

# Detrended Fluctuation Analysis and Adaptive Fractal Analysis of Stride Time Data in Parkinson's Disease: Stitching Together Short Gait Trials

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## Abstract

Variability indicates motor control disturbances and is suitable to identify gait pathologies. It can be quantified by linear parameters (amplitude estimators) and more sophisticated nonlinear methods (structural information). Detrended Fluctuation Analysis (DFA) is one method to measure structural information, e.g., from stride time series. Recently, an improved method, Adaptive Fractal Analysis (AFA), has been proposed. This method has not been applied to gait data before. Fractal scaling methods (FS) require long stride-to-stride data to obtain valid results. However, in clinical studies, it is not usual to measure a large number of strides (e.g., < 100 strides). Amongst others, clinical gait analysis is limited due to short walkways, thus, FS seem to be inapplicable. The purpose of the present study was to evaluate FS under clinical conditions. Stride time data of five self-paced walking trials (25 strides each) of subjects with PD and a healthy control group (CG) was measured. To generate longer time series, stride time sequences were stitched together. The coefficient of variation (CV), fractal scaling exponents  $\alpha$  (DFA) and  $H$  (AFA) were calculated. Two surrogate tests were performed: A) the whole time series was randomly shuffled; B) the single trials were randomly shuffled separately and afterwards stitched together. CV did not discriminate between PD and CG. However, significant differences between PD and CG were found concerning  $\alpha$  and  $H$ . Surrogate version B yielded a higher mean squared error and empirical quantiles than version A. Hence, we conclude that the stitching procedure creates an artificial structure resulting in an overestimation of true  $\alpha$ . The method of stitching together sections of gait seems to be appropriate in order to distinguish between PD and CG with FS. It provides an approach to integrate FS as standard in clinical gait analysis and to overcome limitations such as short walkways.

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## Introduction

The ability to walk is a key component of mobility and is highly related to quality of life. Its assessment enables to get insight into system behaviour and gait disorders. In Parkinson's disease (PD), impaired gait is well documented with patients showing various gait abnormalities [1–4]. From a biomechanical point of view, gait disorders in PD can be characterised by spatiotemporal regulation difficulty e.g., shortened stride length and reduced stride velocity [5,6]. Quantification of within-subject stride-to-stride changes have proven to be promising in terms of characterising gait disturbances in PD [1]. Previous studies have found an increased stride-to-stride variability in patients with PD compared to controls with the tendency of increasing variability with disease severity [7,8]. In general, stride time variability has been shown to be affected by disease and ageing [8–10]. Quantification of stride-to-stride variability requires to measure a huge number of strides - the exact number is not known - than needed when analysing average stride characteristics [11,12].

Concerning the analysis of variability, a new perspective has been established in the last years. Besides the quantification of the amount of variability (e.g., coefficient of variation), the structure has been quantified in order to capture the dynamical properties of the system (for review, see [13,14]). It provides additional information and has been proven sensitive in detecting subtle changes of the system. For instance, [15] could distinguish elderly with more severe gait disorders from healthy age-matched controls by examining gait variability. However, among the subjects with more severe gait disorders, only the structural parameter was able to divide this group into fallers and non-fallers. The combined application of linear and nonlinear tools yields a complementary characterisation of gait variability and how it changes with age and disease [16]. In order to quantify the structure of stride-to-stride variability, Detrended Fluctuation Analysis (DFA) was previously applied [11,15,17,18] and especially with respect to stride time variability in PD [1,2,19]. DFA was introduced by [20] as a method to quantify the fractal dynamics or self-similarity of a time series. The method outputs the scaling exponent  $\alpha$  which can be interpreted in terms of correlations [21,22]. That is,  $\alpha=0.5$  is

