Published in final edited form as:

Gait Posture. 2010 July ; 32(3): 348–353. doi:10.1016/j.gaitpost.2010.06.004.

Re-Interpreting Detrended Fluctuation Analyses of Stride-To-Stride Variability in Human Walking

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Abstract

Detrended fluctuation analyses (DFA) have been widely used to quantify stride-to-stride temporal correlations in human walking. However, significant questions remain about how to properly interpret these statistical properties physiologically. Here, we propose a simpler and more parsimonious interpretation than previously suggested. Seventeen young healthy adults walked on a motorized treadmill at each of 5 speeds. Time series of consecutive stride lengths (SL) and stride times (ST) were recorded. Time series of stride speeds were computed as $SS = SL/ST$. SL and ST exhibited strong statistical persistence ($\alpha \gg 0.5$). However, SS consistently exhibited slightly *anti*-persistent ($\alpha < 0.5$) dynamics. We created three surrogate data sets to directly test specific hypotheses about possible control processes that might have generated these time series. Subjects did not choose consecutive SL and ST according to either independently uncorrelated or statistically independent auto-regressive moving-average (ARMA) processes. However, *cross-correlated* surrogates, which preserved both the auto-correlation and cross-correlation properties of the original SL and ST time series successfully replicated the means, standard deviations, and (within computational limits) DFA α exponents of all relevant gait variables. These results suggested that subjects controlled their movements according to a two-dimensional ARMA process that specifically sought to minimize stride-to-stride variations in walking speed (SS). This interpretation fully agrees with experimental findings and also with the basic definitions of statistical persistence and anti-persistence. Our findings emphasize the necessity of interpreting DFA α exponents within the context of the control processes involved and the inherent biomechanical and neuro-motor redundancies available.

Keywords

Walking; Stride Parameters; Variability; 1/f Noise; Detrended Fluctuation Analysis

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CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

1. INTRODUCTION

Multiple papers describe finding long-range correlations in human walking time series, particularly stride times [1–10]. Such findings imply that each stride depends explicitly on many previous strides by exhibiting stride-to-stride correlations that “decay in a scale-free (fractal-like) power-law fashion” [10]. Most of these conclusions were based on Detrended Fluctuation Analysis (DFA) [10,11]. However, DFA is highly sensitive to yielding false positive results [2,12]. Many *non*-long-range correlated processes yield equivalent results using DFA [12–17]. While more sophisticated analyses (e.g., [2,18]) may better identify the true statistical properties of these signals, one cannot conclude the presence of long-range correlations from DFA alone [2,12,16]. Nevertheless, DFA still provides a valid indicator of the statistical “persistence” or “anti-persistence” in a time series, regardless of the underlying origin [12]. “Persistence” means that deviations in a time series are statistically more likely to be followed by subsequent deviations in the *same* direction (i.e., they “persist” across subsequent data points). “Anti-persistence” means that deviations in one direction are statistically more likely to be followed by subsequent deviations in the *opposite* direction.

Given that human walking [1,4,8–10,19] exhibits statistical persistence, the question becomes what does this mean physiologically? Some authors have argued that statistical persistence reflects an inherently “healthy” system and that statistically uncorrelated or anti-persistent dynamics indicates disease or pathology [6,8,20]. Such conclusions are bolstered by findings that elderly subjects and patients with various central nervous system disorders lose the statistical persistence in their stride times [3,5,8,21–23]. Conversely, however, patients with severe diabetic peripheral neuropathy exhibit *no* such changes [24], despite the fact that peripheral sensory feedback likely plays a significant role in regulating gait cycle timing [25] and variability [26].

A recent computational model of walking dynamics demonstrated that even simple changes in the amplitude of sensory and/or motor noise can yield time series of stride times with a wide range of statistical correlation properties, with no changes in control architecture [27]. This model incorporated a controller that made explicit reference to only *one* single stride in the past. Similar observations were obtained from a model of standing posture [28]. Both models assumed sensory and motor processes were influenced by only white noise [27,28], and thus provide mechanistic explanations for how complex-looking kinematics can arise in the *output* of a neuro-mechanical system, even when neuro-motor *input* fluctuations are Gaussian and white. In the walking model, changes in statistical persistence of stride times due to aging or neuro-degenerative disease could be explained as reflecting changes in neuronal input noise [27].

