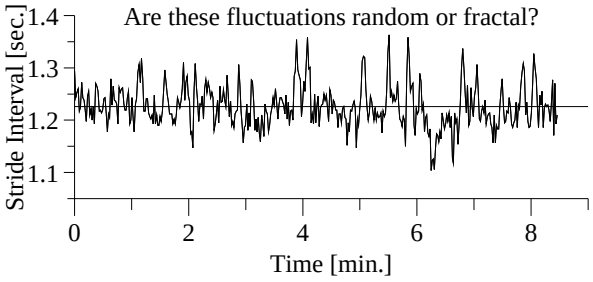

Fig. 1. Stride interval time series of a healthy subject during a walk with constant environmental conditions. While the stride interval appears to be fairly constant, it fluctuates about its mean (the solid line) in an apparently unpredictable manner. The stride interval is a measure of the gait rhythm and is typically defined as the time from heel strike (initial contact) to heel strike of the same foot.

At first inspection, walking appears to be a periodic, regular process. As illustrated in Fig. 1, however, closer examination reveals small fluctuations in the gait pattern, even under stationary conditions [1–6]. One possible explanation for these stride-to-stride variations in the walking rhythm is that they simply represent uncorrelated (white) noise superimposed on a basically regular process. This is what one might expect a priori if one assumes that these subtle fluctuations are merely "noise". A second possibility is that there are finite-range correlations: the current value is influenced by only the most recent stride intervals, but over the long term, fluctuations are random. A third, less intuitive possibility is that the fluctuations in the stride interval exhibit long-range correlations, as seen in a wide class of scale-free phenomena [7–12]. In this case, the stride interval at any instant would depend (at least in a statistical sense) on the interval at relatively remote times, and this dependence would decay in a scale-free (fractal-like), power-law fashion.

To answer this question, we first measured the stride interval in 10 young, healthy men [3–6]. Subjects walked continuously on level ground around an obstacle free, 130 m long, approximately circular path at their self-determined, usual rate for about 9 min. To measure the stride interval, the output of ultra-thin, force sensitive switches was recorded on an ambulatory recorder and heelstrike timing was automatically determined. For a group of ten healthy, young adults, we find long-range correlations with scaling exponent of $\alpha = 0.76 \pm 0.11$ (mean $\pm$ standard deviation) for the original stride interval time series and, after random shuffling, uncorrelated behavior with scaling exponent $\alpha = 0.50 \pm 0.03$; we use the detrended fluctuation analysis (DFA method) for the scaling analysis. Similar results were observed for β, the slope of the line fitted to the Fourier power spectrum. Thus, α and β ($\beta = 2\alpha - 1$) both indicate the presence of long-range correlations and a fractal gait rhythm.

To study the stability and extent of these long-range correlations, we asked 10 young (ages 18–29 years), healthy men to walk for 1 h at their usual, slow and fast paces around an outdoor track. A representative example of the effect of walking rate on the stride interval fluctuations and long-range correlations is shown in Fig. 2. The locomotor