characteristic of an uncorrelated signal and  $0.5 < \alpha < 1.0$  of a persistent signal (positive correlation). In healthy subjects walking under self-paced condition, a fractal scaling index of around  $0.8 - 1.0$  was observed [1,10,23] and higher indices resulted when walking slower or faster than self paced [23]. Values closer to 0.5 reflect a deviation from a healthy state and more random dynamics [9,10,24,25]. It could be shown that PD patients have a DFA scaling exponent close to 0.5 which indicates that stride-to-stride fluctuations are more random and that the long-range scaling behaviour is reduced [1,19]. A simple explanation is that gait of PD patients loses its automatism and fluidity with a break down of memory of the locomotor control system [1]. [19] showed that the  $\alpha$ -value decreases from control group to early PD to later PD patients which underlines the decrease of long-range scaling with disease severity. DFA is just one example to obtain structural information from time series data. Recently, a new method has been proposed, adaptive fractal analysis (AFA) [26–28], which is similar but has a number of advantages over DFA. We would like to point out two of them. First, the most important difference is, that AFA identifies a global smooth trend of the data by combining segments of overlapping windows, whereas in DFA the result of the linear fitting resembles a discontinuous signal with abrupt jumps. Therefore, AFA is not restricted on the signal being stationary. Second, AFA presents a more robust method concerning short time series compared to DFA [26,27]. While DFA is one of the most applied method with respect to stride interval time series, AFA has not been applied for this purpose before. It may be a valuable procedure to compare the results of DFA with AFA fractal scaling outcomes in this context.

The accuracy of the estimation of fractal exponents is reduced related to the length of the time series [29–31]. [30] propose that one needs series of at least  $2^{12}$  data points to get reliable results. However, [32] showed that the loss of accuracy of the estimation in short time series ( $< 2^{10}$ ) is not as dramatic as expected. In clinical gait analysis a few number of strides are typically recorded (e.g.,  $< 20$  strides [33,34]), which can be due to a short walkway (e.g., GAITRite system [35,36]). [36] review that studies differ with respect to the measured number of strides with a reduced reliability when only a few number of strides are analysed. Thus, typical clinical studies of gait are not suitable to the premise of needing long time series for DFA. However, [37] analysed the effects of nonstationarity on DFA, i.e., stitching together segments of data obtained from discontinuous experimental recordings. They found that positively correlated signals with  $0.5 < \alpha < 1.0$ , which can be expected for stride time variability [11], are not affected by the cutting procedure. [1] applied this procedure on experimental data - he analysed gait of subjects walking on a circuit and cut out the turn - resulting in longer time series which only include the straight walking distances. Following these works, the aim of this study is to evaluate the quantification of stride time variability by use of DFA, AFA and the coefficient of variation in patients with Parkinson's disease compared to a healthy control group based on stitching together consecutive walking trials in order to generate longer time series.

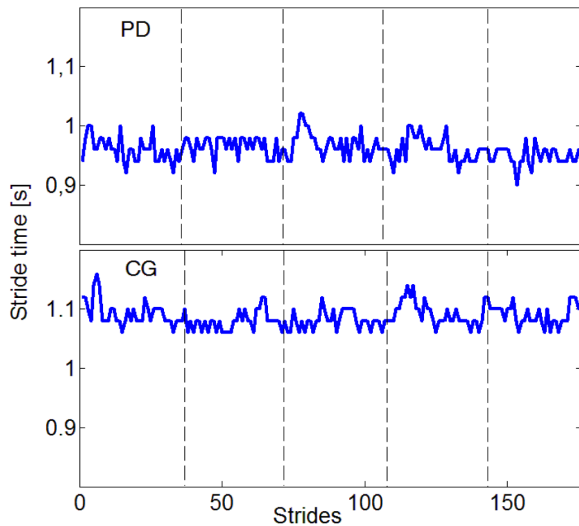
## Materials and Methods

In this study, 19 patients with Parkinson's disease (PD) (age:  $59.5 \pm 10.2$ ; UPDRS:  $36.1 \pm 13.5$ ; Hoehn and Yahr stages I and II) and a control group (CG) of 20 healthy younger subjects (age:  $22.0 \pm 2.7$  years) participated voluntarily and gave written informed consent to the experimental procedure. Gait analysis of PD was part of a larger study protocol which was approved by the ethics committee of the Hochschule Fresenius, University of

Applied Sciences, Idstein, Germany and complies with the scope of the declaration of Helsinki. PD patients with deep brain stimulation, further neurological diseases, orthopaedic impairments, with advanced dementia, and/or inability to walk autonomously were excluded. PD subjects were measured under regular medication (on-state). The subjects were instructed to walk at their individual self-paced velocity along a 49.2 m corridor. Wireless Medilogic© foot pressure insoles were used to evaluate the heel strike time of each foot. The sample rate was set to 50 Hz. With regard to gait initiation [38], recommend to start data collection after two complete gait cycles in order to achieve steady-state walking. [39] found that a 2.5 m distance is sufficient even with frail people. In the present study, the measurement was started after a 5.7 m gait initiation phase when the subjects crossed a predefined line to achieve steady-state walking. Measurement was completed 3.2 m before the end of the corridor, when the subjects crossed a second line, to exclude the gait deceleration phase. One practice walk and five trials were conducted. The heel strike times lead to a right foot and a left foot time series of single stride durations for each trial. There was no evidence for freezing, festination, or common concomitants associated with PD [40]. Data analysis was conducted via Matlab© R2008b.