In a different context, when healthy humans walk over ground in time with a metronome, their stride times become less correlated [9] or anti-persistent [2,4]. It was hypothesized that supraspinal mechanisms “override” normally statistically persistent locomotor behavior [3,9]. A “Super CPG” model [29,30], which naturally produced long-range correlated stride times, replicated this phenomena when driven by periodic external forcing [29]. However, slightly altering this same model’s structure produced these same changes without external forcing [2]. This same phenomenon can also be replicated by changing the feedback gain in a different neuro-mechanical model of walking that only corrects deviations in motor output from *one* previous stride [27]. Most importantly, the *cause* of this loss of persistence here is clearly not “pathological.” Indeed, the notion that loss of correlated structure in metronomic walking is due to *enhanced* supraspinal control [2–4,9,29,30] seems to contradict the idea that the same observed changes are due to *degraded* supraspinal control in patients with central nervous system disease [3,6,8,20,21].

Additionally, none of these models or theories account for the observation that during metronomic walking, while stride *times* change from persistent to anti-persistent, stride *lengths* and stride-to-stride *speeds* remain strongly persistent [4]. Here, we propose a more parsimonious interpretation of these statistical phenomena: namely, output variables that are *not* tightly regulated will exhibit statistical persistence (i.e., small deviations are allowed to persist across multiple consecutive strides), while tightly controlled variables will be uncorrelated or anti-persistent (i.e., small deviations are immediately corrected on subsequent strides). Walking in time with a metronome requires tightly controlling stride time [4]. However, many combinations of stride length and stride speed achieve the exact same stride time, so these parameters do not need to be tightly controlled independently. Walking on a motorized treadmill similarly requires maintaining (on *average*) the same walking speed (i.e., to avoid walking off the treadmill). We hypothesized that in this context, healthy subjects would exhibit significant statistical persistence of both their stride times and stride lengths [1,19], but would tightly regulate their stride-to-stride walking *speed*.

2. METHODS

Seventeen young healthy adults (12M/5F, age 18–28, height 1.73 ± 0.09 m, body mass 71.11 ± 9.86 kg), participated after providing institutionally approved written informed consent. Subjects were excluded if they reported any orthopedic problems or recent lower extremity injuries, exhibited any visible gait anomalies, or were taking medications that may have influenced their walking.

Subjects walked on a level motor-driven treadmill (Desmo S, Woodway USA, Waukesha WI) and wore a safety harness (Protecta International, Houston TX) that allowed natural arm swing [31]. Preferred walking speed (PWS) was determined using a protocol [32] that also allowed for treadmill acclimation and warm-up. Following a 2-minute rest, subjects completed two 5-minute walking trials at each of 5 speeds (80% to 120% of PWS) in pseudo-random order [31]. Subjects rested at least 2 minutes between trials. One trial from each of 4 subjects was discarded due to poor data quality. The remaining 166 trials analyzed ranged from 213 and 334 strides (mean 272 ± 25 strides).

DFA analyses are often conducted on longer time series. When analyzing shorter time series, the trial-to-trial *variance* of the estimates of the DFA scaling exponent, α , increases approximately exponentially [33–35]. However, this does not bias the estimated *mean* value of α [33–35], even for time series as short as 64 samples [35]. We therefore mitigated any effects of increased variance from using shorter time series by averaging DFA α estimates across multiple trials. This was also demonstrated specifically for walking [36].

Movements of five 14-mm markers mounted to each shoe (2nd phalanx, 5th metatarsal, dorsum of the foot, inferior to the fibula, and calcaneus) were recorded continuously at 60 Hz using an 8-camera Vicon motion capture system (Oxford Metrics, UK). All data were processed using MATLAB (Mathworks, Natick MA). Marker trajectories were low-pass filtered at 10 Hz with a zero-lag Butterworth filter. Heel strike was defined from the maximum forward displacement of the heel marker during each stride. Stride times (ST) were calculated as the time interval between consecutive ipsilateral heel contacts. Step lengths were defined as the anterior-posterior distance between the two contralateral heels at each heel contact. Stride length (SL) was calculated by adding the 2 consecutive step lengths composing each stride. Stride speeds (SS) for each stride were then calculated as $SS = SL/ST$.