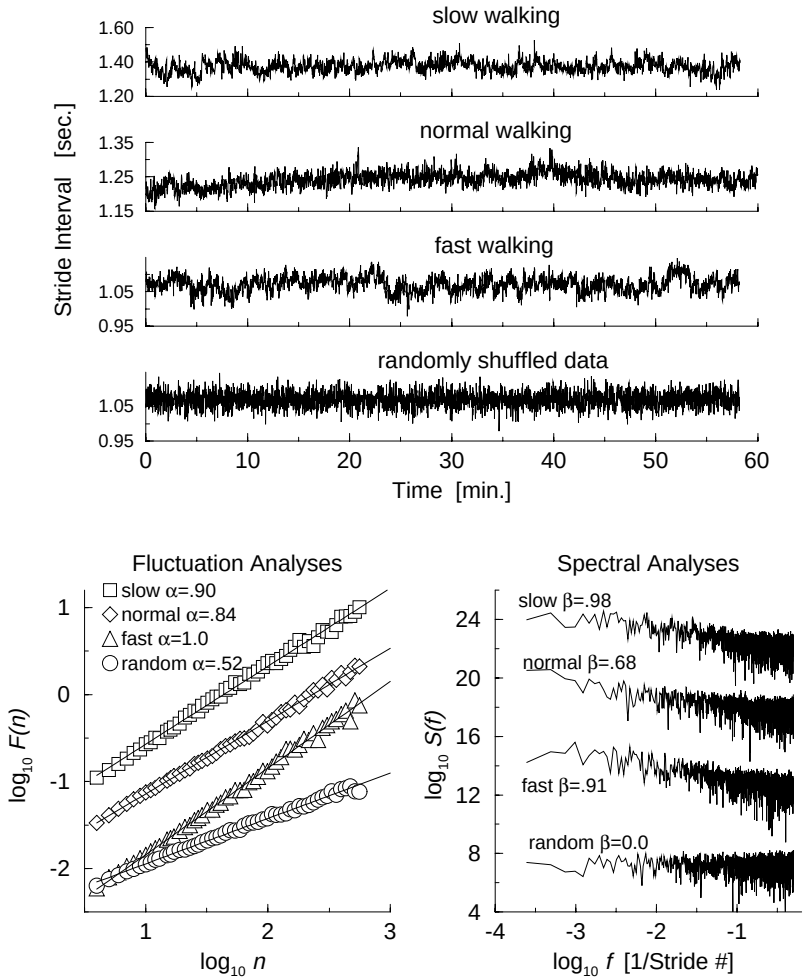

Fig. 2. An example of the effects of walking rate on stride interval dynamics. (Top) One hour stride interval time series for slow (1.0 m/s), normal (1.3 m/s) and fast (1.7 m/s) walking rates. Note the breakdown of structure with random re-ordering or shuffling of the fast walking trial data points, even though this shuffled time series has the same histogram of strides intervals (with the same mean and standard deviation) as the original, fast time series. (Bottom) Fluctuation and power spectrum analyses confirm the presence of long-range correlations at all three walking speeds and its absence with random shuffling of the data. $F(n)$ is the fluctuation size at a given window size, n [9].

control system maintains the stride interval at an almost constant level throughout the one hour of walking (the coefficient of variation was less than 3%). Nevertheless, the stride interval fluctuates about its mean value in a highly complex, seemingly random fashion. However, both DFA and power spectral analysis indicate that these variations in walking rhythm are not random. Instead, the time series exhibit long-range correlations at all three walking rates. The scaling indices α and β remained fairly constant despite substantial changes in walking velocity and mean stride interval.

Consistent results were obtained for all 10 subjects. For all thirty 1 h trials, α was 0.95 ± 0.06 (range: 0.84 to 1.10). Similar results were also observed for the power spectrum scaling exponent β (e.g., for all thirty trials, β was 0.93 ± 0.13). Thus, for all subjects tested at all three rates, *the stride interval time series displayed long-range (fractal-like) correlations over thousands of strides.*

To investigate further the possible mechanisms of this fractal gait rhythm, all 10 subjects were studied under three additional conditions. Subjects were asked to walk in time to a metronome that was set to each subject's mean stride interval (computed from each of the three unconstrained walks). The results during metronomic walking were completely different from those obtained when the walking rhythm was unconstrained. During metronomically-paced walking, fluctuations in the stride interval were, surprisingly, *anti-correlated* in most of the 30 walking trials (the average scaling exponent $\pm$ standard deviation are: total: 0.26 ± 0.24 normal walking: 0.3 ± 0.24 slow walking: 0.2 ± 0.26 fast walking: 0.28 ± 0.22). [2]

These findings indicate that the fractal dynamics of walking rhythm are normally quite robust and intrinsic to the locomotor system. The breakdown of long-range correlations during metronomically-paced walking demonstrates that supra-spinal influences (a metronome) can override the normally present long-range correlations. Since metronomic and free walking utilize the same lower motor neurons, actuators, and feedback, one might speculate further that supra-spinal control (e.g., the brain) is critical in generating these long-range correlations.