One gait trial consists of about 25 strides (data points). By stitching the five trials together, longer data series were constructed with a total number ( $N_{max}$ ) of  $126 \pm 19$  (PD) and  $125 \pm 8$  (CG) data points. The stitching procedure comprised the simple addition of consecutive trials. Suppose  $(x_1, \dots, x_r)$  is the time series of the first trial and  $(y_1, \dots, y_s)$  is the time series of the subsequent trial, then the stitched time series is  $(x_1, \dots, x_r, y_1, \dots, y_s)$  (Figure 1). To quantify the stride-to-stride variability, the coefficient of variation (CV [%]) as well as the fractal scaling exponents  $\alpha$  by means of DFA with linear detrending and  $H$  by means of AFA with quadratic polynomial fits ( $M = 2$ ) were computed (Figure 2). The linear regression is the typical procedure for DFA. In case of AFA [27], propose to use a linear or a quadratic trend, as not every variation of the signal should be captured, leaving enough residuals to analyse further. We created smooth signals for both,  $M = 1$  and  $M = 2$  to visually define the best polynomial order. The linear trends produced inappropriate fits at the edges (region of no overlap), whereas the quadratic polynomials produced more accurate regressions. Time series were integrated prior to the application of DFA or AFA. Technical details of DFA and AFA are extensively described elsewhere (e.g., [20,27,28,32]). After visual inspection of the log-log-plot, scaling exponents were determined as the slope of the linear regression line over the window sizes  $w = 4$  to  $N_{max}/4$  in steps of 2 for DFA and  $w = 5$  to  $N_{max}/2$  (or  $N_{max}/2 + 1$  if the time series has an even number of samples) in steps of 5 in the case of AFA. Concerning the parameter CV, the intra class correlation coefficient ICC(3,1) for each foot was calculated in order to quantify the trial-to-trial reliability.

The parameters (CV,  $\alpha$ , and  $H$ ), were tested for statistically significant differences between the two groups. In case of normally distributed data - proved by the Shapiro-Wilk-Test - the t-Test was applied, and otherwise, the Mann-Whitney-U-Test. The significance level was set to 5%. In addition, linear correlation between CV,  $\alpha$ , and UPDRS was determined by means of Pearson ( $r$ ) or Spearman ( $\rho$ ) correlation coefficient. With respect to DFA and AFA, surrogate data tests were applied to test the null hypothesis ( $H_0$ ) of  $\alpha = 0.5$  (uncorrelated series) [22,41], independently for every subject. Thus, 1000 realizations were generated for each subject and the lower ( $q_{2.5\%}$ ) and upper ( $q_{97.5\%}$ ) sample quantiles were computed (Figure 3). Two different versions were applied: A) the whole time series was randomly shuffled; B) the single trials



**Figure 1. Exemplary stride-to-stride time series of a Parkinson's disease patient (PD) and a healthy subject (CG) after the stitching procedure.** The vertical dashed lines represent the stitching position.

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were randomly shuffled separately and afterwards stitched together. Version B was applied to look for artefacts of the method of stitching together the five trials. The bias ( $Bias(x) = \bar{x}_x - 0.5$ ) together with the mean squared error ( $MSE(x) = s_x^2 + (\bar{x}_x - 0.5)^2$ ) were determined to evaluate the goodness of the estimation. Correlations between  $N_{max}$  and  $MSE(x)$  were conducted to test for significant relations between the length of the time series and the error of the estimation. This is an exploratory study where descriptive  $p$ -values are reported with  $p < 0.05$  considered significant.