Each time series (SL, ST, and SS) was analyzed using Detrended Fluctuation Analysis (DFA) [6,10,11,20], following standard recommendations [24,27]. Details of this algorithm are published elsewhere [6,10,11,20]. Briefly, DFA computes mean square roots of detrended

residuals, $F(n)$, of the integrated time series over a range of segment lengths, n . Here, 50 values of n , evenly distributed between 4 and $N/4$, and linear detrending were used [24,27]. Linear slopes of plots of $\log[F(n)]$ versus $\log(n)$ define the DFA scaling exponent, α [10,11,20]. $\alpha = 0.5$ indicates uncorrelated white noise. $\alpha < 0.5$ indicates anti-persistence: deviations in one direction are more likely to be immediately followed by corrections in the *opposite* direction, consistent with a tightly controlled process. $\alpha > 0.5$ indicates statistical persistence: deviations in one direction are more likely to be followed by deviations in the *same* direction, consistent with a weakly controlled process.

We then analyzed three types of constrained surrogate time series [37,38] to determine how well relatively simple random processes might account for the statistical properties of the experimental data. First, we generated *randomly shuffled* surrogates by taking each original ST and SL time series and independently shuffling the values in random order [10,24,37]. These surrogates tested the hypothesis that subjects controlled their movements by choosing ST and SL that were independent of each other and temporally independent from previous strides.

Second, we generated *phase-randomized* surrogates [37–39] independently for ST and SL. For each time series, we computed the Fourier transform, randomized the phase spectrum, and then computed the inverse Fourier transform [37–39]. These surrogates were designed to preserve the power spectra and autocorrelation properties of each original time series, thus preserving their statistical persistence. These surrogates tested the hypothesis that subjects independently chose their ST and SL as temporally correlated auto-regressive (AR) processes [37,38], or more generally as a nearly equivalent auto-regressive moving average (ARMA) processes (see [37], pg. 81).

Finally, we generated *cross-correlated* phase-randomized surrogates [38,40] *jointly* for the original ST and SL time series from each trial. Here, we simultaneously randomized the phase spectra of both time series the exact same way [40]. These surrogates thus preserved both the *auto*-correlations of ST and SL individually, and also the *cross*-correlations between them. These surrogates tested the hypothesis that subjects simultaneously regulated both SL *and* ST according to a two-dimensional ARMA process [38,40], consistent with adopting a controller that sought to minimize stride-to-stride variations in walking speed (SS).

For each experimental trial, 20 valid surrogates were generated by ensuring the maximum net cumulative distance walked did not exceed the treadmill belt limits (i.e., ± 0.864 m from the center). Surrogates exceeding these limits were discarded so only *valid* surrogates were analyzed. For each valid surrogate, we computed stride speed (SS) time series as $SS = SL/ST$. DFA α exponents were computed for each surrogate time series. For each trial, α was averaged across all 20 surrogates and extracted for statistical analyses. We chose 20 surrogates as sufficient to capture the distribution of α estimates for each set of surrogates [37].

Data were subjected to a 3-factor (Type $\times$ Speed $\times$ Subject) repeated measures, general linear model analysis of variance (ANOVA). Type (Original vs. Surrogate) and Speed (80% –120% of PWS) were fixed factors. Subject was a random factor. Because these surrogates are specifically designed to be random, they should be less (not more) correlated than the original time series. Therefore, we applied a one-sided statistical test [38,41] to determine main effects differences for Type. All statistical analyses were performed in Minitab 15 (Minitab, Inc., State College, PA).

3. RESULTS

SL and ST (Fig. 1A) time series both exhibited extended phases where consecutive values continued increasing or decreasing across multiple consecutive strides before reversing

direction. Moreover, fluctuations in SL appeared largely positively correlated with concurrent changes in ST. In contrast, fluctuations in SS (Fig. 1A) appeared to reverse direction more rapidly and frequently.

These qualitative observations were confirmed by DFA (Fig. 1B). Consistent with previous findings [1,4,9,10,19,24], SL and ST time series both exhibited strong statistical persistence ($\alpha \gg 0.5$). Conversely, SS time series exhibited consistent and statistically significant *anti*-persistence ($\sim 0.4 < \alpha < 0.5$). Thus, at all walking speeds, deviations in both SL and ST were allowed to persist, whereas deviations in SS were rapidly reversed on subsequent strides.

As expected [10,24], randomly shuffling SL and ST (Fig. 2) eliminated all effects of temporal order, yielding statistically uncorrelated time series ($\alpha \approx 1/2$). Likewise, dividing randomly shuffled SL by ST time series yielded surrogate SS time series that were equally uncorrelated (Fig. 2). Differences between original and surrogate time series were highly statistically significant ($p \ll 0.0001$) for all time series at all walking speeds.