2. Changes in fractal dynamics with aging and Huntington's disease

To gain further insight into the basis for this long-term, fractal dependence in walking rhythm, we investigate the effects of advanced age and Huntington's disease, a neurodegenerative disorder of the central nervous system, on stride interval correlations. Using DFA, we compared the stride interval time series (i) of healthy elderly subjects ($n = 10$) and healthy young adults ($n = 22$), and (ii) of subjects with Huntington's disease, ($n = 17$) and healthy controls ($n = 10$).

Fig. 3 compares the stride interval time series for a young and an elderly adult. Visual inspection suggests a possible subtle difference in the dynamics of the two time series. Fluctuation analysis reveals a marked distinction in how the fluctuations change with time scale for these subjects. The slope of the line relating $\log F(n)$ to $\log n$ is less steep and closer to 0.5 (uncorrelated, white noise) for the elderly subject. This indicates that the stride interval fluctuations are more random and less correlated for the elderly subject than for the young subject. Similar results were obtained for other

[2] We find this anti-correlated behavior after integrating the stride interval series and subtracting one from the scaling exponent. This integration procedure is consistent with Ref. [24]. Analysis of the stride interval series without integration yielded uncorrelated random behavior [3–6].

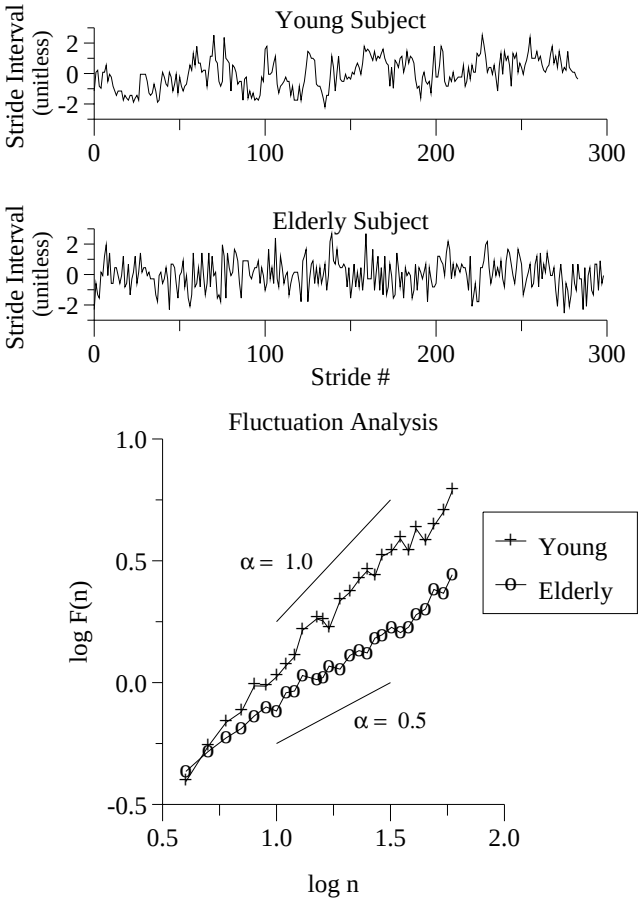

Fig. 3. Example of the effects of aging on the fluctuation analysis of stride interval dynamics. Stride interval time series are shown above and fluctuation analysis below for a 71 year old elderly subject and a 23 year old, young subject. For illustrative purposes, each time series is normalized by subtracting its mean and dividing by its standard deviation. For the elderly subject, fluctuation analysis indicates a more random and less correlated time series. Indeed, α is 0.56 ($\approx$ white noise) for the elderly subject and 1.04 ($\approx 1/f$ noise) for this young subject.

subjects in these groups as well. α was 0.68 ± 0.14 for the elderly subjects versus 0.87 ± 0.15 in the young subjects ($p < 0.003$).