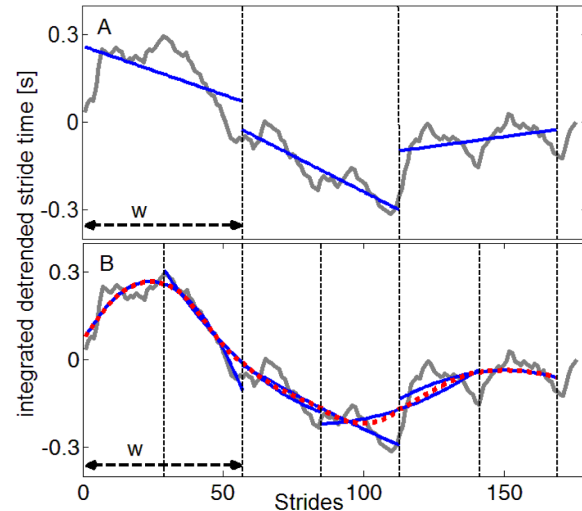
## Results

Results of the right (R) and left (L) stride time variability are presented in the following. Normal distribution was accepted for the data. Results are presented as mean  $\pm$  standard deviation. In order to account for several studies that have shown a close relationship between fractal scaling exponents calculated on stride times and walking speed (e.g., [11,42]), mean velocity of the gait trials (self-paced) were calculated and found to be  $1.44 \pm 0.04$  m/s for PD and  $1.45 \pm 0.04$  m/s for CG.  $H_0$  of being equal sets of walking trials could not be rejected ( $T_{37} = 0.243, p = 0.503$ ). However, the five gait trials exhibited different gait velocities for both groups which was tested by use of oneway repeated-measures ANOVA (PD:  $F_{4,72} = 6.27, p = 0.001$ ; CG:  $F_{4,76} = 17.29, p < 0.001$ ) showing the tendency that gait velocity increases with trial number.

### Stride-to-stride variability

A reduced  $CV_L$  sample mean was observed in PD ( $1.8\% \pm 0.3$ ) compared to CG ( $2.1\% \pm 0.4$ ) which was not significantly different ( $T_{37} = 1.9, p = .067$ ). Concerning  $CV_R$ , a sample mean of  $1.9\%$  ( $\pm 0.4$ ) for PD and  $2.1\%$  ( $\pm 0.5$ ) for CG was observed with no significant differences ( $T_{37} = 1.1, p = .29$ ). ICC(3,1) shows rather poor values in PD (L:  $-0.06$ , R:  $0.34$ ) as well as in CG (L:  $0.23$ , R:  $0.25$ ). CV values of the five gait trials are shown in Table 1.

Concerning the scaling exponent  $\alpha$ , PD showed significantly lower values (R:  $0.8 \pm 0.15$ , L:  $0.79 \pm 0.15$ ) compared to CG (R:



**Figure 2. Polynomial fitting methods of DFA (A) and AFA (B).**

Example of an integrated detrended stride time series (grey) and a window size of  $w = 57$ . In the case of DFA, linear regression lines (blue) are computed within nonoverlapping windows resulting in a discontinuous signal. In the AFA method overlapping windows of  $n + 1 = (w + 1) / 2 = 29$  data points are used. Within each window a quadratic regression line ( $m = 2$ ) is fit to the time series (blue). Afterwards a global smooth and continuous trend (dotted red line) is calculated.

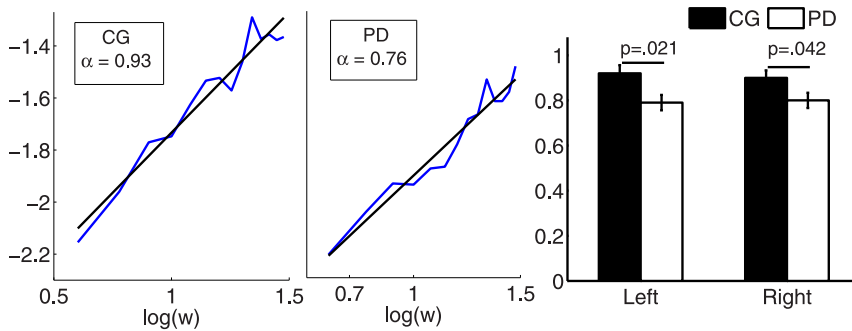
doi:10.1371/journal.pone.0085787.g002

$0.90 \pm 0.15$ , L:  $0.92 \pm 0.16$ ) as presented in Figure 4. In accordance to the results of DFA, AFA exhibited significantly lower values for PD (R:  $0.77 \pm 0.15$ , L:  $0.75 \pm 0.16, T_{37} = 2.5, p = 0.016$ ) in contrast to CG (R:  $0.89 \pm 0.14$ , L:  $0.88 \pm 0.15, T_{37} = 2.5, p = 0.016$ ). An exemplary time series with its global smooth trend and the log-log-plot are shown in Figure 1.