As expected [37], the phase-randomized surrogates (Fig. 3) exhibited strong statistical persistence ($\alpha \gg 1/2$) for both the SL and ST time series. Magnitudes of α for these surrogates were in fact slightly *greater* than for the original data for both SL and ST. These small increases were sufficiently consistent across subjects to be statistically significant for both SL ($p = 0.002$) and ST ($p = 0.001$). Conversely, dividing the phase-randomized SL by ST time series yielded surrogate SS time series that also exhibited strong statistical persistence ($\alpha \gg 1/2$), very different from humans. These differences were highly statistically significant for all SS time series ($p \ll 0.0001$).

Cross-correlated surrogates (Fig. 4) also exhibited strong statistical persistence ($\alpha \gg 1/2$) for both SL and ST time series. Magnitudes of α were again very slightly, but consistently, *greater* for these surrogates for both SL ($p = 0.002$) and ST ($p = 0.001$). However, unlike the phase-randomized surrogates (Fig. 3), accounting for the cross-correlations between SL and ST yielded surrogate SS time series with nearly the same statistical *anti*-persistence ($\alpha < 1/2$) as humans (Fig. 4). Differences between original and cross-correlated SS time series were *not* statistically significant ($p = 0.284$).

The effects of accounting for these different statistical properties of SL and ST time series on the overall control of treadmill walking can be seen by computing maximum distances walked by each surrogate and experimental trial (Fig. 5). These distances quantify how much each trial “wandered” back and forth along the treadmill belt. Only the cross-correlated surrogates (Fig. 5C) exhibited a distribution similar to humans (Fig. 5D).

4. DISCUSSION

There has been great interest in recent years in trying to understand the stride-to-stride correlations observed in human walking [1–10]. However, well-documented limitations of the analytical methods used to assess these statistical properties [2,12–17] raise significant questions about how best to interpret these findings physiologically [4,6,8,20,24,27]. During unconstrained overground walking, stride times, lengths, and speeds all exhibit strong statistical persistence [4]. When these subjects walked in time with a metronome, only stride *times* became anti-persistent [4]. In our study, when subjects walked on a motorized treadmill at constant speed, only stride *speeds* became anti-persistent (Fig. 1B). In each case, the anti-persistent dynamics were exhibited only for that gait variable which required tight control, such that deviations in this variable were followed by rapid corrections. Wherever no such control was required, stride-to-stride deviations went uncorrected and were allowed to persist.

This yields an intuitive and parsimonious interpretation fully consistent with the basic definitions of statistical persistence / anti-persistence [12,14].

The randomly shuffled surrogates (Fig. 2) refute the hypothesis that subjects choose SL and ST that were independent of each other and temporally independent across strides. This was in spite of the fact that this strategy would have been largely successful (Fig. 5A). The phase-randomized surrogates exhibited highly significantly different dynamics than humans for both stride speeds (SS; Fig. 3) and displacements along the treadmill (Fig. 5B). This refuted the alternative hypothesis that subjects choose SL and ST according to statistically independent ARMA processes [37,38].

The phase-randomized and cross-correlated surrogates both exhibited α exponents for SL and ST that were significantly larger than experimental data (Figs. 3–4). For stationary data of infinite length, α is theoretically defined by the slope of the power spectrum plotted on a log-log scale [10,11,24]. Since these surrogates were designed to preserve the power spectra, they should have also yielded the exact same α as their corresponding original time series. They did not because our time series were not infinitely long. This biased the surrogate power spectra towards increased power at low frequencies [38,41], thus generating greater persistence and spurious over-estimates of α . Therefore, these small “statistically significant” differences in the *wrong* direction were *not* grounds to reject the null hypothesis posed by these surrogates [37].

For the cross-correlated surrogates (Fig. 4), the SS time series were statistically indistinguishable from the original data. These surrogates therefore fully replicated the means, standard deviations, and DFA α exponents (within computational limits) of *all* relevant gait variables (SL, ST, and SS), and the displacements along the treadmill (Fig. 5C). These surrogates thus support the hypothesis that subjects controlled their movements by simultaneously regulating both SL *and* ST according to a two-dimensional ARMA process [38,40]. This is fully consistent with the interpretation that subjects specifically sought to minimize stride-to-stride variations in walking speed (SS).