Interestingly, although the correlation properties of stride interval were different in the elderly and young adults, the first moment, the average stride interval, was similar in both groups (elderly: 1.05 ± 0.10 s; young: 1.05 ± 0.07 s). The magnitude of stride-to-stride variability (i.e., stride interval coefficient of variation) was also very similar in the two groups (elderly: $2.0 \pm 0.7\%$; young: $1.9 \pm 0.4\%$). These results show that while α was different in the two age groups, the usual measures of gait and mobility function of these elderly subjects were not significantly affected by age.

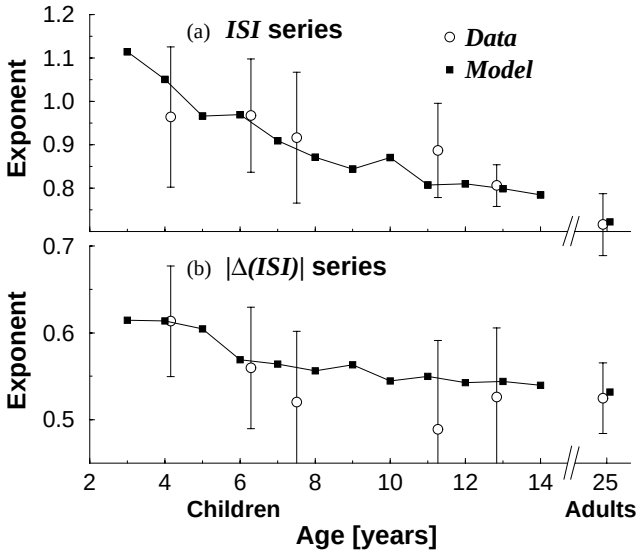

Fig. 4. The scaling exponents of the stride interval series of 50 children between 3 and 13 years old [22,23] and a neural hopping model (see below). To study the effects of maturation, we divide the children into 5 age groups: (i) 3–4 year olds (11 subjects), (ii) 5–6 year olds (10 subjects), (iii) 7–8 year olds (14 subjects), (iv) 10–11 year olds (10 subjects), and (v) 12–13 year olds (5 subjects). We also show data [3–6] from an adult group (10 subjects 1 h long each; ages 20–30 years). For the model simulation, we generate 40 realizations for each value of C; the average value is presented. The age axis for the model follows the relation: age (years) $= C + 2$. (a) The short-range scaling exponents of the original time series both for the data (open circles) and the model (black squares). The exponents calculated for window sizes $6 < n < 13$ strides, decrease with age [3–6]. The scaling exponent obtained by the model decreases monotonically as C increases and is within the error bars of the data. (b) The scaling exponent of the stride interval magnitude series, $|\Delta(stride interval)_i|$. The magnitude scaling exponent decreases with age, indicating a loss of magnitude correlations with maturation. The model exhibits a similar decrease and the simulation is within the error bars of the actual data. The subject-to-subject variability is consistent with the scatter observed in physiologic indices of neural development [13,14].

The scaling exponent α was also reduced in the subjects with Huntington's disease compared to disease-free controls (Huntington's disease: 0.60 ± 0.24; controls: 0.88 ± 0.17; $p < 0.005$). Moreover, among the subjects with Huntington's disease, α was related to degree of functional impairment ($r = 0.78$, $p < 0.0005$), such that the stride-to-stride fluctuations become completely uncorrelated in patients with more advanced disease.

In older adults, we observed a decrease in the fractal scaling exponents. On the other end of the age spectrum, the scaling properties also change as children mature. In contrast to what was observed among older adults, the short-range scaling exponents of young children are *larger* than that seen in healthy young adults and decrease, becoming more adult-like, as children mature [3–6]. We compare the short range scaling exponents for a group of 50 children [22,23] with those of 10 adults [3–6]; this exponent decreases from ~ 1.0 to ~ 0.7 (Fig. 4a).

The magnitude series exponent, a measure of volatility [20],[3] of the stride interval series also decreases with maturation (Fig. 4b). These results suggest that gait pattern of children is more volatile than the usual walking pattern of healthy, young adults.