Correlation between UPDRS, CV, and  $\alpha$  for PD and between CV and  $\alpha$  for CG are presented in Table 2 with respect to right and left stride time variability. No significant correlations were found.

### Surrogate data tests

Surrogate data tests were used to test the null hypothesis of  $H_0 : \alpha = 0.5$  separately for every subject. With respect to surrogate version A,  $H_0$  could not be rejected for 10% of CG and for 26% of PD concerning DFA and could not be rejected for 15% of CG and 32% of PD in the case of AFA. Concerning surrogate version B,  $H_0$  could not be rejected for 30% of controls and for 47% of patients with respect to DFA, and could not be rejected for 30% of CG and 58% of PD. The mentioned percentages were true for both, left and right stride time data. The comparison of the two surrogate versions, A and B, yielded a higher bias or mean squared error when the single trials were shuffled separately and afterwards stitched together. That was, for  $MSE(x)$   $0.009 \pm 0.001$  and  $MSE(H)$   $0.007 \pm 0.0005$  (A) versus  $MSE(x)$   $0.03 \pm 0.02$  and  $MSE(H)$   $0.03 \pm 0.02$  (B). For  $Bias(x)$   $0.044 \pm 0.006$  and  $Bias(H)$   $0.002 \pm 0.01$  (A) versus  $Bias(x)$   $0.14 \pm 0.07$  and  $Bias(H)$   $0.11 \pm 0.09$  (B). In addition, version B yielded increased statistical bounds [ $q_{2.5\%}; q_{97.5\%}$ ] for the acceptance region of  $H_0$ : on average [0.39; 0.72] for DFA and [0.34; 0.67] for AFA(A) versus [0.49; 0.79] for DFA and [0.47; 0.76] for AFA (B) (Figure 3). Concerning version A, smaller MSE values for longer time series were observed: a significant correlation between  $N_{max}$  and  $MSE(x)$  was obtained ( $\rho = -0.96, p < .001$ ). This was not the case with respect to surrogate version B ( $\rho = -0.1, p > 0.05$ ).



**Figure 3. Empirical statistical bounds (2.5% and 97.5% quantiles:  $q_{0.025}$ ,  $q_{97.5}$ ) for surrogate versions A (left panel) and B (right panel).** As an example, the distribution of  $\alpha$  computed from surrogate series of stride time data of one subject is presented with a mean value of  $\bar{x}_\alpha = 0.54$  for version A and  $\bar{x}_\alpha = 0.59$  for version B. The respective mean squared errors are  $MSE = 0.0074$  (A) and  $MSE = 0.014$  (B). doi:10.1371/journal.pone.0085787.g003

**Discussion**

Under clinical conditions, the application of fractal methods to stride time data is often difficult due to the need of long continuous recordings to attain the true value of the scaling exponent. In clinical standard diagnosis, there is often a lack of space which counteracts the evaluation of a large number of strides. Hence, in this study, we examined whether stitching together short sequences of stride time data illustrates a reasonable method to generate sufficiently long time series for the application of fractal methods. To test this procedure, a cohort of PD subjects and a healthy control group were measured. Two fractal methods, DFA and AFA, were applied to stride time series to account for differences between both subject groups. In addition, the CV was calculated as a linear and frequently used parameter of stride time variability data.

CV of stride time data in healthy adults is about 2% [1,18,43] which fits to our results. Interestingly, the data of our PD patients reached lower values which is contrary to the literature [4,8,44–46]. For instance [44], found CV values of 3.3% (non-fallers) to 5.0% (fallers) for PD patients in on-state. Our examination of variability differs from these studies with respect to methodology. Moreover, in the present study, only subjects with low disease severity (low UPDRS score, Hoehn and Yahr stage I and II) were included. [4] report higher values for PD fallers (CV = 5.0) versus nonfallers (CV = 3.3) under medication. In addition, we found that CV of stride time has a low trial-to-trial reliability. Gait data exhibited very poor values of ICC. This is comparable to [47] who found that the coefficient of variation of stride time in healthy older adults ( $n = 59$ ) is attended by a low test-retest reliability.

Others report higher values of CV concerning stride time (e.g., [48,49]). However, these divergences may be due to rather few stride numbers on short walkways (5–18 strides). The present study investigates more strides and it has to be emphasized that gait data has to be collected over a reasonable distance to calculate reliability of stride time data [50]. One can speculate that stitched time series are not suitable to calculate CV of stride time.