The cross-correlated surrogates (Fig. 4) demonstrate that SL, ST, and SS, were coupled in a specific way that demonstrates redundancy, or equifinality [42,43]. To control SS, subjects did not need to tightly control SL or ST. They needed only to ensure that deviations in either variable were “cancelled out” by concomitant changes in the other. Deviations in both individual variables could be allowed to persist with no consequences for SS. We suggest this is precisely what happens in all three cases of unconstrained walking [4], metronomic walking [4], and treadmill walking (Fig. 1). This structure is exactly compatible with the notion that humans apply a “Minimum Intervention Principle” [43] to regulate walking: i.e., they tightly regulate only those variables directly relevant to achieving the task goal, while largely ignoring fluctuations in *irrelevant* variables [42].

All surrogates tested here were constrained to stay on the treadmill belt. However, removing this constraint (i.e., assuming a treadmill of arbitrary length) yielded DFA results that were nearly identical (both qualitatively and quantitatively) to those presented here. Indeed, even for the *unconstrained* case, fewer than 3% of experimental trials yielded cross-correlated surrogates that exceeded the treadmill limits. This is consistent with the notion that any actual human who walked very close to the edge of the treadmill could easily implement a very small amount of *additional* control to prevent such an event.

It has been widely argued that statistically persistent fluctuations are a critical marker of “healthy” physiological function [6,20] and that uncorrelated or anti-persistent fluctuations indicate disease or pathology [6,8,20]. Our results (and those of [4]) directly contradict this. The subjects tested here clearly cannot be simultaneously both “healthy” (according to $\alpha(\text{SL})$

and $\alpha(\text{ST})$) and “unhealthy” (according to $\alpha(\text{SS})$) (Fig. 1B). The loss of statistical persistence in these cases clearly does *not* reflect any degradation of physiological control, but quite conversely reflects an *increased* control effort to achieve the desired task goal [42]. Thus, our findings argue for interpreting DFA results within the context of the control processes involved and the inherent biomechanical and neuro-motor redundancies available to the system [27, 42,43].

The persistent fluctuations observed in physiological time series of healthy individuals likely result from multiple interactions across multiple time scales of the different control components involved, from the motor cortex, to lower brain centers, spinal networks, and afferent feedback [7,10,29,44]. However, it has been argued that high-level central nervous system processes play a *critical* role in shaping the statistical persistence observed in walking, and that breakdown of these mechanisms causes the loss of this persistence in patients with central nervous system disease [3,5,6,8,22]. The present findings suggest the perfectly viable, if nearly opposite, alternative explanation that these patients may instead be exerting *greater* stride-to-stride control over their stride times (and possibly other gait variables) because they are being more “cautious” [22]. Unfortunately, one cannot conclude from cross-sectional comparisons whether the observed changes in gait dynamics occurred because of the pathology itself, or because patients adapted their walking dynamics in response to their physical and/or physiological limitations.

Finally, both sets of interpretations could be true. That is, the shifts toward less persistent gait dynamics in neurologically impaired patients could indeed be caused by *degraded* central control [3,5,8,22], while the changes observed here (Fig. 1B) and by [4] could equally be due to *increased* central control. That is, two very different underlying causes could still have the same statistical effect. Because the only unequivocal thing DFA provides is a statistical characteristic of a measured signal, we cannot and should not confuse any such metrics as indicating (by themselves) any specific underlying *cause* or *mechanism* for generating that signal [12]. What is needed are good first principles mechanistic models [13,14,16] of these behaviors that capture both the relevant neurological [7,29] and biomechanical [27,28] features of human walking that contribute to these observed phenomena. We recently showed that appropriately defined stochastic control models of these walking dynamics can indeed replicate the experimental dynamics described here [45].

Acknowledgments

Partial funding for this project was provided by grant RG-02-0354 from the Whitaker Foundation (to JBD), by NIH grants EB007638 and HD058942 (to JBD), and by NSF grant 0625764 (to JPC). The authors gratefully thank Dr. Hyun Gu Kang for assisting with data collection and Dr. Deanna H. Gates for inspiring the exploration of these questions.

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Figure 1.