3. Modeling the fractal gait rhythm

To investigate the fractal gait rhythm and the mechanisms that might account for it, we attempted to simulate the experimental results. Deterministic and stochastic models have been proposed to understand the underlying regulatory mechanisms of walking. For example, classic "central pattern generator" (CPG) models are based on oscillatory neural activity, where the interaction between neural centers helps regulate gait dynamics [16–19]. These models, however, do not reproduce the observed fractal scaling. A stochastic version of a central pattern generator model generates a fractal stride interval time series, like that seen in healthy young adults [3–6]. However, existing models do not explain observed changes in scaling exponents [3–6], and volatility (magnitude) correlations [20] (see above) that occur during gait maturation from childhood to adulthood.

We propose a stochastic model consisting of a random walk (RW) on a chain, the elements of which represent excitable neural centers [13]. A step of the RW between element i and element j represents the "hopping" of the excitation from center i to center j. The increase of neural interconnectedness with maturation is modeled by increasing the range of "jump" sizes of the RW, since larger jump sizes will allow exploration of more neural centers. This property mimics one aspect of the increasing complexity of the adult nervous system.

Previous studies [21] have identified neural centers with pacemaker-like qualities that fire with frequency f_i, so we represent the network of neural centers by different frequency modes. One mode is activated at a given time ($stride interval \propto 1/f_i$), and the f_i are Gaussian distributed. The model is based on the following assumptions (Fig. 5): Assumption (i) is that the f_i have finite-size correlations, $\langle f_i f_{i+\delta}\rangle / \langle f_i^2\rangle = \mathrm{e}^{-\delta/\delta_0}$. We assume finite-range correlations among f_i because neighboring neurons are likely to be influenced by similar factors [15]. This assumption effectively creates "neuronal zones" composed of neural centers (modes) along the chain with a typical size δ_0. Assumption (ii) concerns the rule followed by the RW process. The active neural center is determined by the location of the RW. The "jump" sizes of the RW follow a Gaussian distribution of width C. Assumption (iii) is that a small fraction of noise is added to the output of each mode to mimic biological noise not otherwise modeled. The output y becomes $y(1 + A\eta)$ where A is the noise level and η is Gaussian white noise with zero mean and unit variance.

[3] Other studies define volatility as the local variance of the signal. Here by volatility correlations we mean correlation in the magnitudes of the series increments.

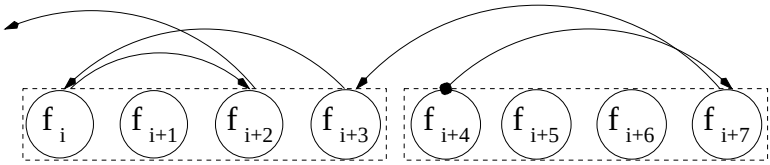

Fig. 5. Illustration of the "neural" hopping model. The values of f_i are not uncorrelated but rather have finite size correlations. Shown is a sequence of four transitions, from mode f_{i+4} to f_{i+7} to f_{i+3} to f_i to $f_{i+2}\ldots$. Larger values of the hopping-range parameter C are associated with larger "jump sizes" along the chain. The neuronal zone of size $\delta_0 = 4$ is indicated by the dashed boxes.

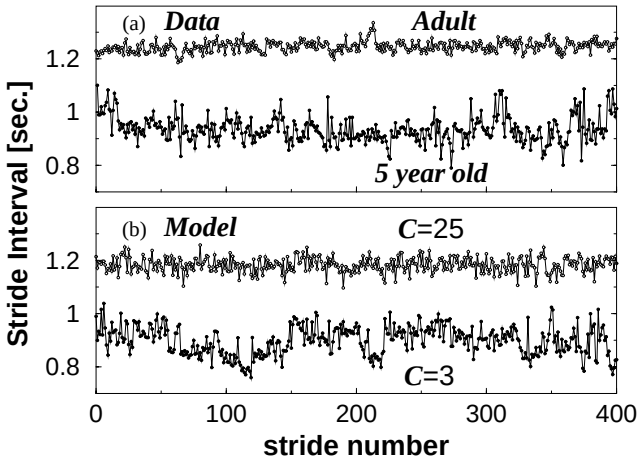

Fig. 6. (a) Examples of stride-interval series of healthy subjects, ages 5 and 25 years. (b) Examples of stride-interval generated by the model. Iterating the model with a small value of the hopping-range parameter ($C = 3$) mimics the stride-interval of young children, while a large value ($C = 25$) mimics that of adults.