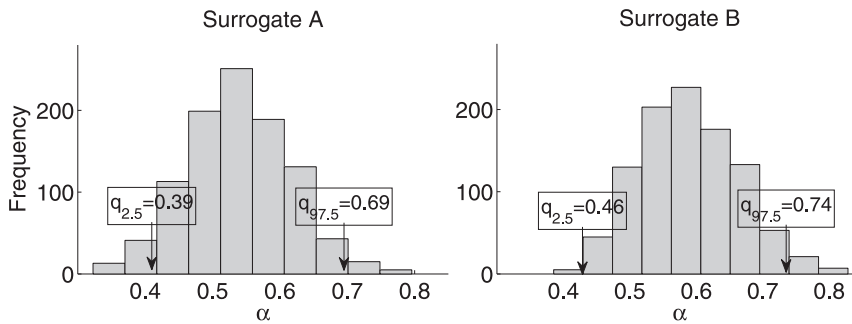
With respect to nonlinear measures, we found significantly lower fractal scaling exponents in PD patients compared to the control group which fits to the literature [1,19,46]. Both fractal scaling exponents  $\alpha$  and  $H$  demonstrated equivalent outcomes with respect to the differentiation of PD and CG. Thus, our results underline the sensitivity of fractal methods even if they are based on stitched time series. It has to be emphasised that different polynomial fits were used for both methods (DFA and AFA). The scope of this article was to evaluate the applicability of fractal scaling methods to discriminate PD from CG. Thus, from a methodological point of view, a comparison between the results has to be drawn carefully. To give consideration to this aspect, underlying polynomial fits should be of the same order.

Gait velocity was significantly different between the trials. However, the mean gait speed of the first trial (slowest velocity) was less than 5% compared to the last trial (fastest velocity). For instance, [42] report their differences between gait speed and the fractal scaling exponent  $\alpha$  (in the range of  $\pm 0.05$ ) on the basis of 20% difference from the comfortable self-paced walking speed. We assume that the changes in walking speed may have a marginal effect on the fractal scaling outcome. Furthermore, we found no significant differences on gait velocity between the subject groups. Nevertheless, we recommend that before analysing different gait trials in order to apply the stitching procedure, three practice trials have to be conducted as we have found later trials to reveal more consistent walking speeds. [46] showed that the DFA fractal exponent can be related to age and disease severity. However, we found no linear correlation between UPDRS, a measure of disease severity, and the fractal scaling exponent  $\alpha$ . Several reasons may account for this phenomenon. It has been proven that fractal methods are sensitive to identify various and subtle information of the systems behaviour [26,51]. Furthermore, UPDRS is a sum score of equally weighted items regarding multiple PD specific symptoms. By contrast, gait is a highly complex motor control performance and therefore, a linear interaction between both methods is unlikely. In addition, no significant linear correlation between UPDRS and CV was found which is in contrast to [4]. Our results of having no correlations between the scaling exponent  $\alpha$  (DFA) and CV support previous findings in PD patients [1] and

**Table 1. Sample mean  $\pm$  standard deviation of the coefficient of variation (CV [%]) of each gait trial.**

Trial	CG		PD	
	Left	Right	Left	Right
1	1.80 $\pm$ 0.51	1.86 $\pm$ 0.49	1.71 $\pm$ 0.31	1.72 $\pm$ 0.40
2	1.77 $\pm$ 0.41	1.76 $\pm$ 0.49	1.65 $\pm$ 0.33	1.48 $\pm$ 0.30
3	1.55 $\pm$ 0.33	1.61 $\pm$ 0.32	1.70 $\pm$ 0.51	1.62 $\pm$ 0.46
4	1.67 $\pm$ 0.32	1.58 $\pm$ 0.29	1.57 $\pm$ 0.33	1.56 $\pm$ 0.37
5	1.65 $\pm$ 0.45	1.65 $\pm$ 0.41	1.70 $\pm$ 0.73	1.50 $\pm$ 0.31

(CG = control group, PD = Parkinson’s disease group). doi:10.1371/journal.pone.0085787.t001



**Figure 4. Results of the fractal scaling exponent  $\alpha$ , determined by DFA, of the Parkinson's disease patients (PD) and the control group (CG).** Example of one subject respectively (left and middle panel). Right panel: sample mean  $\pm$  standard error of  $\alpha$ . The p-value is reported concerning the statistical comparison of the two groups (CG = black, PD = weight). doi:10.1371/journal.pone.0085787.g004

in older adults [52] which may underline that both parameters (linear versus nonlinear) account for different information in the stride time series. No different scaling regions (linear trend of the log-log-plots in DFA and AFA) were found which is in contrast to [53] who found multifractal scaling for both, PD patients and healthy controls.