A: Typical example of the stride length (SL), stride time (ST), and stride speed (SS) time series obtained from a representative subject for a trial where the subject walked at their preferred walking speed (PWS). Both the SL and ST time series exhibited periods where values continued increasing or decreasing across multiple consecutive strides. However, these patterns were not observed in the SS time series (obtained as $SS = SL/ST$). Qualitatively similar results were observed in all subjects. **B:** DFA exponents (α) obtained from all stride length (SL), stride time (ST), and stride speed (SS) time series as a function of walking speed from 80% to 120% of preferred walking speed (PWS). Error bars indicate between-subject $\pm 95\%$ confidence intervals. Subjects exhibited significant stride-to-stride statistical persistence (i.e., $\alpha \gg 1/2$) in

both SL and ST, suggesting that deviations in these measures were not immediately corrected on consecutive strides. Conversely, subjects consistently exhibited slight *anti*-persistence (i.e., $\alpha < \frac{1}{2}$) in stride speeds (SS), suggesting that this measure of walking performance was under tighter control.

Figure 2. Randomly Shuffled Surrogates

Randomly shuffled surrogates were generated for both the SL and ST time series. The surrogate SS time series were then computed as $\text{SS} = \text{SL}/\text{ST}$. By construction, these surrogates exhibited the same means and standard deviations (not shown) as the original walking data. This figure shows the average DFA exponents (α) obtained from all original and surrogate SL, ST, and SS time series as a function of walking speed. Error bars represent between-subject $\pm 95\%$ confidence intervals. The vertical scale is the same as in Fig. 1B. However, the Original and Surrogate data points were shifted slightly to the left or right, respectively, to improve the clarity of the figure. Unlike the experimental trials, these shuffled surrogates exhibited *no*

strong temporal correlations (all $\alpha \approx \frac{1}{2}$) for any of the three variables. Note that the error bars on the surrogate data points are *very* small.

Figure 3. Phase-Randomized Surrogates

Phase-randomized surrogates were generated *separately* for the SL and ST time series. The surrogate SS time series were then computed as $\text{SS} = \text{SL}/\text{ST}$. By construction, these surrogates exhibited nearly the same means and standard deviations (not shown) as the original walking data. This figure shows average DFA exponents (α) obtained from all original and surrogate SL, ST, and SS time series as a function of walking speed. Error bars represent between-subject $\pm 95\%$ confidence intervals. The vertical scale is the same as in Fig. 1B. Original and Surrogate data points were again shifted slightly to the left or right, respectively, to improve clarity. By construction, these surrogates exhibited nearly the same α for SL and ST as experimental trials.

However, *unlike* experimental trials, the surrogate SS time series exhibited strong statistical persistence (i.e., $\alpha \gg 1/2$).

Figure 4. Cross-Correlated Surrogates

Cross-correlated surrogates were generated *jointly* for both the SL and ST time series. The surrogate SS time series were then computed as $\text{SS} = \text{SL}/\text{ST}$. By construction, these surrogates exhibited nearly the same means and standard deviations (not shown) as the original walking data. This figure shows average DFA exponents (α) obtained from all original and surrogate SL, ST, and SS time series as a function of walking speed. Error bars represent between-subject $\pm 95\%$ confidence intervals. The vertical scale is the same as in Fig. 1B. Original and Surrogate data points were again shifted slightly to the left or right, respectively, to improve clarity. By construction, these surrogates exhibited nearly the same α for SL and ST as the experimental trials. However, *unlike* the independently phase-randomized surrogates (Fig. 3), the surrogate

SS time series now exhibited nearly the same statistical *anti*-persistence (i.e., $\alpha < 1/2$) as humans ($p = 0.284$).

Figure 5. Maximum Distances Walked

Histograms of the maximum absolute distances “walked” by all surrogates, and by humans, at all walking speeds. Zero (0) represents the center of the treadmill and the maximum absolute distance to either the front or back edge of the treadmill was 0.86 m. **A)** The vast majority of the randomly shuffled surrogates remained well within the treadmill limits. **B)** The phase-randomized surrogates exhibited the greatest tendency to “wander” along the treadmill, greatly increasing the distances walked. **C)** The cross-correlated surrogates remained closest to the center of the treadmill (0) in spite of retaining the same statistical persistence in both SL and ST as the phase-randomized surrogates. **D)** The distribution of distances walked by human subjects was most closely matched by that of the cross-correlated surrogates (**C**). Note that the

vertical scale in **(D)** is very different because 20 surrogate trials were generated for every 1 trial of human walking.