The model has three parameters δ_0, C, and A. We find that the best agreement with the data is achieved when $A = 0.02$ and $\delta_0 = 25$. In order to simulate changes with maturation, we vary only the third parameter, C, as a function of age, $C = (\text{age} - 2)$ for ages 3–25 years (see Fig. 6 for visual comparison between the data and the model's output). Increasing the hopping range with age is consistent with the fact that neural transmission is not fully developed until the late teens [6].

Briefly, we find that this simple stochastic model captures multiple aspects of gait dynamics and their changes with maturation (see Fig. 4), including: (i) the shape of the probability distribution of the stride interval increments; (ii) correlation properties of the stride interval; and (iii) correlations properties in the magnitudes of the stride interval increments. Further, by varying only a single "hopping-range" parameter, C, a wide array of multifractal dynamics can be generated. The model can also be altered by "knocking out" certain frequency modes (akin to what may occur during very advanced age or in response to neurodegenerative disease). Simulation with drop-out of frequency modes predicts increased gait variability, with (i) increased magnitude

exponents, and (ii) decrease of long-range correlations. Our preliminary analysis of the stride interval series of older adults prone to falls is consistent with this prediction. Generalization of the model to two and three dimensional networks to describe other types of neurological activities is underway.

4. Discussion

Our findings demonstrate that the stride interval time series exhibits long-range, self-similar correlations. This fractal scaling is apparently an intrinsic feature of normal walking rhythm. Scaling exponents obtained using two complementary methods, fluctuation analysis and Fourier analysis, both indicate the presence of long-range, power-law correlations in the gait rhythm during slow, normal and fast walking. With random shuffling of the stride interval, the scaling exponents change to that of an uncorrelated random process. Thus, stride interval fluctuations are not random like white noise, nor are they the outcome of a process with short-term correlations. Instead, the present stride interval is related to the interval thousand of strides earlier and this scaling occurs in a scale-invariant, fractal-like manner.

The presence of a fractal gait rhythm is notable for several reasons: (i) It suggests the presence of a non-trivial long-term dependence ("memory" effect). (ii) Such fluctuations are often associated with a non-equilibrium dynamical system with multiple-degrees-of-freedom, rather than being the output of a classical "homeostatic" process. (iii) Models of the neural basis of rhythmic motor acts (e.g. CPG's) need to be re-examined to account for this previously unanticipated fractal scaling property. (iv) The finding of reduced stride interval correlations with aging and with Huntington's disease parallels the effects of age and disease on the fractal scaling of other processes under neural control [10–12].

Precise elucidation of the factors affecting the fractal dynamics of gait remains to be determined. Nevertheless, we can begin to form an idea of what contributes to this behavior. The drastic change of long-range correlations to long-range anti-correlations during metronomically-paced walking in the same neuro-mechanical system that produces this fractal behavior during normal walking (i.e., in healthy young subjects) suggests: (i) that supra-spinal influences can override the normally present fractal rhythm, and (ii) that this behavior is not simply a result of the neuro-mechanical interaction of a highly complex system.

The alterations in the fractal dynamics of the stride interval with advanced age and Huntington's disease provide additional evidence. Changes in the fractal rhythm in these populations are not simply attributable to reduced gait speed or increased stride-to-stride variability with aging or disease. When healthy young adults walk slowly, the fractal rhythm is not reduced. Moreover, the magnitude of the stride interval correlations was independent of gait speed and stride-to-stride variability. Apparently, stride interval correlations depend on some aspect of the neuro-muscular control system that is not directly related to walking velocity or gait unsteadiness. The stochastic, neural hopping

model suggests that perhaps changes in connectivity and the ability of neurons to communicate with one another contribute to the observed scaling changes in aging and disease. Further investigations will help to elucidate under what conditions and why scale-free human walking becomes a non-correlated random walk.

Acknowledgements

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