The proposed method of stitching together five trials in order to yield longer time series seems to be appropriate in order to distinguish between healthy and pathological gait using nonlinear methods. However, we found higher empirical quantiles and a larger bias, as well as larger mean squared errors concerning surrogate B. One can assume that stitching together the single trials generates a pseudo structure which results in a shift to a positive correlation ( $\alpha > 0.5$ ). Hence, it can be expected that the presented  $\alpha$ -values overestimate the true scaling exponent. This effect was similar in the calculation of  $H$ . However, our results partly concur with  $\alpha$ -values reported in literature e.g., 0.92 for the control group and 0.84 for the PD group [19]. [1] found a fractal scaling exponent of 0.82 for the PD group which was statistically different from the value of the control group. In general, it was found  $0.8 < \alpha < 1.0$  for healthy adults with respect to stride time data [1,10,23], which means that our findings are located at the upper end of the reported range.

In accordance with previous studies [30,31,54], we found a negative correlation between signal length and  $MSE(\alpha)$  which underlines that the error decreases with increasing signal length. This was not true, however, for surrogate version B. Although stitching together the five trials, signal length was smaller than  $2^7$  which resulted in large acceptance regions for  $H_0 : \alpha = 0.5$ . In literature, no consistent recommendations are published concern-

ing the minimum number of strides needed to attain accurate results of gait analysis. [55] propose 400 steps [56], suggest 600 strides, whereas others showed good results of DFA with smaller number of data points [32]. Two methodologically conflicting problems emerge with regard to long time series in gait. First, in clinical standard diagnosis it is hardly possible to measure long distances due to a lack of space and costs required for performing a study [57]. A second problem is the effect of fatigue in patients during prolonged walking. The present study demonstrates a simple procedure which is applicable without these implications. However, from a theoretical point of view, the stitching procedure does not accord to the originally proposed assumption of finding long-range correlations within consecutive strides. The present approach is based on the idea to have measured steady although pathological systems and therefore, sections of the gait process. Therefore, the indicated fractal scaling values are strictly spoken only true for the stitching process. In this study, the true fractal properties of the system (underlying long-term correlations in the signals) were not investigated directly. To account for this aspect, scaling exponents of long continuous recordings of stride time series have to be compared to those scaling values that are obtained by the stitching procedure. Further research has to be undertaken to elucidate the relationship to long continuous data and to get insight into how many sequences should be measured and how many strides should these sequences consist of.

**Conclusion**

The present study demonstrates that applicability of fractal methods in gait analysis is not limited to time series that are collected from prolonged walking conditions. The proposed method enables to create sufficiently long data by stitching together short sequences ( $\approx 25$  strides) to differentiate between a healthy control group and a group of persons with Parkinsons Disease by use of fractal methods. Hence, this approach is useful, for instance, in the context of clinical investigations that are restricted to short walkways. This work provides a first insight into the agreeability between elaborate gait analysis and clinical suitability. It has to be further elucidated which combination between the number and the length of trials will produce the best results. This systematic analysis would be the next step, to establish fractal analysis as a standardised user-friendly method for clinical standard diagnosis.

**Author Contributions**

Conceived and designed the experiments: PS ML CTH. Performed the experiments: PS ML. Analyzed the data: MK PS ML. Contributed reagents/materials/analysis tools: CTH PS ML. Wrote the paper: MK PS ML CTH.

**Table 2. Results of Pearson ( $r$ ) and Spearman ( $\rho$ ) correlation coefficient of right and left stride time data concerning coefficient of variation (CV), scaling exponent  $\alpha$  computed with DFA and UPDRS score.**

Correlation	CG		PD	
	Left	Right	Left	Right
CV, DFA	$r = 0.37$	$r = 0.33$	$r = -0.04$	$r = 0.45$
CV, UPDRS			$\rho = 0.25$	$\rho = 0.3$
DFA, UPDRS			$\rho = 0.17$	$\rho = 0.18$

No significant ( $p > 0.05$ ) correlations were found. CG = control group, PD = Parkinson's disease patients.

doi:10.1371/journal.pone.0085787.t002

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